In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 14-917V

Filed: August 21, 2019

C.P.,	*	To Be Published
	*	
	*	Influenza ("Flu") Vaccine;
Petitioner,	*	Polymyalgia Rheumatica ("PMR");
	*	Seronegative Rheumatoid Arthritis ("RA");
v.	*	Dismissal
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

Christina Ciampolillo, Esq., Conway, Homer, P.C., Boston, MA, for petitioner.
Althea Davis, Esq., U.S. Dept. of Justice, Washington, DC, for respondent.

DECISION[1]

Roth, Special Master:

On September 29, 2014, C.P. ("petitioner") timely filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10, *et seq.*[2] ("Vaccine Act" or "Program"). Petitioner alleges he developed polymyalgia rheumatica ("PMR") and/or seronegative rheumatoid arthritis ("RA") as the result of the influenza ("flu") vaccination that he received on October 14, 2011. Petition at 1.

[1] This Decision has been designated "to be published," which means I am directing it to be posted on the Court of Federal Claims's website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (codified as amended at 44 U.S.C. § 3501 note (2006)). **This means the Decision will available to anyone with access to the internet.** 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] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (1986). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

Many of the facts relevant to this case are undisputed. The parties agree that petitioner was diagnosed with PMR months after his influenza vaccination and that he was subsequently diagnosed with seronegative RA. The issues in dispute are whether the flu vaccine can cause and/or trigger PMR and/or seronegative RA, whether it did so in this case, and whether the timing is medically appropriate.

An entitlement hearing was held on December 11-12, 2017 in Washington, D.C. Petitioner failed to meet his burden to show, by a preponderance of evidence, that it was more likely than not that he developed PMR and/or RA as a result of receiving the flu vaccine. For the reasons detailed below, I find that petitioner is not entitled to compensation.

I. Procedural History

Petitioner filed his petition on September 29, 2014. Petition, ECF No. 1. This case was initially assigned to Special Master Hamilton-Fieldman on September 29, 2014 and was reassigned to me on January 14, 2016. ECF Nos. 4, 32.

Petitioner filed several medical records on October 2, 2014 and a Statement of Completion on October 3, 2014. Petitioner's Exhibits ("Pet. Ex.") 1-18, ECF Nos. 6-9. Respondent filed a status report on November 24, 2014, advising that he had reviewed the medical records that were filed and determined the records to be sufficient to complete his Rule 4(c) Report. Respondent's Status Report ("Resp. S.R.") at 1, ECF No. 13.

On December 29, 2014, respondent filed his Rule 4(c) Report which contained a detailed review of petitioner's medical history. Respondent's Report at 1-8, ECF No. 14. Respondent stated that petitioner failed to provide evidence that he suffered PMR or RA as a result of the flu vaccine. *Id.* at 10. Respondent noted that in May 2013, petitioner's primary care physician recommended that petitioner receive a flu shot in the fall of that year. *Id.* Respondent reasoned that this suggested petitioner's primary care physician did not associate petitioner's PMR and/or RA with the flu vaccine. *Id.* (citing Pet. Ex. 2 at 2-3). Respondent argued petitioner was not entitled to compensation based on his medical records and that without more proof, petitioner's claim should be dismissed. *Id.* Petitioner filed a response to respondent's Rule 4(c) Report on January 7, 2015. Response, ECF No. 15.

A status conference was held on January 21, 2015, after which petitioner was ordered to file an expert report by March 25, 2015. Order, ECF No. 16. After three extensions of time, petitioner filed an expert report from Dr. Kristin Gowin, a rheumatologist, along with supporting medical literature on July 27, 2015. Pet. Ex. 20-21, ECF No. 25. Respondent filed a responsive expert report authored by Dr. Mehrdad Matloubian, an immunologist and rheumatologist, on November 3, 2015 and supporting medical literature on November 13, 2015. Respondent's Exhibit ("Resp. Ex.") A1-A17, ECF Nos. 28-30.

A status conference was held on November 19, 2015 during which Special Master Hamilton-Fieldman requested that, if petitioner decided to file a supplemental expert report, the expert address the triggering action in this case, the homology of RA, the nature of petitioner's

pre-clinical RA, and the issues outlined in respondent's Rule 4(c) Report. Order, ECF No. 30. Petitioner was ordered to file either a supplemental expert report or a status report by January 22, 2016. *Id.* This case was reassigned to me on January 14, 2016. ECF No. 32.

After an extension of time, petitioner filed a supplemental expert report from Dr. Gowin and an expert report with supporting literature from Dr. Michael Gurish, an immunologist, on February 5, 2016. Pet. Ex. 22-24, ECF No. 35. Respondent filed a responsive expert report from Dr. Matloubian and supporting medical literature, on May 4, 2016. Resp. Ex. C1-14, ECF No. 38-40.

A status conference was held on June 30, 2016, during which petitioner advised he intended to file an additional responsive expert report. Order, ECF No. 41. The parties also discussed scheduling an entitlement hearing for October or November 2017. *Id.* Petitioner was ordered to file a responsive expert report by August 1, 2016, and a joint status report proposing entitlement hearing dates by August 29, 2016. *Id.*

On August 29, 2016, petitioner filed a joint status report proposing entitlement hearing dates. Joint S.R., ECF No. 43. An entitlement hearing was scheduled for October 2 and 3, 2017. Prehearing Order, ECF No. 44.

After three extensions of time, petitioner filed a supplemental expert report from Dr. Gurish on October 13, 2016. Pet. Ex. 25, ECF No. 47. Petitioner filed additional medical records on February 8, 2017. Pet. Ex. 26-32, ECF No. 49.

On August 7, 2017, the entitlement hearing was rescheduled for December 11 and 12, 2017. Pre-Hearing Order, ECF No. 50. On October 13, 2017, petitioner filed updated medical records. Pet. Ex. 33-40, ECF No. 51. Petitioner filed his prehearing brief on October 16, 2017, and additional medical literature on November 6, 2017. Prehearing Submission, ECF No. 53; Pet. Ex. 41-44, ECF No. 56.

Respondent filed his prehearing brief and additional medical literature on November 9, 2017. Prehearing Submission, ECF No. 58; Resp. Ex. D-J, ECF No. 59. Petitioner filed a response to respondent's prehearing brief on November 20, 2017. Response, ECF No. 60. The parties filed their joint prehearing submission on December 4, 2017. ECF No. 63. A prehearing status conference was held on December 5, 2017, during which the parties discussed the logistics of the entitlement hearing. Order, ECF No. 64.

An entitlement hearing was held on December 11 and 12, 2017. Order, ECF No. 69. Dr. Gowin and Dr. Gurish testified on behalf of petitioner and Dr. Matloubian testified on behalf of respondent. *Id.* Petitioner's co-counsel appeared via video conference from California, along with petitioner, C.P., and his wife, [redacted], who also testified at the hearing. *Id.* At the conclusion of the hearing, petitioner was ordered to file all pharmacy records from 2010 through 2012, all rheumatology records since March 2017, and all records related to petitioner's November 2017 back surgery, by March 12, 2018. *Id.* Respondent was ordered to file an article referenced by Dr. Matloubian during the hearing that discussed the effect of trauma on the autoimmune system by March 12, 2018. *Id.*

Petitioner filed some of the requested medical records on February 27, 2018, and March 8, 2018. *See* Pet. Ex. 45-50, ECF Nos. 74, 77. On March 12, 2018, petitioner filed a status report advising he needed additional time to obtain the remaining outstanding medical records. Pet. S.R., ECF No. 79. Petitioner was ordered to file all outstanding records, along with a status report advising that the record was complete, by April 16, 2018. Order at 1, ECF No. 80. On March 14, 2018, petitioner filed the outstanding records and a status report advising that the record was complete. *See* Pet. Ex. 51, ECF No. 81; Pet. S.R. at 1, ECF No. 83. The parties were ordered to file their post-hearing briefs by May 14, 2018. Non-PDF Order, dated March 14, 2018. Petitioner and respondent filed their post-hearing briefs on June 20, 2018. *See* ECF Nos. 86-87.

This matter is now ripe for determination.

II. Factual Background

Petitioner was born on August 22, 1956. Petitioner has a complicated medical history both before and after his vaccination which includes type II diabetes, hypertension, high cholesterol, neck pain, tendonitis of the left shoulder, left arm pain, torn rotator cuff of the right shoulder, chronic lower back pain with sciatic pain, numbness and weakness in his legs, hip pain, two arthroscopic surgeries of the left knee, total left knee replacement with resulting deep vein thrombosis ("DVT"), left foot surgery, neuropathic pain, and diabetic neuropathy. *See generally* Pet. Ex. 12-13, 29, 36-38, 40. Over the years, petitioner has undergone chiropractic care, physical therapy, acupuncture and pain management, and received multiple pain injections and pain blocks for his lower back and hip pain. *Id.* He is under the routine care of an ophthalmologist due to his diabetes. *See generally* Pet. Ex. 31. Both petitioner and his wife stated that petitioner was in relatively good health prior to his vaccine and remained physically active due to his physically demanding job as a maintenance worker for a school district. Tr. 6, 27-28; Pet. Ex. 17 at 1; Pet. Ex. 19 at 1. While all of petitioner's medical records were reviewed in this matter, only his relevant medical history is discussed below.

A. Petitioner's Health Prior to Receiving the Allegedly Causal Flu Vaccine

On February 6, 2009, petitioner fell from a ladder onto his left leg while at work as a maintenance worker for his city's school district. Tr. 6; Pet. Ex. 3 at 40-48. An MRI of his left leg revealed a medial meniscus tear of his left knee. Pet. Ex. 3 at 40. Following a course of physical therapy, petitioner underwent arthroscopic surgery of the left knee on June 11, 2009. *Id.* Petitioner filed a workers' compensation claim following his fall from the ladder. Tr. 24.

In the months following petitioner's left knee surgery, he presented multiple times to his primary care physician ("PCP") and orthopedist with continued complaints of worsening knee pain. *See* Pet. Ex. 3 at 32-38, 40-48.

On August 6, 2010, petitioner presented to his PCP with complaints of ongoing left knee pain as well as lower back pain. Pet. Ex. 2 at 25. He had recently been diagnosed with diabetes and was taking Metformin to control his diabetes along with cholesterol and blood pressure medication. *Id.* Due to his continued knee pain, he had been receiving joint injections and a total

knee replacement surgery was discussed. *Id.* An MRI performed in September 2010 revealed a medial meniscus tear, so petitioner's total left knee replacement was scheduled for May 2011. Pet. Ex. 3 at 27. Petitioner's workers' compensation case was reopened in March 2011 due to his progressively worsening knee injury. *Id.* at 32.

A total left knee replacement was performed on May 27, 2011.[3] Petitioner presented for a post-operative orthopedic visit on June 7, 2011 with swelling, arthritis, and chondromalacia[4] of the patella. Pet. Ex. 3 at 7-9. He was still unable to work and was advised to continue physical therapy and wean off his walker. *Id.*; Tr. 7.

On June 21, 2011, petitioner was admitted to Huntington Hospital with complaints of weakness and pain in his left leg. Pet. Ex. 5 at 41-43; Pet. Ex. 13 at 77. He had developed deep vein thrombosis ("DVT")[5] in his left leg requiring surgery and remained hospitalized until June 23, 2011. Pet. Ex. 5 at 42; Pet. Ex. 13 at 77. Petitioner was prescribed a course of blood thinners that he continued to take for months following DVT surgery. *See* Pet. Ex. 2 at 19; Pet. Ex. 6 at 32-33.

On June 30, 2011, petitioner presented to his orthopedist, Dr. Dietrick, for follow up of his left knee replacement and DVT surgery. Pet. Ex. 3 at 2-3. He was taking narcotics for pain management and remained unable to return to work. *Id.* at 3.

On July 25, 2011, Dr. Dietrick issued a report regarding petitioner's work-related left knee injury. Pet. Ex. 6 at 34-37. The report stated that following a fall from a ladder in 2009, petitioner was diagnosed with osteoarthritis, effusion, and probable loose body. *Id.* at 34. He underwent a total knee replacement for his degenerative arthritic condition on May 27, 2011 and his post-operative course was complicated by a blood clot the following month. *Id.* Petitioner had also developed sciatic pain in his lower back after surgery for which he received two epidural injections and was prescribed Dilaudid and Percocet for pain management. *Id.*

From July to September 2011, petitioner presented to his primary care physician and Dr. Dietrick for follow up of his left knee and DVT surgeries. *See* Pet. Ex. 2 at 19; Pet. Ex. 6 at 32-33. At these visits, petitioner continued to complain of sciatica[6] and left knee weakness and pain. *See id.* Petitioner remained out of work. *Id.*

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[3] Records from this surgery were never filed.

[4] Chondromalacia is defined as "softening of the articular cartilage, most frequently in the patella." *Chondromalacia*, DORLAND'S ILLUSTRATED MEDICAL DICTIONARY 352 (32d ed. 2012) [hereinafter DORLAND'S].

[5] Deep vein thrombosis is defined as "thrombosis of one or more deep veins, usually of the lower limb, characterized by swelling, warmth, and erythema." *Deep vein thrombosis*, DORLAND'S at 1923. Thrombosis is defined as "the formation, development, or presence of a thrombus." *Thrombosis*, DORLAND'S at 1923. A thrombus is defined as "a stationary blood clot along the wall of a blood vessel." *Thrombus*, DORLAND'S at 1923.

[6] Petitioner testified he was only experiencing sciatic pain in his left leg rather than through his lower back as discussed by his treating physicians. Tr. 21; *but see* Pet. Ex. 2 at 25; Pet. Ex. 6 at 34.

B. Petitioner's Health at the Time of and After Receiving the Allegedly Causal Flu Vaccine

On October 14, 2011, petitioner presented to his PCP with complaints of right thigh burning, tingling, and numbness that had persisted for approximately one month. Tr. 8; Pet. Ex. 2 at 16; Pet. Ex. 17 at 1. He was diagnosed with entrapment of the lateral cutaneous nerve of the thigh and given Voltaren[7] for pain. Pet. Ex. 2 at 16. He received the allegedly causal flu vaccine at this visit. *Id.* at 16-17, 28.

On October 28, 2011, petitioner presented to Dr. Dietrick for follow up of his left knee replacement. Pet. Ex. 6 at 28. Petitioner was still experiencing numbness and constant pain in his left knee. *Id.* He was noted to walk with an antalgic gait favoring his left leg. *Id.* Dr. Dietrick reported no nerve root tension signs in the left leg but noted atrophy and tenderness on the lateral patella facets with slight lateral tracking of the patellofemoral joint. *Id.* Dr. Dietrick recommended that petitioner attend physical therapy. *Id.* at 29.

Petitioner and his wife testified that in late November 2011, petitioner began experiencing progressive weakness, fatigue, and aches in his shoulders, groin, and back. Tr. 8, 28-29. Petitioner attributed these symptoms to his recent return to work, stating "I was dreading going back to work because I had the major muscle issues . . . It was really hard just getting up and getting started . . . I did physical labor, manual labor, I climbed ladders and everything. It was a killer." Tr. 8-9. Petitioner's wife added he had never complained that way prior to the flu vaccine. Tr. 29; Pet. Ex. 19 at 1-2.

Petitioner returned to Dr. Dietrick on December 7, 2011 with complaints of continued knee pain, popping, stabbing, and numbness. Pet. Ex. 6 at 26. He reported no improvement with physical therapy and at-home exercises. *Id.* Dr. Dietrick advised petitioner to continue with physical therapy but if his pain and discomfort continued, possible arthroscopic surgery or a lateral patellar facetectomy would be considered. *Id.* at 26-27. Petitioner made no additional complaints at this visit. However, he testified that he was experiencing shoulder and hip pain at this visit and at the October 28, 2011 visit but did not mention it to Dr. Dietrick because he was only treating with Dr. Dietrick for his knee. Tr. 24-25.

Petitioner's wife recalled that petitioner called her on December 9, 2011 while she was at work and told her he thought he had the flu. Pet. Ex. 19 at 2. On December 10, 2011, petitioner continued to feel poorly but accompanied her to her employer's holiday party. *Id.* Petitioner was typically very social at these events, but Mrs. [redacted] remembered that petitioner was very quiet at this event; he sat in a corner for most of the event and then left early to go to bed. *Id.*; Tr. 32. Petitioner recalled that he had difficulty walking at this event. Pet. Ex. 17 at 2.

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[7] Voltaren is the brand name for diclofenac sodium, a nonsteroidal anti-inflammatory drug ("NSAID") used to treat pain. *Diclofenac sodium – Drug Summary*, PDR.NET, http://www.pdr.net/drug-summary/Voltaren-XR-diclofenac-sodium-2033.6043 (last visited June 21, 2019).

While petitioner stated that he had returned to work in November, Mrs. [redacted] testified that he returned to work in mid-December and worked through the pain until the school district's Christmas break began. Pet. Ex. 19 at 2-3. She remembered him having difficulty sleeping and being very anxious about working in pain. *Id.* at 3. Mrs. [redacted] stated that petitioner was unable to attend a New Year's Eve party and a family wedding in January 2012 due to his pain and discomfort. *Id.*

Petitioner returned to his PCP on December 29, 2011 with complaints of continued right leg numbness and progressive pain in his legs, shoulders, and groin. Pet. Ex. 2 at 13. He reported that the Voltaren was not working to relieve his pain and discomfort. *Id.* He was noted to have normal range of motion in his upper and lower extremities and diffuse pain over both shoulders and thighs. *Id.* His PCP questioned whether petitioner's myalgias[8] were related to his cholesterol medication. *Id.* Petitioner was directed to discontinue use of this mediation and follow up in two weeks regarding his symptoms. *Id.* Petitioner was given samples of Lyrica[9] for pain management and advised to continue treating with Dr. Dietrick for his paresthesia and knee pain. *Id.* at 13-14.

Petitioner completed a form entitled "History of Work Related Accident/Injury" on January 2, 2012 in which he indicated he had aches and pains outside and in the rear of his left knee, tenderness on the outside and front of the knee, and sharp, stabbing pain on the outside and back of the left knee. Pet. Ex. 6 at 49. No other complaints were reported. *See id.* Petitioner testified that he was experiencing PMR-like symptoms at this time but he did not include those symptoms on this form. Tr. 25.

On January 16, 2012, petitioner presented to Dr. O'Connor, a rheumatologist, with complaints of bilateral shoulder pain, hip pain, stiffness, and low energy which he reported he had been experiencing since December 2011. Pet. Ex. 7 at 65. His family history was significant for diabetes, hypertension, and myasthenia gravis. *Id.* 65-66. Petitioner had a history of alcohol use but quit drinking in 2011. *Id.* at 66. Petitioner had previously been given a Medrol dose pack[10] and noticed improvement of his symptoms. *Id.* Shoulder x-rays revealed moderate loss of joint space at the acromioclavicular joint[11] with osteophytes and a preserved glenohumeral joint space. *Id.* at 65. X-rays of petitioner's hips revealed mild osteoarthritis of both hips. *Id.* Dr. O'Connor noted limited range of motion in both shoulders and hips, but no small joint inflammation. *Id.* at 67. Dr. O'Connor diagnosed petitioner with PMR. *Id.* Additional bloodwork was ordered, and petitioner was prescribed prednisone. *Id.*

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[8] "Myalgia" is muscle pain; "arthralgia" is joint pain. *Myalgia*, DORLAND'S at 150, 1214.

[9] Lyrica is the brand name for pregabalin, an anticonvulsant used to treat neuropathic pain associated with several conditions, including fibromyalgia. *Pregabalin – Drug Summary*, PDR.NET, http://www.pdr.net/drug-summary/Lyrica-pregabalin-467.8329 (last visited June 21, 2019).

[10] This was not noted in the previous PCP records.

[11] The acromioclavicular joint connects the clavicle, or collarbone, to the scapula, or shoulder blade. STEDMAN'S POCKET MEDICAL DICTIONARY 10 (1st ed. 1987) [hereinafter STEDMAN'S].

On January 20, 2012, petitioner returned to Dr. Dietrick reporting that he had tried to return to work but the squatting, kneeling, and climbing ladders and stairs had caused a flare up of his knee pain. Pet. Ex. 6 at 24. He rated his current knee pain as 5-8/10 and noted his knee pain had actually worsened before he had returned to work. *Id.* Dr. Dietrick's assessment of petitioner's condition was lateral retinacular left knee pain, status post-knee replacement, left knee synovitis with overuse, and IT band syndrome. *Id.* Dr. Dietrick recommended that petitioner modify his job duties as he had "overdone it." *Id.* at 25. Petitioner made no complaints of hip or shoulder pain or stiffness during this examination and Dr. Dietrick's examination revealed normal hip range of motion. *Id.* at 24-25.

On February 6, 2012, petitioner returned to Dr. O'Connor reporting his symptoms were alleviated by prednisone but advising he continued to have osteoarthritic pain in his shoulders. Pet. Ex. 7 at 62. Bloodwork revealed an elevated C-reactive protein ("CRP")[12] level and a normal erythrocyte sedimentation rate ("ESR").[13] *Id.* Petitioner's rheumatoid factor and cyclic citrullinated peptide antibody ("CCP")[14] levels were negative/normal. *Id.* Prednisone was continued. *Id.* at 62, 64.

Petitioner presented for a CT scan of his left knee and follow up with Dr. Dietrick on February 27, 2012. Pet. Ex. 6 at 22-23. Due to petitioner's continued complaints of left knee pain, Dr. Dietrick requested the disc of the CT scan to review it to determine if rotation of petitioner's knee implant was the cause. *Id.* at 23.

On March 1, 2012, petitioner returned to Dr. O'Connor. Pet. Ex. 7 at 59. It was noted that at his February 2012 visit, prednisone had been tapered to 15 mg per day and Mobic[15] was prescribed. *Id.* at 60. Petitioner reported continued shoulder pain with limited range of motion of the right shoulder and hip along with symptoms of carpal tunnel on the right hand with stiffness. *Id.* His CRP level was normal. *Id.* Dr. O'Connor wrote it appeared petitioner was developing seronegative rheumatoid arthritis ("RA") with PMR as the presenting sign. *Id.* Mobic was discontinued, and methotrexate prescribed. *Id.* Dr. O'Connor cautioned petitioner against smoking and alcohol use. *Id.* Petitioner's prednisone was increased to 20 mg per day. *Id.*

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[12] C-reactive protein ("CRP") is a protein used to indicate an inflammatory illness. It is elevated in patients with a bacterial infectious disease, tissue necrosis, or an inflammatory disorder. A positive test result indicates the presence, but not the cause, of the disease. *See Mosby's Manual of Diagnostic and Laboratory Tests* 165-66 (Pagana eds., 6th ed. 2018) [hereinafter *Mosby's*].

[13] Erythrocyte sedimentation rate ("ESR") is a non-specific test used to detect illnesses associated with acute and chronic infection, inflammation, and tissue necrosis or infarction. *Mosby's* at 199.

[14] Cyclic citrullinated peptide ("CCP") antibodies are useful in the diagnosis of patients with unexplained joint inflammation, particularly when the patient's rheumatoid factor is negative. These antibodies appear early in the course of RA and are present in the blood of most RA patients. *Mosby's* at 64-65.

[15] Mobic is defined as a "trademark for preparation of meloxicam." *Mobic*, DORLAND'S at 1171. Meloxicam is defined as "a nonsteroidal antiinflammatory drug used in the treatment of osteoarthritis." *Meloxicam*, DORLAND'S at 1126.

On March 14, 2012, petitioner returned to Dr. Dietrick reporting continued numbness and stabbing pain on the outside of his knee with sharp and aching pain in the front and back of his knee. Pet. Ex. 6 at 20. Dr. Dietrick discussed his review of the CT scan as showing mild internal rotation of the femoral component only, with lateral subluxation of the patella. *Id.* at 21. Dr. Dietrick suggested left arthroscopy and debridement with possible lateral retinacular release and lateral patellar facetectomy. *Id.*

Petitioner recalled that during this time, he developed pain in his wrists, hands, feet, and toes that was different from his PMR symptoms. Tr. 10. He described constant aching and tingling in his bones and specifically remembered that in March 2012, his hands were so stiff when he woke up in the morning that he was unable to make a fist. Tr. 11.

On April 5, 2012, petitioner returned to Dr. O'Connor reporting he had decreased his prednisone usage to 10 mg per day was feeling better and believed the methotrexate was unnecessary. Pet. Ex. 7 at 56. Upon examination, Dr. O'Connor noted no joint deformity, heat, swelling, erythema, or effusion of the hands and no edema or cyanosis in petitioner's extremities. *Id.* at 57. Petitioner's seronegative RA was noted to be stable and he was advised to taper his prednisone usage to 5 mg per day for one week and then discontinue use completely. *Id.* at 58.

On April 30, 2012, petitioner presented to Dr. Dietrick for pre-operative examination for lateral patellofemoral compression syndrome. Pet. Ex. 6 at 18. Dr. Dietrick recommended arthroscopic debridement and lateral release in addition to lateral patellar facet patelloplasty, rather than full revision. *Id.* at 19.

On May 2, 2012, petitioner presented to Dr. Wallace, a rheumatologist, for a second opinion. Pet. Ex. 10 at 13. He reported being in good health until the previous year when he underwent a routine left knee replacement that was immediately followed by the development of acute sciatica and a blood clot that required petitioner to take anticoagulants. *Id.* Petitioner reported that in December 2012, he had a sudden onset of achiness in his upper back and neck and "could not move." *Id.* He reported that his bloodwork in January 2012 revealed an elevated ESR level[16] and he was diagnosed with PMR. *Id.* Petitioner reported that his PMR symptoms disappeared immediately after taking steroids; he was initially prescribed 20 mg per day of prednisone by a rheumatologist who eventually tapered him down to 5 mg per day. *Id.* Petitioner stated he still had aches and pains while on 5 mg of prednisone and methotrexate, so he sought a second opinion. *Id.*; Tr. 10-11. At the time of this visit, petitioner reported feeling better but was scheduled for another knee surgery the following week. Pet. Ex. 10 at 13. Dr. Wallace noted no synovitis, cyanosis, clubbing, or edema but did discover approximately one ounce of fluid in petitioner's left knee. *Id.* Dr. Wallace prescribed 20 mg of prednisone per day for one day, and then directed petitioner to take 10 mg per day until his knee surgery. *Id.*

Petitioner underwent total knee arthroplasty for patellofemoral malalignment and lateral plica on May 7, 2012. Pet. Ex. 6 at 38-40.

[16] Petitioner never had an elevated ESR level and he had a mildly elevated CRP level on only one occasion, February 6, 2012. Pet. Ex. 7 at 62, 64.

Petitioner returned to Dr. Dietrick and his PCP three times over the following months for follow up of his left knee surgery. Pet. Ex. 6 at 14-16; Pet. Ex. 7 at 53. He reported persistent knee pain that was relieved by increasing his prednisone dosage to 15 mg per day. Pet. Ex. 7 at 53. He remained out of work due to knee pain. Pet. Ex. 6 at 14-15.

On July 12, 2012, petitioner returned to Dr. O'Connor with continued PMR complaints. Pet. Ex. 7 at 50. Petitioner had been taking methotrexate and prednisone. *Id.* He reported his symptoms worsened when he tapered the prednisone dosage to 5 mg per day, so he increased the dose to 10 mg per day. *Id.* Dr. O'Connor noted that petitioner's recent ESR and CRP levels were normal and there was no joint symptoms or inflammatory synovitis present. *Id.* Petitioner was still out of work due to his ongoing knee pain. *Id.* Dr. O'Connor directed petitioner to taper his prednisone dosage and remain on the methotrexate. *Id.* at 51.

On July 25, 2012, petitioner returned to Dr. Dietrick reporting no change or improvement in his left knee. Pet. Ex. 6 at 12. Dr. Dietrick documented that petitioner had not yet reached maximum medical improvement in his knee. *Id.* He advised petitioner it was unlikely that petitioner could ever return to his current occupation and job duties due to the severe restrictions on his physical abilities related to his knee. *Id.* at 13.[17]

On August 9, 2012, petitioner returned to Dr. O'Connor. At this visit, Dr. O'Connor documented rheumatoid arthritis ("RA") symptoms along with joint swelling, hand pain, bilateral carpal tunnel, and PMR. Pet. Ex. 7 at 47. Dr. O'Connor discovered polysynovitis[18] of petitioner's metacarpophalangeal ("MCP") and proximal interphalangeal ("PIP") joints and wrist with poor distal interphalangeal joints and pain with positive phalens[19] and tinel signs.[20] *Id.* at 48.

Between September and December 2012, petitioner returned to Drs. Dietrick and O'Connor on five occasions with complaints of aches and pains around his left knee. *See* Pet. Ex. 2 at 5; Pet. Ex. 6 at 10; Pet. Ex. 7 at 36-38, 42, 45. He was ordered to taper his prednisone prescription and continue his meloxicam and leflunomide[21] prescriptions. Pet. Ex. 7 at 36. He

[17] During the hearing, petitioner testified that he had been unable to return to work due to his PMR symptoms but later acknowledged his inability to return to work was due solely to his left knee condition. *See* Tr. 9-10, 24.

[18] Polysynovitis is defined as "general inflammation of the synovial membranes." *Polysynovitis*, DORLAND'S at 1494.

[19] Phalen sign is defined as "the appearance of numbness or parasthesias within 30 to 60 seconds during the Phalen test, a positive sign for carpal tunnel syndrome." *Phalen sign*, DORLAND'S at 1714.

[20] Tinel sign is defined as "a tingling sensation in the distal end of a limb when percussion is made over the site of a divided nerve. It indicates a partial lesion or the beginning regeneration of the nerve." *Tinel sign*, DORLAND'S at 1716.

[21] Leflunomide is defined as "an immunomodulator that inhibits pyrimidine synthesis, used as a disease-modifying antirheumatic drug in treatment of rheumatoid arthritis." *Leflunomide*, DORLAND'S at 1017.

noted improvement of his RA symptoms, rating the severity of the symptoms as 3/10. He reported his pain to be mainly sciatic pain. *Id.*

On January 9, 2013, petitioner received a final disability rating for his work-related knee injury. Pet. Ex. 6 at 1-5. He completed a form on that date noting aching in the front, outside and rear of his left knee, sharp pain on the outside and rear of his left knee and up and down his leg, with tenderness in the front and outside of his left knee. *Id.* at 42. Dr. Dietrick concluded that due to his work-related knee injury, petitioner was permanently disabled from his current job and issued a disability rating of 20% of his whole person and 50% of his lower left extremity.[22] *Id.* at 5.

On February 21, 2013, petitioner presented to Dr. O'Connor with complaints of tingling in his hands and thighs and minimal joint swelling. Pet. Ex. 7 at 30. Dr. O'Connor documented that petitioner's sciatic pain had never been evaluated for neuropathy and he was prescribed Neurontin but was unable to take it due to central nervous system changes. *Id.* Petitioner's RA symptoms were noted to be stable with no inflammatory arthritis in petitioner's feet and/or hands noted. *Id.* at 31-32. Dr. O'Connor wrote that there was little development of petitioner's RA or PMR symptoms and his ESR and CRP levels continued to be normal. *Id.* at 32. Dr. O'Connor documented that many of petitioner's complaints were related to his peripheral neuropathy from his diabetes and sciatica. *Id.*

On March 4, 2013, an MRI of petitioner's lumbar spine revealed bulging annuli at L2-3, L4-5, and L5-S1 without impingement. Pet. Ex. 5 at 38-40. On March 7, 2013, an MRI of petitioner's left shoulder revealed a labrum injury with a cyst over the humeral head.[23] *Id.* at 25. On March 11, 2013, an MRI of petitioner's cervical spine showed spondylotic ridge[24] with possible compression on the C7 nerve root. *Id*. at 27.

On March 20, 2013, petitioner returned to Dr. O'Connor. Pet. Ex. 7 at 18. Petitioner was taking methotrexate, low dose prednisone, and Lyrica with lessened pain in his hands. *Id.* Petitioner's bloodwork was normal, and his RA was noted to be stable. *Id.* Dr. O'Connor directed petitioner to increase his Lyrica prescription, but the record contained no explanation for this increase. *Id.* at 20.

On April 8, 2013, petitioner underwent EMG and a nerve conduction study ("NCS") of the upper extremities. Pet. Ex. 5 at 5-18. The EMG/NCS revealed generalized diabetic peripheral neuropathy which had progressed since 2010 and petitioner's diagnosis of diabetes four years prior. *Id.* at 8-9. The doctor opined peripheral neuropathies are predisposed to entrapment neuropathies at the cubital and carpal tunnels as well as subclinical electrical slowing across these regions. *Id.* at 9. Petitioner was noted to have moderate left carpal tunnel syndrome and mild to moderate right carpal tunnel syndrome superimposed on peripheral neuropathy. *Id.* His doctor

[22] Petitioner noted he had frequently been out of work, so he took an early retirement to maintain his health insurance. Pet. Ex. 17 at 3.

[23] Typically, an MRI of a PMR patient would show swelling of the bursae. *See* Resp. Ex. A-2 at 5.

[24] Spondylosis is defined as "dissolution of a vertebra." *Spondylosis*, DORLAND'S at 1754.

concluded that all of petitioner's symptoms discussed at this visit were *unrelated* to PMR and/or RA. *See id.* (emphasis added).

On April 24, 2013, petitioner presented to Dr. O'Connor for follow up. His RA symptoms were noted to be stable. He was directed to continue his current medications. Pet. Ex. 7 at 12.

On May 3, 2013, petitioner underwent caudal epidural and lumbar facet injections at L1-2, L2-3, L4-5, and L5-S1 for sciatic pain. Pet. Ex. 5 at 2-3.

Petitioner presented to his PCP on May 29, 2013 with dermatologic symptoms and skin lesions with abscesses. Pet. Ex. 2 at 2. Petitioner complained of possible shingles on his left shoulder and neck that had persisted for four days. *Id.* He had begun acyclovir the previous day to alleviate his symptoms. *Id.* Upon examination, hypersensitivity along the left shoulder blade was noted but no active rash was present. *Id.* Petitioner was diagnosed with likely prodromal shingles. *Id.* Petitioner's RA was noted to be stable. *Id.*

On June 19, 2013, petitioner received a shingles vaccination without event. *Id.* at 1.

On July 15, 2013, petitioner presented to Dr. O'Connor for instructions on using the Enbrel Sure Clinic Pen.[25] Pet. Ex. 7 at 7. Petitioner tolerated the medication well with no adverse reaction noted. *Id.* No explanation was documented as to why petitioner was prescribed Enbrel as he was asymptomatic at that time. *See id.*

On February 20, 2014, petitioner was diagnosed with a multinodular goiter on his thyroid by his endocrinologist. Pet. Ex. 8 at 44.

On June 23, 2014, petitioner presented to Inland Neurosurgery and Spine with bilateral leg, left ankle, and foot pain. Pet. Ex. 15 at 1-3. He had several epidural injections and had developed burning of the right thigh and anterior shin two years prior. *Id.* at 1. Petitioner also reported his EMG/NCS performed the previous year revealed muscle atrophy that began following his knee replacement in 2011. *Id.* Petitioner reported being diagnosed with RA in 2012 and that his hands constantly ached. *Id.* Petitioner's symptoms were noted to be related to his peripheral neuropathy that had never been worked up for a treatable cause. *Id.* at 3.

In July 2014, petitioner presented to his rheumatologist with tenderness and swelling at the wrists and MCPs. Pet. Ex. 7 at 130-33. His ankles and toes were tender, and he was noted to have symptomatic RA. *Id.* at 132. He was prescribed Remicade.[26] *Id.*

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[25] Enbrel is defined as a "trademark for a preparation of etanercept." *Enbrel*, DORLAND'S at 612. Etanercept is defined as "a soluble tumor necrosis factor receptor that inactivates tumor necrosis factor, used in the treatment of rheumatoid arthritis and juvenile idiopathic arthritis." *Etanercept*, DORLAND'S at 650.

[26] Remicade is a "trademark for a preparation of infliximab." *Remicade*, DORLAND'S at 1623. Infliximab is defined as a "chimeric human-murine immunoglobulin that acts as an anti-tumor necrosis factor antibody." *Infliximab*, DORLAND'S at 937.

On July 22, 2014, petitioner underwent additional EMG/NCS testing due to bilateral upper extremity numbness and tingling. Pet. Ex. 16 at 5-6. He was noted to have mild carpal and cubital tunnel syndrome, mild right C6 radiculopathy, and no brachial plexopathy or myopathy. *Id.* at 5.

On July 29, 2014, petitioner presented to the neurologist with complaints of neck pain, back pain, numbness and tingling of the upper and lower extremities, RA, and PMR. *Id.* at 1. The opinion was petitioner's neuropathy could have been caused by his diabetes. *Id.* at 1. Right C6 radiculopathy and L4-5 root irritation were noted. *Id.* at 2.

Petitioner returned to Inland Neurosurgery on August 12, 2014 for follow up. Pet. Ex. 15 at 23. Petitioner was noted to have a history of demyelinating and axonal peripheral neuropathy and cervical and lumbar degenerative disc disease with shooting pain in his leg. *Id.* Petitioner was diagnosed with peripheral neuropathy, mild right C6 radiculopathy, and a possible torn rotator cuff in his right shoulder. *Id.* at 24. He was directed to continue with Remicade for RA and PMR. *Id.*

On December 10, 2014, petitioner presented for pre-operative examination of his right shoulder. Pet. Ex. 30 at 30. Petitioner was cleared for surgery that was scheduled to take place the following week. *Id.*

On December 11, 2014, petitioner presented for follow up of his RA. *Id.* at 144. He was doing well on Remicade and leflunomide and reported a pain severity level of 2/10. *Id.*

On December 19, 2014, petitioner underwent right shoulder surgery for a torn rotator cuff reportedly resulting from lifting a couch. Pet. Ex. 32 at 66-68. Petitioner subsequently underwent extensive physical therapy for his shoulder following this surgery. *See generally* Pet. Ex. 29.

Between June and September 2015, petitioner presented for follow up of his RA on three occasions. Pet. Ex. 30 at 26, 111, 121. At each visit, petitioner's CRP and ESR levels were normal and his RA was noted to be under control. *Id*.

On September 25, 2015, petitioner underwent dual energy x-ray absorptiometry[27] to evaluate his bone density due to his frequent steroid usage. Pet. Ex. 30 at 92, 95-102. The results of this evaluation were normal. *See id.*

On October 2, 2015, petitioner presented to rheumatologist, Dr. Shinada, at Keck Medicine of USC for additional evaluation of his RA and PMR diagnoses. Pet. Ex. 26 at 8. His history was noted to be significant for diabetes, seronegative RA that began in August 2012, and PMR that began in January 2012. *Id.* Petitioner complained of pain in his hands, wrists, shoulders, and arms with stiffness in the mornings. *Id.* at 17. Petitioner stated he had previously taken Enbrel and methotrexate and was currently taking Arava daily and Remicade infusions every eight weeks. *Id.* He also took Nexium, metformin, glimepiride, Januvia, Invokana, atorvastatin, lisinopril, Lyrica, prednisone 5 mg every other day, various vitamins, and used Andro gel. *Id.* at 16-18. On examination, he had good range of motion in his shoulders, elbows, wrists, and hands with no

[27] Dual energy x-ray absorptiometry is defined as "an imaging technique that uses two low-dose x-ray beams with different levels of energy to produce a detailed image of body components; used primarily to measure bone mineral density." *Duel energy x-ray absorptiometry*, DORLAND'S at 7.

synovitis or edema noted and good range of motion in his hips and knees with some effusion of the left knee and some crepitus and effusion of the right knee. *Id.* at 18. Reduction of prednisone was suggested. *Id.*

On January 18, 2016 petitioner underwent left foot surgery which included distal chevron osteotomy, second plantar plate repair, hammertoe correction with proximal interphalangeal joint arthrodesis, extensor tendon lengthening, and repair of the second extensor tendon plantar with condylectomy around the second metatarsophalangeal joint. Pet. Ex. 32 at 55-57.

Petitioner presented for follow up of his RA twice in March 2016. Pet. Ex. 26 at 69; Pet. Ex. 30 at 22. He complained of increased stiffness at each visit due to inability to receive Remicade injections following his foot surgery. *Id*. He reported taking prednisone and Percocet for pain. *Id*.

On April 5, 2016, petitioner presented to Loma Linda Hospital Pain Management for assessment of his left foot. Pet. Ex. 28 at 13. He had complex regional pain syndrome of the left foot, lumbar radiculopathy, myalgias, arthralgias, stiffness, back pain, and redness and swelling of joints. *Id.* Petitioner was noted to be dependent on opioid therapy and could not function or perform daily activities of living without them. *Id.* at 18. He was ultimately diagnosed with idiopathic progressive polyneuropathy. *Id.* at 21, 36.

Petitioner presented for follow up related to his left foot surgery on April 19, 2016. Pet. Ex. 32 at 20. Petitioner continued to complain of persistent pain despite a sympathetic nerve block performed on April 5, 2016. He continued to experience burning in his forefoot, but he could walk with a substantial amount of pain. *Id.*

Between May 11, 2016 and August 24, 2016, petitioner followed up for his RA on three occasions. *See* Pet. Ex. 30 at 6, 10, 21.

On September 15, 2016, petitioner presented to the orthopedist with complaints of left shoulder and lumbar spine pain. Pet. Ex. 32 at 11. He reported a previous repair of his right shoulder for labral tear with debridement, subacromial decompression, and distal clavicle excision. *Id.* At this visit, he complained of left shoulder pain with reaching in different directions and back pain. *Id.* He was diagnosed with rotator cuff tendinopathy of the left shoulder and degenerative lumbar spondylosis. *Id.*

An MRI of petitioner's lower back performed on September 23, 2016 revealed loss of disc space at L2-3 with new moderate endplate degenerative changes and prominent endplate signal abnormality. Pet. Ex. 30 at 90. There was a progression of retrolisthesis[28] to moderate bilateral neural foraminal narrowing at that level and a posterior disc bulge and ligamentum flavum thickening as well as moderate facet degenerative changes at L3-4. *Id.* at 91. There was superiorly migrated disc material in the left subligamentous space, new from prior study with overall spinal canal stenosis compared to last study. *Id.* Moderate bilateral neural foraminal narrowing was also present. *Id.*

[28] Retrolisthesis is defined as "retrospondylolisthesis," which is defined as "posterior displacement of one vertebral body on the subjacent body." *Retrolisthesis*, DORLAND'S at 1636; *Retrospondylolisthesis*, DORLAND'S at 1636.

On October 3, 2016, petitioner presented to his orthopedist with complaints of lower back pain. Pet. Ex. 32 at 5. It was noted that petitioner's pain seemed to be mechanical rather than neurologic. *Id.* Muscle spasms from T10 to L3 were noted. *Id.* MRIs revealed severe degenerative disc changes, stenosis, and tightness. *Id.* Voltaren and physical therapy were recommended. *Id.* at 6.

On October 24, 2016, petitioner returned to the orthopedist with complaints of episodic acute back pain. *Id.* at 3. He had previously seen improvement with physical therapy and Voltaren. *Id.* He was sent back to physical therapy and directed to continue with Voltaren. *Id.* If necessary, a bilateral L2-L3 root block would be discussed. *Id.*

On October 26, 2016, petitioner returned to his PCP. Pet. Ex. 30 at 1. He was noted to have been initially diagnosed with PMR in January 2012 and treated with prednisone and methotrexate. *Id.* He was ultimately diagnosed with seronegative rheumatoid arthritis after worsening of joint pain and was treated with Enbrel for a year, then switched to leflunomide and Remicade infusions. *Id.* He had a left bunionectomy and tendon repair with continued burning of the left foot and ankle. *Id.* He was taking Percocet for pain and had tendinosis of the left shoulder. *Id.* His active problems at this visit were RA, bicipital tendinitis of the left shoulder, type II diabetes, hypertension, hyperlipidemia, lower back pain, neuropathy, rosacea, PMR, sciatica, and history of shoulder joint pain. *Id.* at 1-2. He was directed to continue his current medications and return in two months for follow up. *Id.* at 5.

As of 2017, petitioner continued to have severe pain in his foot and was diagnosed with reflex dystrophy syndrome of the left foot as well as diabetic neuropathy. Pet. Ex. 34 at 3-4. Sympathetic nerve blocks, Voltaren, and Robaxin were used for pain. *Id.* at 4.

III. The Experts

A. Petitioner's Experts: Dr. Kristin Gowin and Dr. Michael Gurish

Dr. Gowin earned a Bachelor of Arts in Zoology from the University of Miami, an M.D. from the University of Cincinnati College of Medicine, and a Master of Science in clinical epidemiology from the University of Pennsylvania School of Medicine. Tr. 42; Pet. Ex. 21 at 1. In 1990, Dr. Gowin became a resident in internal medicine at the Hershey Medical Center and spent a year on the faculty there as an instructor in the Division of General Internal Medicine. Pet. Ex. 21 at 1. In 1994, she went to the University of Pennsylvania, where she was a fellow in rheumatology and did her degree in clinical epidemiology while there. *Id.*; Tr. 42. She left the University of Pennsylvania in 1999 to pursue private practice. Tr. 42. Dr. Gowin is board certified in rheumatology and was board certified in internal medicine but chose not to re-certify. *Id.*

Dr. Gowin is currently a partner at the Asheville Arthritis and Osteoporosis Center and a sub-investigator in their clinical trial unit. Tr. 41-42, 66; Pet. Ex. 21 at 1. Dr. Gowin actively sees approximately 1,000 patients, the frequency depends on the condition for which they are being seen. Tr. 42-43. Her patients typically suffer from different kinds of arthritis, including rheumatoid arthritis, psoriatic arthritis, and other inflammatory conditions like polymyalgia rheumatica and inflammatory eye disease. Tr. 43. As a sub-investigator, Dr. Gowin contributes patients to clinical

trials but is only involved in the clinical gathering of the scientific data, rather than researching and/or writing articles related to the data. Tr. 68. The drug companies oversee the trials, collection of data, analysis, and reporting of the results. *Id.* The most recent study she is involved in is for the GIACTA trial, researching a treatment for giant cell arteritis. Tr. 69. Dr. Gowin agreed that she has not published a paper since 2001, and the papers she published involved data analysis based on studies of conditions and diseases or statistical analysis of data to assess causal relationship were on gout. Tr. 70-71. Dr. Gowin explained that while she has had some case reports presented at meetings as poster presentations, none were related to the flu vaccine. Tr. 71-72. She has not been involved in cases or investigations related to rheumatic diseases. Tr. 71.

Dr. Gowin opined there is biologic plausibility for the influenza vaccine to cause either onset or flare of RA. Pet. Ex. 20 at 4.[29] She proposed that the vaccine is the initiating event activating T-lymphocytes. *Id.* (citing Pet. Ex. 20-G at 1).[30] Though no single driving antigen has been identified, there may be several that initiate RA in genetically susceptible individuals by molecular mimicry. *Id.* (citing Pet. Ex. 20-G at 1). "The activation of the immune cascade causes ingrowth of blood vessels, recruitment of further immune cells that produce antibodies like the classic RA factor and anti-CCP and proliferation of the lining of the joint (synovium) that eventually causes damages to the cartilage." *Id.* (citing Pet. Ex. 20-G at 4-5). Dr. Gowin submitted that lymphocytes specific for influenza virus have been found in the joints of patients with RA, suggesting that they may participate in onset or flare of the disease. *Id.* (citing Pet. Ex. 20-G at 4-5). "Immunologic changes can be seen even several years prior to the onset of symptoms for patients who will develop rheumatoid arthritis given the appropriate trigger to the immune system. Various viruses or their analogous vaccines have been postulated as triggers for autoimmunity." *Id.* (citing Pet. Ex. 20-H at 1-2).[31]

Dr. Gurish[32] had a Ph.D. in immunology and was an Associate Professor in Medicine in the Division of Rheumatology, Immunology and Allergy at Brigham and Women's Hospital and Harvard Medical School. Pet. Ex. 24 at 1. Dr. Gurish's focus was on mast cell development, trafficking and function in innate and adaptive immune responses, pathways of inflammation with sterile injury, allergic inflammation in lungs and intestine, and parasitic infection. *Id.* He was an instructor of medicine in molecular biology controlling expression of mast cell secretory granule proteases and markers of phenotype diversity. *Id.* Dr. Gurish was a consultant and member of the

[29] Due to the inconsistent filing of medical literature by both parties, there are points in this decision where ECF page numbers are utilized and other times when page numbers from the articles themselves will be utilized.

[30] Peter H. Schur & Gary S. Firestein, *Pathogenesis of Rheumatoid Arthritis*, UPTODATE.COM (Oct. 12, 2011), filed as "Pet. Ex. 20-G."

[31] R.J.W. Venables et al., *Diagnosis and Differential Diagnosis of Rheumatoid Arthritis*, UPTODATE.COM (Mar. 3, 2015), filed as "Pet. Ex. 20-H."

[32] Dr. Gurish passed away on May 31, 2018.

Scientific Advisory Board for MedImmune, Inc.[33] in Gaithersburg, MD and listed himself as an Immunology Expert Witness for the Law Firm of Conway, Homer & Chin-Caplan, P.C. *Id.* at 2.

Dr. Gurish opined that molecular mimicry as well as epitope spreading, bystander activation, and polyclonal activation were responsible for petitioner's development of autoimmunity. Pet. Ex. 23 at 3 (citing Pet. Ex. 23-D at 1043-44;[34] Pet. Ex. 23-E at 85).[35] He explained each mechanism in detail and noted that while the activation of autoimmunity is not well understood, there are several factors that likely contribute to the breakdown of this system, including genetics, environmental exposure to toxins (smoking), infections, or vaccinations. *Id.* at 3-4 (citing Pet. Ex. 23-I at 648-49;[36] Pet. Ex. 23-J at 1192).[37] Dr. Gurish opined that the progression of petitioner's symptoms over the six weeks following the flu vaccine coupled with petitioner's initial positive response to steroids suggested that the underlying disease was of a less aggressive nature (PMR) that eventually amplified into RA. *Id.* at 5. Dr. Gurish concluded that petitioner's rheumatic disease was induced or made clinically evident by the receipt of the flu vaccine as the final significant immunological challenge he experienced prior to the development of PMR, and subsequently RA. *Id.*; Pet. Ex. 25 at 4.

B. Respondent's Expert: Dr. Mehrdad Matloubian

Dr. Matloubian is an Associate Adjunct Professor at the University of California at San Francisco School of Medicine. Resp. Ex. B. at 1. Dr. Matloubian received a Bachelor of Science

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[33] MedImmune, Inc. was the research and development arm of the global drug giant AstraZeneca and manufactures, markets and/or distributes several drugs in the United States including the FluMist Quadrivalent, a live influenza virus vaccine. Andrew Pollack, *AstraZeneca Buys MedImmune for $15.6 Billion*, NEW YORK TIMES (April 24, 2007), https://www.nytimes.com/2007/04/24/business/24drug-web.html. In June of 2009, MedImmune won a Department of Health and Human Services Contract worth $90 million to continue making its seasonal FluMist vaccine and to develop a vaccine targeted specifically at the H1N1 virus. Michael S. Rosenwald, *MedImmune Wins Key Contract to Develop Swine Flu Vaccine*, WASHINGTON POST (June 2, 2009), http://www.washingtonpost.com/wp-dyn/content/article/2009/06/01/AR2009060101608.html?noredirect=on. MedImmune then won a second contract to test its nasal spray flu technology as a viable treatment for H1N1. *Id.* The MedImmune name and branding were recently discontinued in favor of AstraZeneca. Jacob Bell, *AstraZeneca Retires MedImmune Name Amid Sales Turnaround*, BIOPHARMA DIVE (Feb. 14, 2019), https://www.biopharmadive.com/news/astrazeneca-retires-medimmune-name-amid-sales-turnaround/548489/.

[34] Robert S. Fujinami & Michael B. A. Oldstone, *Amino Acid Homology Between the Encephalitogenic Site of Myelin Basic Protein and Virus: Mechanism for Autoimmunity*, 230 SCIENCE 1043 (1985), filed as "Pet. Ex. 23-D."

[35] Carol L. Vanderlugt & Stephen D. Miller, *Epitope Spreading in Immune-Mediated Diseases: Implications for Immunotherapy*, 2 NAT. REV. IMMUNOL. 65 (2002), filed as "Pet. Ex. 23-E."

[36] Nancy Agmon-Levin et al., *Vaccines and Autoimmunity*, 5 NAT. REV. RHEUMATOL. 648 (2009), filed as "Pet. Ex. 23-I."

[37] Nancy Agmon-Levin et al., *Ten Cases of Systemic Lupus Erythematosus Related to Hepatitis B Vaccine*, 18 LUPUS 1192 (2009), filed as "Pet. Ex. 23-J."

degree in biochemistry, a Ph.D., and an M.D. from UCLA. *Id*. He completed his residency in internal medicine and a fellowship in rheumatology. *Id*.; Tr. 176. Dr. Matloubian is an immunologist and board-certified rheumatologist who actively evaluates and treats patients with complex autoimmune diseases, including rheumatoid arthritis, lupus, mixed connective tissue disease, PMR, different types of inflammatory myositis, and vasculitis. Tr. 182; Resp. Ex. B. at 2. Dr. Matloubian also has a Ph.D. in microbiology and immunology with an emphasis in virology. Resp. Ex. B at 2. His post-doctoral training focused on how lymphocytes, or white blood cells, travel from different tissues and different organs, and immune responses to acute and chronic viral infections focusing on both innate and adaptive immune responses. Tr. 177. He is familiar with the mechanics of immunology and how the immune system responds to various antigens. *Id.* He has published several articles on immunology, particularly on lymphocyte trafficking, in peer-reviewed journals. Resp. Ex. B at 6-9. His other research interests include autoimmune disease, interferons, plasmacytoid dendritic cells, antibody responses in chronic viral infection, chemokines, and chronic inflammation. *Id*. at 2.

Dr. Matloubian disagreed that molecular mimicry was the correct mechanism in this case, stating, "For molecular mimicry to be relevant to a disease in question, the natural infection, i.e. influenza virus in this case, should also lead to development of that disease in some people." Resp. Ex. A at 11. He opined there is no evidence associating influenza virus with PMR or RA and that because the components of the vaccine are derived from the influenza virus, "it is highly unlikely that a mechanism such as molecular mimicry can cause arthritis due to this vaccine." *Id.* Dr. Matloubian further highlighted the absence of a causal relationship between the inactivated flu vaccine and the development of inflammatory arthritis. *Id.* at 12; Resp. Ex. C at 7. Dr. Matloubian concluded he strongly believed petitioner would have developed PMR and/or RA even if he had not received the flu vaccine on October 14, 2011. Resp. Ex. C at 7.

The opinions of the experts are set forth in greater detail below.

IV. Findings of Fact

A. Legal Standard Regarding Fact Finding

The process for making determinations in Vaccine Program cases regarding factual issues, such as the timing of onset of petitioner's alleged injury, begins with analyzing the medical records, which are required to be filed with the petition. 42 U.S.C. § 300aa-11(c)(2). Medical records created contemporaneously with the events they describe are presumed to be accurate and "complete" such that they present all relevant information on a patient's health problems. *Cucuras v. Sec'y of Health & Human Servs*., 993 F.2d 1525, 1528 (Fed. Cir. 1993); *Doe/70 v. Sec'y of Health & Human Servs*., 95 Fed. Cl. 598, 608 (2010) ("Given the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law."). This presumption is based on the linked proposition that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in an accurate manner, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs.*, No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl.

Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs*., 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d. 1525 (Fed. Cir. 1993) ("[I]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms. It is equally unlikely that pediatric neurologists, who are trained in taking medical histories concerning the onset of neurologically significant symptoms, would consistently but erroneously report the onset of seizures a week after they in fact occurred.").

Where medical records are clear, consistent, and complete, they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs*., No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—particularly where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health and Human 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) ("It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")). In making contemporaneous reports, the declarant's motivation for accurate explication of symptoms is more immediate, as opposed to testimony offered after the events in question, which is considered inherently less reliable. *Reusser v. Sec'y of Health & Human Servs*., 28 Fed. Cl. 516, 523 (1993).

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. *Vallenzuela v. Sec'y of Health & Human Servs*., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); *see also Eng v. Sec'y of Health & Human Servs*., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (explaining that §13(b)(2) "must be construed so as to give effect also to §13(b)(1) which directs the special master or court to *consider* the medical records [reports, diagnosis, conclusions, medical judgment, test reports, etc.] but does not require the special master or court *to be bound* by them.") (emphasis in original). There are situations in which compelling oral testimony may be more persuasive than written records—for instance, in cases where records are deemed to be incomplete or inaccurate.[38] *Campbell v. Sec'y of Health & Human 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 ("Written records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent.") (quoting *Murphy*, 23 Cl. Ct. at 733 (1991). However, when such testimony is used to overcome the presumption of accuracy afforded to contemporaneous medical records, it must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of*

[38] 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 & Human Servs*., 110 Fed Cl. 184, 203-04 (2013), *aff'd,* 746 F.3d 1334 (Fed. Cir. 2014).

Health & Human Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). A determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu v. Sec'y of Health & Human Servs*., 569 F.3d 1367, 1379 (Fed. Cir. 2009); *Bradley v. Sec'y of Health & Human Serv*s., 991 F.2d 1570, 1575 (Fed. Cir. 1993). The special master must then determine whether to afford greater weight to contemporaneous medical records or testimony given at hearing. This decision must in turn be supported by evidence that it was the result of a rational determination. *Burns by Burns v. Sec'y of Health & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993). Ultimately, "the record as a whole" must be considered. 42 U.S.C. § 300aa-13(a).

B. Polymyalgia Rheumatica and Rheumatoid Arthritis

1. Polymyalgia Rheumatica

Polymyalgia rheumatica ("PMR") is an inflammatory rheumatic disease which commonly presents in patients over the age of 50 with approximately 1 out of every 150 persons over the age of 50 being affected by the disease. Pet. Ex. 43 at 117;[39] Resp. Ex. A-2 at 1053.[40] The cause of PMR is unknown; both environmental and genetic factors are suspected to play a role. *See* Pet. Ex. 43 at 117.

PMR is commonly characterized by startingly abrupt onset of pain and morning stiffness in the shoulders and pelvic girdles. *Id.* "However, distal manifestations, which include peripheral arthritis and distal extremity swelling with pitting edema, may occur in about half the cases." Resp. Ex. A-2 at 1053. Ultrasound evidence of bilateral subacromial/subdeltoid bursitis in the shoulders may represent a hallmark of PMR. *Id.* Elevated inflammatory markers of CRP, ESR, and creatine kinase ("CK") levels have been shown to be the hallmark of PMR. *Id.*; Pet. Ex. 44 at 345-46.[41] Steroids typically have a dramatic effect on the recovery and treatment in PMR patients. Pet. Ex. 43 at 117. Clinical presentation along with dramatic response to steroids is used to make the diagnosis. *See id.*; Pet. Ex. 44 at 346.

2. Rheumatoid Arthritis

Rheumatoid arthritis ("RA") is a common autoimmune disease associated with progressive disability, systemic complications, early death, and socioeconomic cost. Resp. Ex. A-3 at 2205.[42]

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[39] Mashal Salehi, Pavan Luckoor & Ed Marie Sibal-Gomez, *Polymyalgia Rheumatica After Influenza Vaccine*, 8 J. MED. CASES 117 (2017), filed as "Pet. Ex. 43."

[40] Fabrizio Cantini et al., *Shoulder Ultrasonography in the Diagnosis of Polymyalgia Rheumatica: A Case-Control Study*, 28 J. RHEUMATOL. 1049 (2000), filed as "Resp. Ex. A-2."

[41] Kentaro Iwata & Yasushi Mizuno, *A Case of Polymyalgia Rheumatica Following Influenza B Infection*, 8 INTNT'L J. GEN. MED. 345 (2015), filed as "Pet. Ex. 44."

[42] Iain B. McInnes & Georg Schett, *The Pathogenesis of Rheumatoid Arthritis*, 365 N. ENGL. J. MED. 2205 (2011), filed as "Resp. Ex. A-3."

RA is characterized by synovial inflammation and swelling, autoantibody production – including rheumatoid factor and anti-citrullinated protein antibodies ("ACPA") – cartilage and bone destruction, and systemic features including cardiovascular, pulmonary, psychological, and skeletal disorders. *Id.* The pathophysiology of RA is considered a clinical syndrome spanning several disease subsets which each entail several inflammatory cascades in which persistent synovial inflammation and associated damage to articular cartilage and underlying bone are present. Resp. Ex. C-13 at 1094.[43] A key inflammatory cascade includes overproduction and over expression of tumor necrosis factor ("TNF"). *Id.* TNF has many causes including interactions between T and B lymphocytes, synovial like fibroblasts and macrophages. *Id.* This leads to overproduction of cytokines such as interleukin-6 ("IL-6"), which drives persistent inflammation and joint destruction. *Id.*

Individuals with RA are considered either seropositive or seronegative. In seropositive RA, the long-established association with the human leukocyte antigen ("HLA")-DRB1 locus has been confirmed in patients who test positive for rheumatoid factor or ACPA; alleles that contain a common amino acid motif, QKRAA, in the HLA-DRB1 regions, termed the "shared epitope," confer particular susceptibility. Resp. Ex. A-3 at 2206. These findings suggest that some predisposing T-cell repertoire selection, antigen presentation, or alteration in peptide affinity has a role in promoting autoreactive adaptive immune responses. *Id.* Other possible mechanisms include molecular mimicry of the shared epitope by microbial proteins, increased T-cell senescence induced by shared epitope-containing HLA molecules, and a potential proinflammatory signaling function unrelated to the role of the shared epitope in antigen recognition. *Id.* Infectious agents – Epstein-Barr virus, cytomegalovirus, proteus species and E-coli – and their products have been linked with RA through some form of molecular mimicry. *Id.*

While seropositive RA has a presence of autoantibodies, none exist or are known for seronegative RA, suggesting that the pathogenesis or mechanisms that lead to the breakdown of tolerance and generation of autoimmunity in each case may be different. Resp. Ex. C at 3 (citing Pet. Ex. 22 at 2). In practice, seronegative RA is determined by clinical diagnosis and no distinction is made for purposes of treatment because the medications prescribed are the same and are limited. *Id.*

In a new criteria set, classification as "definite RA" is based on the confirmed presence of synovitis in at least one joint, absence of an alternative diagnosis that better explains the synovitis, and achievement of a total score of 6 or greater (of a possible 10) from the individual scores in 4 domains: number and site of involved joints (score range 0-5), serologic abnormality (score range 0-3) elevated acute phase response (score range 0-1), and symptom duration (2 levels; range 0-1). Resp. Ex. A-7 at 2570.[44]

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[43] David L. Scott et al., *Rheumatoid Arthritis*, 376 LANCET 1094 (2010), filed as "Resp. Ex. C-13."

[44] American College of Rheumatology/European League Against Rheumatism Collaborative Initiative, *2010 Rheumatoid Arthritis Classification Criteria*, 62 ARTHRITIS RHEUMATOL. 2569 (2010), filed as "Resp. Ex. A-7."

RA and PMR have similar clinical presentations and it is still debated as to whether they are the same entity. Resp. Ex. A-8 at 1021, 1023.[45] The hall mark of both conditions is morning stiffness or stiffness following extended periods of rest. Resp. Ex. A-5 at 2.[46] . The stiffness in and around the joints may continue for hours and typically improves with activity. *Id.* RA affects the synovial joints and begins in the MCP, PIP, and MTP joints followed by the wrists, knees elbows, ankles, hip and shoulders. *Id.* It may affect the cervical spine but rarely involves the rest of the spine. *Id.* The MTP joints in patients' feet are involved early in the onset of symptoms in almost all RA cases and are second only to the hand in terms of the problems they cause. *Id.* Subluxation of the toes at the MTP joints is common and leads to dual problems of skin ulceration on the top of the toes and painful ambulation because of loss of the cushioning of the pads that protect the heads of the metatarsals. *Id.* at 9.

The onset of RA has been associated with adverse life events with molecular explanations emerging from animal models of inflammation which show a link between the hypothalamic-pituitary- adrenal axis and cytokine production. Resp. Ex. A-3 at 2206-07. "The central nervous system is normally involved in immune regulation and homeostasis, and neuroimmunologic interactions regulate disease development in rodent models of arthritis either locally (several neurotransmitters are expressed in synovitis in RA) or centrally (cytokines are rapidly up regulated in the hypothalamus during peripheral inflammation)." *Id.* Humoral adaptive immunity is integral to RA. *Id.* at 2209. Plasma cells are more widely distributed in the synovium and juxta-articular bone marrow. Plasma cells are not targeted by anti CD20 antibodies and autoantibody levels are variably altered after treatment, these clinical observations suggest that the role of B cells and their progeny in the pathogenesis of RA goes beyond antibody production to include autoantigen presentation and cytokine production. *Id.* at 2209-10.

Smoking, alcohol intake, high BMI, high birthweight, lower socioeconomic status, and periodontal disease increase patient's risk of RA. Resp. Ex. C-11 at 523.[47] Smoking is a dominant risk factor restricted to patients with anti-CCP diseases including seropositive RA. *Id.*

C. The Affidavits and Testimony of Petitioner and His Wife and Onset of Petitioner's PMR and RA

The onset of petitioner's symptoms varies between the medical records and the testimony of petitioner and his wife. Petitioner and his wife both testified that he was relatively healthy before his receipt of the flu vaccination in October 2011. Both initially stated that his pain and morning stiffness began in mid- to late-November 2011. Tr. 6, 27-28. Petitioner associated this with his return to work following knee replacement surgery. Tr. 9-10, 29-30; Pet. Ex. 17 at 2; Pet. Ex. 19

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[45] R. Caporali et al., *Presenting Features of Polymyalgia Rheumatica (PMR) and Rheumatoid Arthritis with PMR-Like Onset: A Prospective Study*, 60 ANN. RHEUM. DIS. 1021 (2001), filed as "Resp. Ex. A-8."

[46] James R. O'Dell et al., *Rheumatoid Arthritis: Introduction*, *in* CURRENT DIAGNOSIS & TREATMENT: RHEUMATOLOGY (John B. Imboden et al. eds., 3rd ed. 2013), http://accessmedicine.mhmedical.com (last visited July 12, 2015), filed as "Resp. Ex A-5."

[47] Laura Hunt & Paul Emery, *Defining Populations at Risk of Rheumatoid Arthritis: The First Steps to Prevention*, 10 NAT. REV. RHEUMATOL. 521 (2014), filed as "Resp. Ex. C-11."

at 1-2. However, Mrs. [redacted] affirmed that petitioner did not return to work until mid-December, just before the Christmas break. Pet. Ex. 19 at 2-3. Further, the medical records prior to petitioner's vaccination document ongoing complaints of neck and shoulder pain, sciatic pain with numbness and tingling in his legs. Pet. Ex. 2 at 16; Pet. Ex. 17 at 1; *see generally* Pet. Ex. 12-13, 29, 36-38, 40. On the day of the allegedly causal vaccination, petitioner presented with complaints of right thigh burning, tingling, and numbness for one month. Pet. Ex. 2 at 16; Pet. Ex. 17 at 1. The medical records after his vaccination consistently document petitioner's reporting onset of PMR symptoms as December 2011, a diagnosis of PMR in January 2012, suspected RA in March 2012 and diagnosis of seronegative RA in August 2012. Pet. Ex. 6 at 67; Pet. Ex. 7 at 47-48; *see* Pet. Ex. 10 at 13; Pet. Ex. 26 at 8. Notably, both petitioner and his wife avoided any testimony regarding his ongoing left knee issue or his being out of work since May 2011 as a result of his work-related knee injury.

In considering petitioner and his wife's testimony in concert with the contemporaneous medical records, the medical records clearly reference onset of the alleged PMR symptoms in December 2011. *See Lowrie*, 2005 WL 6117475, at *20. Petitioner and his wife's testimony carried less weight in this matter due to changes in their testimony when confronted with records filed in this matter. Initially, petitioner and his wife postured that petitioner's pain upon his return to work, pain at the Christmas party, inability to walk, inability to participate in New Year's Eve and a family wedding were the results of his PMR symptoms, only to later concede that the pain was actually related to the ongoing debilitating problems associated with his prior knee surgery. Tr. 9-10, 29-30. Petitioner and his wife also suggested that his early retirement was due to the progression of PMR and RA, when in fact he was determined to be unable to return to his job by Dr. Dietrick due to his knee injury, was assessed with a disability rating by workers' compensation, and recovered an award from workers' compensation as a result thereof. *See id.*; Pet. Ex. 7 at 24. Further, petitioner admitted in his affidavit that he retired early so he could maintain his health insurance because he was out of work for so long with his knee issues. Pet. Ex. 17 at 3.

Ultimately, based on the medical records and testimony, I find that the onset of petitioner's alleged PMR was late December 2011, with seronegative RA suspected in March 2012 and diagnosed in July of 2012.

V. Legal Framework

A. Causation

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health &*

Human Servs., 165 F.3d 1344, 1352 (Fed. Cir. 1999).[48] Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, petitioners must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioners show by preponderant evidence that a vaccination petitioner received caused his or her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs.*, 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each of the *Althen* prongs requires a different showing. Under the first *Althen* prong, petitioner 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 (citation omitted). To satisfy this prong, petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id.* at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde*, 746 F.3d at 1341.

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs.*, 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *id.* at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a

[48] The Vaccine Act also requires petitioners to show by preponderant evidence that the "residual effects or complications" of the alleged vaccine-related injury lasted for more than six months. § 11(c)(1)(D)(i). It is undisputed that this six-month requirement is satisfied in this case.

logical sequence of cause and effect show[s] that the vaccination was the reason for the injury," *Paluck v. Sec'y of Health & Human Servs*., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375).

However, medical records and/or statements of a treating physician's view 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 12(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 & Human Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("[T]here 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 & Human Servs*., 100 Fed. Cl. 742, 749 (2011) (determining it is not arbitrary or capricious for a special master to weigh competing treating physicians' conclusions against each other), *aff'd*. 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs*., 100 Fed. Cl. 119, 136 (2011), *aff'd*, 463 Fed. Appx. 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs*., No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 229, 2011), *mot. for review den'd*, 100 Fed. Cl. 344 (Sept. 29, 2011), *aff'd*, 475 Fed. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires that petitioner establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan v. Sec'y of Health & Human Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id*. However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id*.; *see also Locane v. Sec'y of Health & Human Servs*., 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Finally, although this decision discusses many but not all of the literature in detail which was submitted by the parties, I reviewed and considered all of the evidence submitted including all medical records and literature filed in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs*., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs*., 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

B. Expert Reports and Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human 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 (1991).[49] *Cedillo v. Sec'y of Health & Human Servs*., 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs*., 195 F.3d. 1302, 1316 (Fed. Cir. 1999)).

The *Daubert* factors are usually employed by judges in the performance of their evidentiary gatekeeper roles to exclude evidence that is unreliable and/or could confuse the jury. In Vaccine Program cases, by contrast, these factors are used in the weighing of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human 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 of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742-45. In this case, as in numerous other Vaccine Program cases, *Daubert* has not been employed to determine what evidence should be admitted, but rather to determine whether expert testimony offered is reliable and/or persuasive.

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 & Human 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. 136, 146 (1997)); *see also Isaac v. Sec'y of Health & Human 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 v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1325-26 (Fed. Cir. 2010) ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human 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.").

VI. Analysis

Because petitioner does not allege an injury listed on the Vaccine Injury Table, his claim is classified as "off-Table." As noted above, for petitioner to prevail on an "off-Table" claim, he

[49] The *Daubert* 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).

must show by preponderant evidence that B.L.'s injury resulted from the vaccination at issue. *Capizzano*, 440 F.3d at 1320. Doing so shifts the burden to respondent to show that the injury was caused by factors unrelated to the vaccination. *Deribeaux*, 717 F.3d at 1367.

Application of the *Althen* prongs reveals evidentiary deficiencies in petitioner's claim: (1) petitioner failed to offer a persuasive or reliable medical theory; (2) the theory provided is not applicable to the facts of petitioner's case; and (3) petitioner has not established a medically acceptable timeframe in which his symptoms could have begun or developed.

A. Petitioner has failed to articulate a plausible medical theory causally connecting the flu vaccine and PMR and/or RA.

To satisfy *Althen* Prong I, petitioner must present a "sound and reliable medical or scientific explanation" causally connecting the vaccine to his alleged injuries. *Knudsen*, 35 F.3d at 548. Petitioner's causation theory has several deficiencies.

Dr. Gowin opined there is biologic plausibility for the influenza vaccine to cause either onset or flare of RA. Pet. Ex. 20 at 4. She suggested that the initiating event activating T-lymphocytes could be a vaccine. *Id.* (citing Pet. Ex. 20-G at 1). Though no single driving antigen has not been identified, there may be several that initiate RA in genetically susceptible individuals by molecular mimicry. *Id.* (citing Pet. Ex. 20-G at 1). Dr. Gowin submitted an article that stated lymphocytes specific for influenza virus have been found in the joints of patients with RA suggesting that they may participate in onset or flare of the disease. *Id.* (citing Pet. Ex. 20-G at 4-5). "Immunological changes can be seen even several years prior to the onset of symptoms for patients who will develop rheumatoid arthritis given the appropriate trigger to the immune system. Various viruses or their analogous vaccines have been postulated as triggers for autoimmunity." *Id.* (citing Pet. Ex. 20-H at 1-2). She did not explain how this relates to petitioner, whose RA is classified as seronegative as he does not have a positive rheumatoid factor or anti-CCP.

Dr. Gowin acknowledged that she is not an immunologist but stated that her training and experience in rheumatology is "pretty good clinical immunology training." Tr. 72-73. Dr. Gowin submitted that molecular mimicry is the mechanism by which the flu vaccine caused and/or triggered petitioner's PMR and subsequent RA, stating molecular mimicry is "one of the things that I think people hang their hat on sometimes…where they talk about molecular mimicry as an autoimmune trigger," it's a reliable and plausible theory; "… molecular mimicry, [which] has been put forth as a mechanism for a lot of these reactive syndromes." Tr. 56, 77.

In support of her opinions, Dr. Gowin submitted a case study of an 85-year-old woman with a prior history of diabetes and dyslipidemia who developed a high fever, tested positive for influenza B by rapid influenza test, and was treated with intravenous peramivir.[50] Pet. Ex. 44 at 346-47. After her symptoms resolved, she developed myalgia with tenderness over the trapezius muscles, elbow joints bilaterally, both thighs, particularly on the lateral side, and both calves. *Id.*

[50] Peramivir is a neuraminidase inhibitor indicated for treatment of acute uncomplicated influenza in patients over the age of two who have been symptomatic for no more than two days. *Peramivir*, MEDSCAPE, https://reference.medscape.com/drug/rapivab-peramivir-999307 (last visited June 26, 2019).

at 346. There were no thickened temporal arteries and no tenderness on palpation. *Id.* She had no lymphadenopathy, rash, or arthritis but was ultimately diagnosed with PMR likely attributed to influenza B. *Id.* This article, like all the articles submitted, noted that the etiology of PMR remains unknown. *Id.*

Dr. Gowin submitted that multiple case reports show autoimmune disease onset after influenza vaccine, including cases of RA, PMR, systemic lupus, and reactive arthritis "in an HLA-B27 positive patient."[51] Pet. Ex. 20 at 3 (citing Pet. Ex. 20-A at 572;[52] Pet. Ex. 20-B at 645[53]). Dr. Gowin added there are cases of severe flares of RA with rheumatoid vasculitis in a patient with previously controlled disease and RA flares in a trial noted after influenza vaccines in patients with autoimmune disease. *Id.* Dr. Gowin stated that, though researchers deemed influenza vaccine safe in these patients, 35% had RA flare, 20% of lupus patients had significant flare and 3% of other patients with autoimmune disease had flares, indicating that 21% of patients had flare of their autoimmune disease after influenza vaccination. *Id.* (citing Pet. Ex. 20-D at 53-54[54]). Dr. Gowin conceded that a more recent study on influenza vaccine and safety did not show any significant change in flare rate, but this, Dr. Gowen stated, was because the study was too small, with only 82 patients, of whom 59% were on steroids, which would blunt the immune response. *Id.* (citing Pet. Ex. 20-E at 193-94[55]). Dr. Gowin stated that several other studies did not show flares in patients with existing RA, including a more recent study of 113 cases of RA thought to be associated with influenza vaccination, producing an elevated risk of 1.3, or 36% increased risk. *Id.* at 3-4 (citing Pet. Ex. 20-F at 6594-96[56]). Dr. Gowin opined that the case control portion did not show any increased risk because there were design flaws which failed to adjust for confounding factors, such as smoking, which is a known risk of RA. *Id.* at 4. She further stated that control studies are generally considered less robust evidence than cohort studies. *Id.* The study was underpowered to find small differences between the cases and the controls, and they used the 1988 American

[51] Human leukocyte antigens ("HLA") are antigens present on the surface of white blood cells and on the surface of all nucleated cells in other tissues. *Mosby's* at 274. They are most easily detectable on the surface of lymphocytes. *Id.* HLA testing is used to assist in the diagnosis of certain diseases. *Id.* For example, the HLA-B27 antigen is commonly present in patients with Reiter syndrome, anterior uveitis, and Graves' disease. *Id.* at 274-75.

[52] M.A. Brown & J.V. Bertouch, *Rheumatic Complications of Influenza Vaccination*, 5 AUST. N.Z. J. MED. 572 (1994), filed as "Pet. Ex. 20-A."

[53] D. Biasi et al., *A Case of Reactive Arthritis After Influenza Vaccination*, 13 CLIN. RHEUMATOL. 645 (1994), filed as "Pet. Ex. 20-B."

[54] Anne Herron et al., *Influenza Vaccination in Patients with Rheumatic Diseases*, 242 J. AM. MED. ASSOC. 53 (1979), filed as "Pet. Ex. 20-D."

[55] I. Fomin et al., *Vaccination Against Influenza in Rheumatoid Arthritis: The Effect of Disease Modifying Drugs, Including TNFα Blockers*, 65 ANN. RHEUM. DIS. 191 (2006), filed as "Pet. Ex. 20-E."

[56] Paula Ray et al., *Risk of Rheumatoid Arthritis Following Vaccination with Tetanus, Influenza and Hepatitis B Vaccines Among Persons 15-59 Years of Age*, 29 VACCINE 6592 (2011), filed as "Pet. Ex. 20-F."

College of Rheumatology classification criteria for RA instead of the 2010 new criteria that is more sensitive and specific. *Id.* (citing Pet. Ex. A-7 at 2570; Pet. Ex. 20-F at 6594-96).

Dr. Gowin discussed the Soriano study, stating that it was a large collection of people with onset of PMR after influenza vaccine and two cases in which there was a recurrence following influenza vaccine. Tr. 63-64; Pet. Ex. 42.[57] Soriano looked at PMR and Giant Cell Arteritis ("GCA") which is intertwined in about 10-15% cases and at ten cases in their own clinic in which 50% of the PMR cases had onset after influenza vaccine. Tr. 64-65; Pet. Ex. 42 at 154-55. However, Dr. Gowin ultimately conceded that the study was done in Italy, and the content of the vaccine that was used was not known. Tr. 97. Dr. Gowin was questioned about the Autoimmune/Inflammatory Syndrome Induced by Adjuvants ("ASIA") theory, which was discussed in the study. *Id.* She conceded that she had never heard of ASIA prior to reading this article and does not know enough about it but believes there is some data about adjuvants and arthritis. *Id.* She does not know how flushed out the theory is. *Id.*

Dr. Gowin testified that the question is not the safety of vaccinations for the general public but whether the vaccine can incite the onset of RA in a susceptible individual. Tr. 88. She agreed that the current studies have discounted the possibility but submitted these would be rare events and the studies are underpowered to detect such rare events. Tr. 81, Pet. Ex. 22 at 2. Dr. Gowin submitted that the literature suggests only seropositive RA was studied, likely due to misclassification or case definition. Tr. 82; Pet. Ex. 22 at 2. If the studies counted only seropositive RA, this would result in an underestimation of the number of cases of RA associated with vaccines. *Id.*; Pet. Ex. 22 at 2.

Dr. Gowin stated that there is a known connection between PMR and RA. Pet. Ex. 20 at 4. A prospective study of 116 patients after a one year follow up had a diagnosis change to RA in 20% of those initially diagnosed with PMR. *Id.* (citing Pet. Ex. 20-I at 6-7[58]). She agreed that seropositive and seronegative RA are different, but in clinical practice she makes no distinction between patients with seronegative and seropositive RA for purposes of treatment. *Id.*

Dr. Gurish's theory was based on molecular mimicry, stating that molecular mimicry could cause an interaction by the T cell receptor with that part of the antigen presenting molecule that is exposed. Pet. Ex. 23 at 5.[59] Similar sequences, not necessarily identical, can stimulate the same T cell, known as cross reactivity or T cell degeneracy. *Id.* (citing Pet. Ex. 23-B at 1103-04[60]). This cross reactivity has led to the hypothesis that microbial components can induce a cross reactive

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[57] A. Soriano et al., *Giant Cell Arteritis and Polymyalgia Rheumatica After Influenza Vaccination: Report of 10 Cases and Review of the Literature*, 21 LUPUS 153 (2012), filed as "Pet. Ex. 42."

[58] William P. Docken, *Clinical Manifestations and Diagnosis of Polymyalgia Rhematica*, UPTODATE.COM (Sept. 4, 2014), filed as "Pet. Ex. 20-I."

[59] Dr. Gurish failed to provide specific references to medical literature for multiple propositions in his expert reports. *See* Pet. Ex. 23, 25.

[60] Julie K. Olson et al., *Innate and Adaptive Immune Requirements for Induction of Autoimmune Demyelinating Disease by Molecular Mimicry*, 40 MOL. IMMUNOL. 1103 (2004), filed as "Pet. Ex. 23-B."

response – molecular mimicry – resulting in autoimmunity. *Id.* (citing Pet. Ex. 23-A at 158-59[61]). Mimics occur when linear sequences share a few critical amino acid residues (building blocks of proteins). *Id.* (citing Pet. Ex. 23-A at 158-59).

Dr. Gurish submitted that in addition to molecular mimicry, epitope spreading, bystander activation and polyclonal activation all provide immune responses that could lead to reactivity of self-components.[62] *Id.* at 4 (citing Pet. Ex. 23-E at 85). Dr. Gurish stated that recent evidence has demonstrated that interactions of regulatory T cells and effector T cells in normal lymphoid tissue can be identified, suggesting an ongoing process and that disruption at any time can lead to the appearance of immunity. *Id.* An innate cell called a follicular dendritic cell can drive the production of self-reactive antibodies, providing another explanation for the appearance of pathogenic B cells. *Id.* (citing Pet. Ex. 23-G at A57[63]).

Dr. Gurish noted that genetics, health, and environmental exposure such as toxins, infections, or vaccinations may contribute to the breakdown of the immune system. *Id.* (citing Pet. Ex. 23-I at 85-86). Dr. Gurish stated twin studies prove that the individual nature of the genetic composition of every human which is composed of inherited genes and superimposed epigenetic changes make it virtually impossible to predict how any one individual might react. *Id.* (citing Pet. Ex. 23-H at 978-79[64]). "Thus, the appearance of autoimmunity in one individual such as petitioner, cannot be predicted or ruled out on the basis of population studies conducted to assure the safety of treatments on a large population." *Id.* Like Dr. Gowin, Dr. Gurish submitted that studies that argue vaccines are safe for the public fail to consider individuals who have experienced reactions in these blanket statements. *See id.*

Dr. Gurish cited to literature in support of the view that aging results in more variability of the immune system. Pet. Ex. 25 at 2. The immune system is under constant regulation to allow protective immune response while curtailing autoimmunity. *Id.* (citing Pet. Ex. 25-B at 225-26[65]). The development of autoimmunity is a disruption to this balance. *Id.* (citing Pet. Ex. 25-B at 225-26). The aim of a vaccination is to provoke the immune system but does not guarantee regulation. This regulatory capacity changes constantly over one's lifetime. *Id.* Dr. Gurish argued this

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[61] Michael B.A. Oldstone, *Molecular Mimicry: Its Evolution from Concept to Mechanism as a Cause of Autoimmune Diseases*, 33 MONOCLONAL ANTIBODIES IN IMMUNODIAGNOSIS AND IMMUNOTHERAPY 158 (2014), filed as "Pet. Ex. 23-A."

[62] At hearing, Dr. Gurish stated that epitope spreading is not necessarily relevant to the initiation of PMR but more relevant in the transition from PMR to RA. Tr. 136-38.

[63] Mohey Eldin El Shikh et al., *Breakage of B Cell Tolerance by Antigens on Follicular Dendritic Cells*, 70 ANN. RHEUM. DIS. A57 (2011), filed as "Pet. Ex. 23-G."

[64] Elaine F. Remmers et al., *STAT4 and the Risk of Rheumatoid Arthritis and Systemic Lupus Erythematosus*, 357 N. ENG. J. MED. 977 (2007), filed as "Pet. Ex. 23-H."

[65] Zhiduo Liu et al., *Immune Homeostasis Enforced by Co-Localized Effector and Regulatory T Cells*, 528 NATURE 225 (2015), filed as "Pet. Ex. 25-B."

supported the development of petitioner's autoimmunity after his influenza vaccine, even though he had not experienced such adverse events previously. Pet. Ex. 25 at 2.

Dr. Gurish agreed with Dr. Gowin's opinions; he discussed a case study of a patient who developed RA two weeks after receiving a flu vaccine.[66] Pet. Ex. 23 at 2 (citing Pet. Ex. 20-C at 1-2).[67] The case study noted that "virus-specific antibody-producing B lymphocytes have been isolated from the synovium of patients with RA following influenza vaccination." Pet. Ex. 20-C at 3 (citing Resp. Ex. C-4[68]). According to Dr. Gurish, "The observation of flu-specific B cells in the joints…implies directed migration of antigens to which they are responding in the joints." Pet. Ex. 23 at 2. He noted this indicates a cross reactive self-antigen that may present in the joints leading to reactivity and inflammation and suggested that development of autoimmunity was driven by induction of flu specific B cells in this case. *Id*. (citing Pet. Ex. 20-C at 1-2).

Dr. Matloubian stated that there is no biological evidence linking influenza virus to RA or any other autoimmune disease. Resp. Ex. A at 8. Dr. Matloubian addressed Dr. Gowin's statements about HLA-B27 positive patients, stating people who are HLA-B27 positive are genetically predisposed to develop this type of arthritis usually a couple of weeks after infection with certain microorganisms. *Id.* Six percent of the US population has HLA-B27. *Id.* With the millions of influenza vaccines administered yearly, one would expect to see more cases of vaccine associated rheumatoid arthritis if the association existed. *Id.*; *see* Pet. Ex. 20-B at 645. Further, there is no indication in his records that petitioner is HLA-B27 positive.

Dr. Matloubian addressed the same case study submitted by Dr. Gowin of the smoker with a 12-year history of RA who developed vasculitis two weeks after influenza vaccine. Resp. Ex. A at 9 (citing Pet. Ex. 20-C). Dr. Matloubian noted that she was taking medications known to be associated with vasculitis which could have been the cause of the development of vasculitis independent of the vaccine. *Id.* Dr. Matloubian further pointed out that the authors of the case study concluded that temporal relationship between vaccine and onset of rheumatic disease does not prove cause and effect. Resp. Ex. A at 8-9 (citing Pet. Ex. 20-C at 7).

Dr. Matloubian agreed with Dr. Gowin that epidemiologic studies provide evidence for hypothesis "to ask if there is causal link, mechanistically," between influenza vaccine and PMR/RA and are useful in generating a hypothesis. Tr. 286. However, Dr. Matloubian did not agree that the studies presented in this case were underpowered. *Id.* He pointed out that the Bengtsson article, for example, discussed multiple studies concluding that in combination there was no signal to say there was an association between vaccinations and development of RA. Tr.

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[66] The case study filed as Pet. Ex. 20-C cited to the Pelton article filed as Resp. Ex. C-4, which was also relied upon by Drs. Gurish and Gowen.

[67] P. Iyngkaran et al., *Rheumatoid Vasculitis Following Influenza Vaccination*, 42 RHEUMATOLOGY 907 (2003), filed as "Pet. Ex. 20-C."

[68] B.K. Pelton, A.R. Harvey, & A.M. Denman, *The Rheumatoid Synovial Membrane Participates in Systemic Anti-viral Immune Responses*, 62 CLIN. EXP. IMMUNOL. 657 (1985).

286 (citing Resp. Ex. A-11 at 1833).[69] This article evaluated multiple RA cases and controls and had overall sufficient power greater than 80% to predict relative risk of developing RA after an influenza vaccine as 1.19 – demonstrating the unlikelihood that vaccinations in general could be considered a major risk factor in RA. Resp. Ex. A-11 at 3. The conclusions further noted that no association was found between a high number of vaccines and the development of RA further strengthening the conclusion. *Id.* Dr. Matloubian suggested that rare adverse events following the flu vaccine could be coincidental, stating "We have to look at the mechanism, and there hasn't been a mechanism put forth that makes scientific sense, reliably." Tr. 286-87. There has been no implication of molecular mimicry between influenza virus infection or influenza components and seropositive or seronegative RA. Tr. 289. Thus, Dr. Matloubian concluded that there is no evidence in this case that suggests a plausible mechanism for the induction of seronegative RA that could even lead to a hypothesis that a rare case of seronegative RA could have been triggered by the flu vaccine or flu virus. Tr. 290.

Dr. Matloubian stated that despite the "theoretical simplicity" of molecular mimicry, such a mechanism has rarely been persuasively demonstrated as the cause of autoimmunity, except for rheumatic fever after streptococcal bacterial infection. Resp. Ex. A at 10-11. For molecular mimicry to be relevant, the natural infection must lead to the development of the disease. *Id.* Research of the literature finds no mention of influenza infection being associated with polyarthritis. *Id.* Therefore, since the components of the vaccine are derived from influenza virus, it is "highly unlikely" that a mechanism such as molecular mimicry can cause arthritis due to vaccine. *Id.* (citing Pet. Ex. 20-H at 5). Further, hundreds of millions of people worldwide are affected with influenza each year and there is no epidemic of PMR or RA after the virus itself. Therefore, the theory that molecular mimicry caused petitioner's disease is highly speculative in Dr. Matloubian's opinion. *Id.*

Dr. Matloubian discussed the Pelton article in which lymphocytes specific to a pathogen were found in joints of RA patients as submitted and discussed by both Dr. Gowin and Dr. Gurish. Tr. 251-52; Resp. Ex. C-4. Dr. Matloubian is an expert in lymphocyte trafficking. Resp. Ex. B at 2. He stated that it is not surprising to see a pathogen at an inflammatory site and the findings do not imply the involvement of that pathogen or the lymphocyte specific for it in the pathogenesis of the autoimmune disease. Resp. Ex. A at 11. Natural infections lead to memory lymphocytes, which circulate through the blood and tissues surveying for specific invading pathogens. Tr. 252. The memory lymphocytes would circulate through sites of inflammation such as RA synovium checking to see if their specific pathogen was the culprit. *Id.* The lymphocytes specific to influenza virus found in the joints of those with RA in the study are irrelevant. Resp. Ex. A at 11. Lymphocytes are bystanders, not the culprits, in the inflammation. *Id.* They merely migrate to the area of inflammation wherever chemokines are being expressed, explaining why so many lymphocytes with so many specificities are found in the RA synovium and other sites of inflammation. Resp. Ex. C at 5. The Pelton study showed that the lymphocytes migrated to the joints which are already inflamed; they did not cause the inflammation. Resp. Ex. C-4 at 660.

[69] Camilla Bengtsson et al., *Common Vaccinations Among Adults Do Not Increase the Risk of Developing Rheumatoid Arthritis: Results from the Swedish EIRA Study*, 69 ANN. RHEUM. DIS. 1831 (2010), filed as "Resp. Ex. A-11."

Dr. Matloubian agreed with Dr. Gowin that in practice, no distinction is made between seronegative and seropositive RA, since the medications available for treatment of inflammation are limited. Resp. Ex. C at 3. However, the distinction exists in the pathogenesis of the two diseases. Seropositive RA has a presence of autoantibodies, whereas none exist or are known for seronegative RA, suggesting that mechanisms that lead to the breakdown of tolerance and generation of autoimmunity in each case may be different. *Id.*

The experts agree that there are no known autoantibodies or antigens identified in PMR or seronegative RA so therefore, no sequence of amino acids can be shown to compare to those in the influenza vaccine. Dr. Matloubian stated that if a pathogen does not contain any molecular mimic that is associated with a specific autoimmune disease, that autoimmune disease will not manifest. Tr. 200-01. Dr. Matloubian discussed a 2017 paper in which Dr. Gurish was a co-author. The paper raised the question of whether B cells even play a role in seronegative RA. Tr. 257 (citing Resp. Ex. F[70]). Dr. Matloubian pointed to Dr. Gurish's admission that he did not believe they did and there are no autoantibodies in seronegative RA. Tr. 257. In the paper, using "sophisticated techniques, they define a subset of T cells that provide help to B cells in synovium and blood of patients with rheumatoid arthritis." Resp. Ex. F at 114. T cells were found only in seropositive RA patients, while none were found in seronegative RA; "this underlines that seronegative arthritis and seropositive arthritis are two different diseases." *Id.*; Tr. 257.

Petitioner has failed to file any literature in this case to support a finding that influenza or the influenza vaccine can cause and/or trigger PMR or seronegative RA. Though Dr. Gowin criticized many the studies filed in this matter as underpowered, she agreed that none of the studies demonstrated a clinical likelihood of PMR or RA whether seropositive or seronegative following an influenza vaccine or a bout of influenza. Tr. 297-98. She also acknowledged that millions of people receive the flu vaccine every year, but was unable to provide a single example of a case report or study connecting the vaccine to either PMR or RA. *Id.* Dr. Matloubian pointed out that the literature relied on by petitioner showed an abundance of T-cells in seropositive RA synovium, highlighting the importance of tissue localized T cell/B cell interaction. Tr. 212-13. However, T cells do not exist in seronegative RA, and it is unknown whether there is any T cell/B cell interaction in seronegative RA. *Id*. Dr. Matloubian concluded that there is no known connection between influenza virus and PMR and/or RA. Tr. 227.

Dr. Matloubian added that it is rarely known what the cause of a patient's RA is, with a few exceptions. Tr. 191. For example, it is known that hepatitis B and C have been associated with arthritis, so testing for both conditions is necessary for treatment. *Id.* However, aside from rare instances, there is otherwise no known cause for RA and PMR. Tr. 192.

Dr. Matloubian posited that trauma has been considered a cause of RA and submitted an article at the hearing regarding trauma and RA. Tr. 232-33; *see generally* Resp. Ex. L.[71] Dr. Gowin responded that, clinically, she has never treated or witnessed RA triggered by trauma. Tr. 292. She

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[70] Deepak A. Rao et al., *Pathologically Expanded Peripheral T Helper Cell Subset Drives B Cells in Rheumatoid Arthritis*, 542 NATURE 110 (Feb. 2017), filed as "Resp. Ex. F."

[71] A.W. Al-Allaf et al., *A Case-Control Study Examining the Role of Physical Trauma in the Onset of Rheumatoid Arthritis*, 40 RHEUMATOLOGY 262 (2001), filed as "Resp. Ex. L." This article was filed after the hearing; Drs. Gowin and Gurish did not respond to it.

opined that if the immune system consistently responded to trauma in such a way that RA is triggered, studies would show frequent autoimmune reactions to minor cuts and bruises, and that is not the case. *Id.* Having not had the opportunity to read the article to which Dr. Matloubian was referring, she noted her concern that it is a classic case of recall bias, a system error that is produced by patients' differential recall on the questionnaire between cases and controls. *Id.* She believed that RA patients would be much more likely to recall trauma than those in the control group that did not develop RA. Tr. 292-93. She also submitted that this study was underpowered and has no adjustments for additional variables. *Id.* Dr. Gowin took a literal view – trauma being cuts and bruises – of the article referenced by Dr. Matloubian; however, the article was submitted for purposes of sharing that, when a body is faced with consistent inflammation, such as that suffered by petitioner for months with his knee injury, that constant assault can potentially trigger RA. Tr. 157.

In assessing the reliability and credibility of an expert's opinion, a special master must consider whether the expert offering the opinion is testifying within his training or expertise. *Walton v. Sec'y of Health & Human Servs*., No. 04-503V, 2007 WL 1467307, at *17-18 (Fed. Cl. Spec. Mstr. Apr. 30, 2007) (otolaryngologist not well suited to testify about disciplines other than her own specialty). In recognizing the liberality with which evidence offered in Vaccine Program cases is treated, I read and evaluated all of the testimony of the experts offered and all of the literature filed in this case and gave appropriate weight to whether certain testimony is beyond a particular expert's purview. *See, e.g., King v. Sec'y of Health & Human Servs*., No. 03-584V, 2010 WL 892296, at *78-79 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (finding petitioner's expert far less qualified to offer opinion on general causation issues pertaining to autism than specific issues pertaining to the petitioner's actual medical history, given the nature of the expert's qualifications). Dr. Gowin is a treating rheumatologist who focuses her practice on clinical diagnosis and treatment of rheumatological conditions, including RA. Though Dr. Gowin participates in trials and research related to rheumatological conditions including PMR and RA, she only participates in the clinical side of the trials by recommending patients for a study rather than the research or data gathering. Tr. 43, 67; Pet. Ex. 21 at 1, 8-10. Petitioner's other expert, Dr. Gurish, was an immunologist who focused his practice on mast cell development and the function of innate and adaptive immune responses. Pet. Ex. 24 at 1. Both of petitioner's experts are/were well-credentialed and impressive, but neither expert's practice and research focus on the treatment and development of RA and PMR as heavily as Dr. Matloubian's practice and research. Resp. Ex. B at 2, 6-9. Thus, Dr. Matloubian's testimony and reports were more persuasive than that of Drs. Gowin and Gurish.

PMR, seronegative RA, and seropositive RA are diseases of unknown cause. The literature filed in this matter fails to provide any scientific evidence that either influenza virus or influenza vaccine is associated with PMR, seronegative or seropositive RA. Drs. Gowin and Gurish opined that petitioner's diagnoses of PMR and subsequent seronegative RA are related and were caused by and/or triggered by the influenza vaccine he received. Petitioner provided one case study of an 85-year-old who developed PMR following influenza virus. *See* Pet. Ex. 44 at 345-46. The case study provides no support epidemiologically or mechanistically for the hypothesis that influenza virus was associated with the development of her PMR. *See id.* The authors of the study concluded that the relationship, if there was one at all, was temporal and could not say that she would not have developed PMR in any event.

Moreover, seropositive RA and seronegative RA are different diseases, though clinically they are treated the same. Over 55,000 cases of RA are diagnosed each year. PMR is a disease typical in those who are over 50 years of age and has a usual time of onset in the fall and winter months. The prevalence of these diseases and the onset timing makes a temporal relationship to influenza vaccinations more likely a coincidence. More troubling is that there is known cause of PMR or seronegative RA, and no known autoantibody associated with either condition. The experts all agree that petitioner has PMR and seronegative RA. Therefore, though petitioner's experts provided detailed descriptions of the immune system and molecular mimicry, which could explain a plausible theory for seropositive RA with known autoantibodies, the theory does not apply to PMR or seronegative RA. Petitioner has provided no evidence to suggest what in the influenza vaccine was mimicked to cause or trigger PMR/seronegative RA when neither disease has a known cause or a known autoantibody.

As such, petitioner has failed to satisfy Prong I.

B. Petitioner has failed to demonstrate a logical sequence of cause and effect connecting the flu vaccine to his development of PMR and RA.

In order to sustain his burden under Prong II, petitioner must show that the flu vaccine caused and/or triggered his PMR and/or RA.

Both Drs. Gowin and Gurish opined that molecular mimicry was at play in the development of petitioner's PMR and subsequent seronegative RA diagnoses. Tr. 85-86, 165. However, when asked what in the vaccine was being mimicked in this mechanism, neither expert could provide an answer. Tr. 88, 147-48. Dr. Gowin responded, "I don't think I can give you a specific antigen, Dr. Gurish may be able to do a better job of explaining that." Tr. 88. Dr. Gurish was similarly unable to provide an example of what components in the flu vaccine were being targeted or mimicked during molecular mimicry. Tr. 147-48. Dr. Matloubian stated that while molecular mimicry works in vaccine-associated injuries, since there is no known antibody that causes PMR or seronegative RA, to suggest molecular mimicry as the mechanism in this case would be speculative at best. Tr. 230-31 ("[H]ow do you say something mimics something when you don't know what it is. In [the] case of Guillain-Barre [syndrome], we know the target is the gangliosides on the nerve cells, so then you can say like what mimics ganglioside, but for seronegative RA, we hypothesize that it's some synovial joint antigen, but we don't know what it is.").

Dr. Gowin concluded that petitioner met the diagnosis of PMR based on shoulder and hip girdle pain including arms, shoulders, buttocks, groin and back with stiffness and elevated ESR. Tr. 110. His PMR transitioned into RA with petitioner meeting the 2010 criteria of RA. Other causes of inflammatory arthritis were ruled out. His pre-existing osteoarthritis would not have made him more likely to have an immune reaction to flu vaccine and would not have caused such diffuse pain and elevated markers. His onset was within weeks of the influenza vaccination, consistent with reactive autoimmune syndromes reported in the literature. Petitioner's knee replacement in May of 2011 did not show evidence of synovitis in the joint. In Dr. Gowin's opinion, both temporal relationship and biological possibility exist for the flu vaccine to have triggered the onset of petitioner's RA. Pet. Ex. 20 at 4-5; Pet. Ex. 22 at 2. However, Dr. Gowin's conclusion was not based on accurate information. Petitioner never had an elevated ESR and had

only a mildly elevated CRP on one occasion. Pet. Ex. 7 at 62, 64. All of his other blood work was normal. He had constant moderate pain and swelling of his knee following his knee replacement in May 2011, which continued even after Dr. Dietrick did corrective surgery on his knee. Pet. Ex. 7 at 62; *see generally* Pet. Ex. 7. He had a history of right thigh burning, tingling, and numbness that had persisted for a month prior to the day he received the allegedly causal flu vaccine. Pet. Ex. 2 at 16; Pet. Ex. 17 at 1. Dr. Gowen concluded that onset of symptoms was within weeks of the October 14, 2011 vaccination rather than in December of 2011 as reported by petitioner throughout his medical records.

According to Dr. Gurish, there was no alternative cause for petitioner's development of his rheumatologic condition except the influenza vaccine. Tr. 140. When asked whether a failed knee replacement, with ongoing moderate to severe pain, inflammation and inability to walk would be considered a trauma significant enough to trigger rheumatic disease, he responded that trauma leads to inflammatory response, so he cannot say it could not happen, but he did not see any evidence in this case for that. Tr. 156-57. However, it is the law of the Vaccine Program that evidence of the development of a disease and/or injury temporally following a vaccination is insufficient on its own to establish causation. *Grant v. Sec'y of Health & Human Servs*., 956 F.2d 1144, 1148 (Fed. Cir. 1992). Merely identifying the vaccine as causal because of its existence as a known, pre-onset occurrence is insufficient to establish causation without corroborative record proof demonstrating the "logical sequence of cause and effect" required. *Id*. A wide variety of indirect and circumstantial evidence can support that determination, whether in the form of test results or witness testimony. Thus, Dr. Gurish and Dr. Gowin's opinions of temporal association between the vaccine and petitioner's development of PMR and RA are insufficient to support a finding of entitlement.

Dr. Gowin opined that the influenza vaccine is an antigen vaccine. Tr. 111. When pushed to give an explanation as to how this caused or triggered PMR or RA, she stated, "I don't know that I can give you the specific antigen on the cell or in the vaccine or the adjuvant that it could have even been." Tr. 112. It is of note that the influenza vaccine petitioner received did not contain any adjuvants. Tr. 205.

Several factors are troublesome in this case. Petitioner presented on the day of his vaccination, October 14, 2011, with complaints of symptoms of tingling, burning, and numbness of the left thigh ongoing for weeks that could have been attributed to PMR. Pet. Ex. 2 at 16. Though petitioner and his wife initially testified to the onset of his symptoms in late November, petitioner's physicians' records more persuasively show that his shoulder pain and hip pain arose in mid-December 2011, following his return to work and after having been sedentary since his May knee surgery. Further, his pain and inability to walk during the holiday events in December were ultimately admitted by petitioners to be more related to his knee injury. Tr. 9-10, 29-30. Petitioner repeatedly reported to all his physicians that his symptoms began in December of 2011. On January 20, 2012, petitioner's CRP level was slightly elevated, but the rest of his bloodwork was normal. Pet. Ex. 6 at 64-65. An elevated CRP level is an indication of general inflammation, which petitioner may have had because of his knee injury. Rapid response to prednisone is typical for pain and inflammation, and in fact, petitioner reported to Dr. Dietrick that he had to increase his prednisone following his corrective knee surgery to deal with the pain. Pet. Ex. 7 at 53.

Later records show that petitioner was suspected of having seronegative RA in March 2012, which was confirmed in July 2012 with only one medical record showing joint swelling by Dr. O'Connor, but that was denied by petitioner himself. Whether petitioner had PMR that evolved into seronegative RA or evolving seronegative RA all along, matters little. As set forth at length above, neither PMR nor seronegative RA has a known cause or autoantibody associated with it and neither has been associated with influenza vaccine or virus. Coupled with the fact that the onset of both is typical in those over 50 years of age, which petitioner was, with 55,000 cases of RA diagnosed each year, the temporal association with the influenza vaccine appears to be more coincidence. Petitioner failed to adequately provide a plausible theory for how the flu vaccine could cause or trigger either disease, or that it did so in this case. Petitioner further failed to provide any literature to support a correlation between influenza virus or vaccine and either PMR or seronegative RA. Thus, petitioner has failed to satisfy the requirements of Prong II.

C. Petitioner has failed to show an appropriate temporal relationship between her receipt of the flu vaccine and her development of PMR and/or RA.

To satisfy the third *Althen* prong, petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Human Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

As set forth above, the medical records clearly show that if petitioner did not already have symptoms of PMR and/or seronegative RA on or before the date of his flu vaccine, he consistently reported onset of symptoms as mid-December 2011, with seronegative RA suspected in March of 2012, over two and five months following his receipt of the flu vaccine, respectively. Therefore, petitioner needed to demonstrate an appropriate timeframe for the development of PMR and RA following the flu vaccine to satisfy his burden under Prong III. *Langland*, 109 Fed. Cl. at 443 ("[T]o satisfy the 'proximate temporal relationship' prong of the *Althen* test, petitioners must demonstrate, by a preponderance of the evidence, that the onset of symptoms occurred within a time frame for which it is medically acceptable to infer causation-in-fact….With no reputable theory as to how the vaccination could cause the injury, this exercise is not possible.") (citing *De Bazan*, 539 F.3d at 1352).

The experts' opinions regarding the onset of petitioner's PMR and seronegative RA was at odds with the contemporaneous medical records. Dr. Gowin stated that petitioner's onset was within weeks of his influenza vaccine. Pet. Ex. 20 at 1. Dr. Gurish placed onset at six weeks from vaccination. Pet. Ex. 23 at 5. Thus, both place onset sometime in November of 2011 in reliance on petitioner's affidavit rather than the medical records, which confirm that petitioner reported onset

of symptoms to his physician as December 2011 consistently throughout the record. *See* Pet. Ex. 6 at 65.

Though Dr. Matloubian opined that molecular mimicry was not a plausible biological mechanism for PMR and seronegative RA, he pointed out that generally, it takes T cells 7 to 14 days to reach their peak levels. Resp. Ex. A at 12. During this time, they can provide help to activated B cells, which in turn make antibodies within 7 to 10 days. *Id.* In infections known to cause autoimmunity, the temporal association between inciting infection and development of disease is a couple of weeks, not more than a month. Resp. Ex. A at 12 (citing Pet. Ex. 20-B).

Further, both Dr. Gowin and Dr. Matloubian agreed that individuals can test positive for seropositive RA for years without any clinical symptoms of disease. Both agreed that it is unknown what may trigger the manifestations of clinical symptoms other than smoking in anti-CCP seropositive RA. Tr. 83-84. Dr. Matloubian questioned when a diagnosis should be made, when the autoantibodies are found in the body or when clinical manifestation of disease begins. Tr. 212-13. Therefore, since neither PMR or seronegative RA has any known autoantibodies, when petitioner's disease may have started is unknown, but petitioner's clinical symptoms of PMR, if they were not already present at the time of his vaccination, began by his own reporting in mid-December of 2011, two months after his vaccination. His seronegative RA was suspected in March of 2012 and diagnosed in July 2012.

Petitioner's prior medical history of back pain with associated sciatic pain, neck pain and diabetic neuropathies before, on the day of, and after receiving the flu vaccine are so undefined and insidious in nature, it is impossible to know when the onset of either PMR or seronegative RA truly began. Arguably, petitioner's complaints on the day of the vaccine could have been symptoms of PMR. Alternatively, based on the medical records and the reports by petitioner upon presentation to his doctors, his symptoms of PMR began in mid-December and were diagnosed in January, while his rheumatologist raised the issue of RA in March but did not diagnose it until July because there was no objective evidence to support it. Moreover, while the experts discussed the immune system, how it works and reacts, and their respective theories regarding the flu vaccine and molecular mimicry, they never discussed a timeframe in which that process would result in the manifestation of either disease. Thus, petitioner has failed to sustain his burden under Prong III, as no timeframe was provided.

VII. Conclusion

Accordingly, I find that petitioner has not established entitlement to compensation and his petition must be dismissed. In the absence of a timely filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accordance with this decision.[72]

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master

[72] Pursuant to Vaccine Rule 11 (a), if a motion for review is not filed within 30 days after the filing of the special master's decision, the clerk will enter judgment immediately.