In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 15-1485V
Filed: May 11, 2023
PUBLISHED

BETTINA MCGILL,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

Special Master Horner

Pneumococcal conjugate vaccine ("PCV13"); Influenza ("flu") vaccine; Small fiber neuropathy ("SFN")

Renee J. Gentry, George Washington University Law School, Washington, DC, for petitioners.
Benjamin Patrick Warder, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On December 8, 2015, petitioner filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10-34 (2012),[2] alleging that the 13-valent pneumococcal conjugate ("PCV13") and trivalent influenza ("flu") vaccines that she received on November 7, 2014, caused her to suffer a small fiber nerve injury and interstitial lung disease ("ILD"). (ECF No. 1.) Petitioner later amended her petition to specify that she suffered small fiber neuropathy, ILD, and myofascial pain syndrome caused by her vaccinations. (ECF No. 62, p.2.)

In her post-hearing brief, petitioner indicated that she no longer alleges that the vaccines caused her ILD and further confirmed that her claim is based on a diagnosis of small fiber neuropathy ("SFN"). (ECF No. 148, p. 23 n.7.) Although myofascial pain

[1] Because this decision contains a reasoned explanation for the special master's action in this case, it will be posted on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. *See* 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information the disclosure of which would constitute an unwarranted invasion of privacy. If the special master, upon review, agrees that the identified material fits within this definition, it will be redacted from public access.

[2] Within this decision, all citations to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

syndrome is referenced within the differential diagnosis of some treating physicians, petitioner has not ultimately argued that she met her burden of proof with respect to a myofascial pain syndrome and her expert's causal opinion is specifically predicated on SFN.

For the reasons set forth below, I conclude that petitioner is not entitled to compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make several factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally—and the key question in most cases under the Program—the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable timeframe following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1)(A); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a); § 300aa-13(a)(1)(B).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991). Because small fiber neuropathy is not listed as an injury on the Vaccine Injury Table, petitioner must satisfy this burden of proof.

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause of the injury or condition, but must demonstrate that the vaccination was at least a "substantial factor" in causing the

condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury;" the logical sequence must be supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, [petitioner's] burden is to show by preponderant evidence that the vaccination brought about 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 proximate temporal relationship between vaccination and injury. If [petitioner] satisfies this burden, she is entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine.

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. That expert's opinion must be based upon "sound and reliable" scientific explanation. *Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019) (quoting *Knudsen v. Sec'y of Health & Human Servs*., 35 F.3d 543, 548-49 (Fed. Cir. 1994)). The *Althen* court also indicated that, in finding causation, a Program factfinder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." 418 F.3d at 1280.

II. Procedural History

This case was first assigned to Special Master Dorsey. (ECF No. 4.) Petitioner filed her medical records on a compact disc on May 19, 2016.[3] (ECF No. 5.) On August 4, 2016, respondent filed his Rule 4(c) report, recommending against compensation. (ECF No. 28.) Respondent stated that petitioner's ILD symptoms predated, and therefore could not have been caused by, her vaccinations. (*Id.* at 15-16.) Respondent also indicated that petitioner had failed to provide a reputable

[3] This filing was later stricken from the record to avoid duplicative filings when petitioner submitted additional medical records later in this case. (*See* ECF No. 48; *see also* ECF Nos. 49-54.)

scientific or medical theory, and that she had ignored potential alternate causes of her condition. (*Id.* at 16-18.)

Petitioner moved to substitute attorney Renee Gentry in place of Dan Wilson Bolton III on January 19, 2017. (ECF No. 46.) She subsequently filed additional medical records on February 12, 2017, and expert reports from molecular virologist Judy A. Mikovits, Ph.D., and microbiologist Francis Ruscetti, Ph.D., on June 19, 2017.[4] (ECF Nos. 49-54, 60-61.) Following her filing of expert reports, petitioner filed additional medical records as well as an amended petition alleging her vaccine-caused injuries such as small fiber neuropathy ("SFN"), acute myofascial pain syndrome, and ILD. (ECF Nos. 62, 64.) Respondent then filed responsive expert reports by rheumatologist Chester Oddis, M.D., and immunologist J. Lindsay Whitton, M.B., Ch.B., Ph.D., on October 18, 2017. (ECF Nos. 67, 68.)

On November 29, 2017, Special Master Dorsey issued a Rule 5 Order, preliminarily concluding that petitioner's ILD was a preexisting condition but noting that petitioner may be entitled to compensation for her SFN. (ECF No. 70.) Special Master Dorsey ordered petitioner to file an expert report from a neurologist, updated medical records, and outstanding medical literature cited by her previous experts. (*Id*.) Petitioner filed an expert report by neurologist Carlo Tornatore, M.D., on January 29, 2018. (ECF No. 73.) Petitioner subsequently filed an affidavit regarding her VAERS investigation on February 16, 2018, and updated medical records on March 14, 2018. (ECF Nos. 77, 78.) In response to Dr. Tornatore's report, respondent offered a report by neurologist Jeffrey M. Gelfand, M.D., M.A.S., on July 30, 2018. (ECF No. 85.) On November 5, 2018, petitioner presented a supplemental report by Dr. Tornatore. (ECF No. 89.) Respondent then filed a supplemental report by Dr. Gelfand on April 17, 2019. (ECF No. 96.) In a follow up status conference, Special Master Dorsey indicated that it was probable she would rule in petitioner's favor based on Dr. Tornatore's opinion, but without determining a specific diagnosis. (ECF No. 90.) However, on June 7, 2019, this case was instead reassigned to my docket. (ECF No. 100.)

Thereafter, on August 5, 2019, I issued a scheduling order additionally urging the parties to consider informal resolution, explaining the previously presiding special master's assessment and indicating that I agreed respondent maintained litigative risk. (ECF No. 104.) However, in contrast to the previous special master, I addressed the issue of diagnosis. In particular, I explained that Dr. Gelfand's opinion, asserting that petitioner's upper and lower extremity symptoms may be attributable to different conditions, further complicated the case. (*Id*. at 2-3.) I later referred the case for mediation, but also scheduled an entitlement hearing. A two-day entitlement hearing was scheduled to commence July 14, 2021. (ECF No. 118.) This case was referred to alternative dispute resolution ("ADR") on February 16, 2021. (ECF No. 123.) After multiple ADR conferences did not result in informal resolution, the case was removed from ADR on April 27, 2021. (ECF No. 128.)

[4] As discussed below, Dr. Mikovits's and Dr. Ruscetti's expert reports were later stricken from the record. (*See* ECF No. 142.)

During this period, the parties also continued to refine their presentations. Petitioner filed an additional supplemental report from Dr. Tornatore on July 25, 2019. (ECF No. 103.) Respondent filed a supplemental expert report from Dr. Whitton on April 6, 2020. (ECF No. 111.) Petitioner continued the exchange of expert reports with a supplemental report from Dr. Tornatore on August 9, 2020. (ECF No. 117.) Respondent filed a responsive expert report from Dr. Whitton on November 3, 2020. (ECF No. 119.) Petitioner then filed a final expert report from Dr. Tornatore on January 11, 2021. (ECF No. 121.) Petitioner filed additional medical records on March 25, 2021. (ECF No. 126.) On July 7, 2021, petitioner moved to strike Dr. Mikovits's and Dr. Ruscetti's expert reports and accompanying medical literature (Exs. 25-35). (ECF No. 142.) Petitioner represented that she did not intend to rely on Drs. Mikovits and Ruscetti or their associated literature. (*Id.*) Respondent raised no objection. (*Id.*) Accordingly, Dr. Mikovits's and Dr. Ruscetti's expert reports and accompanying medical literature were stricken from the record.

A two-day entitlement hearing was held on July 14 and 15 of 2021. (*See* Transcript of Proceedings (Tr.), ECF Nos. 145-46.) Petitioner, Dr. Tornatore, Dr. Whitton, and Dr. Gelfand testified. Following the hearing, a status conference was held on August 19, 2021. (*See* ECF No. 147.) I explained that post-hearing briefs were necessary to address three important issues: (i) whether the parties wished to supplement the record with regard to the Viega-Fernandes study discussed during the hearing; (ii) whether there were any relevant substantive differences between petitioner's case and *E.M.*, a recent ruling on entitlement in an SFN case with onset occurring in less than one day post-flu vaccination;[5] and (iii) whether petitioner's lower extremity condition could separately meet the *Althen* test for causation-in-fact in the absence of a finding that petitioner's upper and lower extremity symptoms represent the same condition. (ECF No. 147.) I also instructed the parties to raise any procedural or substantive objections, if any, in reaching the third issue in the context of this case. (*Id.*) Petitioner filed her post-hearing brief on October 18, 2021. (ECF No. 148.) Respondent filed his post-hearing brief on December 17, 2021. (ECF No. 151.) Petitioner filed a reply on January 14, 2022. (ECF No. 152.)

With regard to the Viega-Fernandes study, contemporaneous with her post-hearing brief, petitioner moved for leave to file the Veiga-Fernandes et al. article and an article by Lai et al. article. (ECF No. 149.) Respondent did not object to petitioner's request to supplement the record with the Veiga-Fernandes and Lai articles. (*Id.* at 1; *see also* ECF No. 151, pp. 39-40.) Accordingly, I granted petitioner's motion to supplement the record, and she filed the articles as Exhibits 64 and 65 on October 20, 2021. (Henrique Veiga-Fernandes et al., *Response of Naïve and Memory CD8+ T Cells to Antigen Stimulation* In Vivo, 1 NATURE IMMUNOLOGY 47 (2000) (Ex. 64); Wendy Lai et al., *Transcriptional Control of Rapid Recall by Memory CD4 T Cells*, 187 J. IMMUNOLOGY 133 (2011) (Ex. 65).)

[5] *See E.M. v. Sec'y of Health & Human Servs.*, No. 14-753V, 2021 WL 3477837 (Fed. Cl. Spec. Mstr. July 9, 2021).

The parties offered their competing views of the ruling on entitlement in *E.M*. (*Compare* ECF No. 148, pp. 14-18 *with* ECF No. 151, pp. 40-45.) Ultimately, respondent is persuasive in contending that *E.M.* provides little guidance for this case, because the expert presentation differs significantly from this case and the outcome in *E.M*. cannot be separated from the special master's credibility determination regarding respondent's expert in that case – an expert who has not been presented in this case. (ECF No. 151, pp. 40-45.)

The parties also provided competing views on whether petitioner's lower extremity symptoms could satisfy the *Althen* test standing alone. Although considering the lower extremity symptoms in isolation would change the timing of onset, and therefore also the *Althen* prong three analysis, respondent contends that petitioner's claim would still fail under *Althen* prongs one and two. (ECF No. 151, pp. 45-50.) Petitioner asserts that her lower extremity symptoms would satisfy the *Althen* test if viewed in isolation (ECF No. 148, pp. 18-23.); however, she "asserts that her proper diagnosis is small fiber sensory neuropathy. Ms. McGill's medical records cannot and should not be separated by extremity." (ECF No. 152, p. 1.)

In light of all of the above, this case is now ripe for a resolution of entitlement.

III. Factual History

a. As Reflected in the Medical Records

i. Pre-vaccination

Prior to receiving the flu and pneumococcal conjugate vaccinations on November 7, 2014, petitioner had received at least two flu vaccines and a "pneumonia" vaccine. (Ex. 3, p. 3.) Petitioner's medical history was significant for autoimmune necrotizing myopathy or polymyositis, seropositive nonerosive rheumatoid arthritis ("RA"), neutrophilic dermatitis,[6] gastroesophageal reflux disease ("GERD"), depression, and significant upper respiratory symptoms including coughing, shortness of breath, and "bilateral lung opacities." (Ex. 15, pp. 4, 6, 29, 67-68, 71; Ex. 19, p. 158.) Petitioner initially sought treatment for her myopathy in September 2010, when she described six months of progressive symmetric proximal muscle weakness that primary affected her upper extremities. (Ex. 15, p. 91.) She received intravenous immunoglobulin ("IVIG") therapy and a variety of immunosuppressive drugs including methotrexate, rituximab, prednisone, and CellCept for her autoimmune disorders. (Ex. 15, pp. 67, 83, 91.)

On October 20, 2014, petitioner visited Nizar Chahin, M.D., who noted that in September 2010, petitioner's creatine phosphokinase ("CK"), aspartate

[6] Neutrophilic dermatitis is "[a] type of neutrophilic dermatosis usually seen on the upper body of middle-aged women, characterized by one or more large, rapidly extending, erythematous, tender or painful plaques, with fever and dense infiltration of neutrophilic leukocytes in the upper and middle dermis." *Acute Febrile Neutrophilic Dermatosis*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=69376 (last accessed Nov. 7, 2022).

aminotransferase ("AST"), and alanine transaminase ("ALT") levels were elevated. (Ex. 15, p. 83.) Her myositis profile was negative, she was positive for antinuclear antibodies ("ANA"), rheumatoid factor ("RF"), and cyclic citrullinated peptide ("CCP") antibodies, and negative for antineutrophil cytoplasmic antibodies ("ANCA"). (*Id.*) Dr. Chahin also noted that a September 2010 EMG and nerve conduction study performed on petitioner's right side showed normal sensory and motor responses but fibrillation potentials and positive sharp waves in her right deltoid, biceps, triceps, first dorsal interosseous, vastus medialis and the anterior tibialis. (*Id.*) Dr. Chahin noted that these findings were consistent with an inflammatory myopathy. (*Id.*)

Petitioner presented to her primary care provider, Anna Conterato, M.D., on November 7, 2014, to follow up on her RA and myositis. (Ex. 15, p. 90.) At the time of this visit, petitioner was taking albuterol, a vitamin D supplement, Flexeril, fish oil, Prilosec, tramadol, and CellCept. (*Id.* at 91.) Dr. Conterato noted that petitioner's RA and myopathy were stable, and that petitioner had not had an RA flare "in some time." (*Id.*) During this visit, petitioner received the pneumococcal conjugate and influenza vaccine. (Ex. 2.)

ii. Post-vaccination treatment

Nearly one week later, on November 13, 2014, petitioner presented to Russell J. Coletti, M.D., at the University of North Carolina ("UNC") Internal Medicine Same Day Clinic with a complaint of "burning arm pain." (Ex. 4, p. 2.) Petitioner was concerned that her arm pain was a reaction to her recent vaccinations and reported that six days prior, she had woken up with burning pain from her shoulders to her wrists, which was worse in the left arm. (*Id.* at 3.) She also noted that her symptoms were similar to her prior polymyositis symptoms. (*Id.* at 2.) Dr. Coletti noted that "[t]here does not appear to be any localized skin reaction around the site of the vaccination" and stated that her arm pain was "unlikely to be due to her vaccination." (*Id.*) Upon examination, Dr. Coletti observed pain on abduction of the shoulder, slightly decreased bilateral deltoid strength, and blunted reflexes throughout. (*Id.* at 3.) He ordered petitioner to continue taking Tylenol and using ice packs. (*Id.* at 2.) He also ordered bloodwork to check petitioner's erythrocyte sedimentation rate ("ESR") and CK to rule out a polymyositis flare. (*Id.*)

The following day, petitioner reported to the UNC Emergency Department ("ED") with a bilateral diffuse upper extremity burning sensation, ongoing for one week. (Ex. 4, p. 7.) She reported that the burning sensation began "the same day that she got both her flu and pneumonia vaccines." (*Id.*) She stated that the burning was "only in the arms and nowhere else in the body" and that she had never experienced similar symptoms before. (*Id.*) Her bloodwork from the day prior showed elevated CK levels and a slightly elevated ESR. (*Id.*) Sara Tarjan, M.D., evaluated petitioner and noted that her burning arm pain "may be related to her polymyositis or rheumatoid arthritis although the symptoms are not entirely typical." (*Id.*) Upon examination, petitioner did not reveal any weakness or sensory loss in her upper extremities. (*Id.*) Dr. Tarjan

observed that petitioner's extremities were not swollen or tender to palpation, and that there was no rash. (*Id.* at 11.)

While in the UNC ED, petitioner requested to see attending physician Seth Glickman, M.D. (Ex. 4, p. 12.) Dr. Glickman noted that petitioner had normal vitals and a normal physical exam and that there were no signs of swelling or erythema in her arms. (*Id.* at 13.) He observed mild tenderness to palpation, but normal strength, sensation, and distal pulses in both arms. (*Id.*) His differential diagnosis was a local reaction to immunization versus myositis flare. (*Id.*) He recommended petitioner consult her rheumatologist about taking a steroid burst. (*Id.*) The discharge instructions indicate petitioner's treating physicians were "unsure of the exact cause of [her] arm burning" and that "[i]t may be a flare of [her] RA or polymyositis." (*Id.* at 17.) Petitioner declined steroids. (*Id.*)

Petitioner had a neuromuscular consult with Elizabeth Jovanovich, M.D., on November 18, 2014. (Ex. 4, p. 29.) Petitioner reported that "[s]hortly after injection [she] developed redness and induration on [her] left deltoid" and "some minor erythema on the right side as well though not as severe." (*Id.* at 31.) As of this visit, petitioner reported that the swelling at the injection sites "completely disappeared and that the pain in her right arm ha[d] improved slightly." (*Id.*) On examination there was minimal swelling at deltoids bilaterally, but no evidence of discoloration; her deltoids were tender to palpation bilaterally, worse on the left; and there was no evidence of joint effusions or swelling or discoloration in hands, elbows, feet, or ankles. (*Id.* at 33.) Petitioner's deep tendon reflexes and sensory exam were normal. (*Id.* at 34.) Dr. Jovanovich assessed petitioner with an arthus reaction to her vaccinations. (Id. at 30.) She prescribed a Medrol Dosepak and heat packs for symptomatic relief. (*Id.*)

The next day, petitioner returned to the UNC ED with a chief complaint of arm pain. (Ex. 4, p. 40.) She was treated by nurse practitioner Benjamin Linthicum, who observed decreased reflexes in the upper and lower extremities with no weakness and elevated CK and ESR levels (similar to her previous labs). (*Id.* at 40, 42.) He advised petitioner to continue using steroids and cold/hot compresses and to follow up with rheumatology and internal medicine. (*Id.* at 40.) Petitioner was prescribed Dilaudid for pain management. (*Id.* at 40, 44.)

Petitioner presented to rheumatologist Jake Ritt, M.D., at the UNC Blue Ridge Family Practice on November 20, 2014. (Ex. 4, p. 60.) Dr. Ritt noted that petitioner "did not think [her symptoms were] a flare of her myositis or her RA, but rather a reaction to the vaccination given a week prior." (*Id.*) He noted that the on-call rheumatology fellow started petitioner on gabapentin for possible neuropathic pain during her stay in the ED, but that she was later seen by a neurologist who stopped the gabapentin in favor of corticosteroids for a possible arthus reaction. (*Id.*) Dr. Ritt believed that an arthus reaction "would be unlikely given [petitioner] was injected with two different potential antigens in two different areas (unless the medium the vaccines were carried in were similar and she had a hypersensitivity to that)." (*Id.* at 63.) Instead, he suspected that the more likely cause of petitioner's pain was myofascial pain syndrome "triggered by

the inciting event of the vaccinations." (*Id.* at 63.) He noted that myofascial pain syndrome "is more common in patients with underlying chronic illness and a history of depression." (*Id.*) He prescribed a higher dosage of gabapentin and home exercise to maintain range of motion in petitioner's shoulders. (*Id.*) The same day, petitioner filed a VAERS report noting that she suffered from a "[b]urning sensation in both arms," with an onset of November 8, 2014, at 12:30 AM. (Ex. 17, p. 2.)

Shortly after her visit to Dr. Ritt, on November 20, 2014, petitioner reported to the Duke Health ED with a chief complaint of "medical reaction" and paresthesia. (Ex. 18, p. 1.) Petitioner informed the desk staff that "the pain in [her] arms ha[d] moved to [her] legs." (*Id.* at 3.) Petitioner saw Amy Kumar, M.D., who noted that petitioner was "very upset with the conflicting answers being given to her and many medications being prescribed to her" and noted that petitioner came to the ED to see a neurologist and rheumatologist. (*Id.* at 4.) Petitioner's physical examination did not reveal any significant abnormalities. (*Id.* at 5.) She referred petitioner to outpatient neurology and rheumatology. (*Id.*)

Petitioner was discharged from Duke ED early on November 21, 2014 (Ex. 18, p. 7), but returned the following day with a chief complaint of "generalized pain" (*Id.* at 14). Seth Holt, M.D., and Nicholas Lauerman, M.D., examined petitioner and noted that her burning arm pain had now progressed to her entire body. (*Id.* at 17.) Petitioner noted she was unable to walk as it "cause[d] diffuse pain and . . . generalized weakness" but showed full strength on her focal examination. Drs. Holt and Lauerman explained that petitioner had not been on gabapentin long enough to feel the effects. (*Id.*) The doctors recommended that petitioner continue gabapentin, attempt a trial of Ativan for anxiety, and follow up with her rheumatologist. (*Id.*)

Petitioner then presented to Kimberly Yarnall, M.D., at Duke Primary Care Blue Ridge on November 24, 2014, to establish care. (Ex. 18, p. 29.) At the time of this visit petitioner carried a diagnosis of myositis, depression, GERD, RA, and seasonal allergic rhinitis. (*Id.*) Petitioner informed Dr. Yarnall that she had recently become weak to the point that she was using a walker. (*Id.*) Dr. Yarnall encouraged petitioner to continue taking gabapentin for her nerve pain. (*Id.* at 36.)

On November 25, 2014, petitioner presented to Duke rheumatologist Melissa Wells, M.D., for an evaluation of "burning sensation throughout [her] body." (Ex. 7, p. 2.) Petitioner reported that her pain progressed from being unilateral in her left arm, to bilateral, to her entire body. (*Id.*) She had experienced progressive weakness in her lower extremities and was using a walker after experiencing "a couple of almost falls." (*Id.*) The weekend prior, petitioner had discontinued her narcotic medications and restarted gabapentin. (*Id.*) Upon examination, Dr. Wells observed that petitioner could complete full shoulder abduction with active range of motion, but was unwilling to complete other range of motion tests due to her pain. (*Id.* at 5.) Petitioner exhibited full passive range of motion and bilateral tenderness to palpation on the anterior and lateral shoulders. (*Id.*) Her right knee was tender along the medial aspect, her left knee was diffusely tender along the lateral malleolus, and both knees showed full passive range of

motion. (*Id.*) All other joints were normal. (*Id.*) Dr. Wells observed that petitioner had difficulty following simple instructions such as shrugging her shoulders. (*Id.*) Petitioner was noted to "get anxious and agitated when discussing the course of her illness." (*Id.*) Dr. Wells assessed petitioner with inconsistent diffuse pain, left greater trochanteric bursitis, history of RA and myositis, and chronic immunosuppression. (*Id.* at 6.)

During this visit, Dr. Wells noted that there was no documentation showing a localized reaction at the injection site, and although petitioner originally reported a localized reaction when reporting to UNC ED, she denied any localized reaction at this visit. (Ex. 7, p. 6.) Dr. Wells was "unable to determine if the onset of pain is related to the injection or not." (*Id.*) She stressed however, that she was "not entirely sure" about other potential causes of petitioner's pain because her symptoms did "not follow any typical dermatomal or neurological patterns." (*Id.*) She noted that petitioner's RA and myositis appeared to be well-controlled given that her strength was normal and there were no signs of synovitis. (*Id.*) Dr. Wells could not find any evidence of vasculitis, and petitioner denied any systemic symptoms, which made an underlying inflammatory process less likely. (*Id.*) Additionally, petitioner's presentation was inconsistent with fibromyalgia and GBS. (*Id.*) Dr. Wells suspected that the more likely cause of petitioner's pain was SFN because it "can present in atypical patterns." (*Id.*) Dr. Wells deferred further evaluation to petitioner's neurologist and ordered c-reactive protein ("CRP") and CK labs. (*Id.*)

On December 2, 2014, petitioner visited neurologist Justin Mhoon, M.D., at Duke Health Center with a chief complaint of "numbness." (Ex. 18, p. 64.) Petitioner reported feeling "very unsteady on her feet and as if her legs [were] going to give out." (*Id.* at 66.) On examination, Dr. Mhoon observed full strength bilaterally in petitioner's upper and lower extremities. (*Id.* at 70.) She demonstrated normal pinprick, temperature, vibration, and proprioception in her upper and lower extremities, and had allodynia with light touch over the bilateral upper extremities. (*Id.*) Petitioner's coordination was good but slow, and her deep tendon reflexes were decreased and symmetric bilaterally. (*Id.*) Petitioner staggered upon standing and reported that she felt like she was going to fall, that her knees may give out, and that she could not walk without a walker. (*Id.*) Even when using the walker, Dr. Mhoon noted that petitioner "still takes very awkward steps." (*Id.*) Dr. Mhoon's differential diagnosis included sensory neuritis due to a vaccine reaction, toe axial subluxation, and autoimmune demyelinating disease. (*Id.*) Dr. Mhoon recommended a brain and cervical spine MRI, cervical spine X-ray, and an electromyography ("EMG")/nerve conduction study ("NCS") to evaluate for mononeuritis multiplex, and if all tests were normal, a skin biopsy to evaluate for SFN. (*Id.* at 70-71.) Over a week later, on December 10, 2014, petitioner called Dr. Mhoon complaining that her right arm was "very weak" and that she could not lay it flat, and that her shoulders and back were very painful. (*Id.* at 108-09.)

Petitioner underwent brain and cervical spine MRIs with and without contrast on December 17, 2014. (Ex. 18, pp. 110-11.) Petitioner's cervical spine MRI showed some mildly enlarged cervical nodes, some spurring at the C3-C4 level, and some minor disc degeneration at C3-C4 and C5-C6. (Ex. 5, p. 3.) Petitioner's brain MRI

showed some scattered T2 hyperintense foci involving the white matter that were nonspecific and "could be sequela of prior infection/inflammation including Lyme's disease, minor chronic microvascular disease or demyelinating disease" though none of the lesions revealed restricted diffusion or abnormal enhancement. (*Id.* at 4-5.) Petitioner underwent an EMG and NCS on December 31, 2014, both of which were normal. (*Id.* at 6.) Dr. Mhoon noted that there was no evidence of "a widespread large fiber peripheral neuropathy or myopathy" and that petitioner could undergo a skin biopsy to measure the intra-epidermal nerve fiber density to test for SFN. (Ex. 5, p. 6.)

On December 31, 2014, petitioner presented to Dr. Mhoon to discuss her recent EMG, NCS, and MRI studies. (Ex. 18, p. 133.) Dr. Mhoon noted that petitioner's neurologic exam revealed normal strength and sensation besides allodynia in her upper extremities, and that there was no evidence of peripheral neuropathy or active myositis. (*Id.*) He concluded petitioner may have neuritis or SFN, noting that "if this was a vaccine related neuritis it should improve on its own over time." (*Id.*) Dr. Mhoon stated that petitioner could try tapering her gabapentin dose if her pain did not increase. (*Id.*)

Petitioner underwent skin biopsies of her left thigh and left calf on January 27, 2015. (Ex. 21, p. 1.) Her left thigh biopsy was normal, but her left calf showed significantly reduced epidermal nerve fiber density, consistent with SFN. (*Id.*) There was no evidence of vasculitis or other histological abnormalities. (*Id.*) Dr. Mhoon confirmed this diagnosis via an email to petitioner on February 17, 2015, explaining that SFN is "[o]ften associated with rheumatologic conditions such as yours." (Ex. 13, p. 4.) On March 4, 2015, Dr. Mhoon responded via email to petitioner's questions about her SFN diagnosis. (*Id.* at 3.) In response to petitioner's request for Dr. Mhoon to update her chart to reflect a reaction to the flu and pneumococcal vaccines, Dr. Mhoon wrote, "CANNOT PROVE A DIRECT CAUSATION OF THE VACCINES BUT IF YOU NEED A NOTE AT SOME POINT TO AVOID THE FLU SHOT LET ME KNOW." (*Id.*)

On February 6, 2015, petitioner saw Dr. Yarnall for right shoulder pain, back pain that had moved to her right side, and chest pain. (Ex. 18, p. 178.) Petitioner explained that she had pain in her right side around her rotator cuff for several days. (*Id.* at 182.) Aside from her back and shoulder pain, petitioner's examination was normal. (*Id.* at 183.) Dr. Yarnall ordered chest and shoulder X-rays, referred petitioner to orthopedic surgery, and prescribed tizanidine. (*Id.*) Petitioner's shoulder X-ray revealed a 9 mm sclerotic focus proximal humeral diaphysis, which "in the absence of known malignancy may represent an incidental bone island." (*Id.* at 190.) Petitioner's chest X-ray showed no significant degenerative changes in the thoracic spine; normal pulmonary vasculature; bibasilar interstitial prominence, most conspicuous on lateral radiograph and mildly improved; questionable retrocardiac airspace disease; and no pleural effusion. (*Id.*)

Nearly one week later, on February 12, 2015, petitioner presented to rheumatologist Amanda Nelson, M.D., for a follow up on her RA and myositis. (Ex. 7, p. 15.) Besides the pain that petitioner had reported since her vaccinations, her examination was "entirely unremarkable." (*Id.* at 17.) Dr. Nelson recommended

maintaining the current CellCept dosage and a physical therapy evaluation to increase petitioner's strength and conditioning. (*Id.*) Petitioner was advised to see a pulmonary specialist and began using an albuterol inhaler again. (*Id.* at 15.)

The next day, on February 13, 2015, petitioner presented to Hilda Metjian, M.D., at Duke Medicine. (Ex. 8, p. 2.) Petitioner reported that after experiencing some flu-like symptoms in January, her breathing had not been normal. (*Id.*) Dr. Metjian believed that petitioner's restrictive lung disease is likely related to interstitial lung disease "for her autoimmune process." (*Id.* at 4.) She noted that petitioner had an elevated CK, rheumatoid factor ("RF"), and an interstitial pattern on her chest X-ray. (*Id.*) Petitioner followed up with Dr. Metjian on March 5, 2015, where she received a chest CT that revealed findings consistent with non-specific interstitial pneumonia (NSIP), "most likely autoimmune in etiology." (Ex. 18, p. 237.) Dr. Metjian diagnosed petitioner with ILD, referred her to pulmonary rehabilitation, and ordered petitioner to continue taking CellCept. (*Id.*) Petitioner attended pulmonary rehab sessions from April 13, 2015, to May 7, 2015, during which her pulmonary functions and conditioning slowly progressed. (*See* Ex. 24, pp. 13-129; Ex. 18, p. 419.)

Petitioner followed up with Dr. Mhoon on April 27, 2015, to discuss her ongoing complaints of pain and weakness. (Ex. 6, p. 51.) Petitioner had begun taking alpha lipoic acid in addition to gabapentin and appeared "[o]verall stable." (*Id.*) She explained that she had a recent RA flare and was placed on prednisone, which she discontinued after it caused diarrhea. (*Id.*) Petitioner's examination remained unchanged from prior examinations. (*Id.* at 53-54.) Dr. Mhoon believed that petitioner's SFN "may be sequelae from rheumatoid arthritis" but also considered "vaccination neuritis" as another potential cause. (*Id.* at 60.) He recommended that petitioner continue taking gabapentin and alpha lipoic acid. (*Id.*)

iii. Later medical records

Petitioner has an extensive additional medical history. The remainder of her medical records are less informative with respect to the issues discussed by the experts in this case, but discussion of these records is included in the interest of completeness given that they include additional evaluation of her autoimmune conditions.

Between May and August of 2015, petitioner presented for care numerous times for musculoskeletal complaints relating to her shoulder and wrist. (Ex. 18, pp. 395-409, 442-46, 486, 501-12, 526, 535-46, 638-39; Ex. 9, p. 2-3; Ex. 24, pp. 692-97.) There was some suspicion that petitioner's wrist pain was a flare of RA, but this assessment was not shared by all of the treating physicians. (Ex. 18, pp. 551, 595.) She was diagnosed as having a frozen shoulder. (Ex. 18, pp. 638-39.) She also presented during this period with right leg pain that was consistent with a lumbar spinal disc bulge that was observed on MRI. (Ex. 18, pp. 672, 692, 719, 724, 789.)

On June 2, 2015, petitioner presented to neurologist Samuel Moon, M.D., at Duke Neurology. (Ex. 6, p. 62.) Dr. Moon observed a positive RF and noted that it was

unclear whether petitioner's various symptoms were caused by a single autoimmune disorder. (*Id.* at 66.) Petitioner declined acupuncture due to cost constraints, but Dr. Moon sent a chart message suggesting that petitioner's physical therapy team conduct dry needling, a procedure similar to acupuncture. (*Id*.)

On June 18, 2015, petitioner saw Dr. Nelson at UNC Healthcare to transfer her rheumatology care to Duke. (Ex. 4, pp. 66-67.) Dr. Nelson noted that petitioner's RA seemed to be under control outside of the most recent flare, and that rituximab may be a viable treatment option as she had responded well to it in the past. (*Id.* at 69.) Petitioner's CK levels were improving, and Dr. Nelson considered lowering her CellCept dosage once her active issues resolved. (*Id.* at 69-70.) She ordered repeat CK and liver function labs. (*Id.*) Dr. Nelson advised petitioner to continue her current medications and follow up with her various specialists at Duke. (*Id.* at 70.)

On September 22, 2015, petitioner presented to Melissa Reed, M.D., at Duke Primary Care for flu-like symptoms, including fatigue, nausea, dehydration, and chest pressure. (Ex. 18, pp. 939, 942.) Petitioner was concerned that her symptoms were caused by mold. (*Id.* at 942.) Dr. Reed diagnosed petitioner with autoimmune disease, fatigue, ILD, and anemia. (*Id.* at 946.) She ordered lab work, a thyroid profile, urinalysis, and chest X-rays. (*Id.*) Petitioner's lab work showed that she was less anemic compared to earlier labs, and her chest X-rays did not show pneumonia. (*Id.* at 969.) Dr. Reed believed that petitioner's flu-like symptoms were caused by an autoimmune flare following a viral infection. (*Id.*)

Petitioner reported to the Duke ED on September 26, 2015, with complaints of abdominal pain and fatigue. (Ex. 18, p. 971.) She reported that she had been experiencing generalized abdominal pain all week and that she was unable to eat. (*Id.* at 975.) Petitioner reported trouble walking and left leg spasms, with increased pain. (*Id.*) Petitioner was given a gastrointestinal cocktail of Zofran and normal saline. (*Id.*) Her treating physicians recommended that she continue to take iron supplements to manage her anemia and follow up with her primary care provider. (*Id.* at 978.)

Petitioner followed up with Dr. Yarnall on September 29, 2015. (Ex. 18, p. 1033.) Dr. Yarnall assessed petitioner with GERD, nausea, autoimmune disease, generalized abdominal pain, and prediabetes. (*Id.* at 1007.) Dr. Yarnall noted that the cause of petitioner's nausea was unclear and referred her to a GI specialist. (*Id.*) On October 2, 2015, petitioner presented for initial consult for her GI issues with Melissa Teitelman, M.D., and Sarah Coppolino, PA. (*Id.* at 1018.) PA Coppolino ordered bloodwork for H pylori, an esophagogastroduodenoscopy ("EGD") to assess for esophageal and gastric ulcerations and inflammation, and an abdominal ultrasound to assess for gallstones or chronic inflammation of the gallbladder. (*Id.* at 1024.) Petitioner's abdominal ultrasound was normal aside from some fatty infiltration of petitioner's liver. (*Id.* at 1036.) On November 11, 2015, petitioner later underwent an endoscopy, which revealed that the lumen of the lower third of her esophagus was mildly dilated, but was otherwise normal. (Ex. 18, p. 1226.)

On October 13, 2015, petitioner returned to Dr. Wells. (Ex. 18, p. 1046.) Petitioner reported more frequent and intense RA flares. (*Id.* at 1050.) Dr. Wells did not identify any signs of an active flare on examination. (*Id.* at 1054.) Dr. Wells noted that petitioner's report of unilateral wrist pain is somewhat atypical for RA and more often associated with gout or pseudogout. (*Id.*) Dr. Wells did not observe any signs of chondrocalcinosis on petitioner's X-ray and ordered CK, ESR, CRP, uric acid, and BNP labs to monitor petitioner's RA and screen for gout and hypertension. (*Id.*) She also ordered an electrocardiogram for additional hypertension screening. (*Id.*)

Petitioner returned to NP Aitken on October 14, 2015, for a follow up on her left frozen shoulder. (Ex. 18, p. 1074-81.) Petitioner's exam revealed full strength and range of motion and she was encouraged to continue her physical therapy and home exercises. (*Id.* at 1081.) Petitioner saw Dr. Liu on November 13, 2015, to follow up on her right leg and neck pain. (Ex. 18, p. 1253.) Petitioner reported that she was pain free aside from occasional back pain. (*Id.* at 1256.) Petitioner was walking but had decreased range of motion on her lumbar spine exam. (*Id.*) Dr. Liu recommended a trial of home exercises to help manage petitioner's pain. (*Id.* at 1257.)

On December 2, 2012, petitioner saw Dr. Moon for a follow-up on her neuropathy and immune disorder. (Ex. 18, p. 1288.) Dr. Moon noted that gallstones were found on petitioner's abdominal ultrasound and recommended that she follow up with a GI specialist and consider cholecystectomy. (*Id.* at 1291-92.) Petitioner also returned to the Duke Gastroenterology Clinic on December 17, 2015, and saw PA Coppolino. (Ex. 18, p. 1334.)

On January 5, 2016, petitioner returned to Dr. Wells for left shoulder and left-side neck pain believed to be caused by her myositis. (Ex. 18, p. 1351.) Dr. Wells noted that petitioner was doing very well and showed no weakness or signs of active synovitis. Petitioner declined additional prescriptions for her RA. (*Id.* at 1360.) Dr. Wells ordered CBC, ALT, AST, CK, Creatine, ESR, CRP, Sm antibody, RNP, and anti-SCL70 labs, and recommended that petitioner continue to follow up with her pulmonary specialists. (*Id.* at 1360, 1363.)

On March 2, 2016, petitioner returned to Dr. Moon to follow up on her pain and immune disorders. (Ex. 18, p. 1430.) Petitioner's anemia was borderline, and her pain was variable but seemed to worsen with certain activities. (*Id.* at 1435.) Dr. Moon advised petitioner to rest every afternoon to deal with her chronic fatigue. (*Id.*) Petitioner called Dr. Wells' office on March 7, 2016, complaining of pain and stiffness in her right shoulder and intermittent stabbing pain and stiffness in her right hand. (Ex. 18, p. 1464.) Dr. Wells believed this was an RA flare and recommended either prednisone or the maximum dose of Tylenol and ibuprofen. (*Id.*)

On March 14, 2016, petitioner presented to Dr. Metjian for follow up on her ILD. (Ex. 18, p. 1466.) Petitioner denied any worsening in her ILD symptoms. (*Id.* at 1470-71.) Petitioner's lung volumes were consistent with severe restrictive lung disease, her

carbon monoxide diffusing capacity was substantially reduced, and "the potential effect of anemia on reducing carbon monoxide uptake [could not] be ruled out." (*Id.* at 1473.)

Petitioner returned to Dr. Wells for a follow up of her chronic pain symptoms on April 4, 2016. (Ex. 18, p. 1479.) Petitioner reported that her previous hand pain had resolved but she now suffered from elbow pain for one day, right-side knee pain since March, and occasional back pain. (*Id.* at 1483.) Dr. Wells noted that petitioner was "doing great from a myositis standpoint," but was concerned about underlying RA activity. (*Id.*) Petitioner's examination was generally unremarkable, but she did show some tender joints. (*Id.* at 1488.) Dr. Wells noted that she believed petitioner's right knee pain was due to bursitis and that her elbow pain stemmed from her right shoulder since it mostly occurred with right shoulder movement. (*Id.*) Petitioner's X-ray showed a collapse of the proximal capitate with underlying vague lucencies and extensive sclerosis most consistent with osteonecrosis and collapse, but no definite erosions. (*Id.* at 1493.)

Petitioner presented to David Attarian, M.D., and PA Chanel Copeland at Duke Orthopaedics on April 8, 2016, for evaluation of left shoulder pain. (Ex. 18, p. 1521.) Petitioner reported that her shoulder pain began after her visit to Dr. Wells four days prior and that she was waiting on authorization to fill an indomethacin prescription. (*Id.* 1524.) Petitioner's left shoulder examination showed generalized tenderness to palpation with extremely limited range of motion and strength. (*Id.* at 1525.) PA Copeland assessed petitioner with chronic left shoulder pain and adhesive capsulitis and ordered an increased Toradol injection along with a series of left shoulder X-rays. (*Id.* at 1526.) Petitioner's left shoulder X-ray revealed mild AC joint degeneration. (*Id.* at 1528.)

Petitioner next reported to Dr. Liu on June 10, 2016, for back pain. (Ex. 24, p. 1546.) Petitioner reported that her back pain began following her epidural injection in August 2015, and that it was primarily in her lower back but radiated to her leg occasionally. (*Id.*) She further explained that stretching, Tylenol, and Flexeril helped to alleviate severe pain but that she otherwise experienced a dull pain. (*Id.* at 1550.) Dr. Liu noted that petitioner's pain was likely a symptom of a disc bulge abutting the S1 nerves in the spinal canal and recommended a series of home exercises. (*Id.*)

On July 7, 2016, petitioner reported to Katherine Kaufman, M.D., to follow up on her RA and ILD. (Ex. 24, p. 1580.) Petitioner's myositis seemed to be under control due to her normal strength on examination. (*Id.* at 1586.) Petitioner's ESR and CRP levels were elevated, but not more than usual, Dr. Kaufman recommended Plaquenil to assist with petitioner's RA symptoms. (*Id.* at 1605.)

Petitioner next visited PT Alan Sirk for disability evaluation and testing on September 12, 2016. (Ex. 24, p. 1711.) PT Sirk concluded that petitioner was incapable of performing the physical demands of her target job of Tax-Record Clerk. (*Id.* at 1721-22.) PT Sirk's conclusion was primarily based on petitioner's significant upper extremity impairment and positional intolerance limitations. (*Id.*)

Petitioner followed up on her ILD with Dr. Metjian on September 14, 2016. (Ex. 24, p. 1742.) Petitioner reported ongoing dyspnea with walking upstairs or walking for several minutes and noted that she experienced shortness of breath, tightness in her legs, and numbness in her feet. (*Id.* at 1746.) Dr. Metjian observed that petitioner's lung function had declined, indicating a progressive disease. (*Id.* at 1748.)

On September 19, 2016, petitioner presented to the Duke ED on September 19, 2016, with a chief complaint of "[g]eneralized dysmotility of intestine." (Ex. 24, p. 1773.) Upon initial examination, Kevin Gurysh, M.D., noted that petitioner's labs were remarkable for leukocytosis (high white blood cell count) and electrolyte abnormalities, and that her CT imaging revealed colitis of her transverse colon. (*Id.* at 1786.) Dr. Gurysh considered infectious versus inflammatory processes, and, while less likely, ischemia as the cause of petitioner's GI pain. (*Id.*) A subsequent abdominal angiogram was normal, which ruled out vasculitis, while a colonoscopy and biopsy suggested possible ischemic colitis. (*Id.* at 1794.) Petitioner's bowel functions improved, but her abdominal pain did not, and a repeat CT revealed ongoing inflammation and new enteritis. (*Id.*) On October 6, 2016, a push enteroscopy showed "generalized slow transit consistent with scleroderma-related disease and ulcers most consistent with small intestinal bacterial overgrowth ("SIBO"). (*Id.*) Her discharge summary notes that after ten days of antibiotics and little improvement, inflammatory bowel disease ("IBD") and vasculitis "remained high on the differential." (*Id.*)

On October 20, 2016, petitioner presented to Christopher Eckstein, M.D., at the Duke Neuroscience Center seeking an expert opinion in this case. (Ex. 24, p. 4352.) Dr. Eckstein noted that petitioner's extensive rheumatologic and autoimmune history could predispose her to SFN, but that he "it is possible that the vaccination could have exacerbated her underlying autoimmunity, which would predispose her to the neuropathy, though this would be difficult to prove definitively." (*Id.* at 4358.)

Petitioner did not submit any records documenting her treatment from October 20, 2016, to February 22, 2018.

Petitioner returned to Dr. Kaufman on February 22, 2018, to follow up on her RA. (Ex. 63, p. 47.) Dr. Kaufman noted that petitioner continued to have significantly active RA. (*Id.* at 53.) On March 8, 2018, petitioner saw PA Neil Ewing for bilateral shoulder pain. (Ex. 63, p. 113.) PA Ewing believed that petitioner's shoulder pain was due to a RA flare, as she had stopped taking her RA medications. (*Id.* at 125.) Petitioner saw Dr. Kaufman with complaints of left shoulder, knee, hand, and foot pain on May 24, 2018. (Ex. 63, p. 452.) Dr. Kaufman considered petitioner's pain to be an active RA flare a prescribed a depomedrol injection, with a Medrol Dosepak to be administered if the injection failed to address petitioner's pain. (*Id.* at 457.) She returned to Dr. Kaufman on August 24, 2018. (*Id.* at 524.) Petitioner reported that she was feeling better since starting rituximab, but she continued to show signs of active RA on her examination. (*Id.*) Dr. Kaufman also observed that petitioner's lung and muscle

disease seemed stable. (*Id.*) Petitioner did not exhibit any weakness on her examination, and based on her subjective reports, her ILD appeared stable. (*Id.*)

On October 18, 2018, petitioner visited Dr. Mhoon for a follow up on her SFN. (Ex. 63, p. 660.) Petitioner reported that her neuropathy was overall stable with limited dysesthesias and only occasional burning pain in her arms. (*Id.* at 666.) Dr. Mhoon noted that petitioner's SFN "may have been a reaction to the vaccination or sequelae from rheumatoid arthritis." (*Id*. at 673.)

On December 6, 2018, petitioner visited Lake Morrison, M.D., at Duke Pulmonology for pulmonary function testing. (Ex. 63, p. 785.) Her testing revealed a restrictive process and substantially reduced diffusing capacity, which was the same as her prior pulmonary test results. (*Id.* at 741.) Dr. Morrison noted that petitioner's ILD was generally stable and that there was no need to adjust her medication. (*Id.* at 781, 773.) An updated CT showed progression of asymmetric right greater than left ILD with increased interstitial consolidation and bronchiectasis as compared to her earlier chest CT from March 5, 2015. (*Id.* at 773-74.) This was interpreted as representing an NSIP pattern of fibrosis, most likely in the setting of connective tissue disease. (*Id.*) Petitioner next saw Eugene Friedman, M.D., on January 25, 2019, for additional pulmonary testing, which had remained unchanged from her previous study. (Ex. 63, p. 937.)

Petitioner returned to Dr. Mhoon on October 18, 2019, to follow up on her SFN. (Ex. 63, p. 1506.) Petitioner reported that her SFN seemed stable overall and that she had no significant neuropathic pain other than some cramping in her toes. (*Id.* at 1512.)

Petitioner underwent additional pulmonary function testing on December 2, 2019, which remained unchanged from her previous test. (Ex. 63, pp. 1698-99.) That same day, petitioner followed up on her ILD with Dr. Morrison. (*Id.* at 1729.) Dr. Morrison noted that petitioner was stable from a pulmonary perspective, recommended a twelve-month follow up appointment and additional physical activity. (*Id.* at 1732-33.)

On May 21, 2020, petitioner emailed Dr. Mhoon reporting that she was experiencing a burning sensation in her left arm similar to that which she experienced following her vaccinations in 2014. In response, Dr. Mhoon's assistant recommended an essential oils topical lotion. (Ex. 63, p. 2054.)

The remainder of petitioner's medical records focus on treatment of her frozen shoulder, routine visits, and unrelated illnesses. (*See generally* Ex. 63, pp. 2056-3870.)

b. As Reflected in Petitioner's Affidavits

Petitioner filed her first statement on February 12, 2017. (Ex. 3.) This "affidavit" (signed, but not sworn) was a comprehensive filing and contained a chronology of petitioner's medical treatment, and several statements addressing the basic elements of

a claim for entitlement in the program. Petitioner's extensive description of her specific medical encounters, though reviewed, will not be repeated herein.

Petitioner explains that she received her immunizations on November 7, 2014, at the UNC Internal Medicine primary care clinic. (Ex. 3, p. 2.) She states that she was in fair health prior to the immunizations due to her RA and polymyositis. (*Id.*) She was taking CellCept, Omeperazole for heartburn, and other prescription as needed. (*Id.*) Petitioner notes that her RA and myositis were stable prior to her vaccinations. (*Id.*) She states that her quality of life, independence, and daily living activities were improving, and she was tolerating consistent workouts. (*Id.*) Petitioner received previous flu vaccines in 2012 and 2013 on her doctor's recommendation, and at some point also received a pneumonia vaccination. (*Id.* at 3.)

Petitioner notes that she received the flu vaccine in her left deltoid, and the pneumococcal vaccine in her right. (Ex. 3, p. 3.) Petitioner recounts that she was awakened by a "horrible, burning sensation in both arms," on November 8, 2014. (*Id.* at 4.) From November 8 to November 11, petitioner received at-home care until calling the UNC Nurse Advice Line and being advised to make an appointment with her primary care physician. (*Id.*) Petitioner reports that the burning sensation started in her shoulders and shot down to her forearms. Both of petitioner's arms were sore and swollen at the injection sites, and moving her arms caused pain that woke her from sleep. (*Id.*) Petitioner used Tylenol, Tramadol, Flexeril, and Oxycodone to manage her pain, but none of her medications alleviated the burning sensation. (*Id.*)

Petitioner notes that as of the date she wrote her affidavit, she continued to experience neck, left shoulder, left wrist, and upper back pain described as "painful burning sensation[s], knife stabbing, [and] something crawling on my skin and numbness." (*Id.*)

Petitioner further explains in her affidavit that she is unable to complete light household chores without pain, that she spends much of her time resting because it is how she can get the most relief, that her sleeping patterns are inconsistent, and that her inability to resolve her issues or find a unifying diagnosis has "been very traumatizing, dramatic, and painful for me." (*Id.*)

Petitioner describes the burden that her need for care has placed on her family. (*Id.* at 17.) She explains that she no longer works, has lost her independence, and lacks emotional support due to her distance from family. (*Id.* at 17-18.) Petitioner concludes her affidavit by stating:

> I know my pain and suffering was directly caused by the Flu and Pneumonia vaccines received. The pain started the night of Nov. 7th, 2014 after the shots and has continued for the past 8 months to the present. I believe I had no conditions previous to the vaccine that could have caused the pain I have experienced and continue to experience."

(*Id.* at 18.)

Petitioner filed a brief affidavit on February 16, 2018, explaining that she received the pneumococcal conjugate 3-valient vaccine and influenza vaccine at Duke Medical Clinic on November 7, 2014. She also notes that she "experienced persistent, painful burning sensation in [her] extremities after receiving the vaccination, for which [she] sought medical treatment." (Ex. 48.) She further explains that she filed a VAERS report on December 2, 2014. Finally, petitioner attests that she has not received any additional documentation regarding the investigation of her initial VAERS report. (*Id.*)

c. As Reflected in Petitioner's Testimony

Petitioner testified at the entitlement hearing on July 14, 2021. Petitioner recalled being diagnosed with autoimmune necrotizing myopathy and seropositive RA in 2010. (Tr. 6.) Between 2010 and 2014, before receiving the flu and pneumococcal vaccinations, petitioner testified that her autoimmune necrotizing myopathy caused muscle weakness throughout her body and muscle aches. (*Id.*) She also recalled having pain in her shoulders and back related to sitting or standing for prolonged periods. (*Id.* at 6-7.) Regarding her RA, she recalled experiencing symptoms of swelling and stiffness as well as limited range of motion in her wrists and hands. (*Id.* at 7.) She testified that these conditions made her permanently disabled and unable to work. (*Id.*) Prior to receiving the vaccinations at issue, petitioner testified that her myopathy and RA were stable. (Tr. 7.) Although she still had physical limitations, she was able to cook meals with assistance, wash dished, sweep the floor, do laundry, go for walks, and participate in activities with friends and family. (*Id.* at 7-8.) However, she recalled that her energy levels would "depreciate throughout the day." (*Id.* at 8.)

Petitioner testified that she received the pneumococcal and flu vaccinations in separate arms on November 7, 2014, when she was visiting her primary care provider. (Tr. 8-9, 25.) She recalled that she was not feeling any pain and was able to drive herself to the doctor and walk into the office without using a cane, walker, or wheelchair. (*Id.* at 9.) After leaving the doctor's office, she recalled feeling some soreness. (*Id.* at 9-10.) She testified that she was visiting her son after the appointment but had to "cut the visit short" because the soreness in her arm was getting worse. (*Id.* at 10.) After going to bed, she woke up in the middle of the night screaming and crying "because [she] felt like the inside of [her] body was on fire." (*Id.*; *see also id.* at 25-26.) She described feeling a painful burning sensation in her arms. (*Id.* at 10.)

Over the next week, petitioner could not "get the burning sensation to go away." (Tr. 10.) She self-treated the pain with ice and Tylenol, which did not help. (*Id.*) She could not sleep due to the pain, which was worse at night. (*Id.*) She described the burning in her arms as "sharp in one arm and then it would get real intense in the other one." (*Id.*) At that point, she decided to see the doctor for her symptoms. (*Id.*) As the burning sensation progressed in her arms over the next week, she began feeling it in her back, neck, and shoulders, followed by her legs and feet. (*Id.* at 11; *see also id.* at 29-31 (stating that she "felt like [she] was on fire in different parts of [her] body" and that

the burning pain would move from one part of her body to another).) She remembered telling an ER nurse that the burning sensation was moving down to her leg. (*Id.* at 11.) She also began feeling anxious, scared, and confused. (*Id.*)

Petitioner differentiated the post-vaccination burning sensation from symptoms caused by her myopathy and RA. (Tr. 11.) She explained that with her muscle disease and RA, her muscles would "sting," her hands would "throb," and she would have stiffness in her joints. (*Id.* at 11-12.) Conversely, she described her post-vaccination symptoms as feeling like "the inside of [her] body [was] on fire." (*Id.* at 12.) She noted that her skin was not hot to the touch and that she did not have a fever despite feeling like the inside of her body was on fire. (*Id.*) Petitioner remembered seeing Dr. Mhoon on December 2, 2014. (Tr. 12.) She stated that Dr. Mhoon ordered blood tests, two MRIs, and a skin biopsy to rule out other autoimmune diseases. (*Id.*) She testified that her skin biopsy was positive for small fiber neuropathy, which was the first time she heard that diagnosis. (*Id.* at 13.) She further recalled telling her treating doctors that her SFN symptoms were different from her preexisting conditions. (*Id.* at 13, 33.) She elaborated that when she saw Dr. Mhoon, she told him that she "never felt this before." (*Id.* at 13.)

At the time of the hearing, petitioner testified that she continued to experience symptoms of SFN, though more sporadically. (*Id.*) She explained that she experiences the burning sensation in her arms intermittently. (*Id.*) She explained that the SFN impacted her sleep, caused fatigue, prevented her from being as involved with her children, and made her unable to exercise or drive. (*Id.* at 18.) She described experiencing painful flares, having a frozen shoulder, and having a swollen wrist. (*Id.* at 18-19.) She testified that her condition caused her to lose her independence. (*Id.* at 19.) She further stated that her SFN caused her to suffer depression. (*Id.* at 20.) She averred that her health has not gotten back to the place it was prior to vaccination. (*Id.*)

IV. Expert Opinions

a. Petitioner's Expert, Carlo Tornatore, M.D.

Petitioner filed several reports from neurologist Dr. Tornatore to support her claim. (Exs. 36, 50, 54, 60; ECF No. 89.[7]) Dr. Tornatore also testified during the hearing. (Tr. 35-107, 213-26.) Dr. Tornatore was proffered without challenge as an expert in neurology.[8] (Tr. 41.)

[7] Dr. Tornatore's second report was marked with an exhibit number (26) that was duplicate and out of sequence. It will therefore be referred to by its docket entry – "ECF No. 89."

[8] Dr. Tornatore received his medical degree from Georgetown University School of Medicine in 1986. (Ex. 37, p. 2.) In 1987, he completed an internship in internal medicine at Providence Hospital in Washington, DC. (*Id.*) He completed his residency in Neurology at Georgetown University Hospital in 1990, followed by a fellowship in molecular virology at the National Institute of Health from 1990 to 1994. (*Id.*) He current serves as interim chairman for the department of neurology at Medstar Georgetown University Hospital, as well as chairman of the department of neurology at Georgetown University Medical Center and as Professor of Neurology at Georgetown University Medical Center. (*Id.* at 3.) Previously,

i. Diagnosis

Dr. Tornatore described small fiber neuropathy as an injury to "the unmyelinated C-fibers, thinly myelinated A-deltas, and postganglionic sympathetics," also known as "small fibers." (Ex. 36, p. 8 (citing Anne Louise Oaklander, *Immunotherapy Prospects for Painful Small-fiber Sensory Neuropathies and Ganglionopathies*, 13 NEUROTHERAPEUTICS 108 (2016) (Ex. 38)).) He notes that "the small fibers sense pain and itch, innervate internal organs and tissues, and modulate the inflammatory and immune responses." (*Id.*) He explained that symptoms of the condition include "chronic pain and itch, sensory impairment, edema, and skin color, temperature, and sweating changes." (*Id.*) Small fiber polyneuropathy may also cause "cardiovascular, [GI], and urological symptoms, the neurologic origin of which often remains unrecognized." (*Id.*) According to Dr. Tornatore, the time course and etiology of SFN is "strikingly similar to Guillain-Barre [syndrome ("GBS")]" and "characterized by autonomic and sensory impairment without motor dysfunction that reaches its nadir within a short period of time. (Ex. 36, p. 8.) The monophasic clinical course and frequent presence of a history of antecedent infections suggest a participation of immune mechanisms." (*Id.* (citing Haruki Koike & Gen Sobue, *Autoimmune Autonomic Ganglionopathy*, 53 RINSHO SHINKEIGAKU 1326 (2013) (Ex. 44)).) Skin biopsies are a key diagnostic technique because routine electrodiagnostic studies fail to detect SFN. (Ex. 38, p. 8.) Dr. Tornatore also reports that corticosteroids and immunoglobulins have been effective in treating SFN. (*Id.*)

In response to Dr. Gelfand's assertion that brachial neuritis may provide a better explanation for petitioner's upper extremity symptoms, Dr. Tornatore noted that Dr. Gelfand conceded that "[i]n rare cases, small fiber neuropathy follows a non-length dependent distribution in which symptoms may be manifested predominately in the arms, face or trunk . . . [and that] non-length dependent patterns occur rarely including in presumed dysimmune causes." (ECF No. 89, p. 2.) Dr. Tornatore interpreted this statement to mean that Dr. Gelfand conceded that petitioner's upper extremity symptoms could be consistent with a dysimmune sensory neuropathy. (*Id.*) Dr. Tornatore also addressed Dr. Gelfand's contention that petitioner's lower extremity condition was a comorbidity independent of the process that was causing the burning sensation in her upper extremities. (Ex. 50, p. 3). Although Dr. Gelfand opined that "an acute small fiber neuropathy involving the proximal upper extremities (a non-length dependent pattern) would be atypical" (Ex. J, p. 9), Dr. Tornatore noted that petitioner's skin biopsy report stated that "[o]ccasionally, patients with a length dependent neuropathy can feel symptoms in the hands before the lower extremities" (Ex. 50, p. 3). Thus, Dr. Tornatore concluded that petitioner's upper extremity symptoms are attributable to SFN.

Dr. Tornatore has held positions as vice chairman of the department of neurology at Medstar Georgetown University Hospital, Associate Professor at Georgetown University Medical Center, and Assistant Professor in the department of neurology at Georgetown University Medical Center. (*Id.*) Dr. Tornatore has also published 63 original papers and book chapters on neurology and virology. (*Id.* at 8-14.)

ii. Causation

1. *Molecular Mimicry*

Dr. Tornatore opined that there are two possibilities that would explain petitioner's painful neuropathic sensory symptoms. First, he posited that petitioner was predisposed to autoimmune disorders and developed "[d]e novo" autoimmune small fiber sensory neuropathy shortly after an antigen challenge from her vaccinations. (Ex. 36, pp. 7-8; Tr. 50, 97-98.) Second, he opined that an "indolent, asymptomatic autoimmune small fiber neuropathy . . . was profoundly aggravated" following the antigen challenge from her vaccinations. (Ex. 36, pp. 7-8; Tr. 50, 97-98.) In either event, Dr. Tornatore concluded that "the vaccinations were the inciting factor." (Ex. 36, pp. 7-8.)

With respect to the mechanism by which the vaccinations petitioner received can cause SFN, Dr. Tornatore offered molecular mimicry. (Ex. 36, pp. 8-9.) Dr. Tornatore explained that the process of molecular mimicry triggers an autoimmune response that may result in inflammatory polyneuropathies, including small fiber neuropathy. (*Id*.) Dr. Tornatore opined that when antigens "present on the vaccine share any homology with host antigens . . . the immune response will be directed at both the injected antigens and host antigens, leading to an autoimmune response." (*Id.* at 9 (citing Michael B.A. Oldstone, *Molecular Mimicry, Microbial Infection and Autoimmune Disease: Evolution of the Concept*, 296 CURRENT TOPICS IN MICROBIOLOGY & IMMUNOLOGY 1 (2005) (Ex. 47)).)

Dr. Tornatore further opined that "vaccines have been recognized to trigger autoimmune responses, albeit rarely, that lead to autoimmune response directed against antigens on peripheral nerves, resulting in inflammatory polyneuropathies." (Ex. 36, p. 9 (citing Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979) (EX. 46); J.D. Pollard & G. Selby, *Relapsing Neuropathy Due to Tetanus Toxoid*, 37 J. NEUROLOGIC SCIS. 113 (1978) (Ex. 39)).) Dr. Tornatore also noted that there are numerous case reports of autoimmune peripheral neuropathies following other vaccinations, including rabies, varicella, Lyme, and HPV. (*Id.* (citing Frederic E. Shaw, JR. et al., *Postmarketing Surveillance for Neurologic Adverse Events Reported After Hepatitis B Vaccination*, 127 AM. J. EPIDEMIOLOGY 337 (1988) (Ex. 45); Nizar Souayah et al., *Small Fiber Neuropathy Following Vaccination for Rabies, Varicella or Lyme Disease*, 27 VACCINE 7322 (2009) (Ex. 40); Svetlana Blitshteyn, *Postural Tachycardia Syndrome Following Human Papillomavirus Vaccination*, 21 EUR. J. NEUROLOGY 135 (2014) (Ex. 42)).) According to Dr. Tornatore, it is not only a homology in amino acid sequences where exogenous antigens may induce an autoimmune response, but receptors on B and T cells are now known to recognize peptide sequences that share no homology and in turn mount an autoimmune response on the nervous system through a process known as 'degeneracy.' (*Id.* (citing Don Mason, *A Very High Level of Crossreactivity Is an Essential Feature of the T-Cell Receptor*, 19 IMMUNOLOGY TODAY 395 (1998) (Ex. 41)).)

Dr. Tornatore agreed with Dr. Gelfand's assertion that to prove molecular mimicry, sequence homology between neuronal and vaccinal antigens must be shown. (Ex. 89, p. 2.) However, Dr. Tornatore stressed that sufficient sequence homology has already been demonstrated. Dr. Tornatore cited an article that found homologies between myelin proteins and viral proteins including measles, Epstein-Barr, influenza A and B, and others that cause upper respiratory infections. (*Id.* (citing Ulrike Jahnke et al., *Sequence Homology between Certain Viral Proteins and Proteins Related to Encephalomyelitis and Neuritis*, 229 SCIENCE 282 (1985) (Ex. 51)).) Dr. Tornatore further noted that Dr. Gelfand authored a paper stating that "acute disseminated encephalomyelitis . . . can be preceded by an acute systemic infection or vaccination," acknowledging that vaccination can lead to molecular mimicry-induced autoimmunity. (Ex. 54, p. 5.)

Dr. Tornatore also addressed Dr. Whitton's contention that "[t]here are, however, no clear examples of a human disease caused by molecular mimicry." (Ex. 89, p. 4.) Dr. Tornatore explained that this is incorrect, as rheumatic fever and Sydenham's chorea have been recognized "as the classic examples of molecular mimicry in clinical medicine for over a century." (*Id.*) Dr. Tornatore cited a paper by Carapetis and others, which found that after a group A streptococcal infection ("GAS") of the pharynx in susceptible individuals, "the host response against GAS will trigger autoimmune reactions against host tissues mediated by both Streptococcus-specific antibodies and T cells through a process called molecular mimicry." (*Id.* (citing Jonathan R. Carapetis et al., *Acute Rheumatic Fever and Rheumatic Heart Disease*, 2 NATURE REV. DISEASE PRIMERS 15084 (2016) (Ex. 57)).) He explains that after GAS infections, children may develop antibodies against dopamine receptors, which are cross reactive with streptococcus epitopes. (Ex. 54, p. 5.)

Regarding petitioner's rapid onset, Dr. Tornatore explained that "small fiber sensory neuropathy can occur abruptly." (Ex. 36, p. 8.) Further, he opined that petitioner's previous flu vaccinations could cause "a subsequent re-challenge to mount a brisk and rapid immune response." (*Id.* at 8, 9.) He cited a report from the Institute of Medicine that shows that the latency or lag phase for an autoimmune response following exposure to "many antigens" is between seven and ten days, while "the latency between subsequent exposure to the antigen and the immune response will usually be shorter . . . generally one to three days; the logarithmic phase of the secondary antibody response occurs over the next three to five days." (*Id.* (citing INST. OF MED., EVALUATING BIOLOGICAL MECHANISMS OF ADVERSE EVENTS 51-52 (2011) (Ex. 43)).)

In the context of molecular mimicry, Dr. Tornatore opined that the "secondary immune response" phenomenon explains petitioner's rapid onset. The phenomenon is described as

> a markedly enhance[d] response that is characterized by the accelerated appearance of immunocompetent T and B cells referred to as "memory cells" that collaborate to generate an enhanced production of antibody. The

> latent period is much shorter during the secondary immune response since the memory cells are present at a higher frequency and are available to be stimulated quickly . . . this is the basis for the IOMs recognition that the second exposure to a vaccination may result in a markedly shorter time of onset of an immune response to administration of an exogenous antigen.

(Ex. 50, p. 3.)

Therefore, Dr. Tornatore concluded that it was medically plausible that petitioner's flu and/or pneumococcal vaccine caused her SFN via molecular mimicry.

2. *Interferon Gamma ("IFN- γ") Theory*

In his later reports, Dr. Tornatore offered another explanation for how the vaccines petitioner received can cause SFN in a rapid timeframe. (Exs. 54, 60.) Dr. Tornatore explained that the rapid onset in this case is due to a cytokine reaction producing interferon gamma ("IFN- γ"), a modulator of neuropathic pain. (*See generally id.*) He described an experiment conducted by Dr. Whitton where naïve mice were exposed to viral antigens and an inoculation against that antigen. In the experiment, the mice began producing IFN- γ, a signal that the immune response had been activated, within 6-12 hours. (Ex. 54, p. 1.) The mice were exposed to the same viral antigens and inoculated roughly a year later, and began producing IFN- γ within hours of exposure. (*Id.* at 1-2.) Dr. Tornatore explained that these results show that "within hours of activation, the immune system can produce interferon-gamma, a known mediator of chronic pain . . . consistent with [petitioner's] clinical symptoms." (*Id.* at 2.) Dr. Tornatore theorized that a vaccination could cause the systemic dissemination of viral antigens that could cross into the dorsal root ganglia ("DRG") due to the lack of a blood-brain barrier. (*Id.* at 3.) He discussed how the vaccination could stimulate resident lymphocytes of the DRG that produce IFN- γ, thus resulting in the sensation of pain in a dermatomal distribution. (*Id.*) He asserted that within days, "there will be replication of the lymphocytes with further injury to the DRG ultimately resulting in loss of the sensory projections from the DRG into the extremity . . . ." (*Id.*)

Dr. Tornatore further noted that "the sensory component of the nervous system has a very low threshold to stimulation in the face of a noxious event." (Ex. 54, p. 2.) Citing the diagram in his report, he explains that "there are dermatomes over the extremities that represent the innervation from a single nerve root. If one looks at the C7 nerve, irritation or inflammation of that nerve root will result in sensory changes that involve a significant part of the arm." (*Id.* at 2-3.) Dr. Tornatore explains that the DRG is where the cell bodies of sensory nerves lie, they are very small, and have a unique interface between accumulations of sensory neurons and blood vessels, specifically, that they lack a blood-brain barrier which allows blood borne molecules to directly enter the DRG and interact with neuronal and non-neuronal cells. (*Id.* at 2 (citing Rainer Viktor Harberger et al., *Human Dorsal Root Ganglia*, 13 FRONTIERS IN CELLULAR NEUROSCIENCE 271 (2019) (Ex. 56)).) The non-neuronal cells contained within the DRG

include a group of immune cells consisting mainly of macrophages and T-lymphocytes, with a lower number of B-lymphocytes. (*Id.*) Dr. Tornatore simplifies this concept, noting that in the case of the DRG:

> [W]e have a very small structure with an absent blood-brain barrier that contains not only neurons but also lymphoid and myeloid cells, that can mediate pain and other sensory modalities over a large area. As such, it is biologically plausible that the administration of a vaccine could result in the systemic dissemination of viral antigens that could easily cross into the dorsal root ganglia given the lack of blood brain barrier, stimulate resident lymphocytes of the dorsal root ganglia that then produce IFN-gamma resulting in the sensation of pain in a dermatomal distribution, e.g. the length of the arm, as was the case with [petitioner].

(*Id.* at 3.)

Dr. Tornatore agreed with Dr. Whitton that "within days there will be replication of the lymphocytes with further injury to the DRG ultimately resulting in loss of the sensory projections from the DRG into the extremity, as was the case in Ms. McGill's punch biopsy." (Ex. 54, p. 3.) He stated that he and Dr. Whitton were "in agreement that the immune system has the capability of being stimulated quickly and producing soluble factors such as interferon gamma." (Ex. 60, p. 2.) He further opined that "immune cells can be quickly activated by exogenous antigens to provide sensory symptoms referable to the nervous system." (*Id.* at 3.) Thus, Dr. Tornatore reasoned that because memory T cells can produce IFN- γ within hours of exposure to viral antigen, it is biologically plausible that the vaccines petitioner received caused her to develop SFN 8.75 hours after administration. (*See* Ex. 54, pp 1-2.)

b. Respondent's Experts[9]

i. J. Lindsay Whitton, M.D., Ph.D.

Respondent also filed several reports from immunologist Dr. Whitton to support his position. (Exs. C, M, N.) Dr. Whitton also testified at the hearing. (Tr. 144-213.) He was proffered without objection as an expert in immunology.[10] (Tr. 153.)

[9] Respondent also filed an expert report from rheumatologist Chester Oddis, M.D. (Ex. A.) However, respondent confirmed in his prehearing brief that he did not intend to present live testimony from Dr. Oddis given that he addressed petitioner's claims of ILD and myofascial pain syndrome. (ECF No. 135, p. 3. n.4.) Based on petitioner's intent to proceed solely with her claim for SFN and abandon her claim for ILD and myofascial pain syndrome (ECF No. 148, p. 23, n.7), discussion of Dr. Oddis' report has been omitted from this decision.

[10] Dr. Whitton received his medical degree from the University of Glasgow in 1979, and his Ph.D. from the same university in 1984. (Ex. D, p. 1.) He currently serves as a professor in the department of immunology and microbial science at Scripps Research Institute, and previously held positions as a professor in the department of neuropharmacology, and molecular and integrative neurosciences at Scripps as well. (*Id.*) He is a member of several professional societies including the American

1. *Causation*

a. Molecular Mimicry

Dr. Whitton stated that although that molecular mimicry is a biological phenomenon, it is not a reliable theory for how the flu or pneumococcal vaccines can cause SFN. (Ex. M, p. 10.) Dr. Whitton pointed out that a report from the IOM cited by Dr. Tornatore notes, "[w]hile molecular mimicry is a well-established mechanism in selected animal models, its relevance to human autoimmune disease remains in most cases to be convincingly proven." (*Id.* (quoting INST. OF MED., *supra*, at Ex. 43, p. 12).) The IOM publication further states:

> [W]e found little clinical evidence (e.g., challenge/rechallenge), diagnostic evidence (e.g., presence of antigen or relevant immune complexes in affected tissue), or experimental evidence (e.g., in vitro evidence of cross-reactive T-cells derived from a site of tissue injury) that could be consistent with the hypothesis of molecular mimicry in rare and selected case reports . . . . Based on the literature reviewed, molecular mimicry was not confirmed to be a mechanism leading to the development of the adverse events postvaccination.

(*Id.* (citing INST. OF MED., *supra*, at Ex. 43, p. 13).)

Dr. Whitton also asserted that "it is very difficult to cause disease via molecular mimicry." (*Id.* at 12; *see also* Tr. 157-58 (stating that the term "molecular mimicry" should not be equated with disease, as disease is a distinct step, and it is rare for molecular mimicry to cause disease).) He explained several difficulties in showing that viral infections can lead to molecular mimicry in animals. (Ex. M, pp. 11-12.) Dr. Whitton further noted that he was unaware of any medical literature confirming molecular mimicry as a mechanism by which the flu or pneumococcal vaccines can cause adverse events. (Tr. 167.) Likewise, he was unaware of any sequences in the flu or pneumococcal vaccines that could mimic antigens in small fiber nerves as required for molecular mimicry to occur. (*Id.* at 168.) Thus, Dr. Whitton concluded that neither the flu vaccine nor the pneumococcal vaccine is a recognized cause of SFN. (*Id.* at 197.)

Moreover, Dr. Whitton found it significant that prior to the vaccinations at issue, petitioner had been diagnosed with multiple autoimmune diseases, including ANM, RA, and ILD. He asserted that "SFN is often associated with autoimmune diseases, and some cases have been successfully treated using immunosuppressants." (Ex. C, p. 9.) Thus, Dr. Whitton concluded that petitioner's SFN is more likely "another manifestation

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Association of Pathologists, the American Association of Immunologists, the American Society of Virology, and the American Society of Microbiology. (*Id.*) He currently serves on the editorial boards of three different medical journals and holds the position of editor for the *Virology* medical journal. (*Id.* at 1-2.) Dr. Whitton has published 191 different pieces of medical literature focusing on virology, immunology, and molecular biology. (*Id.* at 2-14.)

of her susceptibility to autoimmune processes" and that petitioner's preexisting autoimmune diseases "provid[e] a likely explanation for her the eventual appearance of SFN." (Ex. C, p. 12.)

Regarding the appropriate timing for molecular mimicry, Dr. Whitton opined that the lag phase for a primary immune response following vaccination is seven to ten days, while the lag phase for a secondary immune response following vaccination is one to three days. (Ex. M, p. 6.) He asserted that both time intervals "far exceed" the 8.75-hour timeframe between petitioner's administration of the flu and pneumococcal vaccines and the onset of the burning sensation in her arms. (*Id.* at 6-8; Tr. 177.) Dr. Whitton noted that he was unaware of any "reliable mechanism" by which vaccinations could trigger SFN in only 8.75 hours. (Tr. 177; Ex. C, p. 12.) Dr. Whitton stressed that autoimmune diseases "take time to develop," and an antibody-mediated autoimmune neurological disease takes days to manifest, even in a previously immunized individual. (Ex. M, pp. 7-9, 12 (discussing how animal models show that an autoimmune neurological disease takes days to weeks to develop, even if the animal had been previously exposed to the inciting antigen).) Thus, even applying the appropriate interval for a secondary immune response, an 8.75-hour onset is too rapid to be plausible. (*Id.* at 7-8.)

Dr. Whitton further described the process of how T cells begin to divide in response to an antigen. (Ex. M, p. 9.) He explained that "T cells can respond to antigen challenge, in vivo, very rapidly – within hours." (*Id.*; *see also* Tr. 180-81 (noting that T cells can make cytokines, such as such as IFN-γ, within hours in response to an antigen challenge).) However, he emphasized that the T cells "do not begin to proliferate for several days (probably ~3-4 days)." (Ex. M, p. 9; Tr. 180-81.) He also clarified that this days-long lag phase applies to memory T cells. (Ex. M, p. 9; Tr. 181.) He elaborated:

> To cause systemic neurological disease, the T cells would have to (i) start to divide (which doesn't happen until ~3 days post-vaccination); (ii) divide multiple times over several more days to reach a sufficient number to cause harm; (iii) migrate to the nerves; and (iv) exert their effector functions thereupon, causing the signs and symptoms of SFN. So, it is implausible to suggest that a locally-injected vaccine could lead to a systemic T-cell mediated disease in only 8¾ hours.

(Ex. M, pp. 9-10 (citing Jason K. Whitmire, Boreth Eam, & J. Lindsay Whitton, *Tentative T Cells: Memory Cells Are Quick to Respond, but Slow to Divide*, 4 PLOS PATHOGENS e1000041 (2008) (Ex. M, Tab 4)).)

Although Dr. Whitton acknowledged that the timeframe may lessen as increasing numbers of booster injections are administered, he contended that "biology dictates that there be a minimum interval of several days." (*Id.* at 12.) Dr. Whitton maintained that petitioner's previous flu vaccinations would not shorten the required lag phase of 1-3

days for a secondary immune response because T cells take three days to start dividing. (Tr. 178.)

b. IFN- γ Theory

Dr. Whitton described Dr. Tornatore's IFN- γ hypothesis as asserting that "vaccine-specific memory T cells, activated after the vaccination, shed sufficient IFN- γ to trigger neuropathic pain." (Ex. N, p. 3.) Thus, he explained that Dr. Tornatore's IFN-γ theory has two parts: (1) antigens are disseminated systemically and enter the DRG where they trigger IFN-γ, causing pain; and (2) the antigen-bearing cells carry antigens into the lymphatics. (Tr. 185-86; *see also* Ex. N, p. 3.) He stated that Dr. Tornatore hypothesized that the IFN-γ caused neuropathic pain in the DRG. (Tr. 187.) He distinguished the IFN- γ theory from molecular mimicry, noting that molecular mimicry necessitates the induction of an immune response and the cross-reaction of the immune response with a self-antigen. (*Id.* at 187-88.) The IFN- γ theory is different from molecular mimicry as it does not involve a host antigen. (*Id.*) Further, Dr. Whitton explained that the IFN- γ theory is not autoimmune as an autoimmune response would require recognition of a self-antigen. (*Id.* at 188.)

Regarding the role of memory T cell responses in Dr. Tornatore's IFN- γ theory, Dr. Whitton explained that "it is a common misunderstanding that memory T cell responses, being 'faster and stronger,' must cause more discomfort / disease to the host." (Ex. N, p. 4.) In contrast, Dr. Whitton explained that "the primary T cell response to infection causes more systemic signs and symptoms than does the memory T cell response, which is faster and more biologically-beneficial." (*Id.*) He stressed that "T cells (both primary and memory) very tightly regulate the production of cytokines, including IFN- γ." (*Id.*) He explained that memory T cells prevent a virus from multiplying, and as a result, "very little virus antigen is produced," and the T cells can therefore terminate cytokine production quickly after a subsequent exposure. (*Id.* at 5.)

For Dr. Tornatore's IFN- γ theory to be feasible, Dr. Whitton opined that the proteins in a vaccination would need to diffuse from the site of administration, enter the DRG, and be taken up by an antigen presenting cell ("APC"). (Ex. N, p. 6.) Once inside the APC, the proteins would be degraded into short epitope peptides that are complexed with major histocompatibility complex ("MHC") molecules into the APC cytoplasm. (*Id.*) The MHC/peptide complexes would then travel to the surface of the APC, where they would activate vaccine-specific T cells in the DRG. (*Id.*) The T cells would then produce IFN-γ that acted on nerve cells through the IFN-γ receptor to cause petitioner's burning sensation. (*Id.* at 6, 9.) Dr. Whitton asserted that this process has never been demonstrated and was purely speculative. (*Id.* at 9; Tr. 191.) Dr. Whitton also noted that Dr. Tornatore did not provide any clinical or experimental support for this theory. (Ex. N, p. 9.)

Dr. Whitton also noted that petitioner's clinical presentation was inconsistent with Dr. Tornatore's IFN- γ theory. He opined that if the bilateral burning sensation in petitioner's arms were caused by the overproduction of IFN-γ by vaccine-specific T-

cells, he would have expected petitioner to have presented with inflammation, redness, and induration at the UNC Same Day Clinic on November 13, 2014. (Tr. 196.) Given that petitioner did not have a fever or swelling in either arm (Ex. 4, p. 3), Dr. Whitton concluded that a vaccine-mediated event was unlikely (Tr. 196; Ex. N, p. 6).

Further, Dr. Whitton argued that even if Dr. Tornatore's IFN- γ theory was possible, it would occur "extraordinarily rarely." (Ex. N, p. 6.) He noted, "Despite billions of doses of vaccine[s] having been administered, there is no recognized risk of antigen diffusing into DRG, causing severe burning neuropathic pain." (*Id.* at 9; *see also* Tr. 196.) Dr. Whitton concluded that Dr. Tornatore abandoned molecular mimicry as a feasible theory and offered "a completely new biological mechanism, antigen diffusing into DRG, for which he presents no evidence, and which makes little biological sense." (Ex. N, p. 8.)

Regarding the appropriate timeframe, Dr. Whitton agreed that the immune system can be activated rapidly following exposure to viral elements. (Ex. N, p. 3.) However, he emphasized the distinction between activation of the immune system and occurrence of neurological injury, which he stressed are "two completely separate biological events." (*Id.* at 2.) Dr. Whitton acknowledged that when a person has memory T cells due to previous exposure to a virus and reencounters that virus, "those T cells very quickly (within hours) are activated by antigen contact a at which point, they immediately produce cytokines (including IFN- γ) that shut down the virus' ability to multiply." (*Id.* at 5; *see also* Tr. pp. 180-81.) Although Dr. Whitton acknowledged that memory T cells can respond to antigen challenge by making cytokines within hours, he explained that those T cells still take about three days to proliferate, after which they begin to "divide explosively and exponentially." (Tr. 180-81.) Thus, although memory T cells can respond to antigen challenge within hours, Dr. Whitton maintained that both primary and memory T cells have a lag phase of about three days, and "the process of division is about the same." (*Id.* (citing Whitmire, Eam, & Whitton, *supra*, at Ex. M, Tab 4).) Thus, Dr. Whitton concluded that Dr. Tornatore's IFN- γ theory would not explain petitioner's rapid onset.

ii. Jeffrey M. Gelfand, M.D., MAS, FAAN

Respondent also filed two reports from neurologist Dr. Jeffrey Gelfand to support his position. (Exs. J, L.) Dr. Gelfand also testified at the hearing. (Tr. 107-139.) He was proffered without objection as an expert in both neurology and neuroimmunology.[11] (Tr. 111.)

[11] Dr. Gelfand received his medical degree from Harvard Medical School in 2006, he completed his internship in internal medicine at the University of California, San Francisco ("UCSF"), followed by a residency in neurology at the same hospital in 2010. (Ex. K, p. 1.) Dr. Gelfand was chief resident from 2009 to 2010 and held a fellowship in multiple sclerosis and neuroimmunology from 2010 to 2012 at UCSF. (*Id.*) He is licensed by the state of California, and board certified in neurology by the American Board of Neurology and Psychiatry. (*Id.*) Dr. Gelfand currently serves as an associate professor of clinical neurology at UCSF, previously he was an assistant professor of clinical neurology and a HS clinical instructor. (*Id.* at 2.) Dr. Gelfand has published 49 peer reviewed articles, 27 review articles, and 6 books and book chapters on various neurological diseases. (*Id.* at 13-19.)

1. Diagnosis

Dr. Gelfand disputed that SFN explains petitioner's symptoms. Although he acknowledged that petitioner's skin biopsy on her leg showed evidence of SFN, he contended that there was no evidence of a concomitant large fiber polyneuropathy. (Ex. J, p. 8.) He opined that petitioner's medical records fail to establish that her acute upper extremity symptoms can be attributed to SFN. (*Id.*) He noted that petitioner's treating physicians raised SFN as a diagnosis based on petitioner's left calf biopsy results but stressed that no evidence supported the presence of other manifestations of SFN, such as autonomic dysfunction. (*Id.*)

Dr. Gelfand further emphasized that petitioner's left calf biopsy showed evidence of SFN, but not her left thigh, which suggests a length-dependent pattern of SFN in her legs. (Ex. J, pp. 8-9; Tr. 121.) However, he noted that petitioner's upper extremity symptoms are incompatible with a length-dependent pattern of SFN because they began in her proximal upper extremities, not her fingertips and hands. (Ex. J, p. 9; Tr. 122.) He acknowledged that small fiber neuropathy may follow a non-length dependent pattern where symptoms manifest primary in the arms, face, or trunk. (Ex. J, p. 9.) However, he emphasized that this is rare, and it is also rare for a patient to have a more typical pattern in the legs and an atypical pattern in the arms. (*Id.*) He stressed that a non-length-dependent pattern would be "atypical, rare, and diagnostically speculative." (Ex. L, p. 1.)

Dr. Gelfand offered neuralgic amyotrophy ("NA"), also called brachial neuritis, as a potential explanation for petitioner's acute bilateral upper extremity pain. (Ex. J, p. 7.) He explained that NA is a brachial plexus neuropathy that involves large fiber peripheral nerves of the brachial plexus innervating the upper extremities and can cause severe bilateral shoulder pain. (*Id.*) Dr. Gelfand opined that petitioner's symptoms may be better attributed to NA given that her bilateral shoulder pain was acute, severe, and out of proportion to any possible weakness. (*Id.*) He distinguished NA from SFN, noting that NA is a "different diagnostic entity than autoimmune SFN, as it involves large and small fiber nerves in the brachial plexus." (Ex. L, p. 1.) Based on the evidence, however, Dr. Gelfand conceded that he could not determine that petitioner's upper extremity condition was more likely than not caused by NA. (Tr. 123.) He testified that he merely wanted to "credit" NA as part of a differential diagnosis for petitioner. (*Id.*) Dr. Gelfand further noted that "the evidence is insufficient to support a vaccine-induced cause of NA, if that is in fact what the diagnosis was." (Ex. L, p. 1.)

Dr. Gelfand acknowledged that petitioner had SFN in her distal lower extremities. (Tr. 132.) Although he conceded that SFN was an "important diagnostic consideration" for petitioner, he did not offer a final diagnosis for petitioner's condition. (*Id.* at 126-27.) He noted that it was difficult to establish a diagnosis "for the etiology of [petitioner's] acute pain syndrome to a more-likely-than-not standard." (*Id.* at 124.) Thus, Dr. Gelfand concluded that petitioner's upper extremity symptoms were not attributable to SFN.

2. Causation

a. Molecular Mimicry

In response to Dr. Tornatore's molecular mimicry theory, Dr. Gelfand noted that Dr. Tornatore did not offer evidence demonstrating how getting another flu vaccination would create an immune response leading to SFN. (Ex. J, p. 10.) Dr. Gelfand also stated that Dr. Tornatore failed to provide sequences in the flu or pneumococcal vaccines that could mimic antigens in small fiber nerves that would demonstrate sufficient homology for molecular mimicry. (*Id.*) He contended that Dr. Tornatore's reports "only offer general statements about antigens that may be in the vaccine or its adjuvants that could cause an inflammatory response generally." (*Id.*)

Further, Dr. Gelfand explained that SFN has several potential causes and associations, including diabetes/prediabetes, endocrine disease (i.e., thyroid abnormalities), vitamin deficiencies (i.e., B12 deficiency), toxic exposure (i.e., alcohol), chronic infection (i.e., HIV), autoimmune disease, paraproteinemia, and inherited or genetic factors. (Ex. J, p. 9.) He also noted that petitioner's history of RA and propensity to autoimmune disease are potential causes of SFN. (*Id.* (citing Astrid J. Terkelsen et al., *The Diagnostic Challenge of Small Fibre Neuropathy: Clinical Presentations, Evaluations, and Causes*, 16 LANCET NEUROLOGY 934, 939 (2016) (Ex. J, Tab 2)); *see also* Tr. 118-20 (stating that individuals with autoimmune disease can be susceptible to other autoimmune diseases, and that SFN can be caused by an autoimmune process).) Additionally, in 30-50% of SFN cases, the underlying cause is unknown. (Terkelsen et al., *supra*, at Ex. J, Tab 2, p. 939.) Further, Dr. Gelfand maintained that neither the flu vaccine nor the pneumococcal vaccine is a recognized cause of SFN. (Tr. 115-16.) He stressed that Dr. Tornatore did not provide medical literature supporting either the flu or pneumococcal vaccine as a cause of SFN, as SFN is a "distinct pathophysiological entity from large fiber neuropathy." (Ex. J, p. 10; Tr. 128.)

Dr. Gelfand also criticized the relevance of the literature offered by Dr. Tornatore. He asserted that none of the literature associated the flu or pneumococcal vaccines with SFN. (Ex. J, pp. 10-11.) Rather, Pollard & Selby (s*upra*, at Ex. 39) involved a large fiber demyelinating neuropathy following the tetanus toxoid; Shaw et al. (s*upra*, at Ex. 45) involved cases of SFN temporally associated with rabies, Lyme disease, and live varicella vaccines; Blitshteyn (s*upra*, at Ex. 42) involved POTS following the HPV vaccine; and Koike & Sobue (s*upra*, at Ex. 44) involved autoimmune autonomic ganglionopathy. (Ex. J, pp. 10-11.) Thus, Dr. Gelfand concluded that Dr. Tornatore provided no evidence to support a causal association between SFN and the flu or pneumococcal vaccines.

With respect to the timing involved in molecular mimicry, Dr. Gelfand opined that the appropriate timeframe for onset of an autoimmune condition following a triggering event would be "over days to a few weeks." (Tr. 133.) He further contended that Dr. Tornatore did not "explain how to reconcile the quite severe and acute onset of

[petitioner's] pain within hours to a day after vaccination and the several days of lag time he discusses as a possible medical theory for the immune response to develop even with prior influenza vaccination." (Ex. J, p. 10.) He elaborated that the onset of petitioner's "clinical syndrome, which came on profoundly within hours to 1 day of vaccination, would be too fast to support a vaccine-induced cause beyond a reasonable degree of medical certainty." (Ex. L, p. 2.)

b. IFN- γ Theory

During the entitlement hearing, Dr. Gelfand briefly addressed Dr. Tornatore's IFN- γ theory. He testified that there was no recognized risk of vaccines causing severe, burning neuropathic pain within mere hours of administration. (Tr. 124.) He further stated that IFN- γ would be routinely expressed as part of many systemic infections and vaccinations. (*Id.* at 137-38.) He explained that acute, burning neuropathic pain is not a routine or expected adverse reaction that is seen with "sort of a standard vaccination" like a flu or pneumococcal vaccination. (*Id.*) Thus, Dr. Gelfand contested the reliability of Dr. Tornatore's IFN- γ theory.

V. Discussion

a. *Althen* prong one[12]

i. Dr. Tornatore's theory includes two necessary components

Dr. Tornatore filed five separate expert reports in this case and also testified during the entitlement hearing. Although he has consistently opined that petitioner suffered vaccine-caused SFN, his theory of causation has evolved. In his initial reports, he asserted that molecular mimicry between vaccine components and peripheral nerve myelin tissue can result in inflammatory polyneuropathies, either by direct homology or by T cell degeneracy. (Ex. 36, p. 9; ECF No. 89, pp. 2-3.) He suggested this could be applied in the context of petitioner's upper extremity symptoms occurring mere hours after vaccination. (*Id.*) However, after being challenged on that theory with respect to timing, Dr. Tornatore stressed that IFN- *γ*, which can be produced by T cells within hours of infection, is "a well-known modulator of pain." (Ex. 54, p. 2.) He theorized that "it is biologically plausible that the administration of a vaccine could result in the systemic dissemination of viral antigens that could easily cross into the dorsal root ganglia given the lack of a blood brain barrier, stimulate resident lymphocytes of the dorsal root ganglia that then produce IFN- [*γ*] resulting in the sensation of pain in a

[12] There is some suggestion from Dr. Tornatore's reports that he could alternatively opine that petitioner's vaccinations significantly aggravated an indolent SFN. (Ex. 36, pp. 7-8.) However, there is no evidence to suggest that petitioner actually was suffering such a condition prior to vaccination. And, in any event, Dr. Tornatore indicated that his theory would still be the same. (Tr. 97-98.) In her post-hearing briefs, petitioner references that aspect of Dr. Tornatore's opinion that posited the possibility that indolent SFN may have been aggravated; however, she couched her claim specifically as meeting the *Althen* test and did not advocate for any separate analysis of significant aggravation under the *Loving* test. Accordingly, it is not necessary to do a separate *Loving* analysis.

dermatomal distribution, *e.g.* the length of the arm, as was the case with Ms. McGill."[13] (*Id*. at 3.) Nonetheless he also continued to maintain his opinion with respect to molecular mimicry. (*Id*. at 4-5.)

During the hearing I asked Dr. Tornatore to clarify the relationship between these two theories. Specifically, I asked him "do these two theories work independently as alternatives or do you need both to explain petitioner's condition?" (Tr. 223.) He responded "[t]hey do *not* work independently." (*Id*. (emphasis added).) He testified that:

> you can have those soluble factors, chemokines, cytokines, gamma interferon produced early on in the immune response that causes one set of symptoms, and then as the immune response continues to develop, you see the full impact of that immune response . . . So somebody may develop, in this case, symptoms, the neuropathic pain, as a result of the gamma interferon, but that gamma interferon is the fingerprint, if you will. It's the canary in the coal mine that there is an autoimmune process via molecular mimicry which then may then smolder and then cause that neuronal demise to happen. So no, they are – I think they're not two separate issues. They are all one mechanism.

(Tr. 223-24.)

ii. Dr. Tornatore's explanation is not sound and reliable

Under *Althen* prong one, petitioner must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (citations omitted). Such a theory must only be "legally probable, not medically or scientifically certain." *Knudsen*, 35 F.3d at 549. Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu*, 569 F.3d at 1378-79 (citing *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1325-26 (Fed. Cir. 2006)). However, "[a] petitioner must provide a 'reputable medical or scientific explanation' for [her] theory. While it does not require medical or scientific certainty, it must still be 'sound and reliable.'"[14] *Boatmon*, 941 F.3d at 1359 (quoting *Knudsen*, 35 F.3d at 548-49).

[13] During the hearing, Dr. Whitton broke Dr. Tornatore's INF- γ theory down into two further hypotheses based on variations in the way Dr. Tornatore expressed it. In his report at Exhibit 54, Dr. Tornatore proposed that systemic dissemination of vaccine antigens could cross into the DRG. (Tr. 184; Ex. 54, p. 3.) In his report at Exhibit 60, Dr. Tornatore indicated that antigen bearing cells from draining lymphatics could reach the DRG. (Tr. 186; Ex. 60, pp. 5-7.) In either event, INF- γ within the DRG is activated to cause neuropathic pain. (Tr. 187.)

[14] In their post-hearing briefs, the parties devote significant attention to their differing views on the question of whether petitioner's burden under *Althen* prong one is limited to establishing a "plausible" or "biologically plausible" theory of causation. (ECF No. 148, pp. 11-13; ECF No. 151, pp. 19, 23; ECF No. 152, pp. 4-5.) The parties appear to use the terms "plausible" and "biologically plausible" interchangeably, which has the effect of placing two Federal Circuit decisions, *Andreu* and *Boatmon*, in

On this particular record, Dr. Tornatore stands alone in seeking to specifically hypothesize that either the flu vaccine or the pneumococcal vaccine can cause SFN. There is, however, at least *some* circumstantial support for this suggestion. As Dr. Tornatore indicated, at least some cases of SFN are considered to be autoimmune. (Oaklander, *supra*, at Ex. 38, p. 1.) Additionally, Dr. Tornatore has submitted literature identifying some other vaccines not at issue in this case as suspected causes of neurologic adverse events, including small fiber neuropathy. (*See* Souayah et al., *supra,* at *Ex*. 40 (case reports of SFN following rabies, varicella, or Lyme disease vaccinations); Shaw et al., *supra,* at Ex. 45 (neurologic adverse events following Hepatitis B vaccine). And, finally, some literature contends that SFN may, in at least some cases, be compared in its presentation to Guillain-Barre Syndrome which has, in turn, been linked to the certain formulations of the flu vaccine. (Oaklander, *supra*, at Ex. 38 p. 6; Schonberger et al., *supra,* at Ex. 46.) With regard to the IFN- *γ* /DRG aspect of Dr. Tornatore's theory, it is also the case that ganglionopathy has been identified as one mechanism by which peripheral sensory neuropathies may manifest. (Oaklander, *supra*, at Ex. 38, p. 2.) Additionally, Dr. Tornatore has support for the proposition, as a general matter, that IFN- *γ* may mediate chronic neuropathic pain. (Mayumi Sonekatsu et al., *Interferon-gamma Potentiates NMDA Receptor Signaling in Spinal Dorsal Horn Neurons via Microglia-neuron Interaction,* 12 MOLECULAR PAIN 1 (2016) (Ex. 55).)

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tension. In *Andreu*, the Federal Circuit accepted a theory as meeting *Althen* prong one based on the lower court's determination that the theory was "biologically plausible." 569 F.3d at 1375. In *Boatmon*, the Federal Circuit rejected a theory that was "at best 'plausible'," as in merely "possible," as not meeting the preponderant evidence standard. 941 F.3d at 1360. Whereas the "biologically plausible" theory in *Andreu* was consistent with a preponderant showing, the Federal Circuit concluded that the *Boatmon* special master had applied a "reasonable" burden of proof that fell below preponderant evidence. *Id*. at 1359. However, both Federal Circuit decisions cite the same standard with respect to petitioner's burden of proof. Both explain that a petitioner's burden of proof is to present a theory that is supported by "reputable medical or scientific explanation." *Andreu*, 569 at 1379 (quoting *Althen*, 418 F.3d at 1278); *Boatmon*, 941 F.3d at 1359 (quoting *Moberly*, 592 F.3d at 1322).) And both decisions further explicitly cite the prior *Knudsen* decision for the proposition that this means that the underlying scientific explanation must be "sound and reliable." *Andreu*, 569 at 1379 (citing 35 F.3d at 548); *Boatmon*, 941 F.3d at 1359 (same). Nothing in *Andreu* implies that the "biologically plausible" theory presented in that case constituted anything less than preponderant evidence or that a theory that is not "sound and reliable" could be considered "biologically plausible." Nor, on the other hand, does *Boatmon* hold that any theory deemed "biologically plausible" is *per se* inadequate to meet the preponderant evidence standard. The Federal Circuit explained in *Knudsen* that "[c]ausation in fact under the Vaccine Act is thus based on the circumstances of the particular case, having no hard and fast *per se* scientific or medical rules." 35 F.3d at 548. Thus, it is not necessary to apply the specific "biologically plausible" terminology referenced in *Andreu*. Nor, however, should an expert's use of the term "biologically plausible" be taken as a fatal concession without regard to context. On the whole, prior Federal Circuit decisions have instead focused on contrasting petitioner's preponderant burden of proof against either the merely "possible" or "plausible," which is insufficient, and "scientific certainty," which is too burdensome. *E.g. Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1322 (Fed. Cir. 2010)(explaining at turns first that petitioners argued in favor of "something closer to proof of a 'plausible' or 'possible' causal link between the vaccine and the injury, which is not the statutory standard" and then that petitioners also argued that the term "cause-in-fact" implies scientific certainty "[b]ut this court has regularly used that term to describe the causal requirement for off-Table injuries and has made clear that the applicable level of proof is not certainty . . .")

Based on all of this, Dr. Tornatore contends that he is "not making a stretch" in opining that the flu vaccine can be the cause of SFN. (Tr. 76.) However, this is not ultimately persuasive. Considering the record as a whole, the above points at best set the stage for his theory to be *possible* without evidence supporting it as *probable*. In contrast to Dr. Tornatore's opinion, Dr. Gelfand and Dr. Whitton both contest that either the flu or pneumococcal vaccines are recognized causes of SFN and Dr. Gelfand further suggests the notion is speculative. (Tr. 115-16 (Gelfand); Tr. 57 (Whitton).) A close examination reveals for the reasons discussed in the sections that follow that there are too many unsupported gaps in Dr. Tornatore's opinion for it to be considered sound and reliable. 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 v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (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 rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. Appx. 999 (Fed. Cir. 2013).

iii. Dr. Tornatore's comparison of SFN to GBS is unpersuasive

While Dr. Tornatore is correct that there is some overlap between GBS and SFN, he is not persuasive in equating the causes of GBS to the causes of SFN. In contrast to GBS, SFN is generally considered to be caused by underlying systemic conditions, including autoimmune conditions, diabetes, and toxic causes. (Oaklander, *supra*, at Ex. 38, p. 5.) Only a subset of idiopathic SFN cases is suspected to result from tissue-specific dysimmunity in a manner that could potentially be compared to GBS. (*Id*. at 6.) However, even in that context, nothing on this record confirms the implicated tissue is necessarily the same. Whereas Dr. Tornatore specifically discuses myelin tissue as the likely target of autoimmune attack in the context of post-vaccination GBS (Tr. 55), the small fibers are largely not myelinated.[15] The small fibers consist of both unmyelinated C-fibers and "lightly" or "thinly" myelinated A-delta fibers. (Oaklander, *supra*, at Ex. 38, p. 1; Tr. 96-97.) In an ordinary clinical setting (including this case), the specific small fiber(s) at issue in SFN are not distinguished. (Tr. 96-97, 135-37.) Dr. Tornatore has not provided evidence establishing that SFN itself is a demyelinating condition.

At base, Dr. Tornatore effectively asserts that because GBS in some instances affects the small fibers, then whatever causes GBS can also cause SFN. (Tr. 78.) In his testimony, however, Dr. Tornatore appeared to acknowledge that SFN is associated with other autoimmune processes, including GBS, "for reason that are unclear." (Tr. 84-85.) Indeed, in his first report, Dr. Tornatore quotes the literature as explaining that:

[15] As noted above, Dr. Tornatore additionally asserts a role for an immune process affecting the DRG. In that regard, he notes that the DRG is also "very loosely" myelinated. (Tr. 96-97.) However, he confirmed the DRG aspect of his theory is cytokine driven rather than proposing molecular mimicry. (Tr. 92-94.)

> The best-known peripheral neuropathies are those affecting the large, myelinated motor and sensor fibers. These have well-established immunological causes and therapies. Far less is known about the somatic and autonomic "small fibers"; the unmyelinated C-fibers, thinly myelinated A-delas, and postganglionic sympathetics."

(Ex. 36, p. 8 (quoting Oaklander, *supra*, at Ex. 38, p. 1.) Moreover, Dr. Gelfand persuasively urges that even if GBS can sometimes include small fiber symptomology, it is still defined by its large fiber involvement, which is necessarily absent in SFN.[16] (Tr. 114-15, 135-37.) Thus, on the whole, Dr. Tornatore is unpersuasive on this record in suggesting that the cause(s) of GBS represent significant evidence concerning the causes of SFN.[17]

iv. Molecular mimicry is not supported on this record

Setting aside the flawed analogy to GBS, Dr. Tornatore has not otherwise offered evidence supporting homology between components of the vaccine(s) at issue in this case and the tissues affected by SFN despite specifically invoking molecular mimicry. Nor is there evidence of record to support cross-reaction or development of disease from a relevant mimic. Nor has Dr. Tornatore offered evidence more broadly implicating either the flu or pneumococcal vaccine(s) as causes of SFN. Considering the body of

[16] For a more thorough discussion of the many variants of GBS, *see Swaiss v. Sec'y of Health & Human Servs.*, No. 15-286V, 2019 WL 6520791, at *12-18 (Fed. Cl. Spec. Mstr. Nov. 4, 2019). In *Swaiss*, the special master concluded, based on evidence not included within this record, that petitioner had established that a form of immune-mediated SFN could be said to represent a "small fiber GBS variant." *Id.* at 18. Importantly, however, the *Swaiss* special master discussed "GBS" as an "umbrella term" for which the nosological limits remain unclear. *Id*. at 14. This uncertainty does not assist petitioner in meeting her burden. Rather, it underscores that the "S" in GBS stands for "syndrome." Considered as a group, the GBS variants are generally believed to have a multitude of both clinical presentations and causes; however, the degree to which a cause of one condition placed under that umbrella could be said to apply to all conditions under the umbrella is an open question. The association between the flu vaccine and GBS is largely understood to be based specifically on a large fiber demyelinating form of polyneuropathy. While the Schonberger study did not specifically define GBS, it did specifically explain that subject cases were screened to require objective evidence of muscle involvement. (Schonberger et al., *supra*, at Ex. 46, p. 3.) Thus, cases of SFN, any pure sensory variant, or the "small fiber GBS variant" accepted in *Swaiss,* necessarily would have been excluded.

[17] Another special master has also previously reached the same conclusion. *E.g. Fantini v. Sec'y of Health & Human Servs.,* No. 15-1332V, 2022 WL 1760730, *22 (Fed. Cl. Spec. Mstr. May 2, 2022) (explaining that "[t]he fact that reliable science establishes an association between GBS and the flu vaccine . . . does not inerrantly lead to the conclusion that SFNs can also be deemed to be similarly-associated, given the facial differences in the nature of these conditions . . ." and further concluding molecular mimicry was not supported.); *accord Mason v. Sec'y of Health & Human Servs*., No. 17-1383V, 2022 WL 600415, at *26 (similarly explaining in a different context that "I am unwilling to find that Petitioner has preponderantly established a causal relationship between the flu vaccine and CIDP merely because that theory has been accepted in the Program's past. Review of prior relevant cases suggests that more often than not, that determination has been based upon the faulty supposition that GBS and CIDP are two sides of the same coin."); *but see Jones v. Sec'y of Health & Human Servs.,* No. 15-1239V, 2018 WL 7139212, at *13 (Fed. Cl. Spec. Mstr. Dec. 21, 2018) (accepting that vaccines, including the flu vaccine, can cause SFN based on analogy to GBS).

literature he cites as a whole, it serves mainly to underscore the speculation involved in Dr. Tornatore's reliance on molecular mimicry. Dr. Tornatore has submitted literature that merely shows that various autoimmune targets have been suspected in various autoimmune injuries, none of which implicate the vaccines at issue in this case as causes of the SFN at issue in this case.[18] This leaves his application of molecular mimicry to this case wholly unsupported. Respondent's experts are persuasive in cautioning against unsupported inferential leaps in the context of molecular mimicry. In particular, Dr. Whitton explained that molecular mimicry cannot in itself be equated with disease (Tr. 157), that disease-causing molecular mimicry is much rarer than molecular mimicry itself (Tr. 204), that molecular mimicry involves a specific interaction that is not generalizable across differing contexts[19] (Tr. 165-66), that molecular mimicry is not the cause of all autoimmune disease (Tr. 204-05), and that autoimmune disease should not be assumed to require any external trigger at all (*id*.).

"Of course, petitioners are never required to establish mechanism – but they often attempt to do so, and therefore it is reasonable to evaluate their success in in their effort." *Howard v. Sec'y of Health & Human Servs*., No. 16-1592V, 2022 WL 4869354, at *24 (Fed Cl. Spec. Mstr. Aug. 31, 2022). There is no debate in this case that molecular mimicry is, in general, a genuine hypothesis to explain at least some autoimmune conditions. However, as has been observed in prior cases, "[t]hough molecular mimicry is a generally accepted scientific principle, mere invocation of the scientific term does not carry a petitioner's burden in a Program case." *Deshler v. Sec'y of Health & Human Servs*., No. 16-1070V, 2020 WL 4593162, at *20 (Fed. Cl. Spec. Mstr. July 1, 2020) (citing *Forrest v. Sec'y of Health & Human Servs.*, No. 14-1046V, 2019 WL 925495, at *3 (Fed. Cl. Spec. Mstr. Jan. 18, 2019)). Here, Dr. Tornatore's inability to substantiate the proposed molecular mimicry is significant because he has not otherwise been persuasive in suggesting that his theory is supported circumstantially. Moreover, because Dr. Tornatore confirms that his INF- *γ* ganglionopathy theory does not operate independently of this molecular mimicry theory, his references to INF- *γ* do not cure his inability to persuasively support the molecular mimicry aspect of this theory.

Dr. Tornatore does suggest that the concept of T cell degeneracy can explain the presence of molecular mimicry without the need to identify a specific homology.

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[18] When prompted during the hearing to discuss the salient points of his cited literature with respect to molecular mimicry, Dr. Tornatore highlighted the following: Exhibit 47 is a study by Oldstone that examined molecular mimicry in rabbits between polymerase of hepatitis B and myelin-related protein (Tr. 54-55); Exhibit 39 is a case report by Pollard and Selby examining post-tetanus GBS (Tr. 55-56);Exhibit 46 is a study by Schonberger, et al., finding epidemiologic evidence of GBS following vaccination with a swine flu vaccination. (Tr. 55); and Exhibit 40 is a paper by Souayah, et al., presenting five case reports of SFN following vaccination with either rabies, varicella, or Lyme disease vaccinations (Tr. 56). Dr. Tornatore himself characterized this as reflecting evidence relating to a "hodgepodge" of vaccines. (Tr. 56.)

[19] That is, Dr. Whitton explained that in the case of GBS one specific bacterium (Campylobacter jejuni) has been shown to have caused GBS via molecular mimicry. Otherwise, the epidemiological evidence supporting the swine flu vaccine as a cause of GBS does not in itself demonstrate molecular mimicry. To suggest that all cases of GBS are explained by molecular mimicry is an unsupported "leap." (Tr. 166.)

Specifically, he opines that "[r]eceptors on B and T cells that were once thought to have a high level of specificity for individual foreign antigens are now known to recognize peptide sequences that share no homology. . . Hence, microbiological antigen from a bacteria or virus which bears no similarity to nervous system antigen could active a B or T cells receptor which would then cause the B or T cell to mount an autoimmune response in the nervous system." (Ex. 36, p. 9 (citing Don Mason, *A Very High Level of Crossreactivity is an Essential Feature of the T-cell Receptor*, 19 IMMUNOLOGY TODAY 395 (1998) (Ex. 41).) According to Dr. Tornatore, this concept, referred to in the cited literature as resulting in "unfocused cross reactivity," allows him to rely on evidence relating to a "hodgepodge" of vaccines so long as they are ultimately implicated in the same outcome. (Tr. 56.)

If accepted, the degeneracy concept *might* suggest that it is less important to demonstrate a specific homology in those cases where some other evidence suggests that a vaccine can cause a particular autoimmune injury. However, even if I accepted both Dr. Tornatore's reliance on T cell degeneracy and his reliance on analogy to GBS, I would still conclude that this would be too speculative on this record to provide preponderant support for petitioner's theory. In the absence of some evidence specific to SFN, accepting T cell degeneracy as the primary support for Dr. Tornatore's opinion would mean effectively accepting that anything is possible – that any vaccine can be said to cause any autoimmune condition without any definable limit.[20]

v. IFN- *γ* is not supported as a post-vaccination initiator of SFN

Turning to the other aspect of Dr. Tornatore's theory, even though Dr. Tornatore has support in identifying IFN- *γ* as a possible *mediator* of neuropathic pain, nothing in the available literature suggests, as Dr. Tornatore urges, that it would be an *initiator* of SFN symptoms. Rather, the primary study Dr. Tornatore cites stresses that the effects of IFN- *γ* on the dorsal horn are unclear. That study discusses IFN- *γ* as an enhancer rather than initiator of neuropathic pain, suggesting only that it may be a causative agent with respect to the chronicity of neuropathic pain. In fact, the authors discuss the production of IFN- *γ* in the dorsal horn as a *consequence* of otherwise manifesting nerve injury. (Sonekatsu et al, *supra*, at Ex. 55, p. 8 (Fig. 8).) Nothing in Dr. Tornatore's supporting literature evidences the idea that IFN- *γ* would function as Dr. Tornatore

[20] Regardless, Dr. Tornatore's application of the degeneracy concept does not appear to be sound for several other reasons. First, the article he relies on for this concept presents mathematical analyses only and disclaims the ability to determine whether the type of "unfocused" cross reactivity proposed actually predominates in the body. Second, the author further hypothesizes that this "unfocused" T cell cross reactivity, if it is occurring, would likely reduce the number of autoreactive T cells resulting from a foreign antigen. (Mason, *supra*, at Ex. 41, p. 9.) This appears to propose that cross reactivity may be more common, but far less significant, than previously thought. That idea is more supportive of Dr. Whitton's testimony that cross reactive autoimmune responses do not invariably lead to disease (Tr. 156-57, 188-89) than it is supportive of Dr. Tornatore's theory. Dr. Whitton explained that while molecular mimicry rarely causes disease, molecular mimicry is not in itself rare. (Tr. 204.) And, in any event, even while discounting the need for specific homology, this paper still stands for the proposition that autoimmune disease results from cross reactivity. Yet there is still no persuasive evidence that such cross reactivity is occurring in the present context. Therefore, Dr. Tornatore's theory of T cell degeneracy would still be speculative without more

suggests in the overall context of his theory, constituting the preceding "fingerprint" of a later developing vaccine-caused autoimmune nerve damage. In contrast, Dr. Gefland testified that while IFN- *γ* is part of an inflammatory response, including in response to vaccination, he disagrees that there is evidence to support IFN- *γ* as a cause of acute, burning neuropathic pain. (Tr. 138.) Dr. Whitton likewise suggests that Dr. Tornatore's specific explanation of how this would happen has never been demonstrated to actually occur. (Tr. 191.) He further stresses that INF- *γ* is commonly produced by the body in response to vaccination and infection, and yet neuropathic pain is not seen as a typical response. (Tr. 194-95.)

Dr. Whitton explains that rather than continuously producing INF- *γ*, T cells produce cytokines only upon contact with an antigen. Thus, the degree of cytokine response produced by a vaccination is more limited compared to a replicating viral infection. (Ex. N, p. 5.) For example, respondent filed a study by Cohen, et al., that demonstrated that INF- *γ* was elevated following administration of the Smallpox vaccine.[21] (Jeffrey I. Cohen et al., *Kinetics of Serum Cytokines after Primary or Repeat Vaccination with the Smallpox Vaccine,* 201 THE J. OF INFECTIOUS DISEASES 1183 (2010) (Ex. N, Tab 2, p. 1).) However, subjects experienced only transitory symptoms including fatigue, lymphadenopathy, myalgia, headache, pruritis, loss of appetite, chills, and fever. (*Id*. at p. 2 (Fig. 1), 3 (Tab. 1).) According to Dr. Whitton, if Dr. Tornatore's DRG/ INF- *γ* theory were correct, these study subjects should have experienced neuropathic pain. (Tr. 192.) Also notable, while INF- *γ* was associated with fatigue, lymphadenopathy, and myalgia, it was not associated with pruritis, which is the only symptom observed in the study that would appear to implicate the sensory small fibers. (Cohen et al., *supra,* at Ex. N, Tab 2, p. 6 (Tab. 3).) A single study of 42 subjects standing alone is inadequate to refute Dr. Tornatore's theory; however, it does strongly suggest Dr. Tornatore's theory is less likely to be accurate in a real-world context, reducing its overall reliability.

vi. Conclusion as to *Althen* prong one

For all the reasons discussed above, petitioner has failed to preponderantly establish that either the flu vaccine or pneumococcal vaccine can cause SFN. Additionally, because Dr. Tornatore has confirmed that the two parts of this theory – molecular mimicry and IFN- *γ* – do not work independently, and because I have determined that neither aspect of the theory is sufficiently supported, this analysis prevents petitioner from meeting her burden under *Althen* prong one regardless of whether one examines her condition as relating to her upper extremities alone, her lower extremities alone, or both together.[22]

[21] However, it is stressed that "[t]he smallpox vaccine is associated with more serious adverse events than any other live attenuated vaccine in use today," suggesting caution would be needed in generalizing these findings to any other vaccination. (Cohen et al., *supra*, at Ex. N, Tab 2, p. 1.)

[22] Prior cases present a mixed record with respect to whether the vaccines at issue in this case can cause SFN. *Compare Fantini,* 2022 WL 1760730, *22 (petitioner did not satisfy *Althen* prong one with respect to SFN allegedly caused by the flu vaccine.); *Todd v. Sec'y of Health & Human Servs.,* No. 15-860V, 2020 WL 727973, at *22 (Fed. Cl. Jan. 8, 2020) (finding that petitioner did not meet *Althen* prong one with

b. *Althen* prong two

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1280) (stating that "medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'"). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. *See* Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (stating that "there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). Ultimately, petitioner may support her claim either through her medical records or by expert opinion. § 300aa-13(a)(1).

i. Treating physician opinions do not preponderantly support petitioner's claim

In this case, it is clear that petitioner's treating physicians took seriously the *suspicion* of vaccine causation. However, careful review of the entire body of medical records shows that the treating physicians were equivocal in their opinions and never actually *concluded* that petitioner suffered vaccine-caused SFN. Ultimately, when considering the records as a whole, petitioner's treating physicians did not express a view that petitioner's SFN symptoms were more likely to be vaccine-caused than they were to be sequela of her preexisting autoimmune conditions.

When petitioner first reported her upper extremity symptoms to Dr. Coletti at a same-day clinic, she described them as similar to her preexisting polymyositis. (Ex. 4, p. 2.) Dr. Coletti confirmed the lack of any local injection site reaction and concluded that vaccine-causation of her symptoms was "unlikely." (*Id*.) The next day, an emergency

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respect to demonstrating a theory that the flu vaccine can cause SFN) *with E.M.,* 2021 WL 3477837, at *36 (finding the flu vaccine can cause SFN where petitioner's expert demonstrated homology between components of the flu vaccine and alpha 3 AChR, which is associated with SFN, and respondent countered only by citing a lack of epidemiologic support); *Jones.,* 2018 WL 7139212, at *13 (accepting that vaccines, including the flu vaccine, can cause SFN based on analogy to GBS); *Doe v. Sec'y of Health & Human Servs.,* 2007 WL 3120297 (Fed. Cl. Spec. Mstr. Oct. 18, 2007) (finding the flu vaccine caused petitioner's SFN). Of those cases finding the flu vaccine can cause SFN, I note that the prior *E.M.* case in particular had a substantially more detailed showing of molecular mimicry to support petitioner's theory under *Althen* prong one and that showing was *not* predicated on homology to myelin proteins.

department physician, Dr. Tarjan recorded petitioner's history of a post-vaccination onset, but concluded that her symptoms may be related to either her polymyositis or her rheumatoid arthritis. (*Id.* at 7.) Another emergency department physician, Dr. Glickman, was the first to include a reaction to immunization in a differential diagnosis set against a myositis flare. (*Id*. at 13.) However, the discharge summary confirms the physicians were "unsure" of the cause of her symptoms, maintaining they may be related to her polymyositis or rheumatoid arthritis. (*Id*. at 17.) Subsequently, petitioner saw a neuromuscular specialist, Dr. Jovanich, who assessed an arthus reaction to vaccination. (Ex. 4, p. 30.) However, when petitioner then followed up with a rheumatologist, Dr. Ritt, he felt that an arthus reaction was "unlikely" and instead suggested a more likely explanation would be myofascial pain syndrome triggered by vaccination. (*Id*. at 63.) Neither physician indicated any suspicion of SFN. By November 25, 2014, petitioner saw a second rheumatologist, Dr. Wells, now complaining of symptoms in both her upper and lower extremities. Dr. Wells was the first to suggest petitioner's symptoms may be due to SFN; however, she was "unable to determine if the onset of pain is related to the injection or not." (Ex. 7, p. 6.)

Thereafter, petitioner began consulting with neurologist Justin Mhoon. Of all of petitioner's treating physicians, Dr. Mhoon's causal assessment is the most thoroughly documented and the most consistent with petitioner's claim; however, it is also highly equivocal. When petitioner first presented to Dr. Mhoon on December 2, 2014, he provided a differential diagnosis including both sensory neuritis due to a vaccine reaction and an unspecified form of autoimmune demyelinating disease. (Ex. 18, p. 70.) Later, however, after petitioner had undergone additional testing, including EMG and NCS that ruled out large fiber peripheral neuropathy, Dr. Mhoon changed his differential to either neuritis or SFN, but noted that "if this was a vaccine related neuritis it should improve on its own over time." (*Id*. at 133.) Once Dr. Mhoon confirmed SFN based on petitioner's skin biopsy, he indicated that SFN is "[o]ften associated with rheumatologic conditions such as yours" and further indicated when pressed by petitioner's follow up questioning that he "cannot prove a direct causation of the vaccines." (Ex. 13, pp. 3-4.) In subsequent records, Dr. Mhoon continued to equivocate as to whether petitioner's condition was explained as sequela of her pre-existing rheumatoid arthritis or as a separate vaccine-caused neuritis.[23] (Ex. 6, p. 60; Ex. 63, p. 673.)

In October of 2016, petitioner additionally saw Dr. Eckstein at the Duke Neuroscience Center, who provided an opinion comparable to that of Dr. Mhoon. (Ex.

[23] In her post-hearing brief, petitioner stresses that Dr. Mhoon recommended that petitioner avoid flu and pneumococcal vaccinations in the future. (ECF No. 148, pp. 33-34 (citing Ex. 6, p. 60).) Petitioner argues the decision to withhold vaccination can be probative evidence. (*Id*. (quoting *Andreu*, 418 F.3d at 1376).) While this general proposition is true, it is important to note that the *Andreu* court specifically considered the fact that the referenced treating physician testified and, consistent with his determination to withhold vaccination, never disclaimed having a causal opinion favoring vaccine causation. *Andreu*, 418 F.3d at n. 3. In contrast, Dr. Mhoon's records in this case include an explicit statement that he cannot reach a conclusion that petitioner's vaccines caused her condition. (Ex. 13, pp. 3-4.) Dr. Mhoon's additional indication that it may be "reasonable" for petitioner to avoid vaccination does not contradict that explicit causal opinion nor does it in any way help to resolve the equivocation evident throughout Dr. Mhoon's records.

24, p. 4358.) When pressed by petitioner with regard to whether her SFN was vaccine-caused, he stressed that petitioner's preexisting autoimmune disease predisposed her to SFN, but added that "I *suppose it is possible* that the vaccinations could have exacerbated her underlying autoimmunity . . . though this would be difficult to prove definitively." (*Id*. (emphasis added).)

These treating physician opinions do not preponderantly support petitioner's claim. *Accord Stapleford v. Sec'y of Health and Human Servs.,* No. 03-234V, 2009 WL 1456441, at *17 n.24 (Fed. Cl. Spec. Mstr. May 1, 2009) (explaining that medical records may include notations where a physician "may well be indicating a *question* in the physician's mind whether there is a causal relationship, or a *suspicion* that there might be a causal relationship. However, that is quite different from an indication that such physician has reached a *conclusion* concerning a causal relationship") (emphasis in original), *aff'd*, 89 Fed. Cl. 456 (Fed. Cl. 2009). When viewing the record as a whole, the treating physician's opinions supporting vaccine-causation are tentative at best, and largely fail to move beyond suspicion. Moreover, apart from recognizing temporality, they are not consistent in either diagnosis or rationale. However, "[a] treating physician's recognition of a temporal relationship does not advance the analysis of causation." *Isaac v. Sec'y of Health and Human Servs*., No. 08-601V, 2012 WL 3609993, at *26 (Fed. Cl. Spec. Mstr. July 30, 2012).

ii. Dr. Tornatore is unpersuasive in opining petitioner's vaccinations are a necessary trigger of her SFN

Because I have separately determined that the onset of upper extremity symptoms in this case is too fast relative to vaccination to allow for a causal inference (*see Althen* three, below), Dr. Tornatore's specific reliance on the abruptness and short latency of the upper extremity symptoms to arrive at his causal assessment renders his opinion less persuasive. Importantly, but for Dr. Tornatore's stressing of an abrupt onset, both parties' experts otherwise agree that petitioner's SFN is consistent with her preexisting autoimmune conditions,[24] a point that is also repeatedly raised by the treating physicians. In fact, Dr. Tornatore's seeming hesitation in completely ruling out an indolent and asymptomatic SFN predating the vaccination, despite himself placing symptom onset post-vaccination, underlines this very point. (Ex. 36, pp. 7-8; Tr. 97-98.)

It should also be noted that the Cohen, et al., study presented by Dr. Whitton introduces a degree of tension between Dr. Tornatore's theory and petitioner's own clinical history. Dr. Tornatore relies primarily on the INF-*γ* aspect of this theory to explain the minimal latency between vaccination and onset of SFN. However, while experimental studies filed in this case do show that memory T cells produce INF-*γ* more

[24] Specifically, *see* Tr. 114 (Dr. Gelfand explaining SFN is associated with rheumatologic and other autoimmune diseases); Tr. 97-98 (Dr. Tornatore agreeing "we totally recognize that primary autoimmune diseases whether Sjorgren's or rheumatoid or lupus, can have small fiber sensory neuropathy as a part of that general autoimmunity that the patient has . . ."); Tr. 70-71 (Dr. Tornatore acknowledging SFN can be autoimmune in nature but discussing likelihood of vaccine being a logical trigger); Tr. 82-83 (Dr. Tornatore disagreeing with Dr. Mhoon that SFN was sequela to RA due to abrupt onset); Tr. 84-85 (acknowledging people with RA can develop SFN).

quickly than naïve T cells, in Cohen, et al.'s human study, only first-time vaccine recipients mounted a significant INF- *γ* response *at all*. (Cohen et al., *supra,* at Ex. N, Tab 2, p. 5 (Fig. 3(A)).) Dr. Whitton explains that this is because the "more biologically beneficial" memory T cell response generally produces fewer cytokines before the antigen at issue is neutralized. (Ex. N, pp. 3-5.) Thus, the proposed INF- *γ* response Dr. Tornatore relies upon (perhaps counterintuitively) is less likely in the scenario of a repeat exposure and recall response, which is the scenario actually present in this case.

In any event, Dr. Tornatore is not persuasive in suggesting that an abrupt onset in itself distinguishes petitioner's SFN as more likely to have been triggered by vaccination. This also helps to explain why petitioner's treating physicians were unable eliminate petitioner's preexisting autoimmune conditions from their differential diagnosis despite the abrupt onset. In asserting that onset of SFN within hours of vaccination is medically reasonable, a significant part of Dr. Tornatore's rationale is that the small fibers are exquisitely sensitive and that the symptoms of a SFN would be the very first to be felt or recognized by a patient even in a broader neuropathy. (Tr. 51-52.) However, this rationale would apply equally regardless of the underlying cause of petitioner's SFN. Indeed, contrary to Dr. Tornatore's stated view, the literature he provides suggests that SFN related to preexisting autoimmunity may be due to sensory ganglionopathies, which can be distinguished from distal sensory axonopathies both by patchy and often proximal symptoms and by the more rapid onset. (Oaklander, *supra*, at Ex. 38, p. 2; Jinny Tavee & Lan Zhou, *Small Fiber Neuropathy: A burning Problem*, 76 CLEVELAND CLINIC J. OF MEDICINE 298 (2009) (Ex. A, Tab 3, p. 4).) For example, Sjogren syndrome in particular is known to result in sudden unexplained neuropathic pain from ganglionitis. (Oaklander, *supra*, at Ex. 38, p. 2.) Notably, consistent with this description, the experts' disagreement as to petitioner's correct diagnosis in this case has been driven in significant part by her rapid and atypical (*i.e*. not length dependent) onset of symptoms. In contrast, Dr. Whitton additionally stresses that petitioner's onset of SFN was not accompanied by any of the clinical signs of a robust cytokine response as would be likely if Dr. Tornatore's theory was accurate. (Tr. 56.)

iii. Petitioner cannot meet *Althen* prong two based on her lower extremity symptoms alone

Both petitioner and Dr. Tornatore contend first and foremost that her upper and lower extremity symptoms should together be viewed as constituting SFN and should not be separated. (Tr. 89-90; ECF No. 152, pp. 1-1.) Additionally, following the hearing, I ultimately conclude that Dr. Gelfand's testimony regarding the nature of petitioner's upper extremity symptoms is too equivocal to cast substantial doubt on the SFN diagnosis favored by petitioner as a unifying explanation of the upper and lower extremity symptoms. (Tr. 123, 126-27, 132.) If one were to look exclusively at the lower extremity symptoms, then the later timing of onset would potentially be consistent with an inference of vaccine causation. However, this would detract from Dr. Tornatore's stated rationale that a particularly "striking" and "abrupt" onset of upper extremity symptoms helps inform his overall assessment of a logical sequence of cause-and-effect implicating petitioner's vaccinations despite otherwise having conditions

predisposing her to SFN. (Ex. 60, pp.1- 2.) In any event, without more, a medically acceptable onset is not persuasive as a means to assert vaccine-causation. *Devonshire v. Sec'y of Health and Human Servs.,* No. 99-031V, 2006 WL 2970418, at *19 (Fed. Cl. Spec. Mstr. Sept. 28, 2006) (medical expert's "*post hoc ergo proptor hoc* reasoning…has been consistently rejected by the Court and is 'regarded as neither good logic nor good law'") (quoting *Fricano v. U.S.,* 22 Cl. Ct. 796, 800 (1991) (emphasis in original)). The Federal Circuit has explained that "neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149). Thus, even limiting the question to petitioner's lower extremity symptoms, her preexisting autoimmune conditions would still constitute a likely explanation for petitioner's SFN based on both the treating physicians' observations and the expert opinions presented in the case.

iv. Conclusion as to *Althen* prong two

For all these reasons, petitioner has not preponderantly established *Althen* prong two. This analysis prevents petitioner from meeting her burden under *Althen* prong two regardless of whether one examines her condition as relating to her upper extremities alone, her lower extremities alone, or both together.

c. *Althen* prong three

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

In this case discussion of two periods of onset is warranted. Petitioner began experiencing upper extremity symptoms of SFN within hours of her vaccination. Specifically, the subject vaccines were administered at a medical appointment that began at about 3:30pm (Ex. 15, p. 93) and she reported onset of symptoms occurring either the "same day" (Ex. 4, p. 7) or as waking her in the middle of the night (Ex. 4, p. 2; Tr. 25). In her VAERS report she placed onset at 12:30AM. (Ex. 17, p. 2.) Thus, for purposes of discussing causation, the experts generally relied upon an onset of about 8-

9 hours. (Tr. 74, 103 (Dr. Tornatore); Tr. 126 (Dr. Gelfand); Tr. 177 (Dr. Whitton).) Petitioner's initial treatment records confirmed her condition was limited to her arms (Ex. 4, p. 7) and she later reported as of November 20, 2014, that her symptoms had moved to her legs (Ex. 18, p. 3). Thus, onset of lower extremity symptoms is placed at about twelve days post-vaccination.

i. Upper extremity onset

1. *Molecular Mimicry*

With respect to his molecular mimicry theory, Dr. Tornatore opined that the onset of petitioner's burning sensation of her arms 8-9 hours after vaccination is medically appropriate because of petitioner's history of vaccination. (*See* Ex. 36, p. 8.) He explained that petitioner's previous flu vaccinations could cause a rapid recall response." (*Id.* at 8, 9.) He further opined that the secondary or memory immune response phenomenon explains petitioner's rapid onset as "the memory cells are present at a higher frequency and are available to be stimulated quickly." (Ex. 50, p. 3.) In support of his contention, he cited an IOM report that he argued shows that a "second exposure to a vaccination may result in a markedly short time of onset of an immune response to administration of an exogenous antigen." (*Id.* (citing INST. OF MED., *supra*, at Ex. 43).) After the hearing, petitioner also supplemented the record with the Veiga-Fernandes article (Veiga-Fernandes et al., *supra*, at Ex. 64.) The Veiga-Fernandes study found that in immunocompromised mice, "[b]lastogenesis occurred early in memory cells; increased size was detected by 8 h[ours] and all cells had become blasts 24 h[ours] after in vivo transfer." (*Id.* at 48.)

Dr. Whitton contested Dr. Tornatore's rapid recall response theory, noting that the IOM explained there is a lag phase of 7-10 days for a primary immune response and 1-3 days for a memory immune response. (Ex. M, p. 6.) He emphasized that both of those lag phases "far exceed" the nearly 9-hour interval between petitioner's vaccine administration and the onset of the burning sensation in her arms. (*Id.* at 6-8; *see also* Tr. 177.) Dr. Gelfand Similarly opined. (Ex. J, p. 10; *see also* Ex. L, p. 2.) According to Dr. Whitton, the so-called "lag phase" is not likely to be injurious. (Tr. 174.)

Dr. Whitton also persuasively explained that his murine immunology research further confirms that 8-9 hours is too rapid for a vaccine to cause disease via molecular mimicry. (Ex. M, pp. 9-10.) Although he acknowledged that T cells can respond to antigen challenge within hours, he stressed that the cells do not begin to proliferate for several days. (*Id.*) He further explained that this "lag phase" also applies to memory T cells. (*Id.*) Thus, for a vaccine to cause disease via molecular mimicry, the T cells would have to begin dividing over a period of about three days post-vaccine, then divide several times over more days to reach the required number to cause harm, travel to the nerves, and "exert their effector function thereupon, causing the signs and symptoms of SFN." (*Id.* (citing Whitmire, Eam, & Whitton, *supra*, at Ex. M, Tab 4).) Dr. Whitton further explained that animal models show that even if an animal had been previously exposed to the inciting antigen, it takes "days or weeks" for an autoimmune neurological

disease to develop. (*Id.* at 12.) Although he acknowledged that the time interval between vaccination and disease may decrease as booster injections are administered, "biology dictates that there be a minimum interval of several days." (*Id.*; *see also* Tr. 178.)

Based on Dr. Whitton's detailed explanation of how T cells respond to antigen challenge, 8-9 hours is not a medically appropriate timeframe for a vaccine to cause an autoimmune injury via molecular mimicry. Thus, Dr. Tornatore's molecular mimicry theory is incompatible with an onset of 8-9 hours post-vaccination.[25]

2. *IFN- γ Theory*

Dr. Tornatore also opined that in the context of his IFN- γ theory, "the immune system has the capability of being stimulated quickly and producing soluble factors such as interferon gamma." (Ex. 60, p. 2.) He elaborated that memory T cells "can be quickly activated by exogenous antigens to produce sensory symptoms referable to the nervous system." (*See id.* at 3.) Dr. Tornatore further indicated that an animal study conducted by Dr. Whitton supported his opinion because the mice exposed to viral antigens and an inoculation against that antigen began producing IFN- γ within 6-12 hours. (Ex. 54, p. 1 (citing Whitmire, Eam, & Whitton, *supra*, at Ex. M, Tab 4).) Thus, Dr. Tornatore opined that "within hours of activation, the immune system can produce interferon-gamma, a known mediator of chronic pain . . . consistent with [petitioner's] clinical symptoms." (*Id.* at 2.) After the hearing, petitioner presented the Lai study in support of this theory. (Lai et al., *supra,* at Ex. 65.) The Lai study examined IFN- γ production and found that memory T cell activation occurred within 6-8 hours. (*Id.* at Ex. 65, p. 135 (Fig. 1).)

As discussed under *Althen* prong one, Dr. Tornatore's suggestion that post-vaccination IFN- γ would be an initiator or "fingerprint" or an autoimmune SFN is largely

[25] While I find Dr. Whitton persuasive regarding the need for a latency period greater than the 9 hours seen in this case, I additionally note that I do not treat Dr. Whitton's explanation of "days to weeks" long latency as constituting a bright line. *Accord Paluck v. Se'cy of Health & Human Servs*., 786 F.3d 1373, 1383-84 (Fed. Cir. 2015) (finding that "[t]he special master further erred in setting a hard and fast deadline" for onset and noting that the medical literature filed in the case "do not purport to establish any definitive timeframe for onset of clinical symptoms."). For example, in a prior case I have given at least some weight to the recall response as favoring a more rapid onset for an adaptive immune response and resulting autoimmune injury. That case involved GBS occurring one day post-vaccination. *Harris v. Sec'y of Health & Human Servs*., No. 18-944V, 2023 WL 2583393 (Fed. Cl. Spec. Mstr. Feb. 21, 2023). However, the difference between a 1-day onset and a less than 9-hour onset still remains significant. In that prior case I observed that "[w]hile the petitioner in this case has preponderantly satisfied *Althen* prong three despite an atypically rapid onset of just one day, this is a close call and the conclusion is a function of the specific record in this case. The conclusion that a one-day onset is medically appropriate for post-vaccination GBS is not unprecedented, but neither is it the norm. Of the few GBS non-Table claims adjudicated in the Program where onset occurred earlier than three days after vaccination most have not succeeded." *Harris*, 2023 WL 2583393, at *35; *see also Rowan v. Sec'y of Health & Human Servs.*, No. 17-760V, 2020 WL 2954954, at *19 (Fed. Cl. Spec. Mstr. Apr. 28, 2020) (concluding that "[p]etitioner's claim must be dismissed because it has not been demonstrated that the flu vaccine could cause GBS in a 30 to 36-hour timeframe).

unsupported on this record. Moreover, as discussed with respect to *Althen* prong two, Dr. Whitton demonstrated that only a primary vaccine exposure, and not a secondary exposure leading to a recall response, is likely to generate meaningful levels of IFN- γ. Thus, Dr. Tornatore's reliance on experimental studies showing memory T cells to generate IFN- γ more quickly is not persuasive in the context of this case.

However, even if this aspect of the theory were better supported, Dr. Whitton also explains that the process would still be unlikely to unfold within 8-9 hours. Dr. Whitton emphasized that activation of the immune system and occurrence of neurological injury are distinct biological events. (Ex. N, p. 2.) Neither Dr. Whitton's own article (Whitmire, Eam, & Whitton, *supra*, at Ex. M, Tab 4.) nor the Lai article (Lai et al., *supra,* at Ex. 65) support Dr. Tornatore's opinion because they merely show that T cells can respond to antigen challenge within a matter of hours. (Ex. N, pp. 3, 5; Tr. 180-81.) According to Dr. Whitton, the Whitmire study Dr. Tornatore relies upon actually shows that memory T cells are "quick to respond but slow to divide," as is required to cause neurological injury. (Whitmire, Eam, & Whitton, *supra*, at Ex. M, Tab 4.) This would likewise affect the overall production of IFN- γ. Thus, for example, the above discussed Cohen, et al, study that measured IFN- γ levels in actual human vaccinees following smallpox vaccination found both that cytokine levels, including IFN-γ, "typically" did not begin to rise until 4-5 days post-vaccination and that symptom presentation correlated to cytokine level over time. (Cohen et al., *supra,* at Ex. N, Tab 2, p. 6, 7 (Figs. 4(A)-(B)).) The Cohen study is the only evidence of record in this case that addresses the timing of IFN- γ production in actual patients. It suggests that the mouse models must be viewed with caution.

Thus, petitioner has not established that it is medically reasonable to conclude that petitioner's vaccinations caused her to suffer SFN within just 8-9 hours of administration due to IFN- γ induced neuropathic pain. Accordingly, petitioner has failed to satisfy her burden under *Althen* prong three based on the assumption that her upper and lower extremities represent the same condition.

3. *Comparison to E.M.*

Special masters are not bound by the decisions of other special masters and are also not obligated to distinguish prior cases. *Boatmon*, 941 F.3d at 1358 (explaining that "[t]o the extent the Court of Federal Claims required that special masters cite and distinguish decisions of other special masters, it was incorrect.") Nonetheless, in this case it might be helpful. Similar to this case, petitioner's expert in *E.M.* relied on recall response to explain how memory T cells can cause SFN within four to six hours post-vaccination. 2021 WL 3477837, at *42. This was based in significant part on citation to two papers, Lai, et al., and Schonberger, et al., both of which this petitioner has also filed in this case. The Lai paper addresses the time for memory T cells to respond to an antigen and the Schonberger is an epidemiologic paper examining incidences of post-Swine flu vaccine GBS.

The *E.M*. special master credited petitioner's expert's reliance on these articles at least in part because (1) he had superior credentials in immunology as compared to respondent's neurology expert and (2) respondent's expert had changed his assessment of onset during the hearing, prompting her to observe that she would not give his opinion "much, if any, weight." *Id*. at *43. In this case, Dr. Whitton – who is well qualified to opine and who does not present any credibility issue comparable to what was seen in *E.M*. – has offered expert opinion evidence thoroughly and persuasively rebutting Dr. Tornatore's opinion with regard to the timing of onset. I have also explained above why this rebuttal applies specifically to the Lai paper and, with regard to the Schonberger paper, have explained why I do not find GBS to be a helpful analog with respect to vaccine causation. Even accepting *arguendo* that comparison to GBS could be informative, the Schonberger article provides only scant evidence of any rapid onset and lacks the granularity that would be necessary to demonstrate a mere hours-long onset in any case, let alone that causation could be ascribed in such outlier cases. Without more it would be unpersuasive in this case even absent Dr. Whitton's testimony.

ii. Lower extremity onset

Alternatively, if one were to assume that only petitioner's lower extremity symptoms were confirmed to be a part of her SFN as Dr. Gelfand opined, then *Althen* prong three would no longer present an issue. Under that scenario, respondent acknowledges that an onset of SFN twelve days after vaccination would be medically appropriate to infer causation. (ECF No. 151, p. 49, n.17.) However, the analysis above confirms that petitioner has likewise failed to meet *Althen* prongs one and two, which means that petitioner still cannot be compensated. *Hibbard v. Sec'y of Health & Human Servs.*, 698 F.3d 1355, 1364-65 (Fed. Cir. 2012) (holding the special master did not err in resolving the case pursuant to prong two when respondent conceded that petitioner met prong three).

VI. Conclusion

Petitioner has clearly suffered and she has my sympathy. Moreover, it is understandable that she would come to personally believe that pain arising so close in time to her vaccination would be related to those vaccinations. However, for all the reasons described above, petitioner has not preponderantly demonstrated that she actually suffered a vaccine-caused injury and is therefore not entitled to compensation. Accordingly, this case is dismissed.[26]

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master

[26] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.