In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 15-1451V

(to be published)

CAILEN MCKOWN, Petitioners, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Corcoran Filed: July 15, 2019 Entitlement Decision; Human Papillomavirus ("HPV") Vaccine; Postural Orthostatic Tachycardia Syndrome ("POTS"); Eczema; Autoimmune Diseases

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DECISION DENYING ENTITLEMENT[1]

On December 1, 2015, Cailen McKown filed a Petition under the National Vaccine Injury Compensation Program (the "Vaccine Program"[2]), alleging that two doses of the Human Papillomavirus ("HPV") vaccine she received on March 20, 2013, and September 3, 2013, respectively, along with a Hepatitis A vaccine received on March 20, 2013, caused her to suffer postural orthostatic tachycardia syndrome ("POTS") and skin rashes (including eczema). Petition (ECF No. 1) ("Pet.") at 1-2.

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

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended at 42 U.S.C. §§ 300aa-10–34 (2012)) (hereinafter "Vaccine Act" or "the Act"). All subsequent references to sections of the Vaccine Act shall be to the pertinent subparagraph of 42 U.S.C. § 300aa.

A hearing in this matter was held on September 26-27, 2018. After consideration of the record and testimony provided at hearing, I find that Petitioner is not entitled to a compensation award. As discussed in more detail below, Petitioner has not demonstrated with reliable scientific and medical evidence that the HPV or Hep A vaccines could be, or were, causative of her POTS (or worsening of POTS or her preexisting eczema/skin symptoms). Petitioner's experts failed to offer a persuasive, reliable medical theory in support of her claim, and were unpersuasive in their attempt to establish that Petitioner's POTS was the rare neuropathic variant that could be autoimmune in nature.

I. Factual Background

Pre-Vaccine Health History

Ms. McKown was born on January 14, 2000, and received routine vaccinations throughout her childhood without any reported adverse events. Ex. 1 at 13-14. Her medical history was significant for eczema (treated with Elidil), diffuse dry skin, and recurrent fever blisters, which she experienced one to three times per month since the age of six months. *Id.* at 32-34 (12/1/2010 ten-year-old well-adolescent visit), 36 (2/25/2011 eleven-year-old well-adolescent visit), 38.

Around the time Petitioner received the first dose of the HPV vaccine, she had already displayed symptoms pertaining to the injuries at issue in this case, in addition to eczema. Thus, on March 20, 2013, at Petitioner's thirteen-year-old well-child visit, her mother, Mrs. Tabatha McKown, reported to treaters that Petitioner had recently experienced lightheadedness with position changes and two episodes of syncope in the prior three months, both associated with position changes and without heart palpitations. Ex. 1 at 44. Ms. McKown's history of dry skin was also noted, but she was otherwise deemed to be well. *Id.* The pediatrician's impression was neurocardiogenic syncope, and Petitioner was told to increase fluids and salt in her diet, with a referral to a cardiologist to follow up on the possible cause of these symptoms. *Id.*

Receipt of HPV Vaccine Doses

Petitioner received the first HPV vaccine dose (along with the Hep A vaccine) on March 20, 2013, at the aforementioned well-child visit. Ex. 1 at 14, 44. No adverse reaction was noted at the time, and there is no recorded instance of any reaction to the first HPV dose within a month of its administration. Then, on April 26, 2013 (about five weeks following vaccination), emergency medical service providers transported Ms. McKown to the emergency room at Eastside Medical Center in Snellville, Georgia, from a local yogurt shop, where, following an upset stomach, she had stood up and then immediately began feeling lightheaded, along with a severe headache. Ex.

8 at 6-16. Petitioner passed in and out of consciousness for brief intervals several times, and she was hyperventilating when EMS responders arrived. *Id.* at 6, 12.

At Eastside Medical Center, the treating ER physician noted that Ms. McKown had reportedly experienced similar symptoms in the past, observing that she had been recently advised by her primary care physician ("PCP") to follow up with a cardiologist. Ex. 8 at 6, 12. Laboratory results, an EKG,[3] and a head CT scan[4] were normal, with the exception of revealing left sphenoid sinus disease. *Id.* at 8-10, 24-25. The ER physician diagnosed Petitioner with sinusitis (which he noted could explain her headaches and dizziness), and vasovagal syncope, discharging her that same day. *Id.* at 10.

Three days later Ms. McKown followed up with her PCP, Dr. Melissa Magill. Ex. 1 at 47. Petitioner's mother recounted Petitioner's recent syncope symptoms at the yogurt shop, noting that she had been "lightheaded" and "woozy," and "trembly," but not jerking, drifting in and out of consciousness for five to ten minutes. *Id.* Following an exam, Dr. Magill assessed Petitioner with a "known history of neurocardiogenic syncope[,]" and recommended that she increase fluid and salt intake. *Id.*

Cardiology Assessment

On May 1, 2013, Ms. McKown saw Dr. Kenneth Dooley, a cardiologist at Sibley Heart Center in Atlanta. Ex. 7 at 5-7. Dr. Dooley noted that Petitioner reported having passed out a total of two to five times previously, most recently the week before. *Id.* Petitioner provided some additional details about the circumstances of three of the times she had experienced syncope: first, three years prior after her father had reprimanded her for poor behavior; second, approximately six months prior while watching a veterinary procedure; and third, five days prior, in the yogurt shop incident. *Id.* at 5. She also stated that she felt that her symptoms were worsening. *Id.* Upon exam, Dr. Dooley noted that Petitioner almost passed out when asked to sit up. *Id.* at 6. Her supine blood pressure was noted to be 104/64 (heart rate: 68), and her standing blood pressure was 90/52

[3] An EKG (or "electrocardiogram") is a noninvasive graphic tracing that records the electrical activity of the heart muscle. *Dorland's Illustrated Medical Dictionary* 597, 599 (32nd ed. 2012) (hereinafter *Dorland's*). Each beat of the heart is triggered by an electrical impulse (generated from cells in the heart), and the EKG records the timing and strength of these signals. *See Electrocardiogram*, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/ekg/about/pac-20384983 (last accessed on June 19, 2019). EKGs are used to detect or diagnose common heart problems (including irregular heart rhythm, blocked arteries, structural problems, heart attacks, or heart disease). *Id.*

[4] A CT (or "computerized tomography") scan combines a series of X-rays taken from different angles around the body and uses computer processing to create cross-sectional images of the bones, blood vessels, and soft tissues inside the body. *See CT Scan*, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/ct-scan/about/pac-20393675 (last accessed on July 19, 2019). CT images are more detailed than a plain X-ray. *Id.* This type of scan is used to quickly examine patients who may have wide-spread internal injuries, and can be used to diagnose or monitor diseases such as cancer, heart disease, lung nodules, and liver masses. *Id.*

with a pulse of 100. *Id.* Dr. Dooley diagnosed her with "syncope, possibly vasovagal in origin[,]" noting that her history and symptoms were most suggestive of simple fainting. *Id.* He scheduled Petitioner for a tilt table test[5] and recommended she be screened for epilepsy as well. *Id.*

Petitioner had a follow up appointment with Dr. Dooley two months later, on July 1, 2013. Dr. Dooley noted that the tilt table test conducted in late May 2013 had been positive for POTS. Ex. 7 at 8-9; *see also* Ex. 55. Orthostatic readings taken in conjunction with the test revealed a significant increase in heartrate (when tilted from the supine position to the standing position), followed by complaints of dizziness and blurry vision (after approximately eight minutes at standing). Ex. 55 at 5. Ms. McKown's blood pressure then dropped significantly (minimum cuff pressure of 57/32) and her heartrate slowed significantly (into the low fifties) as the tilt returned to flat. *Id.* Within five minutes after returning to the supine position, her rates returned to normal. *Id.* Dr. Dooley discussed the results of the test, and noted that Petitioner continued to have short syncopal episodes. Ex. 7 at 8-9. He prescribed Fludrocortisone (0.1 mg, once daily) in addition to the Valtrex she was currently taking. *Id.* Dr. Dooley also advised Petitioner to return in six months (or sooner if the medication did not work), and to have her mother notify him if she experienced additional episodes. *Id.*

Repeated Skin-Related Symptoms

A week later, on July 8, 2013, Petitioner's mother called Dr. Dooley's office to report that Petitioner had stopped taking Fludrocortisone, thinking it had caused her to develop a rash and hives, although these symptoms resolved on their own after treatment with Benadryl. Ex. 7 at 33. A week later, Petitioner's mother called again to report that her hives had returned after she drank a large glass of milk, causing Petitioner's PCP to diagnose her with a dairy allergy. *Id.* at 31. Notes from the telephone call also indicated that Ms. McKown's PCP suspected her pre-existing eczema may have been a manifestation of her dairy allergy. *Id.* Petitioner thereafter restarted Fludrocortisone, with no initial notable problems at that time. *Id.* On July 23, 2013, however, her mother called a third time to report that Petitioner was now suffering from worsening headaches, and was not "feel[ing] like herself" (i.e., "feeling very tense"), although without any repeat syncopal episodes. *Id.* at 30. Dr. Dooley recommended that Ms. McKown start Midodrine as an alternative to Fludrocortisone. *Id.*

Petitioner went back to her PCP at Lawrenceville Pediatrics on July 25, 2013, now complaining of worsening eczema, a rash on her neck, back, and arms, and insect bites. Ex. 1 at

[5] A tilt table test is used to evaluate syncope by measuring heart rate and blood pressure in response to the body's change in position. *Tilt Table Test*, Mayo Clinic, https://www mayoclinic.org/tests-procedures/tilt-tabletest/about/pac-20395124 (last accessed July 5, 2019). The patient begins by lying flat on a table for around 15 minutes. *Id.* The table is then quickly tilted upright to change the body's position from lying down to standing up. *Id.* The table generally remains upright for 45 minutes to allow the doctor to monitor the patient's cardiovascular response. *Id.*

50. The pediatric nurse practitioner noted that Ms. McKown's eczema flared when the weather was hot, causing her to feel itchy and scratch her skin. *Id.* It was also noted that Petitioner had been diagnosed with POTS recently and was waiting to see an electrophysiologist. *Id.* The nurse practitioner advised Petitioner to see an allergist, and recommended treatment with various over-the-counter antihistamines and ointments. *Id.* A few weeks later, on September 3, 2013, Petitioner presented again to Lawrenceville Pediatrics with complaints relating to a sore throat and congestion. *Id.* at 15-17. She was diagnosed with sinusitis, and now received her second HPV vaccine dose, with no noted adverse reaction. *Id.* at 17.

Progression of Skin Symptoms and Efforts to Treat Them

In the ensuing months in the fall of 2013, Ms. McKown's skin condition continued to worsen. In November 2013, she had four dermatology appointments at Georgia Skin Cancer and Aesthetic Dermatology in Athens, Georgia, for treatment of an itchy rash all over her body that was reported to have been present for months. Ex. 2 at 11-12, 13-16, 20, 25-26. Following examination, the treating dermatologist (Dr. Jeffrey Moulton) diagnosed her with atopic and contact dermatitis and eczema. *Id.* at 25-26. Dr. Moulton recommended that Petitioner take Clobetasol and/or Vaseline to mitigate her symptoms (in addition to avoiding strong cleansers, scented detergents, and excessive hand washing). *Id.* at 25. Patch testing was also performed, which was positive for numerous chemicals. *Id.* at 11-12, 13-16, 20. No instances of syncope or POTS-related symptoms are reported in the medical records from this time period, however, and Ms. McKown was noted to be alert, oriented, and in "no acute distress." *Id.* at 11, 13, 25-26.

One month later, on December 4, 2013, Ms. McKown sought yet another opinion regarding her skin condition. She presented to Dr. Deidre Crocker, at the Allergy & Asthma Center of Conyers in Monroe, Georgia, complaining of worsening eczema and a rash. Ex. 3 at 2-4. At this visit, Petitioner informed Dr. Crocker that she had always had mild eczema, but that her symptoms had worsened in May 2013 – around the same time she was diagnosed with POTS. *Id.* at 2. The rash was aggravated by hot water and sweat, and a variety of treatments (steroid injections, Benadryl, and changes in shampoos/lotions) had proven ineffective. *Id.* Increased itching was also noted with ingestion of wheat products. *Id.*

On exam, Petitioner displayed multiple scaly, red eczema patches on the neck, arms, and trunk. Ex. 3 at 2-3. Her diagnosis remained "contact dermatitis and other eczema, due to unspecified cause." *Id.* at 3. Dr. Crocker expressed the view, however, that Ms. McKown's skin condition was likely not related to POTS. *Id.* at 4 ("POTS is not an immune disease so do not feel that is triggering eczema but possible she has had some st[r]essors contributing to increased eczema . . . [a]lso possible she may have developed some allergic sensitivities triggering her eczema"). *Id.* She advised Petitioner to avoid a number of dermatitis allergens, and prescribed Mometasone cream. *Id.*

Ms. McKown had a follow-up appointment with Dr. Crocker two weeks later, on December 19, 2013. It was reported at this time that her eczema had improved, but she still was experiencing significant itching and a rash. Ex. 3 at 5-6. Her food diary also revealed that every time she ingested corn products she broke out in hives, with a milder reaction to wheat and other products. *Id.* at 5. Blood work conducted on the following day was negative for celiac disease, but allergy testing was positive for several foods and environmental substances, including fruits, oat, carrot, barley, sweet potato, corn, and wheat. *Id.* at 11-13. During this blood testing, Ms. McKown also had an episode of hypotension, hypoxia, and tachycardia during the blood draw. Ex. 1 at 18; Ex. 20 at 1. She was reported to have passed in and out for ten minutes until her vitals came back to normal. Ex. 20 at 1. But this reaction to the testing did not result in any further medical treatment, emergency or otherwise.

2014 Treatment of Skin and POTS-related Symptoms

On January 2, 2014, Petitioner saw Dr. Crocker again, now reporting that the steroidal treatment she had been prescribed the prior month had cleared her skin over the holiday, but that she had experienced flares after playing with dogs and ingesting a wheat cracker. Ex. 3 at 8-9. Her physical exam was normal but for multiple scaly, erythematous patches of eczema on the face, neck, elbows, knees and legs. *Id.* at 9. Additional testing showed strong positives to cat, dust mites, tree pollen, and grass pollen (as well as certain foods including wheat, corn, and soy). *Id.* Ms. McKown was again prescribed oral Prednisone. *Id.* at 9.

One week later Ms. McKown presented to Dr. Seth Marcus, at GI Care for Kids in Atlanta for treatment of abdominal pain (with specific concern for possible "allergic gut"), syncope, and food allergies. Ex. 12 at 5-9; Ex. 1 at 18-21, 75. Similar to the health course described above, Dr. Marcus noted that Petitioner had experienced minor syncopal episodes in the past, culminating in her May 2013 complete loss of consciousness for twenty to thirty minutes along with other associated symptoms. Ex. 12 at 5-6. He noted that these episodes usually arose with stressful or exciting events, appeared to be more frequent during her menstrual cycle, and generally occurred two to three times per month. *Id.* at 5-6. A history of eczema was also noted, along with intermittent hives and worsening of her eczema in May 2013 (although the contemporaneous record does not really record increased complaints of eczema before July 2013). *Id.* at 6. Consistent with Dr. Crocker, Dr. Marcus expressed uncertainty as to whether Petitioner's syncopal episodes could be related to her atopic disease or were an independent phenomenon. *Id.* at 8-9 ("I am unaware of a known relationship between POTS and atopy, histamine release, hypereosinophilic syndrome or eosinophilic gastrointestinal disease"). *Id.* at 9. He did however opine that her syncope was likely unrelated to dietary exposures, with the exception of her most recent event that had occurred one day after exposure to wheat. *Id.* Labs again ruled out celiac disease. Ex. 5 at 85.

On January 27, 2014, Ms. McKown had a follow-up visit with her cardiologist, Dr. Dooley, seeking clearance for her upcoming colonoscopy and endoscopy to help assess the cause of her ongoing GI symptoms. Ex. 7 at 10-11, 34. Petitioner now stated she was having one to two episodes of syncope per month, the last of which was on December 29, 2013, lasting around twenty-five minutes and resulting in stiffness and eye-rolling (although this incident is not reflected in any immediately contemporaneous record). *Id.* Dr. Dooley noted, however, that Petitioner had not been taking her POTS medication for months, out of the concern that it exacerbated her rash. *Id.* The colonoscopy and upper GI endoscopy were performed by Dr. Marcus on January 31, 2014. Ex. 12 at 19-20. Biopsies showed no significant abnormalities except for mild esophagitis with rare eosinophils in the distal and mid esophagus. Ex. 5 at 79-84.

On February 21, 2014, Petitioner saw another immunologist, Dr. Karen Freedle, at Emory Children's Center in Atlanta for a secondary evaluation of her skin condition. Ex. 1 at 76-79, 85-87; Ex. 5 at 42-72. Consistent with past treaters, Dr. Freedle's impression was atopic dermatitis, chronic urticaria of unclear etiology, chronic nasal and ocular symptoms, intermittent GI complaints, and multiple food allergies. Ex. 1 at 76. Laboratory results conducted during the visit were also normal, with the exception of a mildly elevated sedimentation rate and positive ANA at 1:40 with a homogeneous pattern. Ex. 5 at 76-78.

On March 5, 2014, telephonic records establish that Petitioner's mother called Dr. Dooley about a recent study in a journal indicating that POTS could be caused by an autoimmune disorder, and asked whether Petitioner's treatment would change in light of such findings. Ex. 6 at 25. She reported that Ms. McKown had been tested for lupus and many allergies, her last syncopal episode was in December, and that most of her other medications had been eliminated. *Id.* Dr. Dooley noted that he had not heard POTS was autoimmune, but that he could refer Petitioner to a neurologist to explore this concern. *Id.*

Over the course of the next few months, Ms. McKown's adverse skin symptoms continued to worsen. On April 7, 2014, Petitioner again saw her PCP, Dr. Magill, complaining of the same rash, but with concerns for possible staph infection. Ex. 1 at 22-24. On examination, Petitioner had diffuse dry skin with "punched out" lesions to the forehead, elbow, wrists, ankles, and knees. *Id.* at 23. Her diagnosis remained eczema, and Dr. Magill recommended that she continue taking clindamycin and acyclovir. *Id.* That same month Petitioner had a follow-up with Dr. Freedle on April 21, 2014. Ex. 5 at 35-37. Her examination was normal except for open, excoriated areas of skin. *Id.* at 36. A skin biopsy taken at this visit from her right arm revealed subacute spongiotic dermatitis. Ex. 1 at 93-96.

On May 12, 2014, Ms. McKown saw a different dermatologist, Dr. Zakiya Rice of Children's Healthcare of Atlanta, regarding her rash and sores. Ex. 4 at 65-70. The history from this visit again identified onset as May 2013, with heat causing aggravation of symptoms. *Id.* at

65-66. The examination revealed eczematous papules with excoriations, but well-hydrated skin. *Id.* at 68. Dr. Rice assessed Petitioner with "spongiotic dermatitis, steroid responsive," and recommended that she restart topical steroids and taper oral Prednisone. *Id.* At a follow-up appointment on June 2, 2014, Dr. Rice noted that Petitioner continued to use Prednisone due to her persist rash. *Id.* at 50-57. A biopsy taken during the visit was consistent with eczema. *Id.* at 55.

The following month, Petitioner saw her PCP, Dr. Magill, for her fourteen-year-old well visit on June 4, 2014. Ex. 1 at 25-30. She now reported that she had been experiencing joint pain in the shoulders, upper back, wrists, and fingers since October 2013 (in addition to the skin rashes/eczema and syncopal episodes described above) – although, as the somewhat exhaustive record review above reveals, no prior treaters had been informed of these newly-reported symptoms. *Id.* at 25. Her examination was positive for persistent diffuse dry skin with lichenification on the elbows and an erythematous rash on the face (consistent with her ongoing skin symptoms). *Id.* at 27. Hives were also reported as a current problem (attributable to overheating, embarrassment, and emotion), but were not noted on exam. *Id.* at 25, 27. By June 12, 2014, Ms. McKown had completed steroid treatment, but her skin was reportedly still red and burning, the hives and sores were becoming more severe, and her joints were reported to be throbbing. Ex. 4 at 32-33; Ex. 5 at 137-38.

Treatment of Joint Pain

On June 30, 2014, Ms. McKown saw a rheumatologist, Dr. Kelly Rouster-Stevens, at Children's Healthcare of Atlanta, for evaluation of her joint complaints. Ex. 21 at 1-3; Ex. 23 at 21-24. Dr. Rouster-Stevens noted that Petitioner had been diagnosed with POTS in May 2013, and then a few months later developed a rash associated with pruritus and burning. Ex. 21 at 1. The medical history from this visit also states that since her last syncopal episode in December 2013, Petitioner had experienced some improvement in her POTS symptoms, but had developed joint pain, and the persistence of her skin rash (initially thought to be related to allergies) was proposed to possibly have some alternative explanation. *Id.* On examination, Ms. McKown displayed eczema patches and multiple tender trigger points in her back and extremities, but no joint swelling or warmth. *Id.* at 3. Dr. Rouster-Stevens opined that Petitioner likely did not have lupus, arthritis, myositis, or any other chronic rheumatologic process, suggesting instead that her exam was most consistent with amplified musculoskeletal pain syndrome. *Id.* Lab tests performed at this time did not support a diagnosis of lupus or other autoimmune connective tissue disease. *Id.* at 4, 7-8. Dr. Rouster-Stevens ultimately recommended that Petitioner be evaluated in the pain clinic. *Id.* at 10.

Roughly one month later, Ms. McKown had follow-up appointments with Drs. Freedle and Rice on July 21, 2014. Ex. 5 at 20-22; Ex. 4 at 16-21. Dr. Freedle noted that Petitioner's rash responded well to steroids, but that she stopped taking them due to how they made her feel mentally, and that overall she felt she was not experiencing true improvement on this treatment

course. Ex. 5 at 20. Her visit with Dr. Rice revealed the same persistent symptoms noted above. Ex. 4 at 16-21. At this visit, Petitioner and her mother also reported they "worried about this startign [sic] with Gardasil vaccination 3/2013 and 9/2013[,]" but Dr. Rice offered no opinion regarding vaccine causation. *See id.* Otherwise, Dr. Rice noted that a naturopathic consult had been scheduled for July of that year – at which time Petitioner planned to seek further treatment. *Id.*[6]

Cleveland Clinic Evaluation

In mid-August 2014, Petitioner obtained multiple evaluations at the Cleveland Clinic in Ohio in an attempt to ascertain the etiology for her constellation of symptoms. None of these Cleveland Clinic treaters concluded that her symptoms were vaccine-related, and few saw any relationship between the different symptoms she was experiencing.

First, on August 13, 2014, Ms. McKown saw dermatologist Dr. Joan Tamburro, D.O. Ex. 11 at 5-11. The health history taken during the visit indicated that Petitioner reported she had been itchy with sores all over her body since October 2013, shortly after receiving a second HPV vaccine dose. *Id.* at 5. Her POTS diagnosis and joint pain were also noted. *Id.* at 6. After examination and consideration of Petitioner's history, Dr. Tamburro diagnosed her with atopic dermatitis (consistent with the diagnosis of past treaters) which had not been adequately treated. *Id.* at 7. Dr. Tamburro found no evidence to support a diagnosis of connective tissue disease, determining only that Petitioner likely had an "angry back" (based on previous patch testing) which explained the numerous allergy positives. *Id.* Exam notes also indicated that Dr. Tamburro discussed with Petitioner and her mother that a "persistent rash from Gardasil [wa]s unlikely." *Id.* Lidex and topical ointments were prescribed for her symptoms. *Id.*

Second, on the following day Petitioner saw two Cleveland Clinic allergists, Drs. Maria Blanch and Velma Paschall, for an alternative evaluation of what appeared to possibly be chronic urticaria. Ex. 11 at 12-23. Both physicians noted that Petitioner had a history of eczema since childhood that had become more severe beginning the summer of 2013. *Id.* at 12. Episodes of hives (or urticaria) were also noted to be triggered by heat, stress, showers, and animals. *Id.* Upon exam, it was determined that Petitioner's symptoms were most consistent with severe, chronic urticaria that was resistant to standard antihistamine treatment, recommending instead an alternative, newly-approved treatment (Xolair). *Id.* at 14. Following lab testing, Dr. Paschall sent a letter to

[6] In late July 2014, Ms. McKown sought naturopathic care for her persistent symptoms described herein (including skin rashes, hives, dizziness, and joint pain). *See* Ex. 19 at 6-8 (visit to Oconee Natural Healthcare Center in Watkinsville, GA). Ms. McKown's treater, Dr. Wyler Hecht, reviewed her health history and recommended treatment with various supplements including Sea Cure (a white-fish dietary supplement), Immuno PRP spray (an immune-balancing spray), Immuno PRP powder (immune-balancing vitamin supplement), and Curacel (a plant-based vitamin for cell growth) to aid her symptoms. *Id.* at 8. Dr. Hecht's notes also indicated that Petitioner's mother felt "as though the Gardasil vaccine triggered the reaction in her daughter[,]" but he offered no opinion regarding causation. *See id.* at 6.

Petitioner's mother (dated September 19, 2014) in which she expressed the view that it was "unlikely that [Petitioner] has autoimmune chronic urticaria" because both her anti-IgE and anti-IgE receptor antibody tests were negative. Ex. 22 at 1.

Third, on August 15, 2014, Petitioner saw Cleveland Clinic rheumatologist Dr. Andrew Zeft for a second evaluation for onset of joint symptoms and persistent rash. Ex. 11 at 24-28. The examination revealed no objective evidence of arthritis. *Id.* at 28. Dr. Zeft opined that her arthralgia was mechanical in etiology, and that she had amplified pain from muscle spasm and trigger of muscle spasm secondarily due to pain and itching from her skin lesions. *Id.* He thus recommended treatment of her primary skin lesions, and knee stretching and strengthening exercises. *Id.*

Subsequent Treatment

The following September through November 2014, Ms. McKown began treatment with Dr. Phillip DeMio[7] in Worthington, Ohio. Ex. 14 at 12. At this time, Petitioner's mother reported to Dr. DeMio that she had received a diagnosis of "Gardasil Syndrome (recently spoke with Lloyd to confirm and Dr. Hitch in Athens)," along with POTS, chronic hives, and dermatitis. *Id.* She also specifically told Dr. DeMio that Dr. Rice (the dermatologist Petitioner saw in the spring of 2014) had determined that "Gardasil triggered all this but . . . won't go on record to say so." *Id.* at 22. (As noted above, however, the official records from Petitioner's visits to Dr. Rice do not corroborate this assertion – and, if anything, undermine it).

Petitioner's first visit with Dr. DeMio included a discussion of her health history and a physical exam. Ms. McKown was noted to be "calm and "coop[erative]" on exam. Ex. 14 at 12. Eczema and hives were present at the time of the visit, and her POTS diagnosis was noted. *Id.* at 12, 25. Dr. DeMio assessed Petitioner with "adverse rxn to [G]ardasil," noting that she had "reported this to many prior practitioners" in the past (albeit ones who wouldn't "stand for any suggestion of va[ccine] injury"). *Id.* at 25, 27. Dr. DeMio recommended that Ms. McKown begin self-administered vitamin B-12 injections to treat her symptoms. *Id.* at 59-60. Lab testing revealed a positive IgM titer for Lyme western blot (but negative IgG), and normal results for toxic/essential elements and metals. *Id.* at 36-37, 38-52.

On November 4 through 6, 2014, Mrs. McKown called to report a decline in Petitioner's condition, but noted that the B-12 injections had helped "tremendously" to give her more energy. Ex. 14 at 30. In a handwritten note, Dr. DeMio indicated a "Dx: "Lyme [disease], [and] what components of current [symptoms] are directly from Lyme: based at least in part on index's response." *Id.* at 31. Notes taken during the phone consultation reveal that Dr. DeMio

[7] Dr. DeMio's website indicates that his practice focuses on medical testing and treatment for autism, AD/HD disorders, and Lyme disease. *See About Dr. DeMio*, http://drdemio.com/dr-phillip-demio/ (last accessed on June 19, 2019).

recommended that Petitioner continue with the B-12 injections. *Id.* He also prescribed ultra-low dose Naltrexone capsules, Hydroxychloroquine, Doxycycline, Cefdenir, and Valtrex. *Id.* at 53-58. At a follow up visit in May 2015, Dr. DeMio ordered blood work relating to static encephalopathy, metabolic disorder, mineral deficiency, and autoimmune disorder. Ex. 9 at 5-6. The results, however (including sedimentation rate, iron, c-reactive protein, ammonia, ANA, and lupus), were normal and negative for autoimmune disorders. *Id.* at 7-12.

From August 2014 to April 2015, Ms. McKown began attending counseling and started physical therapy for her joint pain. A counseling note from September 2014 indicated that she had "learned she no longer has a POTS diagnosis" but rather her symptoms were "actually a part of the illness related to Gardasil." Ex. 18 at 7. Notes from October of that year also report that the B-12 injections were having a positive effect, causing Petitioner to feel much better. *Id.* at 16-17, 19-20. In November 2014, her counselor noted that she had been diagnosed with Lyme disease (presumably on Dr. DeMio's suspicion), and had started antibiotic treatment in response. *Id.* at 20-23. Petitioner's physical therapy notes from the following April indicated that she reported her joint and muscles aches were caused by "Gardasil vaccine/Lyme disease." Ex. 15 at 6. Her health history included concerns for "Gardasil syndrome[,]" Lyme disease, and POTS. *Id.* at 7.

Over the next few months, Ms. McKown's health course began to improve. The following year, on March 4, 2016, she returned to her PCP for a sixteen-year-old well-adolescent visit at Lawrenceville Pediatrics. Ex. 24 at 2-8. The evaluating nurse practitioner noted that Ms. McKown's mother reported that Petitioner was "still recovering" (presumably from the symptoms discussed herein), but no current illnesses or chronic problems were noted, with a normal exam. *Id.* at 2, 4. The only current medication reported was Mometasone ointment, and Petitioner was not on any supplements at the time of the appointment. *Id.* at 2. An additional PCP visit on March 17, 2016, indicated that Ms. McKown presented with complaints of flu-like symptoms, fever, and moderate nasal congestion (though no medications were prescribed). *Id.* at 9-11. No further medical records have been filed.

II. Fact and Expert Witnesses

A. *Petitioner's Witnesses and Hearing Evidence*

1. Mrs. Tabitha McKown

Mrs. McKown, Petitioner's mother, testified about her health history and symptomatology course following receipt of doses of the vaccines at issue herein. Tr. at 11-97; *see also* Ex. 63 (narrative statement). Prior to receiving doses of the HPV vaccine, Petitioner was healthy, active, and energetic. Tr. at 12. She was training for a triathlon, and enjoyed biking, swimming, and running. *Id.* In support, Mrs. McKown offered multiple photographs of Petitioner (between in

September 2012 and January 2013) showing her interacting with friends and enjoying the above-noted activities. *Id.* at 13-15.[8]

As noted above, Petitioner had multiple pre-existing medical conditions which bear on the injuries alleged in the present matter (specifically, dry skin and eczema). Mrs. McKown acknowledged this fact but maintained that her daughter's symptoms were exacerbated after her receipt of doses of the HPV vaccine. Tr. at 15, 61; *see also* Ex. 7 at 31 (dated July 15, 2013). She described Petitioner's childhood eczema as mild to moderate (consisting of dry patches on the folds of the arms and backs of the legs). Tr. at 15, 60-61. Following the administration of Vaseline or steroid cream, the patches would resolve rather quickly, and before vaccination Petitioner never experienced eczema on the face, chest, or back. *Id.* at 15-16.

Mrs. McKown also acknowledged that Petitioner had experienced syncopal episodes (and lightheadedness with position change) in the past. Tr. at 15-16. She attributed such instances of lightheadedness to Ms. McKown's triathlon training, dehydration, and low blood pressure. *Id.* at 16. Mrs. McKown disputed the accuracy of a record from March 2013 indicating that Dr. Magill had noted that Petitioner experienced two syncopal episodes in the last three months, maintaining that she had reported to Dr. Magill the episodes of syncope had occurred over a three-year period (consistent with reports made to Dr. Dooley), and were triggered in ways distinguishable from other instances (e.g., following a reprimand for poor behavior or the sight of blood). *Id.* at 16-17.

According to Mrs. McKown, Petitioner's overall health began to decline rapidly following her first dose of the HPV vaccine on March 20, 2013. Tr. at 17.[9] She experienced a "pretty quick[]" onset of migraines thereafter, though Mrs. McKown could not recall the exact date they started. *Id.* Five weeks later, Petitioner experienced a syncopal episode while at a yogurt shop with friends. *Id.* at 17-18. The event resulted in EMS transporting Petitioner to the local emergency room. *Id.* at 17. Mrs. McKown recalled that her daughter was diagnosed with POTS within weeks of the yogurt shop episode. *Id.* Thereafter, her syncopal episodes became more severe (i.e., lasting anywhere from a few seconds to thirty minutes) and occurred more frequently. *Id.* at 17-18.

[8] The photographs offered in support of Ms. McKown's pre-vaccine health (as well as a number also purporting to demonstrate the progression of her skin-related symptoms) were not filed prior to hearing. Tr. at 12-13. On direct examination of Mrs. McKown, Respondent objected to the photographs being entered into evidence. *Id.* at 29-31. I permitted their admission over this objection, although I cautioned counsel that the evidence had to be properly authenticated once filed. Petitioner's counsel filed the photographs on October 31, 2018 (roughly one month following the hearing), but has not authenticated them. *See* Exs. 73-74. The photos are also undated.

[9] At hearing, Mrs. McKown testified that she initially declined to have her daughter vaccinated against HPV, due to her age. Tr. at 35. At eleven years old, she felt Petitioner was not highly at risk for HPV, especially given her personal circumstances (i.e., she was homeschooled and not sexually active). *Id.*

Petitioner subsequently developed new allergies, dramatic exacerbation of her skin condition (including hives and oozing sores that would cover her body), and joint pain after the second HPV dose. Tr. at 18-19, 27. Her migraines became more intense, and there were days when her leg pain was so severe she could not get out of bed. *Id.* at 19.[10] Treaters tested her for multiple conditions including lupus. *Id.* at 27. Her syncopal episodes continued through the remainder of 2013. Mrs. McKown recalled an episode of syncope that required a trip to the emergency room at Clearview Medical Center in December 2013. *Id.* at 92-93. During this episode, Petitioner's pulse and oxygen levels dropped low enough to (in her words) trigger a "code blue" alert, although the source of this characterization of the incident was the phlebotomist responsible for drawing Petitioner's blood. *Id.* at 92-93.[11]

Mrs. McKown further posited that Petitioner's symptoms worsened in January 2014. Tr. at 28, 54. Her joint pain and fatigue escalated, she could not concentrate on her school work, and she had no energy and had trouble sleeping at night. *Id.* The pain triggered by her skin symptoms and joint pain required morphine treatment (which was ineffective). *Id.* She also needed assistance with various personal care tasks (including ambulating and bathing). *Id.* Petitioner would also wear long-sleeved clothing to hide the affected areas of skin and hide her face in photographs. *Id.* at 38-39, 54.

Next, Mrs. McKown recalled the cardiology appointment with Dr. Dooley that she attended with Petitioner in May 2013 (around the time she was tested for POTS). Tr. at 64-65. At that time, Petitioner had reported an episode of syncope lasting twenty-five minutes. *Id.* at 64-65. Mrs. McKown testified that these syncopal episodes occurred "dozens" of times. *Id.* at 65. During the episodes, Petitioner would experience feelings of cold, her eyes would roll back in her head, and at times she would convulse. *Id.* at 66. Mrs. McKown acknowledged that Dr. Dooley (along with Dr. Magill) recommend that Petitioner schedule an appointment with a neurologist. *Id.* at 67-68. But Mrs. McKown decided against a neurology consult because she believed POTS to be strictly a cardiac condition, fearful that it could be misdiagnosed as "epilepsy" and other conditions. *Id.* at 67.[12] She also did not want to subject her daughter to endless testing. *Id.* at 69-71.[13]

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[10] Notably, however, Respondent submitted evidence revealing that Ms. McKown participated in a 5K race on November 16, 2013. Tr. at 58.

[11] On cross, Respondent pointed out that Ms. McKown had submitted no records memorializing this allegedly drastic drop in her vitals apart from a hand-written note (filed as Ex. 20 and dated October 9, 2014) prepared by the phlebotomist responsible for the blood test that immediately triggered the purported reaction. Tr. at 92-93. The only other documentation in the medical record noting a significant change in vitals is Ex. 55 (her tilt table test results from May 2013). *Id.* at 93.

[12] Mrs. McKown also testified that she did not recall Dr. Dooley suggesting that Petitioner's POTS was *not* autoimmune in nature (despite the suggestion in the medical record to the contrary). Tr. at 68-69; *but see* Ex. 7 at 25.

[13] According to Mrs. McKown, Petitioner's treaters at the Cleveland Clinic also did not consider neurology testing to be necessary for further evaluation of her condition. Tr. at 68. She did, however, admit later in her testimony that she

As Mrs. McKown recalled, Petitioner's skin condition continued to worsen during June 2014. Tr. at 59-60. At this point, Mrs. McKown posited that her daughter was taking six to eight Zyrtec daily in attempts to combat the rashes and sores. *Id.* at 59-60. According to Mrs. McKown, Petitioner's treaters even suggested that she begin six weeks of chemotherapy (given the persistent nature of the symptoms) – which prompted her to seek out second opinions regarding the cause of her condition (and appropriate treatment). *Id.* at 24-25, 60. (There is no record support for this recollection, however).

In July 2014, Petitioner was examined by a rheumatologist, Dr. Rouster-Stevens, at Atlanta Children's for evaluation of her joint pain, and assessed with "amplified musculoskeletal pain syndrome." Tr. at 71-72. Lupus and other autoimmune tissue disease were both ruled out based on lab results. *Id.* at 74.[14] Mrs. McKown testified, however, that she could not recall the particular diagnostic opinion offered at this visit, remembering only that Dr. Rouster-Stevens advised Petitioner to "go back to immunology" (presumably given her skin condition and congruent allergy testing) or seek treatment at a pain clinic. *Id.* at 72. As she recalled, Petitioner's diagnosis often varied depending on what type of specialist evaluated her symptoms, leading her to feel as if the family was being shuffled from specialist to specialist with no resulting concrete explanation for her condition. *Id.* at 74-75.

Mrs. McKown also testified about the Cleveland Clinic evaluations, starting with rheumatologist Dr. Zeft in August 2014. Tr. at 76-77. Dr. Zeft diagnosed Petitioner with amplified musculoskeletal pain (consistent with Dr. Rouster-Stevens) for which he recommended specialized exercises and pain management. *Id.* at 76-78. Ultimately, Mrs. McKown acknowledged these recommendations, but decided against scheduling a pain clinic evaluation. *Id.* at 75. Petitioner did, however, participate in some exercises (such as yoga, for example) to help with her joint pain. *Id.* at 77.

Mrs. McKown next discussed Petitioner's appointment with allergist Dr. Paschall. Tr. at 78, 80. Records from this visit indicate that Dr. Paschall assessed Ms. McKown with "chronic idiopathic urticaria" and discussed the appropriate treatment protocol for the illness, ruling out autoimmune chronic urticaria. *Id.* at 84. Despite the record evidence, Mrs. McKown could not recall any treater opining that Petitioner had chronic urticaria. *Id.* at 78. She also could not remember if her daughter took any of the medications prescribed for such a condition, though she

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requested a neurologic evaluation at the Cleveland Clinic (along with testing relating to aluminum levels in the blood), but was rebuffed. *Id.* at 69-71.

[14] Despite the lack of testing evidencing typical autoimmunity markers, Mrs. McKown maintained that Petitioner's treaters informed her that her elevated ANA levels indicated she had some form of autoimmune disease. Tr. at 74 ("I was told that a positive ANA means there is some type of autoimmune somewhere").

allowed for the possibility (given the multitude of treatments that were recommended to treat her symptoms). *Id.* at 79-80. She also admitted that Petitioner had tried multiple topical steroid creams (along with oral steroids) intermittently in the past to assist with her symptoms. *Id.* at 59-60, 83-84. She could not recall if Petitioner was currently using any ointments at the time of her Cleveland Clinic evaluation. *Id.*

According to Mrs. McKown, it was at the Cleveland Clinic that Petitioner's treaters first proposed a connection between the HPV doses she received and her onset of POTS (despite record evidence to the contrary). Tr. at 22, 24-25.[15] Later on in her testimony, Mrs. McKown acknowledged that she and she daughter learned of the term "Gardasil syndrome" through online research, and that they began to discuss the concept with treaters – although, as the medical record reveals, the idea was generally not embraced. *Id.* at 31-32, 89. Two treaters in particular – Dr. Rice (Ms. McKown's dermatologist at Emory) and Dr. Freedle – refused to treat Petitioner further after the suggestion was made that her HPV doses had resulted in her condition. *Id.* at 31-32, 85, 87-88. When confronted with medical records evidencing treater opinions that the HPV vaccine was likely *not* the cause for her condition, Mrs. McKown posited that these treaters were simply unwilling to admit the possibility that the vaccine could have caused Petitioner's symptoms. *Id.* at 85, 86-88.

Mrs. McKown also testified about Petitioner's treatment with Dr. DeMio. Tr. at 32, 85. She indicated that Dr. DeMio was one of the only physicians who shared her suspicion that her daughter's symptoms were caused by the HPV vaccine. *Id.* at 85-86, 90.[16] He started Petitioner on a "recovery protocol" which included the paleo diet, antibiotics, and various supplements, and which Mrs. McKown alleged had resulted in a "slow improvement" of Petitioner's symptoms. *Id.* at 32, 85. Over a period of months thereafter, Petitioner's skin cleared (though Mrs. McKown testified that she continues to experience flares presently). *Id.* at 33. Her syncopal episodes were controlled (i.e., she experienced only one every few months or so). *Id.* She was also able to compete in 5K races by April of that year. *Id.* at 58. As of 2018, Petitioner was successfully attending college, although she purportedly continues to experience syncopal episodes three to four times per week that can last up to thirty minutes. *Id.* at 33. Stress continues to trigger flare-ups of her skin condition and her headaches continue to be severe. *Id.* at 33-34.

[15] Notes from the Cleveland Clinic evaluations actually suggest that Ms. McKown was able to control her POTS symptoms through behavior modifications (including physical movements like squeezing her hands or completing exercises to help with blood flow). Tr. at 90-91.

[16] At hearing, Mrs. McKown also testified that one of Petitioner's pediatricians at Lawrenceville Pediatrics, "Tammie," allowed for the possibility that the HPV vaccine caused her condition, but could not recall this treater's full name. Tr. at 87-88. Respondent pointed out that at the time of the hearing, Petitioner had submitted no records evidencing an evaluation from a physician with that first name. *Id.* Given the discrepancy, counsel indicated that Petitioner would filed update records following the conclusion of the hearing. *Id.* at 88-89. However, no additional information has been filed.

2. Photographic and Social Media Evidence

To illustrate her contentions about the course of Petitioner's skin-related symptoms, Mrs. McKown offered photographs purportedly taken on various dates ranging from 2013 through 2015, many of which had been posted to social media. Tr. at 21-26; *see* Ex. 74. As she described, one photo was taken close-in-time to Ms. McKown's receipt of the second dose of HPV (in September 2013), and showed redness and hives scattered over the arms and trunk following exposure to water. Tr. at 26; *see* Ex. 74 at 9. Multiple filed photos were taken around December 2013 during the Cleveland Clinic consultations (and showed inflamed patches of skin on the face and neck). Tr. at 22-23; *see* Ex. 74 at 3-8. Yet another photo from (mid-April or May 2015) evidenced a skin eruption after spending time in the sun. Tr. at 21; *see* Ex. 74 at 1.[17]

In reaction, Respondent produced additional photographic evidence (spanning the length of the relevant time period), all of which tended to show that Petitioner's skin appeared to be relatively clear at various points throughout 2013 through 2018. Tr. at 41-52; *see* Exs. P and Q.[18] These photographs were taken from Ms. McKown's Facebook account, with timestamps revealing that they were posted on various dates between April 2013 and 2018 (although this obviously does not establish when a particular photo was actually taken). For example, in a photo posted in mid-November 2013 (or two months following Petitioner's receipt of the second dose of HPV), Ms. McKown's skin appears to be clear and she was not wearing any long-sleeved or high-necked clothing to cover her skin. Tr. at 41-42. Mrs. McKown explained, however, that her daughter would routinely repost photos to Facebook that were taken pre-vaccination (and likely did so in November of that year). *Id.* at 41-42. Three photos taken in August 2014 and January 2015 also appear to show Petitioner wearing short-sleeved clothing (with no noticeable skin rashes on her face or arms). *Id.* at 42-43. In response, Mrs. McKown testified that Petitioner often edited photos she posted to social media during her illness in order to remove any evidence of a rash (a practice Mrs. McKown witnessed her doing throughout 2014). *Id.* at 43-44, 93-94.

Respondent also offered photos posted on various dates in late 2015 through early 2018, at which time Petitioner's skin symptoms began to subside. Tr. at 45-48. A photo dated from April 2018 depicted Ms. McKown on the beach with family members. *Id.* at 49. Her skin appears to be

[17] As previously stated, the photographs (filed as Ex. 74) were not filed prior to hearing, although Petitioner's counsel indicated that they would be filed following the hearing (with evidence confirming the dates they were taken). Counsel did so, but did not offer evidence to confirm the dates upon which they were taken. *See generally* Ex. 74. I can thus only rely on the testimony offered by Mrs. McKown (which tends to suggest they were taken following Petitioner's receipt of the second dose of HPV in September 2013 through mid-2015). I largely do not, however, find that the photos do more than illustrate vaguely the existence of eczema flares in this period – a contention not fully disputed, and one that even if deemed correct does not aid Petitioner in establishing that the HPV vaccine caused the flares.

[18] Respondent also filed evidence that Petitioner participated in 5K races in November 2013 and April 2015. *See* Ex. R.

clear in the photo, and Mrs. McKown admitted at hearing that the sores her daughter had experienced previously were no longer a problem at this time. *Id.* She did, however, maintain that the photograph showed evidence of a rash or "redness" on her hands and back. *Id.* Overall, after late 2015, Mrs. McKown posited that Petitioner would have "good days" where her skin remained clear, but she continued to experience breakouts on and off for the last three years. *Id.* at 50-54.

3. Dr. Carlo Tornatore

Dr. Tornatore authored one expert report and also testified at the entitlement hearing on Petitioner's behalf. *See* Ex. 56, filed on July 31, 2018 (ECF No. 42-2) ("Tornatore Rep."). He opined that Petitioner developed POTS (along with a significant aggravation of preexisting eczema) following her receipt of HPV vaccine doses on March 20, 2013, and September 3, 2013. Tr. at 120-21, 135; Tornatore Rep. at 14. In the alternative, if Petitioner's POTS preexisted her vaccinations, then the vaccines likely significantly aggravated that condition. Tr. at 135; Tornatore Rep. at 14.[19]

Dr. Tornatore is a board-certified neurologist. *See* Exhibit 57, dated July 31, 2018 (ECF No. 42-3) ("Tornatore CV"). He graduated from Cornell University with a Bachelor of Arts in Neurobiology, and attended Georgetown University, where he received a Master of Science in Physiology. Tr. at 98; Tornatore CV at 2. He subsequently graduated from medical school at Georgetown University School of Medicine, completing a residency in the Department of Neurology at Georgetown University Hospital. Tr. at 98; Tornatore CV at 2. He also completed a fellowship in molecular virology and genetics at the National Institute of Health in Bethesda, Maryland. Tr. at 98; Tornatore CV at 2. Dr. Tornatore has published multiple articles addressing cell biology and pathology of demyelinating disorders. Tornatore CV at 8-20. Currently, he serves as the Chair of the Department of Neurology at Georgetown University Medical Center, and Interim Chair of the Department of Neurology at Medstar Georgetown University Hospital. Tr. at 98-99; Tornatore CV at 3. He also serves as a director of the neurology clerkship program at Georgetown University Hospital (which is responsible for training roughly 200 medical students per year). Tr. at 99; Tornatore CV at 3. During his tenure at Georgetown, Dr. Tornatore also developed the Neuroimmunology and Multiple Sclerosis Center. Tr. at 99; Tornatore CV at 3. He also serves as an ad hoc reviewer for several neurology journals. Tr. at 102; Tornatore CV at 7.

Dr. Tornatore is not board certified in immunology or dermatology. Tr. at 133, 139. His CV also does not evidence any subspecialty in autonomics (or reference any publications or memberships associated with such a specialty). *Id.* at 133-34, 135. In his current practice, he primarily treats multiple sclerosis ("MS") patients (some of whom have congruent autonomic

[19] Upon questioning by the Court, Dr. Tornatore stated that his opinions herein (along with those contained in his expert reports) were wholly independent from those offered by Drs. Mikovits and Ruscetti. Tr. at 188.

issues). *Id.* at 100, 103. Dr. Tornatore estimated that roughly 600 of his MS patients have POTS (attributable to various primary illnesses, including deconditioning, cardiovascular issues, or autonomic problems). *Id.* at 137-38. He acknowledged, however, that he is not an expert in POTS or the autonomic nervous system. *Id.* at 137-38. He also has not performed any testing related to POTS (i.e., a title table test), but does have some familiarity with orthostatic reading measures. *Id.* at 138. In addition, Dr. Tornatore has not treated any MS/POTS patients with dermatologic problems similar to those describe herein. *Id.* at 103, 136-37, 140.

Dr. Tornatore characterized POTS as dysfunction of the autonomic nervous system[20] resulting from dysregulation of the blood vessels responsible for adjusting the heart rate when the body changes position. Tr. at 103. Patients suffering from POTS typically experience increased heart rate, accompanied by a drop in blood pressure, due to the heart's attempt to compensate for lack of blood supply to the brain (which can result in dizziness when moving from a lying down position to a standing one, for example – as evidenced by tilt table testing). *Id.* POTS can also present secondarily to central nervous system disorders (like MS, for example). *Id.* Other symptoms can include lightheadedness, syncope, and dizziness (something Dr. Tornatore deemed characteristic of the disease, but not evidence of it). *Id.* at 156-58. The filed literature suggests several different triggers can cause an individual to develop POTS, including viruses, bacteria, deconditioning, and genetics. *See* C. Gibbons, et al., *Structural and Functional Small Fiber Abnormalities in the Neuropathic Postural Tachycardia Syndrome*, PlosOne (2013), https://doi.org/10.1371/journal.pone/0084716, filed as Ex. 61 (ECF No. 47-5) ("Gibbons").

In addition, Dr. Tornatore proposed that POTS can be autoimmune in origin. Tr. at 103-04. An autoimmune disease or disorder features an individual's immune system reacting hyperactively, attacking self antigens (at the same time it is reacting to foreign infectious agents). Dr. Tornatore relied on the presence of certain autoantibodies in patients diagnosed with POTS as evidence of its autoimmune nature, offering literature (including that authored by Respondent's expert, Dr. Gibbons) that he said established this finding. *See generally* Gibbons at 1-10. The Gibbons paper analyzed twenty-four POTS patients (along with ten healthy controls) to define the neuropathology and clinical parameters of neuropathic POTS (as compared to the non-neuropathic variants). Gibbons at 1. While Gibbons concluded that various subtypes of POTS display overlapping symptoms, neuropathic POTS patients (experiencing "sudomotor dysfunction") could also exhibit symptoms similar to those seen in post-ganglionic dysautonomia. *Id.* at 6-7. Based on the above, Dr. Tornatore concluded that there is a specific target antigen associated with neuropathic POTS) – the "postganglionic proteins" or "ganglia" (which have shown to be virally-associated with "postganglionic cholinergic dysautonomia"). Tr. at 122-23.

[20] Dr. Tornatore defined the autonomic nervous system as the primary regulator of involuntary physiologic processes (i.e., sweating, bowels, bladder, and respiratory functions). Tr. at 103.

In Dr. Tornatore's opinion, POTS can also be associated with the onset of various skin eruptions (including eczema and atopic dermatitis). Tr. at 119-20, 181. Dysfunction in the autonomic nervous system can, he proposed, also cause dysregulation (or inflammation) in the somatosensory nerves in the skin. *Id.* at 117-18, 119. Specifically, neural signaling by the sensory nerves (or "neuroimmune signaling") can produce neurogenic inflammation, thereby resulting in the nervous system playing an "active role in inflammation" – attacking the skin directly, and leading to eczema and atopic dermatitis. *Id.* at 118-19. He claimed that Ms. McKown's skin biopsy from April 2014 (which evidenced markers for inflammation) supported his conclusion. *Id.* at 152-53.

On cross, Dr. Tornatore admitted that he had offered no literature directly supporting his proposition that eczema could be autoimmune in derivation instigated by vaccination. Tr. at 120-21, 153-54. He nonetheless emphasized that it was reasonable to associate a skin eruption (or outbreak) with other evidence of autoimmune disease, given that both occur congruently with an underlying inflammatory process. *Id.* at 124, 154-55. In support, Dr. Tornatore referenced a single piece of literature, which he posited shows that eczema can be induced by certain antibodies. *Id.* at 153; *see* T. Voisin, et al., *Neuro-Immune Interactions in Allergic Diseases: Novel Targets for Therapeutics*, 29 Int'l Immnol. 247 (2017), filed as Ex. 72 (ECF No. 55-4) ("Voisin").[21] Voisin, however, discussed *allergic* inflammation and the interplay between immune cells/inflammatory mediators as "neurotransmitters" or signalers (which, by way of "cross-talk," mediate the immune response to allergens). Voisin at 1. It also made no mention of POTS (nor does it attempt to relate autoimmune disease to allergies, or connect POTS to eczema). Dr. Tornatore otherwise offered no evidence that the autoantibodies he discussed as associated with a neuropathic form of POTS could also be implicated in causing eczema. Tr. at 153-54.

Dr. Tornatore went on to discuss what Petitioner's actual medical records revealed, in an effort to bulwark the reasonableness of his theory. First, he noted that Petitioner's tilt table testing (completed in May 2013) confirmed she suffers from POTS. Tr. at 169-70, 180. Her subsequent episodes of prolonged unconsciousness[22] (i.e., anywhere from ten to thirty minutes) thereafter were also in his view likely related. *Id.* at 165-67, 168-70. Dr. Tornatore could not confirm, however, which POTS variant best described Petitioner's specific symptoms. *Id.* He theorized that Ms. McKown likely had a form of autoimmune "neurogenic" or "neuropathic" POTS (despite any treater evidence so opining). *Id.* at 180-81. In support of this determination, Dr. Tornatore pointed to Petitioner's worsening eczema flares (in conjunction with accompanying "skin color changes") as evidence of this finding. *Id.* at 181. He maintained as well that the Voisin article supported his

[21] Voisin was filed the day before the hearing. ECF No. 55 (confirming Voisin was filed on September 25, 2018).

[22] The fact that one of these prolonged episodes was observed only by a phlebotomist did not alter Dr. Tornatore's opinion. Tr. at 165-68. Respondent also noted that these observed episodes were not contemporaneously recorded in the record (but rather were dictated in letters thereafter or described by Petitioner's mother only). *Id.* at 165-68.

opinion (given the association of neuroimmune signaling with the pathophysiology of allergic diseases, such as forms of eczema). *Id.*

On cross, Dr. Tornatore also posited that "acrocyanosis" – which he described as bluish-colored limbs – is a "hallmark" of neurogenic POTS. Tr. at 549-50. As he explained, bluish coloring in the limbs is evidence of some "neurogenic issue" by which the nerves compress the capillaries. *Id.* at 549-50. Dr. Tornatore pointed to an instance in the record where one of Petitioner's treaters noted such an occurrence. *Id.* at 550 (citing Ex. 21 at 1), 556. In so maintaining, he again invoked Voisin, which discussed a 1901 study revealing that nerve stimulation could result in "vasodilation" (or a change in limb color). *Id.* at 554; Voisin at 3. As discussed earlier, however, Voisin makes no mention of POTS at all (and it thus does not consider whether limb discoloration is a hallmark of neuropathic POTS). Nonetheless, Dr. Tornatore maintained that evidence of nerve stimulation (and the resulting bluish coloring or "flushing" of the limbs noted in the medical records) was enough to conclude the best diagnosis was likely neurogenic/neuropathic POTS. Tr. at 556.[23]

In reaction to Respondent's argument that Petitioner's POTS was more likely attributable to "hypovolemia" or dehydration, Dr. Tornatore referenced various lab reports in Petitioner's records tending to suggest that the relevant markers for hypovolemia were documented as normal over the course of her illness. Tr. at 556-59. Hypovolemia (or dehydration caused by low blood volume), he posited, is best evidenced by an elevated BUN[24] to creatinine measurement[25] (i.e., evidence of blood volume depletion). *Id.* at 559. But, based on his own understanding of the science, Dr. Tornatore suggested that low levels of creatinine could "falsely elevate" the BUN-creatinine ration (due to diet or GI issues) – thereby rendering the marker somewhat unreliable at times as a diagnostic tool. *Id.* at 558-59. Even so, absent any irregular BUN-creatinine ratio, a diagnosis of chronic dehydration (associated with POTS) could not be reliably supported in his reading of the record. *Id.* at 559-60.

Dr. Tornatore next maintained there was a plausible biologic mechanism by which the HPV vaccine could cause POTS: molecular mimicry. Tornatore Rep. at 13-14. Molecular mimicry

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[23] Apart from the above, Dr. Tornatore posited that Respondent's expert, Dr. Gibbons, offered some evidence that a "lack of sympathetic tone" in the limbs could result in blood vessel constriction and pooling – thereby causing some discoloration similar to that noted above. Tr. at 556-57. He could not, however, point to any literature cited by Respondent relating this specific symptom to POTS. *Id.* at 557-58.

[24] A BUN (or "blood urea nitrogen") test is a blood test used to measure the amount of urea nitrogen in the blood. *See Blood Urea Nitrogen (BUN) Test*, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/blood-urea-nitrogen/about/pac-20384821 (last accessed on June 19, 2019). Urea nitrogen is a chemical waste product that is typically removed from the body through the kidneys. *Id.* A higher than normal BUN test can suggest that the kidneys or liver may not be working properly. *Id.*

[25] Creatinine is a chemical waste product produced by muscle metabolism. *See Creatine Test*, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/creatinine-test/about/pac-20384646 (last accessed on June 19, 2019). Properly functioning kidneys filter creatine from the blood. *Id.* A creatine test – which measures the level of creatinine in the blood – can thus indicate kidney irregularities. *Id.*

occurs when the body is exposed to an environmental factor (such as a vaccine or infection) which results in a cross-reaction between autoantibodies (produced by the body in response to that external factor) and a self structure in the body that the foreign antigen has mimicked. In so proposing, Dr. Tornatore relied on scientific literature establishing homology between protein components of the HPV vaccine and certain human protein structures. Tr. at 170-71, 547-49; D. Kanduc & Y. Shoenfeld, *Inter-Pathogen Peptide Sharing and the Original Antigenic Sin: Solving Paradox*, 8 Open Immunol. J. 16 (2018), filed as Ex. 69 (ECF No. 53-3) ("Kanduc").[26] Kanduc examined a nine-protein sequence from HPV16, and recorded amino acid sequence similarities to the human proteome at the pentapeptide level, concluding that the proteome contains twenty-nine pentapeptides also found in the HPV16 vaccine – and thus ample mimics for molecular mimicry to have occurred. Kanduc at 16.

Dr. Tornatore struggled, however, to specify *where* in the body this autoimmune cross-reaction was purportedly occurring. At most, he proposed that the immune response triggered by receipt of the HPV vaccine caused a cross reaction directed at the body's peripheral nerves (or some "neuronal element" or epitope). Tr. at 120-21, 171. He also made some suggestion that Kanduc supported a conclusion that the triggered autoimmune response could be directed against the human septin-9 protein. *Id.* at 171; Kanduc at 21. He did not, however, offer literature evidencing homology between the HPV vaccine components and any specific tissues in the autonomic nervous system. Tr. at 172. And Kanduc referenced no studies or models indicating such a cross reaction could occur (in the context of either target). Dr. Tornatore nonetheless admitted at hearing that he relied solely on Kanduc to establish that components of the HPV vaccine could interact with septin-9 (or some other "crossreactive target") and result in POTS (though, he posited that he could offer more support if given additional time).[27] *Id.* at 171-72, 173, 548; *see* Kanduc at 21. He maintained, however, the Kanduc paper provided enough support to conclude such a similarities existed (given the Program's "more probable than not" preponderance standard). Tr. at 172.[28]

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[26] On cross, Respondent raised concerns regarding the reliability of Kanduc (given that it was published in the *Open Immunology Journal*, which Respondent proposed was a disreputable, "predatory" journal). Tr. at 174. Dr. Tornatore, however, posited that the circumstances of Kanduc's publication (or the reputation of the authors) did not weigh heavily on his assessment of the findings discussed in the article. *Id.* at 175. In his view, the authors simply performed proteomic searchers (which he deemed "totally objective"), confirming the possibility of homology between the HPV vaccine components and self structures. *Id.* at 175-76.

[27] Kanduc, like Voisin, was filed the night before the hearing. *See* ECF No. 53 (confirming Kanduc was filed on September 25, 2018).

[28] In so stating, Dr. Tornatore stepped outside of his demonstrated medical expertise and into the shoes of the person designated to determine the law in this proceeding – here, the special master. But it is inadvisable for medical or scientific experts to so comment on the relevant legal standard, as I have noted in other cases. *See, e.g.*, *Chinea v. Sec'y of Health & Human Servs.*, No. 15-95V, 2019 WL 1873322, at *19, 31 n.42 (Fed. Cl. Spec. Mstr. Mar. 15, 2019), *appeal docketed*, No. 15-95V (Fed. Cl. Apr. 15, 2019).

This inability to establish more than vague contours of how homology between the HPV vaccine and self proteins might explain a putative autoimmune attack went beyond this narrow issue, however. Dr. Tornatore offered little in the way of evidence suggesting that the HPV vaccine has ever been shown from a reliable experiment or study to be pathogenic in the manner proposed (i.e., to trigger an autoimmune process sufficient to produce the symptoms relevant to this case). Rather, he relied solely on the fact that Kanduc supported a finding of "human homology" between HPV vaccine protein components and host proteins – without evidence suggesting the alleged cross-reactivity could result in an adverse disease process. At most, Dr. Tornatore referenced Sydenham's chorea (a known autoimmune disease characterized by rapid, jerking movements) to explain how an immune response directed against a bacterial infection can "overshoot" and also lead to different illnesses (rheumatic heart disease, for example). Tr. at 110. As he described, in the case of Sydenham's chorea, the body develops an immune response to strep bacteria – and the autoantibodies produced in response cross-react with host proteins – thus, causing direct damage to the heart and brain. *Id.*

Along those same lines, and as another mechanistic explanation in the alternative to molecular mimicry, Dr. Tornatore briefly discussed the concept of "T cell receptor degeneracy." Tr. at 105-07, 189-90; Tornatore Rep. at 14. According to Dr. Tornatore, the body lacks an "army of T cells" capable of defending against the "millions, if not billions" of antigens it is exposed to on a regular basis. *Id.* Thus, it would be impossible for one specific T cell to be directed at one single antigen. *Id.* at 105, 566. Rather, T cells are "degenerate" – meaning they can recognize several different antigens (and mount an immune response against those antigens) without having to have identifiable homology to every possible foreign antigen, including amino acid chains whether associated with a particular virus or vaccine. *Id.* at 105, 546.

Dr. Tornatore characterized the degeneracy concept as either a primary or secondary adaptive response, but distinguished it from the more typical underlying mechanisms offered in the Program (i.e., bystander activation and epitope spreading). Tr. at 189. In his view, T cell degeneracy involves antigen stimulation the of the "same identical T cell clone[,]" rather than by acceleration of an ongoing immune process by local activation of antigens presenting as a result of the cross-reactivity. *Id.* But he stressed that because Kanduc established that the HPV vaccine has specific homology with self-protein sequences, T cell degeneracy was not a necessary mechanistic explanation for his theory in this case. *Id.* at 189 ("we have that specificity for HPV protein sequence and human sequences to say . . . you don't need the T cell degeneracy"), 546. Though, upon further questioning, Dr. Tornatore posited it was possible that both had occurred in this case – protein cross-reactivity led to the onset of POTS, while T cell degeneracy contributed to the exacerbation of Ms. McKown's skin symptoms (in which case it is possible the eczema worsening was either directly aggravated by the nervous system or stimulated by the underlying adaptive process attributable to POTS). *Id.* at 547.

Dr. Tornatore further suggested that Petitioner's subsequent re-exposure to antigens in the second dose of the HPV vaccine could have played some role in the molecular mimicry process.

Tr. at 107-09. Because of the homology identified between components in the HPV vaccine and self sequences, nonspecific T cells could attack self structures/amino acid sequences (following immune system stimulation by the vaccine) in addition to those cells responding specifically to the vaccine – thereby resulting in a more amplified and inherently more rapid cellular response. *Id.* at 107-08. Just as a vaccine booster response is intended to assist the body in developing immunologic memory to certain antigens, Dr. Tornatore posited that memory developed from exposure to a second dose of HPV could cause the cross-reactivity to occur at a much faster pace. *Id.* at 108-09; *see* Kanduc at 21.

Admittedly, Dr. Tornatore conceded, Kanduc offered multiple possible sequence mimics that could share homology with numerous self proteins in the entire human genome. Tr. at 112, 552. To account for such a large amount of protein sequence similarities, Dr. Tornatore posited that genetic risk factors also play a part in how the immune system "overshoot[s]" or reacts in response to vaccine-induced autoantibodies. *Id.* at 112-13. Dr. Tornatore offered rheumatologic disorders as a pertinent illustration. *Id.* Patients with spondyloarthropathies, for example, have elevated levels of HLAB-27 (an abnormal haplotype protein located on the immune cells). *Id.* These proteins (produced as a result of a genetic rheumatologic disorder) inhibit the body's ability to regulate the immune system – thereby resulting in an increased risk for autoimmunity. *Id.* at 113. Thus, regardless of the number of possible shared sequences, Dr. Tornatore seemed to suggest that a patient's genetics will always play *some* role in susceptibility to develop an autoimmune disorder. *Id.* at 190-91, 552-53. He did not indicate, however, what (if any) genetic abnormality contributed to Petitioner's onset of symptoms. *Id.* at 552.

Dr. Tornatore next discussed the evidence (or "combination of things") in the medical records that he maintained supported the conclusion that Petitioner likely experienced chronic inflammation (and resulting immune dysfunction) post-vaccination. Tr. at 127, 179-80. First, Dr. Tornatore pointed to Petitioner's POTS – a disease which (as discussed above) "can be autoimmune" in some circumstances. *Id.* at 177, 180. He also asserted that eczema "is inflammatory in nature" which further evidenced some systemic involvement. *Id.* at 177. Indeed, Petitioner's biopsy from April 2014 revealed inflammation in the skin. *Id.* at 153, 178, 180. Otherwise, Dr. Tornatore referenced allergy testing – which revealed elevated levels of IgE to various food and outdoor allergens. *Id.* at 179 (citing Ex. 1 at 65-66), 180. Even so, Dr. Tornatore admitted on cross that elevated IgE is *not* specific to autoimmunity directly, but could suggest a patient has a "propensity for a hyperimmune state." *Id.* at 179. He also agreed that Petitioner's records contained no test results establishing the existence of any specific antibodies to neuronal antigens – though he deemed such testing insignificant. *Id.*

Apart from the above, Dr. Tornatore concluded that the HPV vaccine doses Petitioner received were likely responsible for instigating her condition given their close temporal relationship to her reported onset (and/or worsening) of syncope and rash symptoms. Tr. at 125, 127, 177, 553. In his view, Petitioner's health course and the "tempo" of her symptoms "changed dramatically after vaccination, particularly the second vaccination in September." *Id.* at 125. She

at most experienced situational syncope before her receipt of the HPV vaccine doses, with worse and longer instances thereafter. *Id.* at 125-26. Her preexisting eczema also worsened following her second dose of HPV (as evidenced by the multiple doctor visits she attended over the course of her illness). *Id.* at 125-27. Dr. Tornatore also cited to various treater statements indicating their recognition that Petitioner's symptoms had worsened in the months following her vaccine doses. *Id.* at 125 (citing Ex. 1 at 49), 129 (citing Ex. 3 at 2-4).

While stressing the significance of the above, Dr. Tornatore was dismissive of the fact that testing performed on Petitioner did not reveal other common, objective indicators of systemic inflammation (including the CRP[29] and ESR[30] rates). Tr. at 110-11. In his view, such measurements are not always elevated in autoimmune disease patients because not all immune responses result in protein production detectable in the blood. *Id.* at 110-11. In multiple sclerosis and myasthenia gravis, for example, inflammation is directed at the nervous system tissue – and the CRP and ESR testing typically reveal normal results for its patient population (given that antibodies produced in response to such immune-mediated conditions are deposited in the tissue, *not* the blood). *Id.* at 111-12.[31] In addition, Petitioner experienced worsening eczema at the same time that her CRP and ESR rates were normal (a point conceded by Respondent's expert). *Id.* at 111. Thus, as Dr. Tornatore posited, the underlying pathogenesis of an autoimmune disease process could not be discredited in this case even if some classic tests for inflammation had not been satisfied. *Id.* at 177-78.

Dr. Tornatore also acknowledged that Ms. McKown's medical records evidenced a pre-vaccination history of two syncopal episodes – but he deemed them "situational" or "vasovagal" in nature, and thus distinguishable. Tr. at 128, 161-63; *see also* Tornatore Rep. at 14. In his view, both instances of past syncope were accompanied by a "significant emotional" component (i.e., a reprimand for bad behavior and exposure to blood). Tr. at 162. They were therefore not likely associated with her subsequent autoimmune-instigated POTS, given the above-noted situational components, the rarity of such occurrences, and lack of other evidence suggest a preexisting underlying autonomic problem. *Id.* at 128, 162, 163 (noting "these episodes were 2 ½ years apart"). By contrast, Ms. McKown clearly experienced more syncopal episodes following her receipt of two HPV vaccine doses. *Id.* at 129, 162-63. However, if her pre-vaccine syncopal episodes *could*

[29] CRP is a test used to measure inflammation in the body. *C-Reactive Protein Test*, Mayo Clinic, https://www.mayoclinic.org/tests-procedures/c-reactive-protein-test/about/pac-20385228 (last accessed June 20, 2019). It measures the amount of C-reactive protein in the blood via a simple blood test. *Id.* The results can indicate a patient's risk for infection or heart disease, for example. *Id.*

[30] ESR is a blood test used to show inflammatory activity in the body. *Sed Rate (Erythrocyte Sedimentation Rate)*, Mayo Clinic, https://www mayoclinic.org/tests-procedures/sed-rate/about/pac-20384797 (last accessed June 20, 2019). It measures the distance red blood cells fall in a test tube in one hour. *Id.* The further the cells descend in the tube, the greater evidence of an existing inflammatory response of the immune system. *Id.*

[31] For further support, Dr. Tornatore posited that the best indicator of a multiple sclerosis diagnosis is an MRI. Tr. at 179. Bloodwork for multiple sclerosis patients is typically normal and does not reveal the presence of systemic inflammation. *Id.*

be related to POTS, then Dr. Tornatore proposed that the HPV vaccine doses likely aggravated it given the increased incidence of post-vaccination symptoms. *Id.* at 128-29, 161-62; Tornatore Rep. at 14.

As to the timing of the onset of Petitioner's POTS symptoms, Dr. Tornatore maintained that she had experienced her first "true" symptoms of syncope on April 26, 2013 (or four to five weeks post vaccination) at the yogurt shop, and that such timing was medically reasonable. Tr. at 155, 164; Tornatore Rep. at 14. For support, he relied solely on a single epidemiologic study – involving a totally different disease. *See* L. Schonberger, et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. Epidem. 105 (1979), filed as Ex. 60 (ECF No. 47-4) ("Schonberger"). Schonberger's authors studied the incidence of onset of GBS following receipt of the swine flu vaccine, concluding that such an increase had occurred mostly within a five-week period thereafter (although it was possible the risk interval could extend nine to ten weeks). Schonberger at 110.

By contrast, Dr. Tornatore acknowledged outright that Petitioner's eczema most likely began pre-vaccination. Tr. at 142. But he felt the symptoms she experienced in this time period were less recurrent or "episodic" – with the "tempo" of incidents increasing after the second HPV vaccine dose in September 2013. *Id.* at 149-50, 151. Despite record evidence suggesting that Petitioner's eczema had actually been deemed "recurrent" or "severe" in the past, Dr. Tornatore maintained that records from close-in-time to her receipt of the vaccine clearly described an exacerbation of symptoms. *Id.* at 144-45, 146, 148-49. Dr. Tornatore also pointed to instances in the record where other treaters categorized Petitioner's skin condition as "coming and going" (or at times normal). *Id.* at 146-48. Ultimately, Dr. Tornatore could not pinpoint the exact day and time Ms. McKown's skin symptoms worsened, but maintained that the skin rashes progressively worsened over a six-month period (beginning in June or July 2013 and later resulting in an exacerbation or flare-up in September). *Id.* at 151.

For support on the reasonableness of the eczema exacerbation timeframe, Dr. Tornatore again referenced Schonberger. Tr. at 116-17. He posited that literature on the topic of ADEM also supported a conclusion that autoimmune diseases can occur between forty-eight hours and thirty days following vaccine administration (though, he offered no literature directly addressing eczema in this context, or why literature pertaining to GBS or some other clearly neuropathic condition could also be applied to eczema). *Id.* at 117. Based on Schonberger, Dr. Tornatore also explained that an initial immune response mediated by T cells could be quick (especially in the context of an anamnestic response), but could also linger for a period of two months or even longer. *Id.* at 116. Given the amount of time it takes to develop immune memory, Dr. Tornatore posited that an autoimmune response would occur in a similar timeframe (i.e., within a sixty-day period or earlier if the patient has previous exposure to the same vaccine antigens). *Id.* at 116-17.

2. Drs. Judy Mikovits and Francis Ruscetti

Drs. Mikovits and Ruscetti[32] prepared two reports in support of Petitioner's claim. *See* Expert Report, dated Dec. 2, 2016, filed as Ex. 25 (ECF No. 18-2) ("First Mikovits Rep."); Expert Report, dated July 19, 2017, filed as Ex. 51 (ECF No. 30-2) ("Second Mikovits Rep."). Only Dr. Mikovits testified at hearing, however.[33] Dr. Mikovits offered the opinion that the two doses of HPV, in conjunction with the Hep A vaccine, caused Petitioner to develop POTS and atopic dermatitis. Contrary to Dr. Tornatore, however, Dr. Mikovits posited that Ms. McKown's medical records did not offer persuasive evidence that she indeed suffered from recurrent skin rashes (characterized as atopic dermatitis) prior to her receipt of the above-noted vaccines – thus, her opinion does not appear to implicate a significant aggravation theory (at least as it applies to Petitioner's skin condition).

Dr. Mikovits is a consultant with MAR Consulting Inc., a group she co-founded, and serves as an advisor for a private equity investment company. Curriculum Vitae, filed as Ex. 26 (ECF No. 18-3) ("Mikovits CV") at 1. She received her undergraduate degree in biology from the University of Virginia, and a Ph.D. in molecular biology and biochemistry from George Washington University. *Id.* at 4. Dr. Mikovits did not attend medical school, however, and is not a licensed medical doctor. Tr. at 247. She thus has no direct experience treating skin diseases or autonomic disorders.

From 1992 to 1994, Dr. Mikovits was a post-doctoral fellow in molecular virology at the National Cancer Institute, Lab of Genomic Diversity, subsequently serving as a staff scientist at the National Cancer Institute, Lab of Leukocyte Biology, from 1994 to 1998. Mikovits CV at 1-2. Thereafter, from 1999 to 2001, she served as a Lab Director at the Laboratory of Antiviral Drug Mechanisms, a division of the National Cancer Institute. *Id.* at 3. Dr. Mikovits worked in various capacities at several biotechnology start-up companies from 2002 to 2006, and conducted research at the Whittemore Peterson Institute for Neuroimmune Disease ("Whittemore") from 2006 to 2011, studying diseases with inflammatory components and environmentally acquired immune

[32] At hearing, Dr. Mikovits testified that both she and Dr. Ruscetti separately reviewed the medical records and case filings prior to preparing their joint expert report. Tr. at 224. They assumed the factual assertions contained in Respondent's Rule 4(c) Report were correct. *Id.* at 225. She further stated that she and Dr. Ruscetti worked together to prepare the report, relying on their respective areas of expertise in substance (for Dr. Ruscetti: dendritic cells and the adaptive immune system; for Dr. Mikovits: mast cells and microglia involvement in disease pathology). *Id.*

[33] According to his CV, Dr. Ruscetti received his B.S. in biology from Boston University, followed by a Ph.D. in microbiology from the University of Pittsburgh. *See* Curriculum Vitae, filed as Ex. 27 (ECF No. 18-4) at 1. From 1972 to 1975, Dr. Ruscetti served as a research instructor at the University of Pittsburgh School of Medicine. *Id.* He worked for Litton Bionetics as a cell biologist from 1975 to 1978. *Id.* In 1978, he joined the National Cancer Institute, and presently serves as Principal Investigator for the Leukocyte Biology Section. *Id.* at 1-2. He is also an Adjunct Professor of Biochemistry and Molecular Biology at George Washington University. *Id.* at 2. Dr. Ruscetti serves on the editorial board of *Stem Cells*, and his CV lists multiple authored publications. *Id.* at 2-33.

deficiency. Mikovits CV at 2; Tr. at 221. She has not conducted research since 2012, but now works as a consultant. Mikovits CV at 1.

Significant and alarming elements of Dr. Mikovits's professional history were pointed out at hearing that greatly diminished her credibility as an expert. In particular, while at Whittemore Dr. Mikovits was accused of stealing laboratory materials, arrested, and fired from her position. Tr. at 218, 265-66. In addition, a paper Dr. Mikovits published in *Science* was later retracted because the results could not be replicated, causing the journal to lose confidence in the report and the validity of its conclusions. *Id.* at 216, 258-59. Dr. Mikovits nevertheless has continued to reference the article on her consulting website without disclosing its retraction. *Id.* at 265. Dr. Mikovits has also published works and given presentations which dispute the safety and value of vaccines, imply a link between vaccines and autism, and/or recommend a moratorium on vaccine administration generally. *Id.* at 250-51, 255, 256-58. She has collaborated with an anti-vaccination publisher and co-author, and regularly speaks at the "AutismOne" conference. *Id.* She has also supported a ban on all HPV vaccines and attacked the Vaccine Program as biased. *Id.* at 255.

Like Dr. Tornatore, Dr. Mikovits testified about Petitioner's claimed vaccine-caused diagnoses of POTS and atopic dermatitis, as well the possible causal connection between the HPV vaccine (and Hep A) and her disease onset. Although the opinions she offered regarding the vaccines at issue in the case and the appropriate causal mechanism differed from those offered by Dr. Tornatore, she similarly opined that Ms. McKown's onset of POTS and atopic dermatitis flares were caused by an autoimmune process (or reaction) that was likely vaccine-induced.

Dr. Mikovits began by discussing Petitioner's May 2013 POTS diagnosis. Although Dr. Mikovits has never treated POTS patients, she filed literature defining POTS as a heterogeneous disorder characterized by abnormal increments in heart rate upon assumption of the upright posture, accompanied by orthostatic intolerance and other secondary symptoms. *See, e.g.*, L. Brinth, et al., *Orthostatic Intolerance and Postural Tachycardia Syndrome as Suspected Adverse Effects of Vaccination Against Human Papilloma Virus*, 33 Vaccine 2602 (2015), filed as Ex. 42 (ECF No. 20-7) ("Brinth").[34] Though the cause of POTS is unknown, the onset of its symptoms can occur following febrile illness, pregnancy, immunization, sepsis, surgery, or trauma. Brinth at 2603; *see also* E. Benarroch, *Postural Tachycardia Syndrome: A Heterogenous and Multifactorial Disorder*, 87 Mayo Clin. Proc. 1214 (2012), filed as Ex. 28 (ECF No. 19-2) ("Benarroch") (50 percent of POTS cases have an antecedent viral infection, and 25 percent have prior familial

[34] Dr. Mikovits expert report stated – incorrectly – that lightheadedness and syncope were not associated with POTS. Second Mikovits Rep. at 2. She also cited literature positing the same. *See* S. Blitshteyn, *Postural Tachycardia Syndrome Is Not Caused by Deconditioning*, 6 Pulm. Circ. 401 (2016), filed as Ex. 66 (ECF No.49-4) (letter to the editor). At hearing, she corrected herself and stated she should have said those symptoms were simply not "diagnostic" of the condition. Tr. at 271.

history); First Mikovits Rep. at 5-6.[35] Dr. Mikovits also suggested that POTS could be related to a genetic susceptibility. Tr. at 280-81.[36]

Dr. Mikovits also characterized POTS as an autoimmune disease. Tr. at 243-44. As she broadly posited, the "heterogeneity" of the disease suggests it is neuroinflammatory in nature. *Id.* at 229; *see also* S. Blitshteyn, *Autoimmune Markers and Autoimmune Disorders In Patients With Postural Tachycardia Syndrome (POTS)*, 24 Lupus 1364 (2015), filed as Ex. 64 (ECF No. 49-2) ("Blitshteyn"). In support, Dr. Mikovits cited to instances in the medical records where various treaters noted that Petitioner had, post-vaccination, experienced symptoms often associated with an underlying inflammatory process (including pain, brain fog, weakness, and skin eruptions). Tr. at 287, 292.[37] And she pointed to lab results from December 2013[38] (revealing increased levels of IgE and Lyme disease) as evidence of ongoing inflammation. *Id.* at 287-88. She did, however, acknowledge that the more traditional biomarkers for inflammation – which she defined as "mast cell mediators" – were either documented as normal (i.e., CRP and ESR) or never tested (i.e., cytokine levels). *Id.* at 287-88.

Dr. Mikovits went on to argue that relevant literature establishes that at least "half" of all POTS cases have some neuropathic basis. Tr. at 276-77; M. Thieben, et al., *Postural Orthostatic Tachycardia Syndrome: The Mayo Clinic Experience*, 82 Mayo Clin. Proc. 308 (2007), filed as Ex. 54 (ECF No. 30-5) ("Thieben"); Benarroch at 1215-16. Thieben was a retrospective study of 152 POTS patients seen at the Mayo Clinic over eleven years, and considered the data and test results obtained during treatment. Thieben at 308. It hypothesized that a particular autoantibody (the ganglionic acetylcholine receptor) was associated with neuropathic cases of POTS, although by its own terms the article does *not* propose or embrace the contention that POTS is in *all* (or even most) cases an autoimmune disease. *Id.* Indeed, as observed in Benarroch (a review article aimed at cataloging the pathophysiology and subtypes of POTS, including the neuropathic and hyperadrenergic variants), Thieben only found that *fourteen percent* of all POTS patients considered in the study presented with evidence of a ganglionic acetylcholine receptor antibody

[35] During her testimony, Dr. Mikovits asserted that POTS is not caused by deconditioning (a contention directly contrary to what the medical community better versed in POTS understands to be the case in many instances). Tr. at 245; *compare* Benarroch at 1216-17.

[36] Dr. Mikovits spent some time at hearing discussing the MTHFR methylation defect (which she suggested was present in Petitioner's family history). Tr. at 280-81. But she could not point to a medical record confirming that Ms. McKown possessed this particular defect, nor did she offer evidence associating it with POTS. *Id.* at 281.

[37] Upon further questioning, Dr. Mikovits admitted that she relied heavily on the fact of the POTS diagnosis as proof that Petitioner had experienced post-vaccination neuroinflammation. Tr. at 292.

[38] Presumably, Dr. Mikovits is referring to Ex. 1 at 65-66 (dated from December 2013) – which is consistent with Dr. Tornatore's testimony. Dr. Mikovits did not reference a particular date during her testimony, however. *See* Tr. at 287-88.

(which could indicate an immune cause of neuropathic POTS in some cases, but hardly even close to half. Benarroch at 1215-16.

Dr. Mikovits further relied on various studies associating POTS with other autoimmune conditions, such as autoimmune inflammatory syndrome induced by adjuvants ("ASIA")), and chronic fatigue syndrome ("CFS"). Tr. at 243-45; *see, e.g.*, S. Dahan, et al., *Postural Orthostatic Tachycardia Syndrome (POTS) – A Novel Member of the Autoimmune Family*, 25 Lupus 339 (2016), filed as Ex. 65 (ECF No. 49-3) ("Dahan"); S. Cerpa-Cruz, et al., *Adverse Events Following Immunization With Vaccines Containing Adjuvants*, 56 Immuno. Res. 299 (2013), filed as Ex. 30 (ECF No. 19-4) ("Cerpa-Cruz"); G. Giannotta, et al., *Vaccines and Neuroinflammation*, 3 Int'l J. Public Health Safety 1 (2018), filed as Ex. 68 (ECF No. 53-2) ("Giannotta"). Dr. Mikovits posited that forty percent of POTS patients congruently suffer from other autoimmune conditions (like those noted above) – though no specific piece of literature directly supports such an assertion. Tr. at 272.

In addition, Dr. Mikovits referenced various review articles that "reported" occurrence of "severe somatoform dysautonomic and neuropathic syndromes" following receipt of the HPV vaccine, all of which she posited result in symptoms consistent with or overlapping POTS. Tr. at 228-29; First Mikovits Rep. at 7; *see* B. Palmieri, et al., *Severe Somatoform and Dysautonomia Syndromes After HPV Vaccination: Case Series and Review of Literature*, SpringerOnline (2016), doi 10.1007/s12026-016-8820-z, filed as Ex. 32 (ECF No. 19-6)[39]; S. Aratani, et al., *Murine Hypothalamic Destruction With Vascular Cell Apoptosis Subsequent to Combined Administration of Human Papilloma Virus Vaccine and Pertussis Toxin*, Scientific Reports (2016), doi:10.1038/srep36943, filed as Ex. 43 (ECF No. 20-8); M. Martinez-Lavin, *Hypothesis: Human Papillomavirus Vaccination Syndrome—Small Fiber Neuropathy and Dysautonomia Could be Its Underlying Pathogenesis*, Clin. Rheum. (2015), doi:10.1007/s10067-015-2969-z, filed as Ex. 34 (ECF No. 19-8); Brinth at 2602.

Dr. Mikovits next discussed the purported association between Petitioner's skin symptoms, which she characterized as atopic dermatitis, and an autoimmune process. Tr. at 277-79. Atopic dermatitis, in her view, is also immune-mediated – and thus distinct from eczema, for example. *Id.* at 230, 277-78. While both conditions are of the same disease family, atopic dermatitis is a subgroup that is inflammatory in nature, while eczema is not. *Id.* at 278. To support the purported neuropathic connection between Petitioner's skin symptoms and POTS, Dr. Mikovits referenced Voisin (discussed earlier by Dr. Tornatore), which concluded that immune cells/inflammatory mediators can act as "neurotransmitters" or signalers (by way of "cross-talk,"), and thereby mediate allergic inflammation. *Id.* at 230; Voisin at 1. Dr. Mikovits concluded that "cross-talk" between neuronal receptors and immune cells can cause skin inflammation – as evidenced by an onset of itching. Tr. at 230.

[39] Exhibit 33 is a corrected version of this article.

Based on her review of the record and scientific literature filed in support, Dr. Mikovits posited that the biologic process responsible for Ms. McKown's onset of symptoms post-vaccination likely originated with dysregulation or "overstimulation" of the innate immune system (which she defined as comprising the body's "first responders" during an immune response). Tr. at 196, 227, 289; First Mikovits Rep. at 8. Autoimmunity, in her view, is initiated by an "overactive immune system" in which various innate immune mediators (including dendritic cells, mast cells, and microglia) respond to some "perceived threat" (or foreign agent), thereby causing excessive inflammation in the body. Tr. at 237-38; *see also* First Mikovits Rep. at 8. The innate system is capable of recognizing foreign antigens quickly and building a memory response. First Mikovits Rep. at 8.

Dr. Mikovits next turned to the role a vaccine could play in causing such innate system overactivity. As. Dr. Mikovits explained, the components of the HPV and Hep A vaccines (both of which contain aluminum as an adjuvant) dysregulate brain microglia (and mast/dendritic cells at the mucosal cell surface) – thereby causing chronic neuroinflammation in a genetically susceptible patient. Tr. at 226-27, 233, 281-82; First Mikovits Rep. at 8-9. After stimulation by a vaccine, the innate system triggers the overproduction of inflammatory cytokines. Tr. at 227, 233. Such an overproduction of cytokines could thereafter become chronic, when microglia – non-neuronal glial cells located in the brain that also play an immune defense role – are primed to become a source of excess cytokine production, often in reaction some prior insult (such as vaccination). First Mikovits Rep. at 8-9. In most cases, patients confronted with a cascade of proinflammatory cytokines can self-regulate the overproduction via a normal immune system response (with the help of T cells). Tr. at 230-40. Those who experience dysregulation, however, typically have some form of a genetic susceptibility to autoimmunity (though puberty and/or a traumatic injury could also spur on such a response). *Id.* at 240-41.

Dr. Mikovits specifically implicated the IL-1 beta cytokine (which she stated "controls the local pro-inflammatory cascade") as responsible for preventing protective immunity from becoming destructive. Tr. at 230-31. Dr. Mikovits posited that scientific literature on the topic shows that inhibiting IL-1 beta production can prevent adverse cardiac events from occurring (though, she did not file any to corroborate her statements). *Id.* at 231. Based on the above, Dr. Mikovits concluded that POTS (which is cardiac driven) is likely caused by an inflammatory response brought about by the overproduction of IL-1 beta. *Id.*

The receipt of multiple vaccines, Dr. Mikovits maintained, could amount to "damage at a distance" whereby the immune system cannot adequately respond to the above and properly self-regulate. Tr. at 232, 295. Inflammatory cytokines can thus more quickly cross from the periphery (where they originate) and into the central nervous system (thereby interacting with brain microglia), breaching the blood-brain barrier with the help of macrophages. *Id.* at 295. Dr.

Mikovits posited that histidine and polysorbate 80 (both HPV vaccine ingredients) were also likely responsible for this breach. *Id.* at 233. And she allowed for the possibility that "virus-like particles" contained in the vaccines can "package pieces and parts of other DNA and other retroviruses" could contribute to the breach, but she could not be more specific as to how (or even what literature existed that reliably could support such a contention). *Id.* at 234; *see also* First Mikovits Rep. at 10.

Along those same lines, Dr. Mikovits discussed the various "excipient[]" components (i.e., preservatives or adjuvants) contained in the HPV and Hep A vaccines – which she deemed responsible for such as response. Tr. at 231-32; First Mikovits at 10-11. In particular, Dr. Mikovits implicated the aluminum adjuvant[40] (which is present in both the HPV and Hep A vaccines) as causative of the dysregulation of the cytokine balance discussed above. Tr. at 232; R. Gherardi, et al., *Aluminum Adjuvants of Vaccines Injected Into the Muscle: Normal Fate, Pathology, and Associated Disease*, 100 Morphologie 85 (2016), filed as Ex. 49 (ECF No. 21-5); F. Liang & K. Lore, *Local Innate Immune Response in the Vaccine Adjuvant-Injected Muscle*, 5 Clin. & Trans. Immunol. 74 (2016), filed as Ex. 41 (ECF No. 20-6); C. Exley, et al., *When an Aluminum Adjuvant Is Not an Aluminum Adjuvant Used in Human Vaccination Programmes*, 30 Vaccine 2042 (2012), filed as Ex. 39 (ECF No. 20-4) (Letter to the Editor); C. Exley, et al., *Aluminum-Based Adjuvants Should Not Be Used as Placebos In Clinical Trials*, 29 Vaccine 9289 (2011), filed as Ex. 38 (ECF No. 20-3) (Letter to the Editor).

She also referenced literature discussing the concept of ASIA[41] (even though Petitioner has explicitly indicated it is not part of her causation theory)[42], and mast cell activation disorder – which, in her view, establishes that merely 400 micrograms of alum is enough to "cripple[e]" the microglia – thereby resulting in a disease state characterized by ongoing inflammation. Tr. at 232, 281, 289-90; First Mikovits Rep. at 11; *see* L. Tomlijenovic, et al., *Postural Orthostatic Tachycardia With Chronic Fatigue After HPV Vaccination as Part of the "Autoimmune/Auto-Inflammatory Syndrome Induce By Adjuvants": Case Report and Literature Review*, J. Invest. Med. (2014), doi:10.1177/2324709614527812, filed as Ex. 29 (ECF No. 19-3); M. Frieri, et al., Mast Cell Activation Syndrome: A Review, 13 Curr. Allergy Asthma Rep. 27 (2013), filed as Ex. 40 (ECF No. 20-5). The chronic aspect of such a response is attributable to the alum "stay[ing] in

[40] At hearing, Dr. Mikovits asserted that the specific aluminum adjuvant present in the HPV and Hep A vaccines has never been tested for safety despite being used in vaccines for over 80 years. Tr. at 281.

[41] On cross, Respondent pointed out that ASIA is not recognized by the World Health Organization as playing a role in the causation of adverse events related to vaccination. Tr. at 272-73. Dr. Mikovits acknowledged the finding, but maintained that the ASIA is consistent with the theory that an autoimmune disease can be triggered by various external agents. *Id.* at 273.

[42] *See* Pre-Hearing Reply, dated Sept. 21, 2018 (ECF No. 52) ("Respondent spends a good portion of their Pre-Hearing memorandum addressing the ASIA theory. However, while Drs. Mikovits and Ruscetti reference ASIA, neither they nor Dr. Tornatore assert ASIA as their theory of causation.").

the macrophage" for "more than one year." Tr. at 289.[43] This interaction (coupled with the components of her theory discussed below) helps to create the overall autonomic dysfunction Petitioner alleges to have experienced post-vaccination. *Id.* at 289-91.[44]

While maintaining that the autoimmune process resulting in Ms. McKown's injuries originated from an innate immune response, Dr. Mikovits posited that the adaptive system (via molecular mimicry) also likely plays a role in the overall cascade of autoimmune dysfunction. In addition to sequential homology (between vaccine-induced autoantibodies and self proteins), Dr. Mikovits explained that "conformational epitope[s]" expressed on the cell surface (in response to the insulting foreign antigen) are actually responsible for the cross-reactivity. Tr. at 234-35. For example, the Hep A vaccine contains a "cell line" called MRC-5 (a human cell line from aborted fetal tissue) that shares homology with endogenous human retroviruses. *Id.* at 231. The overlapping mimicry thus stimulates neuroinflammatory pathways and results in some adverse disease process. *Id.* In the context of the HPV vaccine doses, Dr. Mikovits posited that the Kanduc paper (also referenced by Dr. Tornatore) establishes 200 potential immunogenic and cross-reactive epitopes to HPV vaccine antigens. *Id.* at 235. This process can also be hastened by subsequent exposure to the same vaccine antigens, which would be consistent with a "challenge-rechallenge" response. *Id.* at 235-36.

B. *Respondent's Witnesses*

1. Dr. Christopher Gibbons

Dr. Gibbons filed one written report in this matter and testified at hearing. Tr. at 303-443; Expert Report, dated May 4, 2018, filed as Ex. D (ECF No. 35-1) ("Gibbons Rep."). Based upon the record as a whole and his review of the scientific literature, Dr. Gibbons posited that vaccines do not cause POTS (nor did they do so in this case).

Dr. Gibbons currently serves as a staff neurologist at Beth Israel Deaconess Medical Center in Boston, Massachusetts. Curriculum Vitae, filed as Ex. O (ECF No. 54-1) ("Gibbons CV"); Tr. at 303. In addition to the above, he holds a number of clinical and teaching positions: Associate

[43] Dr. Mikovits also made some suggestion that the chronic inflammation (or driver of the "disease engine") associated with immune system disorders can also be caused by "oxidative stress" or "reactive nitrogen species". Tr. at 244. Such concepts were not discussed in depth – but have been offered in prior Program cases without success. *See, e.g., Bast v. Sec'y of Health & Human Servs.*, No. 01-565V, 2012 WL 6858040, at *6 (Fed. Cl. Spec. Mstr. Dec. 20, 2012) (rejecting theory that seizures resulted from vaccine-induced mitochondrial dysfunction associated with oxidative stress), *mot. for review den'd*, 117 Fed. Cl. 104 (2014).

[44] On cross, when confronted with evidence that humans *ingest* far more aluminum per day than the considerably smaller amounts contained in a vaccine, Dr. Mikovits posited that ingesting is not comparable in effect to injecting cells directly with the adjuvant. Tr. at 282. She did not offer any scientific or medical evidence to corroborate the assertion, however.

Professor of Neurology, Director of the Neurocutaneous Laboratory, Associate Director of the Autonomic Laboratory, and Director of the Neuropathy Clinic at Beth Israel's Joslin Diabetes Center. Tr. at 303. He received his undergraduate degree from Dartmouth College, followed by a and medical degree from Albert Einstein College of Medicine. Gibbons CV at 1; Tr. at 309. He completed a neurology residency at Johns Hopkins Hospital in Baltimore, Maryland, and a fellowship thereafter in clinical neurophysiology (with a subspecialty in autonomic disorders) from Beth Israel. Tr. at 309.

Over the course of his career, in his clinical practice Dr. Gibbons has repeatedly evaluated and treated patients with immune-mediated conditions, including autonomic disorders, peripheral nerve disease, neuropathies, and small fiber neuropathies. Tr. at 303-04. Due to the overlap between the autonomic and peripheral nervous systems, his treatment focus has been in patients with Parkinson's disease (who also display dysautonomia), multiple system atrophy, and diabetic neuropathy. *Id.* at 304-05. He also oversees a neurocutaneous skin biopsy lab in which he (and his students) evaluate biopsy results to determine nerve involvement. *Id.* at 305. He treats patients with POTS (and routinely conducts tilt tabling testing). *Id.* at 306-07, 340. In addition, Dr. Gibbons serves on the board of various journals focused on autonomic issues, including *Autonomic Neuroscience: Basics and Clinical*. *Id.* at 308. Dr. Gibbons estimated that he spends roughly half of his time treating patients (while the remaining half is used for teaching and research). *Id.* at 305-06, 311. He does not have specialty training in the fields of immunology, dermatology, or epidemiology, however. *Id.* at 399.

Dr. Gibbons began his testimony by describing the primary functions of the autonomic nervous system (along with defining descriptive terms used to describe autonomic nervous system irregularities, such as "dysautonomia" and "autonomic damage"). Tr. at 315. Dr. Gibbons defined the autonomic system as the "subconscious" part of the nervous system responsible for controlling or regulating the body's involuntary functions (including breathing, heart rate, blood pressure, sweating, urination, and defecation). *Id.* "Dysautonomia" or "autonomic dysfunction," he posited, is a vague term used in the literature to refer to a "perceived problem" with the autonomic system, which may or may not be associated with some function in the autonomic system itself. *Id.* at 315-16.

In contrast, "autonomic damage" refers to a specific, identifiable injury to the autonomic system. Tr. at 316-17. For example, Parkinson's disease is a condition linked to autonomic damage. *Id.* at 317. As Dr. Gibbons explained, Parkinson's can be attributed to a particular protein in the body ("alpha-synuclein"), the presence of which is identifiable by both microscopic imaging and testing, and which causes direct damage to the autonomic system. *Id.* Vasovagal syncope (or presyncope symptoms: dizziness, lightheadedness, palpitations, or blacking/greying out), on the other hand, does not necessarily reflect the existence of damage to the autonomic system, but can

instead constitute a "normal, completely physiologic, typical response" by an otherwise-healthy autonomic nervous system. *Id.* at 316, 336-37.

Dr. Gibbons next defined POTS and its usual symptoms and course. POTS is characterized by a "heart rate increase that goes up over time beyond what we would consider normal," resulting in a number of symptoms (lightheadedness, dizziness, palpitations, tremulousness, feelings of presyncope, and vasovagal syncope). Tr. at 317-18, 322; *see also* R. Freeman, et al., *Consensus Statement on the Definition of Orthostatic Hypotension, Neurally Mediated Syncope and the Postural Tachycardia Syndrome*, 161 Autonomic Neurosci. 46 (2011), filed as Ex. D, Tab 1 (ECF No. 35-2); Gibbons Rep. at 3-4. Skin rashes, in his view, are typically not a presenting symptom of POTS. Tr. at 436-37.[45] In addition, vasovagal syncope is not by itself diagnostic of POTS (or its inevitable result). *Id*. at 322, 414, 416 ("people with POTS don't have to faint").

POTS is classified as a "syndrome" due to the various etiologies (some of which can be autoimmune) associated with its onset, including deconditioning, chronic illness, and/or autonomic neuropathy. Tr. at 317; Gibbons Rep. at 3-4.[46] As Dr. Gibbons explained, POTS can be an "end result of many potential avenues," including a secondary reaction to a variety of diseases, like diabetes, thyroid dysfunction, renal dysfunction, or multiple sclerosis. Tr. at 318. The diagnostic criteria for POTS require (a) an increase in the heart rate of more than 30 bmp or more from the supine to standing position (or 40 bmp or more if under twenty years of age), (b) symptoms that are consistent with such a diagnosis (as describe above), and (c) a continuation of symptoms for greater than six months. *Id.* at 318-19. POTS is most common in young women (generally teenagers up to their twenties). *Id.* at 319.

POTS can be divided into various subgroups with overlapping symptoms (Tr. at 402), including hypovolemic, hyperadrenergic, and neuropathic. As Dr. Gibbons explained, all the variants are distinguishable based on laboratory or clinical testing. Tr. at 322, 402. Hypovolemic POTS is characterized as POTS "related to low blood pressure" (occurring, for example, when an individual exercises excessively without proper fluid intake). *Id.* at 320-21. The hypovolemic variant can also present secondarily to various illnesses associated with deconditioning (including myalgic encephalomyelitis and CFS). *Id.* at 419-20. Hyperadrenergic POTS is related to an adrenaline response (i.e., an increase in norepinephrine). *Id.* at 321. According to Dr. Gibbons, this

[45] As already noted, Petitioner's experts relied on Voisin as establishing "cross talk" between nerves resulting in onset of skin rashes that could in turn demonstrate how skin conditions like eczema were the product of the same autoimmune process that allegedly was causing a person to experience POTS. Tr. at 423-25. In response, Dr. Gibbons deemed the theory "interesting" but claimed that it did not stand as reliable evidence establishing a reliable medical association between such conditions (nor evidence that the nerves were indeed damaged in the process). *Id.* at 423, 433.

[46] At hearing, Dr. Gibbons disputed any suggestion that POTS is more often than not caused by neuroinflammation. Tr. at 325-26.

subgroup is more easily differentiated in autonomic testing. *Id.* Hyperadrenergic POTS can occur congruently with autoimmune disease (for example, type I diabetes or limb encephalitis), though in such cases a patient will experience *other* severe symptomology consistent with the primary disease process in question. *Id.* at 314, 357. This subgroup can also be antibody-mediated or instigated by the presence of toxic substances. *Id.* at 325.

Finally, Dr. Gibbons discussed neuropathic/neurogenic POTS – a variant he deemed (based upon his direct experience) "quite rare." Tr. at 321-22, 377. Dr. Gibbons described this subgroup as an immune-mediated form of POTS resulting in "some sort of damage to the autonomic nervous system" which impairs the typical autonomic functions, resulting in the tachycardia associated with the illness. *Id.* at 321, 324-35.[47] Neuropathic POTS is typically associated with a particular antibody (the "ganglionic receptor antibody") that targets the acetylcholine receptor in the autonomic ganglia, resulting in autoimmune ganglionopathy. *Id.* at 358. High amounts of the ganglionic receptor antibody can cause profound autonomic failure (resulting in a total loss of control of blood pressure, heartrate bowels, bladder, etc.). *Id.* Based on his knowledge of the literature, Dr. Gibbons posited that the ganglionic antibody is the only clinically relevant antibody associated with neuropathic POTS. *Id.* at 358. He could not identify any other antibody targeted at the heart,[48] but added that he was aware of ongoing research on the topic, although he deemed it preliminary. *Id.* at 358, 375-76, 396.[49]

In addition to the above, Dr. Gibbons questioned the significance of various pieces of literature offered by Petitioner's experts to support the conclusion that *all* (or most) forms of POTS are neuropathic in nature. In so doing, Dr. Gibbons emphasized the absence of evidence establishing that the POTS she experienced was consistent with the immune-mediated form of the condition. Benarroch, for example, discusses the more common POTS subtypes (hypovolemic and hyperadrenergic), but allows that POTS can be immune-mediated (as Petitioner proposes). Tr. at 361. Dr. Gibbons, however, posited that Benarroch actually supported his earlier conclusion that the *only* known antibody associated with neuropathic version of the condition is the ganglionic acetylcholine receptor antibody – with no other subtype of the disease demonstrated "convincingly" to be immune mediated. *Id.*

Thieben, in his view, followed the same pattern: establishing that neuropathic POTS is a rare subtype specifically involving antibody-mediated, neuronal damage. Tr. at 364. As Dr.

[47] A further subset of neuropathic variant is "cholinergic" POTS. Tr. at 324. The cholinergic variant results from damage to the "sympathetic cholinergic system" associated with gastrointestinal symptoms (and can be associated with abnormal sweating and nerve damage). *Id.* at 324, 370-71.

[48] Dr. Gibbons posited, however, that structural damage to the heart directly could cause POTS. Tr. at 396-97.

[49] As Dr. Gibbons explained, it is difficult to determine if elevated titers of certain circulating antibodies are clinically relevant absent (a) evidence that high titers are associated with a disease, and (b) evidence that removing those antibodies prompts a recovery. Tr. at 376.

Gibbons explained, Thieben provided evidence that a small number of POTS patients (6 out of 42 or 14 percent) presented with the above-described ganglionic antibody in low positive values. *Id.* at 363. But this percentage appeared high to Dr. Gibbons, as he has seen only *one* POTS patient with the same ganglionic antibody in the last ten years of his clinical practice. *Id.* In addition, those percentages have never been reproduced in further research. *Id.* at 364, 442-43. Thieben's authors similarly made clear that actual damage to the autonomic nervous system did not explain every POTS subtype. *Id.*

Dr. Gibbons next discussed the Blitshteyn paper. Tr. at 364-65. Blitshteyn's authors tested POTS patients for certain autoimmune biomarkers (e.g., ANA, TTG, SS-A antibodies, etc.), concluding (based on their positive readings) that autoimmunity must be common in POTS generally. *Id.* at 365. Dr. Gibbons, however, criticized Blitshteyn for the inclusion criteria used in conducting its analyses. *Id.* at 366. He noted that the study appeared to have self-selected for patients who suffer from both POTS and an additional autoimmune disease. *Id.* at 366-67. It was thus no surprise that many of the studied patients also tested positive for autoimmune biomarkers. *Id.* at 367. More importantly, many biomarkers tested for in Blitshteyn had no known association (in the causation context) to the patient's underlying disease process – and therefore their presence did not establish a causal link. *Id.* at 367. Thus, Blitshteyn could not, in Dr. Gibbons's opinion, be invoked to support a conclusion that POTS is generally autoimmune in nature (as Petitioner's causation theories implied). *Id.* at 368.

Dr. Gibbons also addressed the relationship between neuropathic POTS and vasovagal syncope of the kind relevant to this case. In his experience, neuropathic POTS actually *does not* result in, or even feature, syncope. Tr. at 322. Rather, syncope is typically a product of a properly functioning autonomic nervous system – thus, patients with autonomic damage would likely not manifest syncope symptoms. *Id.* at 323, 394, 421 (describing syncope is a "normal physiologic response"). Rather, the progressive, neuropathic variant of POTS results in orthostatic hypotension *without* the tachycardia typically associated with the non-neuropathic subgroups. *Id.* at 323-24. In fact, neuropathic POTS should feature a reduction in syncope (given the resulting damage to the autonomic system). *Id.* at 349. All in all, Dr. Gibbons concluded that healthy patients can have vasovagal syncope with *no* corresponding autonomic damage. *Id.* at 434, 436.

Relying on his review of the medical records, Dr. Gibbons characterized Petitioner's variant of POTS as most likely hypovolemic rather than neuropathic. Tr. at 326, 394. Dr. Gibbons could not identify any evidence in the record to suggest or corroborate the propriety of a neuropathic or hyperadrenergic POTS diagnosis. *Id.* at 394. He further opined that Ms. McKown's onset of POTS was not caused or significantly aggravated by either (or both) of her HPV vaccine doses. *Id.* at 326, 394. In his practice, Dr. Gibbons regularly treats POTS patients (some of whom have received the HPV vaccine and some who have not), and has seen no variances in disease course. *Id.* at 429-30. Overall, he felt that some of Petitioner's overall course could be attributable

to deconditioning (in light of her physical fitness routines[50]) and/or genetics (related to her height[51]). *Id.* at 355-56.

In support, Dr. Gibbons referenced record evidence tending to suggest that Petitioner's POTS onset was unrelated to any autoimmune process and did not display what he would expect to see if her POTS was neuropathic. No clinical test results indicated that she was experiencing any underlying autoimmune disease process. Tr. at 396. A ganglionic receptor antibody test (which could support a neuropathic variant of the condition) was never conducted. *Id.* In addition, as noted above, Dr. Gibbons posited that symptoms indicative of neuropathic harm would be "striking" in nature (including symptoms such as: fixed/dilated pupils, invariant heart rate, gastroparesis, and urinary retention). *Id.* at 439 (describing an event where "essentially every component of the autonomic nervous system shuts down"). But there was no record evidence that Petitioner experienced any such drastic symptoms. And other than Dr. DeMio, none of Petitioner's treaters proposed autoimmune disease to explain her POTS. *Id.* at 356.

Rather, Ms. McKown's course was more consistent with the hypovolemic variant of the condition. Tr. at 332-33. In support, Dr. Gibbons referenced records detailing her hospital visit in April 2013 (following the syncopal incident at the yogurt shop). *Id.* at 332-33. The orthostatic vitals taken at the time indicated that Petitioner's blood pressure was stable during the visit. *Id.* at 332. Her heart rate did increase significantly upon standing, but her syncopal symptoms resolved following intake of a liter of fluids. *Id.* The fact that Petitioner's symptoms ceased following fluid intake suggested to Dr. Gibbons that she was suffering from the hypovolemic variant. *Id.* at 332-33. In addition, laboratory testing completed during her hospital visit in late April 2013 revealed an elevated BUN to creatine ratio of 20:1. *Id.* at 333. He considered such a high ratio as indicative of dehydration (which would explain the resolution of symptoms following fluid intake). *Id.*[52]

In addition, Dr. Gibbons referenced Petitioner's cardiology appointment with Dr. Dooley (at which time she was officially diagnosed with POTS). In his view, the description of her symptoms taken during this visit represented a "classic array" of those seen with POTS/vasovagal

[50] Deconditioning did not necessarily mean that a person had been physically inactive. Dr. Gibbons referenced the fact that astronauts (who are physically fit and trained) can develop POTS after returning to earth. Tr. at 355-56 ("[c]ouch potato[es]" are not the only ones at risk").

[51] Dr. Gibbons suggested that the literature strongly supports an associations between POTS and having a small stature. Tr. at 355. This point was not, however, developed at hearing or in the parties' filings, and I therefore do not give it substantial weight in Respondent's favor (or against Petitioner).

[52] Apart from the above, Dr. Gibbons acknowledged that the ER record also evidence a complaint of GI discomfort. Tr. at 331. He thus allowed for the possibility that Petitioner's GI symptoms could have caused her syncopal episode. *Id.*

syncope patients that he had treated in the past. Tr. at 335.[53] Ms. McKown's orthostatic vitals again revealed a table blood pressure reading with an increase in heartrate (from 68 to 100) upon position change – which he deemed as "not quite meeting [POTS] criteria," though not ruling it out either (as it was close enough to the diagnostic measures). *Id.* at 335. Tilt table testing, however, confirmed the diagnosis thereafter. *Id.* at 338-39, 342 ("[s]he fainted with her heart rate dropping, her blood pressure dropping, and then she recovered"). Based on the above, Dr. Gibbons eliminated the hyperadrenergic variant as a possible explanation, given that her blood pressure readings were "pretty much the same or lower" (i.e., they didn't go up). *Id.* But this record did not support the neuropathic variant, because Petitioner had experienced syncope upon position change – which indicated the *absence* of autonomic system damage. *Id.* There was also no evidence of direct cardiac damage indicated on Petitioner's EKG. *Id.* at 432. Thus, Dr. Gibbons concluded that the hypovolemic variant was the form best supported by record evidence. *Id.*

Other records generated subsequent to Petitioner's POTS diagnosis were also in Dr. Gibbons's view consistent with what would be experienced by a typical patient with non-neuropathic POTS. Tr. at 345. For example, Dr. Gibbons referenced a record from January 2014, in which Ms. McKown complained of "minor near-syncopal episodes" with worsening episodes now resulting in loss of conscious (for twenty to thirty minutes) and abnormal eye movement. *Id.* As Dr. Gibbons explained, recurrent episodes of syncope reflected a typical POTS course. *Id.* at 346.

Petitioner's purported prolonged episodes of unconsciousness, however, were in Dr. Gibbons's view inconsistent with a POTS diagnosis (regardless of the subtype involved). Tr. at 346, 349. Prolonged unconsciousness, Dr. Gibbons reasoned, is instead associated with inadequate blood flow to the brain or a brain irregularity (i.e, "both [brain] hemispheres . . . are not working"). *Id.* at 346. Dr. Gibbons suggested that these prolonged episodes could possibly be attributable to an underlying seizure disorder, but Petitioner's presentation was more consistent with vasovagal syncope. *Id.* at 347. Moreover, episodes of unconsciousness extending twenty to thirty minutes would typically be associated with "significant cerebral deficits" which Petitioner did not experience. *Id.* As a result, Dr. Gibbons proposed it most likely that these purported episodes of prolonged unconsciousness were caused by other mechanisms (such as: hyperventilation, fear, anxiety, fright, worrying, etc.) in which patients are "just unwilling to return to the situation" that caused the episodes, and therefore did not conform to *any* form of POTS, neuropathic or otherwise. *Id.* at 348-49.

[53] This record also indicated that Ms. McKown (or her caretakers) expressed a concern for "recurrent" syncopal episodes in years past, but Dr. Gibbons could conclude only that those episodes were at least consistent with her more recent syncopal symptoms in April 2013. Tr. at 334, 405, 433. Later in his testimony, however, he opined that POTS patients with a predisposition of fainting (in response to blood being drawn, for example) are more commonly placed in the hypovolemic or hyperadrenergic categories. *Id.* at 344.

Along those same lines, Dr. Gibbons noted that more recent records from 2014 indicated that Petitioner had been experiencing one to two episodes of POTS-related syncope per month. Tr. at 350; *see* Ex. 7 at 10. The frequency of the syncopal episodes, Dr. Gibbons posited, was also consistent with non-neuropathic POTS (which, as noted earlier, he would expect to diminish, not increase, syncopal episodes). Tr. at 350-51. By July 2014, records indicated Petitioner's POTS symptoms were improving, *but* she had now experienced a new onset of joint pain. *Id.* at 351 (citing Ex. 21 at 1). Dr. Gibbons reported that symptoms of pain are not typically indicative of POTS, unless the pain was attributable to some other underlying disorder. *Id.* at 352. POTS, in his view, is not associated with any form of pain syndrome. *Id.* He therefore also discounted this symptom as POTS related in any form.

With respect to timing, Dr. Gibbons opined that Petitioner's POTS symptoms preceded receipt of the HPV vaccine. He pointed to the records referencing pre-vaccination syncopal episodes (for which Petitioner was referred to the cardiologist in the first place – on the very day she received the first HPV dose). Tr. at 326 (citing Ex. 1 at 44). Records from March 2013 indicated that Ms. McKown reported three syncopal episodes with position change (along with lightheadedness) before the visit – which Dr. Gibbons classified as "classic" POTS symptomatology. *Id.* at 327-28.[54] Dr. Gibbons ultimately placed onset around six months prior to her receipt of the first dose of HPV. *Id.* at 328, 342-43. He also referenced earlier-in-time orthostatic measurements taken during her wellness checks at ages eleven through fourteen. *Id.* at 329. Heart rate measurements taken at this time evidenced resting heart rates ranging from 104 beats per minute to 84 – measures he deemed high for a pediatric patient who was also actively training for triathlons. *Id.* In his view, heart rate ranges in the 80-100 range suggests something was occurring even though it might not be overtly clinical. *Id.* at 330.

Dr. Gibbons also addressed Dr. Tornatore's arguments that Petitioner's pre-vaccination syncopal episodes were distinguishable because they were merely "situational" – a concept he deemed to be *not* a "good medical term." Tr. at 403. Rather, situational syncope and vasovagal syncope are in his experience essentially synonymous concepts. *Id.* (describing "situational" syncope as "vasovagal syncope related to a situation"). Later on in his testimony, however, he admitted that certain "situations" could trigger vasovagal syncope (for example, having blood drawn or receiving a reprimand for bad behavior, as is alleged to have occurred with Petitioner). *Id.* Ultimately, he proposed that the earlier instances were most likely associated with, rather than distinguishable from, Petitioner's later course. *Id.* at 326-38, 342-43.

Apart from the above, Dr. Gibbons also questioned the strength of independent evidence purportedly establishing a connection between the HPV vaccine and POTS. Brinth, for example, identified young women in Denmark who had recently received the HPV vaccine and were then

[54] If the prior episodes of syncope were ignored, Dr. Gibbons maintained that he would place onset of Petitioner's POTS in May 2013, given the symptoms reported during the visit described above. Tr. at 410-11, 412-13.

referred to a syncope clinic due to reported orthostatic intolerance symptoms. Brinth at 2602-05. Its authors observed 21 cases of POTS out of 35 studied individuals. *Id.* at 2605. Significantly, however, as Dr. Gibbons pointed out, *all* of the patients tested had suspected their POTS was caused by the HPV vaccine – and were in fact referred to the trial for that reason. Tr. at 381. In his view, given the self-selection bias in the inclusion criteria for studied individuals, no firm relationship could be drawn from Brinth's results. *Id.* at 382.

In addition, Dr. Gibbons noted that Brinth's conclusions (which he admitted caused some "concern" in the medical community regarding vaccine causation) prompted the European Medical Agency ("EMA") to conduct a follow-up study, in which it analyzed over 60,000 reports of onset of POTS following the receipt of the HPV vaccine. Tr. at 383-85; Assessment Report: Human Papillomavirus (HPV) Vaccines, EMA (2015), filed as Ex. D, Tab 13 (EC 36-4) ("EMA Report"). The EMA report found that the incidence rate for the number of patients with post-vaccination POTS (1 reported per 10,000) proved to be *smaller* than predicted, resulting in an assessment that disputed associating the HPV vaccine to POTS. Tr. at 385; EMA Report at 38-39. Dr. Gibbons admitted on cross examination, however, that the EMA report was based on some post-marketing surveillance (a passive reporting system informing manufacturers of the incidence rate) which, Petitioner posited, could have resulted in deflated numbers of reported incidence (as low as one percent). Tr. at 427-28.

Dr. Gibbons also offered other literature supporting his contention that the HPV vaccine likely plays no role in the development of POTS. Tr. at 387; J. Skufca, et al., *Incidence Rates of Guillain Barre (GBS), Chronic Fatigue/Systemic Exertion Intolerance Disease (CFS/SEID), and Postural Orthostatic Tachycardia Syndrome (POTS) Prior to Introduction of Human Papillomavirus (HPV) Vaccination Among Adolescent Girls in Finland*, 3 Papillomavirus Research 91 (2017), filed as Ex. D, Tab 14 (ECF No. 36-5) ("Skufca"). Skufca is a Finish study which cataloged onset of various autoimmune disease following receipt of the HPV vaccine beginning in 2013 (although it admittedly included reports from years prior, from 2002 to 2012, when the HPV vaccine was not administered). Skufca at 91. Skufca's authors noted a significant increase in reported POTS cases the year *prior* to the vaccine being administered. Tr. at 387. Around this same time, medical awareness of POTS increased (i.e., outreach campaigns were conducted to inform physicians of the condition and diagnoses increased). *Id.* Thus, in Dr. Gibbons's view, the increase in reported cases could be attributable merely to greater awareness of POTS, as opposed to an incidence rate increase attributable to vaccination. *Id.* at 387-88, 392.[55]

In addition, Dr. Gibbons offered a wide-in-scope review article on POTS's purported association with HPV vaccine. Tr. at 392-93; *see* B. Butts, et al., *Human Papillomavirus Vaccine*

[55] On cross examination, Dr. Gibbons acknowledged that Finland stopped administering the HPV vaccine around 2012. Tr. at 429.

and Postural Orthostatic Tachycardia Syndrome: A Review of the Current Literature, J. Child Neuro (2017), doi:10.1177/0883073817718731, filed as Ex. J (ECF No. 43-5) ("Butts"). Butts cataloged various items of literature (including peer-reviewed articles, government statements, and medical advisory committee notes – some of which were filed in the present matter) in an attempt to gauge the strength of studies purporting to associate the HPV vaccine with POTS. Butts at 1, 3. Its authors acknowledged the existence of case reports detailing the onset of POTS post-vaccination, but ultimately determined that the existing epidemiologic evidence did not support a causal connection. *Id.* at 7-10; Tr. at 392-93.

2. Dr. Andrew MacGinnitie

Dr. MacGinnitie served as Respondent's second expert, offering two written reports in the matter and testifying at hearing. Expert Report, dated Mar. 28, 2017, filed as Ex. A (ECF No. 23-1) ("First MacGinnitie Rep."); Expert Report, dated May 5, 2018, filed as Ex. C (ECF No. 34-1) ("Second MacGinnitie Rep."). Based on his review of the record, Dr. MacGinnitie opined that the HPV and Hep A vaccines Petitioner received did not exacerbate her subsequent eczema flares thereafter, and from an immunologic standpoint could not cause eczema or POTS. Tr. at 454, 455.

Dr. MacGinnitie is an attending physician and the Clinical Director for the Division of Immunology at Boston Children's Hospital in Boston, Massachusetts. Curriculum Vitae, filed as Ex. B (ECF No. 26-1) ("MacGinnitie CV"); Tr. at 444-45. He is also an Associate Professor of Pediatrics at Harvard Medical School. Tr. at 445. Dr. MacGinnitie received his undergraduate degree from Yale University, followed by both a medical degree and Ph.D. from the University of Chicago. *Id.* at 445. He thereafter completed his residency, followed by a fellowship in allergy and immunology at Boston Children's. *Id.* at 445. He is board certified in pediatrics and allergy and immunology, and has been in practice as an allergist/immunologist since 2004. *Id.* Further, he has seen patients with various immunologic diseases, including reactions to vaccines. *Id.* Ninety percent of his patients are children. *Id.* at 446-47. He estimated that he spends two-thirds of his time treating patients in a clinical setting. *Id.* at 450.

In his practice, Dr. MacGinnitie treats many allergic conditions (including eczema/ atopic dermatitis, food and environmental allergies, and urticaria). Tr. at 447. He diagnoses roughly 150-160 eczema patients per year. *Id.* at 447-48. Dr. MacGinnitie also performs research and has produced roughly thirty-five publications on the topic of food allergies (or associated conditions). *Id.* at 452. And he has authored a paper on the effects of vaccination on patients with egg allergies. *Id.* at 451. Dr. MacGinnitie serves as a medical reviewer for various journals, including *Allergy*

and Immunology. *Id.* at 452. As he acknowledged at hearing, however, Dr. MacGinnitie is not a neurologist (or dermatologist), and he has not treated patients with POTS. *Id.* at 503-04, 505-07.[56]

Dr. MacGinnitie began his testimony by defining the various medical terms relevant to Ms. McKown's skin condition as described herein, including eczema/atopic dermatitis – which he deemed interchangeable (Tr. at 508) – as compared to urticaria. Eczema is an erythematous, dry, flaky rash that can persist from hours to days or months, and can chronically relapse. Tr. at 460-61. Roughly ten to twenty percent of the population experiences eczema at some point in life, making it a fairly common occurrence. *Id.* at 461. Ninety percent of patients are diagnosed as infants, and some eventually outgrown the condition. *Id.* Eczema can worsen seasonally or flare unexpectedly, when skin is dry (or in the midst humid conditions). *Id.* Hormones can also increase the likelihood of a flare-up. *Id.* Due to the above, eczema can be difficult to treat. *Id.* Eczema is not a true allergic response, but patients diagnosed with eczema are more susceptible to developing food allergies and asthma later in life. *Id.* at 460. As Dr. MacGinnitie explained, symptoms of inflamed, abraded skin (followed by exposure to various foreign antigens) increase the likelihood later-onset allergies. *Id.* Vaccines, in his view, are not considered in the medical community to be causal of eczema. *Id.* at 462-63.

Dr. MacGinnitie opined that eczema is not an autoimmune condition. Tr. at 462, 501, 544. It is also not understood to be a presenting symptom of POTS (as he understands the disease). *Id.* at 463. Later on in his testimony, however, Dr. MacGinnitie did acknowledge that the condition is inflammatory (i.e., mediated by T cells) at least as it relates to the skin – and thus, certain immune-modifying drugs, like Prednisone, can be used to effectively treat it, as was the case herein. *Id.* at 509-10. He maintained, however, that eczema can only be associated with distinct "subtypes" of inflammation. *Id.* at 523. He also posited that the underlying inflammation associated with the condition is not systemic in nature. *Id.* at 462.

On cross examination, Dr. MacGinnitie acknowledged that the innate immune system likely plays some role in the development of eczema (given the increase in eosinophils associated with the condition). Tr. at 514. He also agreed that cytokines can "drive[]" an "inflammation axis" for eczema. *Id.* at 527. Certain drug therapies for eczema exist that are aimed at specific cytokine receptors, further underscoring the innate system's connection. *Id.* at 514, 524. Dr. MacGinnitie agreed that cytokines can be involved in the pruritus (i.e. itching) associated with eczema. Tr. at 521. Itching, Dr. MacGinnitie posited, could be caused by cytokines stimulating the relevant nerve fibers in the skin. *Id.* at 515-17. Thus, nerves are involved in eczema's symptoms, given that by

[56] On cross-examination, Petitioner's counsel questioned Dr. MacGinnitie about the various grants he received (from the government and pharmaceutical companies), which enabled him to complete research. Tr. at 504-05. I do not, however, find that Dr. MacGinnitie's receipt of such funding reduced his credibility or suggested bias – any more that I would find that Dr. Tornatore's frequent appearances on behalf of petitioners in the Vaccine Program is a *per se* basis for finding him not credible as a general matter.

definition, pruritus is a "phenomenon mediated in the CNS." *Id.* at 522. In so stating, he refuted the suggestion that histamines cause itching (given that antihistamines do alleviate eczema symptoms). *Id.* at 514.[57]

Urticaria, by contrast, is a term used interchangeably with "hives" or "welts." Tr. at 449. As Dr. MacGinnitie described, urticaria refers to raised skin lesions that usually "come and go" in response to a specific allergen (for example, peanuts). *Id.* at 449-50. Urticaria can also be chronic in nature – whereby the accompanying lesions persist for six weeks (or longer), often without any explanation. *Id.* at 450. In a typical urticarial response, the body's mast cells release histamine (often instigated by exposure to an irritant/allergen), causing hives in response. *Id.* at 466-67. The initiating allergen, however, is not itself the direct cause of hives. *Id.* at 466-67. Based on his review of the medical literature, Dr. MacGinnitie knew of no studied association between vaccination and onset of hives. *Id.* at 467. He allowed for the possibility, however, that hives could be autoimmune in rare circumstances (i.e., where particular antibodies – antithyroid autoantibodies, for example – are directed at the mast cells). *Id.* at 475, 544.[58]

Dr. MacGinnitie described an allergy as an "inappropriate immune response to a harmless environmental stimuli" (for example, tree pollen, cat dander, or peanut protein). Tr. at 463. Typical immune responses include sneezing, watery eyes, or anaphylaxis (a more extreme reaction). *Id.* Allergies are typically diagnosed via serum-specific IgE testing or "RAST" – though such testing can also produce false-positives. *Id.* at 465. A patient's prior health history (following exposure to suspected antigens) can also be helpful in evaluating for allergies. *Id.* An "intolerance," however, is not the same as an allergy. *Id.* at 464. Allergies, he posited, require systemic involvement (as evidenced by an overall worsening of the response). *Id.* For example, a patient who is "lactose-intolerant" will exhibit milder symptoms than a patient with a true milk allergy. As noted earlier, allergic responses can result in more extreme symptoms (i.e., anaphylaxis). *Id.* at 465.

Dr. MacGinnitie proposed that allergies are not associated with a predisposition for infection, autoimmune disease, or severe autonomic dysfunction. Tr, at 468. Similarly, Dr.

[57] On cross examination, Petitioner's counsel presented Dr. MacGinnitie with two articles, neither of which were filed prior to hearing nor discussed during examination of Petitioner's own experts. *See* G. Yosipovitch & A. Papoiu, *What Causes Itch in Atopic Dermatitis*, 8 Curr. Allergy & Asthma Reps. 306 (2008), filed as Ex. 75 (ECF No. 61-4); J. Hamilton, et al., *Dupilumab Improves the Molecular Signature In Skin of Patients With Moderate-to-Severe Atopic Dermatitis*, 134 J. Allergy Clin. Immunol. 1293 (2014), filed as Ex. 76 (ECF No. 61-5). Counsel argued that the papers established that an "overexpression" of interleukins or neutrophils could cause eczema itch. Tr. at 514, 515-17. Based on his on-the-spot review of the article, however, Dr. MacGinnitie posited that interleukins or neutrophils might only be "mediators" of pruritis, rather than causal of it. *Id.* at 517-20. The Hamilton article, counsel posited, reveals that eczema can be effectively treated by certain anti-inflammatory drugs (including Dupilumab which targets the Th2-centered inflammatory axis). *Id.* at 523-27. Indeed, Dr. MacGinnitie agreed that anti-inflammatories can successfully treat the condition. *Id.* at 527. In so agreeing, however, Dr. MacGinnitie maintained that anti-inflammatories like Dupilumab are commonly associated with eczema and food or environmental allergies. *Id.*

[58] Along those same lines, Dr. MaGinnitie posited that it is uncommon for patients to present with *both* eczema and urticaria (given the distinctions described above). Tr. at 509. It is even less common for eczema/urticarial patients to also have POTS. *Id.* Dr. MacGinnitie testified that he has never treated a patient with all three conditions. *Id.*

MacGinnitie testified that he knew of no causal link between vaccines and onset of allergies (a theory, he noted had been considered in the past, but not persuasively established). *Id.* at 466. As he explained, he has treated patients who experience an allergic reaction to a vaccine, but *none* who experience a worsening of allergy symptoms post-vaccination. *Id.* Indeed, he recommends that his allergy patients receive vaccinations (particularly those with asthma), as vaccines can prevent infections (and subsequent asthma exacerbations). *Id.*

In light of Petitioner's claim that her skin symptoms worsened following receipt of the HPV vaccine (along with some form of immune system dysregulation), Dr. MacGinnitie reviewed the relevant medical records for any evidence consistent with such a response. Leading up to Ms. McKown's syncopal episode at the frozen yogurt shop, Dr. MacGinnitie found no record evidence she possessed an abnormal immune system (beyond the possibility she had pre-existing environmental allergies). Tr. at 468. At most, the records established that Petitioner clearly had eczema (most likely in a mild form) as an infant, along with various preexisting environmental allergies. *Id.* at 467, 469. He also observed that a CT scan taken during the April 2013 hospitalization revealed evidence of a sinus infection (which could be a result of her allergy symptoms). *Id.* at 488.

Thereafter, Dr. MacGinnitie agreed, the post-vaccination records evidenced a worsening of eczema (and onset of hives) around May 2013 and into the summer. Tr. at 468. But he described Ms. McKown's eczema course as a "classic" case (i.e., it was "within the range of what's typically seen" with eczema patients). *Id.* at 512. Any worsening Petitioner experienced could be attributed to the "waxing and waning" nature of eczema – which, he posited, is normal for patients with the condition. *Id.* at 468-69. Petitioner's April 2014 skin biopsy was also consistent with eczema, along with the photographic evidence offered on the day of hearing. *Id.* at 473 (citing Ex. 1 at 93), 477.

The relevant medical records also establish that Petitioner tested positive for various environmental allergies around this time (including grass, cat dander, tree pollen, dust mite, and ragweed). Tr. at 470-71 (citing Ex. 3 at 11; Ex. 1 at 42). Dr. MacGinnitie opined that these allergies likely pre-dated her receipt of the vaccine doses at issue herein. *Id.* at 469, 471. By contrast, Dr. MacGinnitie could not say for certain if Petitioner's food allergy testing clearly evidenced positive values. *Id.* at 470-71. He deemed the majority of her allergy testing (related to strawberry, peach, oat, and so on) to be false-positives. *Id.* at 471.

During the Cleveland Clinic visit in August 2014, Petitioner was diagnosed with chronic urticaria (which was thought to have begun in the summer of 2013). Tr. at 473-74 (citing Ex. 11 at 12-14; Ex. 1 at 33), 532-33. The photographic evidence offered at hearing also revealed evidence of the condition. *Id.* at 478. Dr. MacGinnitie acknowledged this diagnosis, and noted that the treating physician categorized the condition as idiopathic (thus could not identify a cause). *Id.* at

474.[59] As noted earlier, Dr. MacGinnitie posited that physicians are rarely able to determine the cause of chronic urticaria (as it can be a diagnosis of exclusion, unrelated to environmental triggers). *Id.* But the testing completed during this visit (including CBC, anti-IgE, anti-IgE receptor antibodies, tryptase,[60] thyroid function, and inflammatory markers) clearly ruled out any autoimmune basis for the urticaria. *Id.* at 475-76, 545; *see* Ex. 22.

Along those same lines, Dr. MacGinnitie could find no clear etiology for the episodes of joint pain noted in Petitioner's medical record. Tr. at 478. Indeed, her rheumatologic work-up at the Cleveland Clinic also did not identify a clear trigger. *Id.* at 479 (citing Ex. 11 at 24-28). As Dr. MacGinnitie explained, the Cleveland Clinic treaters seemed to first attribute Ms. McKown's joint pain to her skin condition, but then later distinguished the two (or at least determined that the pain was secondary). *Id.* at 480. There was also no evidence of actual arthritis (or inflammation in the joints) versus "arthralgias" (or complaints of joint pain). *Id.* at 479. Even so, Dr. MacGinnitie posited that eczema and urticaria usually did not result in secondary joint pain or present with arthralgias. *Id.* at 479. Indeed, Dr. MacGinnitie testified that he has never treated a patient with eczema or urticaria *also* suffering from related joint pain (along with arthralgias, and myalgias). *Id.* at 511.

Before the Cleveland Clinic work-up, Petitioner's appointment with Dr. Rouster-Stevens in early July 2014 resulted in a diagnosis of "amplified musculoskeletal pain syndrome." Tr. at 481 (citing Ex. 21). Notably, however, Dr. Rouster-Stevens determined the condition was not autoimmune in nature. *Id.* at 481. According to Dr. MacGinnitie, the only proof of autoimmunity he could identify at this visit was a low ANA titer – which, he considered an unremarkable finding, and likely a low positive in any event. *Id.* Lupus and connective tissue disorder were also ruled out. *Id.*

All in all, based on his review of the record in its entirety, Dr. MacGinnitie could not conclude that Petitioner was experiencing an underlying inflammatory process. Tr. at 482. As he explained, the best markers for ongoing inflammation are the ESR and CRP (both of which remained relatively stable throughout Petitioner's symptoms course). *Id.* Her February 2014 lab test results showed only a slighted elevated ESR (which Dr. MacGinnitie deemed "unlikely of clinical importance" at the time) given that almost forty percent of the population have slightly elevated ANA (many of which do not have an underlying systemic disease). *Id.* at 483 (citing Ex.

[59] On cross examination, Petitioner offered some suggestion that the urticarial lesions were related to a medication she had taken in the past (Fludrocortisone). Tr. at 534. Dr. MacGinnitie acknowledged that the records indicated a concern for a reaction to the medication, but he posited that the chronic nature of symptoms described therein made it unlikely that Fludrocortisone triggered its onset. *Id.* at 535-36, 537.

[60] Along those same lines, Dr. MacGinnitie noted that Petitioner tested negative for mast cell activation disorder or mastocytosis (which he defined as an increased in the mast cells resulting in various symptoms including: flushing, urticaria, POTS, cognitive fog, and anaphylaxis). Tr. at 485, 486 (citing Ex. 11 at 21). Mast cell activation is measured by analyzing a patients tryptase levels. *Id.* at 485, 486; *see* C. Akin, *Mast Cell Activation Disorders*, 2 J. Allergy Clin. Immunol. Pract. 252 (2014), filed as Ex. A, Tab 21 (ECF No. 25-1).

1 at 77); *see* M. Satoh, *Clinical Interpretation of Antinuclear Antibody Tests in Systemic Rheumatic Diseases*, 19 Mod. Rheumatol. 219 (2009), filed as Ex. A, Tab 9 (ECF No. 23-10). Moreover, lab results from tests conducted in late summer 2014 were also negative for the traditional inflammatory markers. Tr. at 484. Dr. MacGinnitie agreed with Dr. Tornatore's earlier point that a patient could still have an autoimmune disease even absent positive inflammatory markers, but he would not opine that this was true for non-CNS illnesses of the kind relevant to this action. *Id.* at 482.

Dr. MacGinnitie next turned to the medical theories of causation proffered by Petitioner in support of her claim. As to Dr. Mikovits's theory, Dr. MacGinnitie noted that he found her opinion confusing. Tr. at 455. From what he could understand of it, Dr. Mikovits seemed to allege that various vaccine components (in combination) can lead to immune dysfunction in both the adaptive and innate systems. *Id.* Thus, Dr. Mikovits posited that the aluminum adjuvant component, for example, is "taken up" by macrophages, which then migrate to the CNS and "differentiate" into the microglia. *Id.* Retroviral DNA (another vaccine contaminant) was also mentioned in her theory, though Dr. MacGinnitie knew of no causative mechanism to support such a theory. *Id.* Dr. Mikovits similarly relied on the concept (explained primarily by Dr. Tornatore in reliance on the Kanduc paper) that molecular mimicry between HPV components and self sequence proteins could have contributed to the purported autoimmune process resulting in Petitioner's injuries. *Id.*

But Dr. MacGinnitie found Dr. Mikovits's theoretical components wholly unpersuasive and scientifically unreliable. For example, he maintained that he knew of no evidence supporting a causal connection between retroviral DNA and the onset of *any* disease process. Tr. at 455-56. As he explained, ten to fifteen percent of a person's own DNA is retroviral, rendering it unclear how the tiny amounts contained in vaccines would have any adverse effect (given that humans are routinely exposed to it in larger amounts via skin, gut, and everyday infections). *Id.* at 455-56.

Dr. Mikovits's aluminum adjuvant[61]/mimicry component of her theory, was, in his view, also scientifically unreliable. Dr. MacGinnitie found no evidence in the literature filed in this case that immune system macrophages can actually absorb the aluminum component in vaccines sufficient to cause injury, let alone migrate through the CNS and into the brain microglia, given the fact that the adjuvant originates in the muscle or subcutaneous tissue after injection. Tr. at 456-57, 498-99. Moreover, such a theory did not explain *how* such mechanisms could later result in POTS or eczema (or any medically acceptable disease process for that matter). *Id.* at 456-57, 500-01. And Dr. MacGinnitie greatly doubted the sense behind the theory that aluminum in vaccine could be pathogenic at all. As he explained, adjuvants have been used in vaccines for over one hundred years, and the applicable scientific studies have found no evidence that aluminum (or any other adjuvant) is unsafe. *Id.* at 497. Vaccines also contain miniscule amounts of aluminum

[61] Dr. MacGinnitie explained that adjuvants are included in vaccine ingredients to increase the immune response (or activate a low level of inflammation, so the immune response is higher). Tr. at 497-98.

(around 225 micrograms) in comparison to the amounts ingested/inhaled by humans on a daily basis. *Id.* at 498.[62]

Dr. MacGinnitie allowed that proinflammatory cytokines could both trigger and propagate a disease process in certain circumstances. Tr. at 540. He took issue, however, with Dr. Mikovits's suggestion that cytokines likely played a role in the disease/symptoms course relevant herein. *Id.* As he explained, the articles offered by Petitioner in support of such a theory show no evidence of a pathologic process. *Id.* In Dr. MacGinnitie's understanding, propagating (or mediating) a disease is not the same as *triggering* it. *Id.* He also pointed out that a cytokine response is an immediate product of the innate system's activation (and therefore not an inherently chronic process). *Id.* at 540. At most, cytokines can trigger some initial inflammation which would only later lead to a more "long-term" response in the adaptive system, rather than continuing to multiply in unabated form. *Id.* Thus, in Ms. McKown's case, an innate immune response even involving cytokine upregulation would not inherently cause the onset of an onset of injury months following activation (given the rapidity nature of its initial response). *Id.* at 532-33.

Dr. MacGinnitie similarly critiqued the joint mimicry/T-cell degeneracy theories proffered by Dr. Tornatore as inconsistent and vague. He began by noting that both could not be simultaneously explanatory. Tr. at 457. As he understood it, the concept of degeneracy involved T cells not specific to a presenting antigen nevertheless recognizing multiple antigens, leading to cross-reactivity[63] without molecular mimicry – a process that would be contradictory to a molecular mimicry mechanism (as purportedly supported by Kanduc) involving homology between specific HPV vaccine components and self proteins. *Id.* at 457, 489, 542. If degeneracy best explained the mechanism herein, then *any* T cell could be responsible for the alleged cross-reactivity, but it would be impossible to say which[64] – rendering degeneracy as an explanatory mechanism counter to the specificity underlying the mimicry theory. *Id.* at 489.

Even so, Dr. MacGinnitie disputed the reliability of degeneracy as a possible mechanism herein. Because in his view any T cell response would be "primary" (i.e., an immediate response attributable to the innate system), the fact that a T cell is stimulated initially by one antigen does not mean that the now activated T cell will by default respond to a "clone" thereafter (as Dr.

[62] On cross examination, Petitioner argued that injection with aluminum is not the same as ingesting or inhaling the substance. Tr. at 529. In response, Dr. MacGinnitie suggested a recent (unfiled) paper suggests that patients receiving immunotherapy are injected with higher doses of aluminum than in vaccination, and yet do not experience increased autoimmunity. *Id.* at 529-30. He agreed upon further questioning, however, that such evidence does not rise to the level of epidemiologic evidence in trustworthiness. *Id.* at 530.

[63] Bystander activation, by contrast, involves "one activated T cell activating other T cells" Tr. at 544.

[64] On cross examination, however, Dr. MacGinnitie agreed that humans "can't generate T cells to every possible antigen." Tr. at 542. He also agreed that there is reliable scientific support for the concept that T cells can recognize different distinct antigens even with a lack of sequence homology. *Id.*

Tornatore suggested). Tr. at 543.[65] For support, Dr. MacGinnitie referenced Kanduc, which he posited actually reveals that subsequent exposure to vaccine antigens results in a *less* robust response to variant flu wild virus types thereafter – as if memory of specific, original antigens "outcompete[s]" future antigens. *Id.* at 543.

As for the molecular mimicry theory as a mechanistic descriptor applicable herein, Dr. MacGinnitie did not dispute that Kanduc[66] correctly identified homology between the HPV vaccine and certain self proteins peptide sequences. Tr. at 489-90. The particular peripheral nervous system protein (septin-9) identified as having been the target in Kanduc, however, has *not* been established by independent reliable scientific or medical evidence to have anything to do at all with the pathogenesis of POTS. *Id.* at 490. Rather, septin-9 is related to a genetic disorder. *Id.* Moreover, Kanduc made no attempt to show that a human (or mouse) injected with HPV proteins could even generate an immune response to septin-9 in a controlled setting. *Id.* Thus, the theorized association between homologous peptides was lacking in sufficient scientific evidentiary support to deem it reliable. *Id.* at 489-90.

Along those same lines, Dr. MacGinnitie maintained that the homologies observed in Kanduc actually undermine the reliability of molecular mimicry as the most likely mechanism for Petitioner's injuries, citing literature that demonstrated the prevalence of homology between foreign antigens and amino acid sequences in the human body. Tr. at 490-92; B. Trost, et al., *Bacterial Peptides Are Intensively Present Throughout the Human Proteome*, 1 SelfNonself 71 (2010), doi:10.4161/self.1.1.9588, filed as Ex. G (ECF No. 43-2) ("Trost"). Trost identified various bacterial proteomes (both pathogenic and nonpathogenic) in an attempt to catalog homologous structures shared between the proteomes and self proteins. The study ultimately identified over 50,000 "ninemers" (or nine amino-acid regions of homology) or mimics shared between the proteomes and one-third of the human proteome. Tr. at 491. If molecular mimicry theory was indeed causally mechanistic for autoimmune disease in most cases, the "widespread overlap" between viral and bacterial proteins "would predict that autoimmune disease should have a much higher incidence than actually observed" (both in the total number of individuals affected and the number of autoimmune pathologies per individual). *Id.* at 492. Dr. MacGinnitie thus found it difficult to reconcile how molecular mimicry could *always* be considered potentially explanatory for onset of autoimmune disease, absent some other, more specific evidence suggesting it explained a particular disease process. *Id.* at 492-93.

[65] Indeed, Dr. MacGinnitie maintained that the body inherently "deletes" T cells that respond strongly to self antigens. Tr. at 493.

[66] Dr. MacGinnitie expressed some concern regarding Kanduc's publication credentials. Tr. at 489. In his view, the article was published in a "predatory" journal. *Id.* Predatory journals are considered open-access (i.e., they charge authors a price to publish the article) and are not effectively peer-reviewed prior to publication. *Id.* at 458-59. As he stated at hearing, both Kanduc (published in the *International Journal of Public Health and Safety*) and the Giannotta paper (published in *Bentham Open*), are in his view, predatory in nature. *Id.* at 459.

Overall, Dr. MacGinnitie argued that both proffered mechanisms relied on a similar assumption: that *any* stimulus from a foreign antigen could potentially cause an autoimmune disease. Tr. at 458. But if either mechanism was as broadly reliable as asserted, the incidence in autoimmune disease should be "orders of magnitude higher" (an increase unsupported by the available scientific literature) – suggesting that other factors (apart from "potentially autoreactive" T cells in isolation) are more likely involved in the relevant mechanism. *Id.* at 458, 544. In Dr. MacGinnitie's view, "across the range of immune stimuli that humans are exposed to on a daily basis, immunization is really not particularly notable" as such a potential factor. *Id.* at 487.

Dr. MacGinnitie went on to evaluate some of the evidence offered by Petitioner purportedly establishing a direct causal connection between the HPV vaccine and POTS, like case studies. Tr. at 493-94, 499-500. Although he allowed that such evidence had some limited reliability, the more sophisticated epidemiologic studies[67] considering such a relationship have consistently shown no discernible increase in POTS following HPV vaccine administration. *Id.* at 493-94. As a result, Dr. MacGinnitie felt it was more likely that any association between HPV vaccine and POTS observed in case reports was coincidental – attributable to the fact that the HPV vaccine is administered around the same time that POTS typically develops (in a young female population). *Id*.

One large-scale surveillance study performed in the U.S., for example, followed approximately 189,00 patients vaccinated with the HPV vaccine (between 2006 and 2008) to determine the likelihood of developing an autoimmune disease thereafter. Tr. at 494-95; C. Chao, et al., *Surveillance of Autoimmune Conditions Following Routine Use of Quadrivalent Human Papillomavirus Vaccine*, 271 J. Int'l Med. 193 (2012), filed as Ex. A Tab 6 (ECF No. 23-7) ("Chao"). Chao (funded but not authored by pharmaceutical manufacturer Merck & Co.) was a peer-reviewed observational study analyzing a database comprised of the medical histories of approximately 189,000 women in California to determine whether the studied population had developed a variety of autoimmune conditions after receiving the HPV vaccine. Chao at 194. The researchers compared the results of the studied vaccinated population with unvaccinated, similarly-situated individuals, in order to compare incidence ratios for the identified autoimmune conditions, but ultimately found no association between onset of autoimmune disease and receipt of the HPV vaccine. *Id.* at 194-95. Although Chao's authors did not specifically include POTS in the study criteria, Dr. MacGinnitie maintained that the article still cast doubt the concept that a vaccine can "break[] tolerance" sufficient to cause an autoimmune disease. Tr. at 495.

Dr. MacGinnitie also referenced two additional large-scale epidemiologic studies focusing on the HPV vaccine. Tr. at 495; L. Grimaldi-Bensouda, et al., *Autoimmune Disorders and Quadrivalent Human Papillomavirus Vaccination of Young Female Subjects*, 275 J. Int'l Med.

[67] Dr. MacGinnitie acknowledged that epidemiologic studies can never completely disprove causation, but he nevertheless felt they provide better evidence of causality (or the lack thereof) than the associations observed in case reports. Tr. at 494.

398 (2014), filed as Ex. A, Tab 7 (ECF No. 23-8) ("Grimaldi-Bensouda"); S. Block, et al., *Clinical Trial and Post-Licensure Safety Profile of Prophylactic Human Papillomavirus (Types 6, 11, 16, and 18) L1 Virus-Like Particles*, 29 Pediatric Infect. Dis. J. 95 (2010), filed as Ex. A, Tab 8 (ECF No. 23-9) ("Block"). Grimaldi-Bensouda was a French case control study of roughly 1,800 patients (22 percent of whom had received the HPV vaccine), which measured the onset of specific autoimmune diseases (including: ITP, CN/MS, GBS, lupus, rheumatoid arthritis, type I diabetes, and autoimmune thyroiditis) following HPV vaccine administration. The Grimaldi-Bensouda authors found no increased rate of autoimmunity following vaccination. Tr. at 496. The Block study monitored over 21,000 female patients administered HPV in five clinical trials, and beyond some evidence of injection site pain following administration, observed no association with any subsequent autoimmune phenomena. Tr. at 496; Block at 100.[68] As with Chao, however, Dr. MacGinnitie acknowledged on cross examination that neither Grimaldi-Bensouda or Block included POTS (or dermatologic conditions) when measuring for autoimmunity. Tr. at 528. Even so, given the lack of evidence associating HPV vaccine administration with other autoimmune conditions, Dr. MacGinnitie posited that it was unlikely that POTS (which Petitioner has alleged was autoimmune in nature with respect to herself) could be similarly associated.

III. Procedural History

Ms. McKown filed her Petition on December 1, 2015. Pet. at 1. Following the filing of pertinent medical records, Respondent filed the Rule 4(c) Report on June 28, 2016 (ECF No. 15), contesting Petitioner's right to an entitlement award. I thereafter ordered the parties to file expert reports in support of their respective positions. Petitioner filed an initial report from Drs. Mikovits and Ruscetti on December 2, 2016 (ECF No. 18). Respondent's initial report from Dr. MacGinnitie was next filed on April 14, 2017 (ECF No. 23). Supplemental expert reports were filed on July 19, 2017 (ECF No. 30) and May 4, 2018 (ECF No. 34), respectively. Congruently, on May 4, 2018, Respondent filed a report authored by Dr. Gibbons (ECF No. 35), plus Dr. MacGinnitie's supplemental report.

Thereafter, Petitioner indicated in her initial pre-hearing brief that she intended to call Dr. Tornatore as a witness at hearing (and file a report in the matter). *See* Brief, dated July 13, 2018 (ECF No. 40) at 1-2. This suggestion was made *long* after the original expert report filing had concluded (and *without* my direction or approval). Petitioner did not indicate *why* she intended to offer a supplemental report in the matter (though it likely was viewed as necessary to remedy

[68] On direct examination, Dr. Mikovits suggested that clinical trials conducted prior to the release of the HPV vaccine did not include a trial completed with a non-aluminum placebo. Tr. at 496. In response, Dr. MacGinnitie posited that Block used a non-aluminum placebo to measure for autoimmunity but found no evidence of an increase. *Id.* at 496-97.

issues relating to the qualifications of Drs. Mikovits and Ruscetti).[69] Nonetheless, Petitioner filed the report from Dr. Tornatore on July 31, 2018 (ECF No. 42), roughly two months prior to hearing.

I scheduled the matter for hearing on September 26-27, 2018. The hearing took place as scheduled, and included testimony from the experts identified above (along with testimony from Petitioner's mother). Following the hearing's conclusion, the parties submitted post-hearing briefs. The matter is ripe for adjudication.

IV. Applicable Legal Standards

A. *Claimant's Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury" – i.e., an injury falling within the Vaccine Injury Table, corresponding to one of the vaccinations in question and also occurring within a statutorily-prescribed period of time – or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; *see also Shalala v. Whitecotton*, 514 U.S. 268, 270 (1995) (quoting 42 U.S.C. § 11(c)(1)(C)(i)); *Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006).[70] Petitioner in this case asserts only a non-Table claim.

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

[69] By this time, other special masters had authored opinions in different cases critiquing the quality of the opinions offered by Drs. Mikovits and Ruscetti (and/or expressed their view that the testimony offered was unpersuasive in the causation context). *See, e.g.*, *McCabe v, Sec'y of Health & Human Servs.*, No. 13-570V, 2018 WL 3029175 (Fed. Cl. Spec. Mstr. May 17, 2018); *Barker v. Sec'y of Health & Human Servs.*, No. 16-1554V, 2018 WL 2772454 (Fed. Cl. Spec. Mstr. May 11, 2018); *Dominguez v. Sec'y of Health & Human Servs.*, No. 12-378V, 2018 WL 2225540 (Fed. Cl. Spec. Mstr. Apr. 2, 2018).

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

When a Table Injury claim is successfully established, causation is presumed. 42 C.F.R. § 100.3. Table claims must satisfy with evidence the specific elements of the relevant claim, including the definitions of terms set in the Qualifications and Aids to Interpretation (the "QAI"). Section 14(b). Case law underscores that, to obtain the benefit of the presumption of causation associated with a Table claim, the claim's requirements must be strictly construed. *Miller v. Sec'y of Health & Human Servs.*, No. 02-235V, 2015 WL 5456093, at *24 (Fed. Cl. Spec. Mstr. Aug. 18, 2015) (requiring petitioner to satisfy the "strict Table definition" of encephalopathy).

For a non-Table claim, proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In such circumstances, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner asserting a non-Table claim must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health & Human Services*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury."

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

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Human Servs.*, 121 Fed. Cl. 230, 245 (2015), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017).

In discussing the evidentiary standard applicable to the first *Althen* prong, many decisions of the Court of Federal Claims and Federal Circuit have emphasized that petitioners need only establish a causation theory's biologic plausibility (and thus need not do so with preponderant proof). *Tarsell v. United States*, 133 Fed. Cl. 782, 792-93 (2017) (special master committed legal error by requiring petitioner to establish first Althen prong by preponderance; that standard applied only to second prong and petitioner's overall burden); *Contreras*, 121 Fed. Cl. at 245 ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)); *see also Andreu*, 569 F.3d at 1375. At the same time, there is contrary authority from the Federal Circuit suggesting that the preponderance standard applied when evaluating a claimant's overall success in a Vaccine Act claim also bears on the first *Althen* prong. *See, e.g.*, *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1350 (Fed. Cir. 2010) (affirming special master's determination that expert "had not provided a 'reliable medical or scientific explanation' *sufficient to prove by a preponderance of the evidence a medical theory* linking the [relevant vaccine to relevant injury]") (emphasis added). Regardless, one thing remains: petitioners always have the burden of establishing their Vaccine Act claim *overall* with preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell*, 133 Fed. Cl. at 793 (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct – that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be

weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 136 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *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 also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id*. at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & 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).

B. *Law Governing Claims of Significant Aggravation*

Besides arguing that the HPV and/or Hep A vaccines directly caused her injuries, Petitioner's experts have also allowed for the possibility that the vaccines significantly aggravated her preexisting eczema or previously-undiagnosed POTS. Where a petitioner so alleges, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *See generally Loving v. Sec'y of Health & Human Servs*., 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Secretary of Health & Human Services*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6)

> a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

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

Subsumed within the *Loving* analysis is the requirement to evaluate the likely natural course of an injured party's preexisting disease, in order to determine whether the vaccine made the petitioner worse than he would have been but for the vaccination. *Locane v. Sec'y of Health & Human Servs.*, 685 F.3d 1375, 1381–82 (Fed. Cir. 2012) (upholding special master's determination that petitioner had failed to carry her burden of proof in establishing that her preexisting injury was worsened by the relevant vaccine); *Hennessey v. Sec'y of Health & Human Servs.*, No. 01-190V, 2009 WL 1709053, at *41-42 (Fed. Cl. Spec. Mstr. May 29, 2009), *mot. for review den'd*, 91 Fed. Cl 126 (2010). The critical point of examination is thus "whether the change for the worse in [petitioner's] clinical presentation was aggravation or a natural progression" of the underlying condition. *Hennessey*, 2009 WL 1709053, at *42.[71] The Federal Circuit has upheld the determinations of special masters that worsening was not demonstrated in connection with establishing a petitioner's overall preponderant burden of proof for a non-Table causation-in-fact claim. *See, e.g., Snyder/Harris v. Sec'y of Health & Human Servs.*, 553 F. App'x 994, 999-1000 (Fed. Cir. 2014); *Locane*, 685 F.3d at 1381-82.[72]

The mere fact a vaccine might "trigger" a transient negative response in an individual with an underlying condition or disease is not proof of worsening if that individual would be expected to experience a similar overall course regardless. *Faoro v. Sec'y of Health &Human Servs.*, No. 10-704V, 2016 WL 675491, at *27 (Fed. Cl. Spec. Mstr. Jan. 29, 2016), *mot. for review den'd*, 128 Fed. Cl. 61 (Fed. Cl. Apr. 11, 2016) (finding that "the vaccinations would not have changed her clinical course and thus, the vaccinations did not significantly aggravate her preexisting

[71] The legislative history of the Vaccine Act strongly supports interpreting "significant aggravation" as requiring a claimant to establish that a vaccine rendered a preexisting condition qualitatively worse than it would have been otherwise – not simply that the affected individual experienced a post-vaccination symptom that contrasts with the individual's comparatively better pre-vaccination health. *See* H.R. Rep. No. 99-908, at 15 (1986) ("This [significant aggravation] provision does not include compensation for conditions which might legitimately be described as pre-existing (e.g., a child with monthly seizures who, after vaccination, has seizures every three and a half weeks), *but is meant to encompass serious deterioration* (e.g., a child with monthly seizures who, after vaccination, has seizures on a daily basis" (emphasis added)).

[72] This is consistent with the fact (well recognized by controlling precedent) that evidence of "worsening" relevant to Respondent's alternative cause burden may reasonably by evaluated by a special master in determining the success of a petitioner's prima facie showing. *Snyder/Harris*, 553 F. App'x at 1000, *quoting Stone*, 676 F.3d at 1380 ("no evidence should be embargoed from the special master's consideration simply because it is also relevant to another inquiry under the statute"); *see also de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("[t]he government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner's evidence on a requisite element of the petitioner's case-in-chief").

condition"). This point has been emphasized in a subcategory of Program cases involving the claim that a child's Dravet syndrome (a rare seizure disorder now understood to be caused by the SCN1A gene mutation) was significantly aggravated by vaccination. *Faoro*, 2016 WL 675491, at *1. In such cases, special masters have repeatedly determined that petitioners failed to show that a child's expected outcome would have been different but for the vaccination – even though it was not disputed that the child's first major seizure had been triggered by vaccination. *Id.* at *2 ("[a]lthough H.E.F.'s vaccinations may have caused a low-grade fever or otherwise triggered her first seizure, neither the initial seizure nor her vaccinations caused or significantly aggravated her Dravet syndrome and resulting neurological complications"); *see also Snyder/Harris*, 553 F. App'x at 1003 (special master was not arbitrary in finding that petitioners' expert failed to show that the child's outcome would have been different had he not received the vaccinations at issue).

C. *Law Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as "the results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such a determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"); *Rickett v. Sec'y of Health & Human Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs*., No. 11-685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10,

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

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs*., No. 03-1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Human Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd*, 968 F.2d 1226 (Fed. Cir.), *cert. den'd sub nom. Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight")).

However, there are situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Human Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

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

2014). In deciding whether to afford greater weight to contemporaneous medical records over contrary testimony, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

D. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc*., 509 U.S. 579, 59-96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592-95).

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

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen*, 618 F.3d at 1347 (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl.

Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339).

E. *Consideration of Medical Literature*

Both parties relied on significant amounts of medical and scientific literature to support their respective positions. I have reviewed all of the medical literature submitted in this case, although my decision does not discuss each filed article in detail (nor would it be reasonable to require a special master to do so – especially in a case like this, where far more literature than was necessary has been filed). *Moriarty v. Sec'y of Health & Human Servs.,* 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted).

ANALYSIS

I. Overview of POTS, Eczema, and Relevant Prior Decisions

The parties do not dispute the validity of Petitioner's POTS diagnosis, or that she suffered from eczema before vaccination. Some discussion of what is known about these illnesses/conditions, and/or how they have been addressed in prior cases, will help elucidate some of the grounds for my decision in this case.

POTS

POTS is a *subset* of orthostatic intolerance, not a wholly separate clinical entity. Benarroch at 1214-15. It is marked by an increase in heart rate, or tachycardia, caused by a change in body position from the supine position to the upright position, without an accompanying increase in blood pressure, and results in feelings of dizziness and lightheadedness, as well as fatigue, headache, and exercise intolerance. Gibbons at 1-2; Benarroch at 1214. Thus, a claimant alleging a vaccine injury of POTS is arguing that the relevant vaccine has done *something* to the autonomic system sufficient to cause a chronic aberrant response to orthostatic change. The relevant literature does not indicate that POTS has ever been associated with onset of skin rashes or the exacerbation of an existing skin condition. *See generally* Thieben; Butts.

There are several POTS variants with different possible etiologies, although research has not conclusively established any one as explanatory for the majority of POTS cases. Benarroch at 1214. POTS is often the secondary result of other conditions – lower limb blood pooling, hypovolemia (meaning decreased blood plasma), or deconditioning due to inactivity (as Dr. Gibbons best explained). Thieben at 308. Two other proposed etiologies for POTS are a hyperadrenergic state (meaning elevated norepinephrine concentrations) leading to tachycardia, or

a neuropathic variant resulting from autonomic nerve fiber damage. *Id.* at 308, 313; Benarroch at 1214-15. POTS is more commonly experienced by women. Benarroch at 1214.

A small subset of POTS cases may be autoimmune-mediated. *See, e.g.*, Thieben at 311, 313 (noting that six of forty-two patients tested positive for a particular ganglionic antibody); Gibbons at 1-2; Benarroch at 1215-16. It appears from more recent studies, however, that researchers have not embraced autoimmunity as the most likely explanation for POTS in the majority of individuals, and even Thieben allows that autoimmune-implicated POTS would *not* be the most common way in which it occurs – and if it did, would be accompanied by evidence of "sympathetic denervation." Thieben at 312-13; *see also* Butts at 957. Importantly, an individual suffering from an autonomic neuropathy mediated by autoimmunity would have a number of presenting symptoms, *in addition to* orthostatic tachycardia, revealing harm to the autonomic nervous system. Gibbons at 5-7.

I have had the opportunity several times to consider Vaccine Program claims alleging that POTS, or other forms of orthostatic intolerance, was attributable to vaccination. *See generally Yalacki v. Sec'y of Health & Human Servs.,* No. 14-278V, 2019 WL 1061429 (Fed. Cl. Spec. Mstr. Jan. 31, 2019), *appeal docketed*, No. 14-278V (Fed. Cl. Mar. 4, 2019); *Johnson v. Sec'y of Health & Human Servs.*, No. 14-254V, 2018 WL 2051760 (Fed. Cl. Spec. Mstr. Mar. 23, 2018); *Combs v. Sec'y of Health & Human Servs.*, No. 14-878V, 2018 WL 1581672 (Fed. Cl. Spec. Mstr. Feb. 15, 2018). In all, I have found that the petitioner had not met the burden of proof – primarily because the claimant failed to successfully establish, with preponderant evidence, a reliable scientific association between *any* vaccine and POTS, let alone the HPV vaccine.

In *Johnson*, a young woman alleged that the HPV vaccine caused her POTS that had been diagnosed several years after receipt of the vaccine. *Johnson*, 2018 WL 2051760, at *7 n.11, *26 n.35. In determining that the petitioner had not established a reliable medical causation theory, I found that she failed to demonstrate that POTS is more often than not autoimmune in origin. *Id.* at *24-25. I also determined that the petitioner's overall disease course, measured from the date of the alleged causal vaccination to the time her symptoms were thought to possibly reflect POTS, was simply too meandering and lengthy to deem it a medically reasonable timeframe for vaccine causation. *Id.* at *22-25.

Combs, by contrast, involved only the claim that a young woman developed syncope well after receipt of the HPV vaccine. *Combs*, 2018 WL 1581672, at *1. Respondent offered persuasive evidence about the autonomic nervous system and discussed the same kind of orthostatic intolerance issues raised in this case. After a hearing and full consideration of the evidence and expert testimony, I found that the petitioner had not established that her condition arose from damage to the autonomic nervous system (or brain microglia as alleged), since there was no such evidence in the record to support that contention, nor that her syncope was likely vaccine-caused. *Id.*

In *Yalacki*, a petitioner alleged that the Hep B vaccine caused her to suffer from POTS and/or chronic fatigue. *Yalacki*, 2019 WL 2019 WL 1061429, at *1. In contrast to the above-noted cases, Respondent in *Yalacki* raised valid concerns regarding the diagnosis best supported by the medical record (thereby suggesting petitioner had neither disease) – although there was credible evidence supporting both injuries as having occurred. *Id.* at *35. Even so, in determining that the young woman in *Yalacki* had not established a reliable medical theory of causation, I found that she had not established that her POTS course (assuming she in fact had it) was consistent with what would be expected to occur for the neuropathic variant of POTS – something she had to establish in order for the vaccine to have been causal. *Id.* at *34. Petitioner's significant orthostatic symptoms prior to vaccination also suggested that her POTS, if it existed, *predated* vaccination, but was not exacerbated by it. *Id.* at *37.

Such decisions certainly do not dictate the outcome of this case. However (and given the congruity of evidence offered in such cases with that filed herein), they do demonstrate the existing lack of up-to-date, persuasive scientific evidence associating the HPV vaccine with significant orthostatic intolerance (beyond recognized, close-in-time reactions like syncope, which is itself a Table claim for certain vaccines (42 C.F.R. § 100.3 VIII(C) (2018)), as well as the kind of hurdles a petitioner faces in attempting to obtain an entitlement award based on such a theory. Too often, such claimants propose a theory dependent on establishing (a) that their POTS was the neuropathic, autoimmune-mediated variant, and (b) that the relevant vaccine could initiate the process resulting in POTS, when both the medical record, and existing medical and scientific literature, do not support either contention.

Eczema

Program cases alleging eczema/atopic dermatitis as the injury have typically involved allegations of other disease processes as well. The majority of petitioners have combined an eczema injury claim with the argument that food allergies, or developmental/social delays (and autism), were also vaccine-caused, but none have succeeded. *See, e.g.*, *Gilmore v. Sec'y of Health & Human Servs.*, No. 17-2026V, 2019 WL 1468203 (Fed. Cl Spec. Mstr. Feb. 26, 2019) (dismissed for failure of proof); *A.W. v. Sec'y of Health & Human Servs.*, No. 15-1568V, 2018 WL 1150730 (Fed. Cl. Spec. Mstr. Feb. 1, 2018) (food allergies and eczema not caused by trace amount of food proteins in vaccines). A few eczema cases (some of which are combined with other injuries) have, however, resulted in settlement. *See, e.g.*, *Williams v. Sec'y of Health & Human Servs.*, No. 15-1224V, 2019 WL 994570 (Fed. Cl. Spec. Mstr. Jan. 29, 2019); *Parker-Winter v. Sec'y of Health & Human Servs.*, No. 13-150V, 2014 WL 657714 (Fed. Cl. Spec. Mstr. Jan. 23, 2014).

II. Petitioner Has Not Established Her Claim with Sufficient Preponderant Evidence

A. *Petitioner's POTS and Eczema Most Likely Preceded Her March 2013 Vaccinations*

Although Ms. McKown argues in the main that the HPV vaccine caused her injuries, she also (primarily through the testimony of Dr. Tornatore) proposed that the vaccine could have exacerbated pre-existing POTS or her long-standing but mild eczema. The existing medical record best supports a significant aggravation claim for most of her claimed injuries, because that record suggests both conditions existed at the time Petitioner received the first HPV vaccine dose in March 2013.

There is no dispute that Petitioner had already been diagnosed with mild eczema before receiving the HPV vaccine, and had been treated for it since a young age. Accordingly, Petitioner can only succeed in a claim arguing that her eczema was significantly aggravated by vaccination through satisfaction of the *Loving* prongs. (I do, however, treat urticaria separately below as a potentially direct vaccine-caused injury, based on testimony at hearing from Dr. MacGinnitie distinguishing it from eczema, and the fact that Petitioner does not appear to have suffered from hives before receipt of the HPV vaccine).

Petitioner's POTS-based claim also must be considered as a significant aggravation claim in light of the medical record. The literature filed in this case (along with existing Program caselaw) supports the conclusion that POTS progresses over a several-month period, and often precedes formal diagnosis for some time given the difficulties in discerning its symptoms. *See* Tr. at 319; Benarroch at 1214 (describing POTS as "chronic"); Thieben at 309 (POTS inclusion criteria required the presence of symptoms for "more than" three months); *see also Johnson*, 2018 WL 2051760, at *22. Thus, the very fact that Ms. McKown received her POTS diagnosis in May 2013 from Dr. Dooley – within two months of receiving the first HPV vaccine dose – is almost too close in time to her receipt of the first HPV vaccine dose to be deemed causal.

More significantly, the record establishes by May 2013 Petitioner had already experienced *three* syncopal occurrences, two of which preceded the first HPV dose. And Dr. Tornatore (whose knowledge of POTS from treating MS patients unquestionably does not render him an expert in POTS or the autonomic nervous system) was unpersuasive in his effort to distinguish these earlier episodes as phenotypically different from what otherwise would be deemed incidents of vasovagal syncope. Indeed, these two prior incidents were raised as a concern *at the time* Petitioner received her first HPV vaccine dose, and were also the basis for her referral to the cardiologist, Dr. Dooley, who provided a POTS diagnosis that was substantiated in the proper manner (with a tilt table test). *See* Ex. 1 at 47; Ex. 7 at 5. Accordingly, I find on this record that it is more likely than not that

Petitioner's POTS began before March 2013 – meaning any claim that the HPV vaccine played a role in her subsequent course is properly analyzed under *Loving.*[73]

B. *Petitioner Has Not Established a Plausible Causation Theory* (*Loving Prong 4)*

1. Petitioner's Experts Were Unpersuasive or not Credible

Before discussing the specific merits of Petitioner's theory, it is appropriate to note the significant competence and/or qualifications gap separating both sides' experts. Respondent offered two experts, one of whom (Dr. Gibbons) possessed direct practice experience treating patients with POTS (and more generally evaluating autonomic disorders associated with the condition). Both were credentialed, well-qualified to speak on the issues in dispute, and offered compelling and persuasive testimony, especially in their willingness to concede points that were helpful to Petitioner.

Petitioner, by contrast, initially relied on the report jointly authored by Drs. Mikovits and Ruscetti – but then seems to have decided to hedge her position by adding Dr. Tornatore close in time to the hearing. Having now heard Dr. Mikovits testify, the prudence of that decision is fairly evident.

Other special masters have harshly criticized the reliability and validity of the opinions Drs. Mikovits and Ruscetti have offered – some going so far as to refuse to compensate them for their time, based on the determination that their opinions were unhelpful in resolving a particular case. *See, e.g., Dominguez v. Sec'y of Health & Human Servs.*, No. 12-378V, 2018 WL 3028975 (Fed. Cl. Spec. Mstr. May 25, 2018) (awarding Dr. Mikovits a rate of only $75 per hour for work completed on an expert report after determining she did not assist resolution of the matter); *McCabe v. Sec'y of Health & Human Servs.*, No. 13-570V, 2018 WL 3029175, at *20-21 (Fed. Cl. Spec. Mstr. May 17, 2018) (criticizing Dr. Mikovits for diagnosing medical conditions, absent any medical license or training, and incorrectly stating medical definitions); *Barker v. Sec'y of Health & Human Servs.*, No. 16-1554V, 2018 WL 2772454, at *1-2 (Fed. Cl. Spec. Mstr. May 11, 2018) (critiquing Drs. Mikovits and Ruscetti for proffering theories based on "misleading references to medical literature"); *Rogero v. Sec'y of Health & Human Servs.*, No. 11-770V, 2017 WL 4277580, at *23-24 (Fed. Cl. Spec. Mstr. Sept. 1, 2017) (stating Dr. Mikovits's opinion regarding vaccine causation was "never clearly or coherently explained" at hearing), *mot. for review den'd*, slip op. (Fed. Cl. Jan. 11, 2018), *aff'd*, 748 F. App'x 996 (Fed. Cir. 2018).

At hearing, Dr. Mikovits lived up to her reputation. She was dismissive and conclusory in attempting to explain away the many embarrassing episodes from her professional life. Indeed, the mere fact that she had to recount them at all harmed her credibility from the outset. *See, e.g.*, Tr. at 217 ("[Whittemore] fired me . . . [and] I get a letter from a lawyer saying you've stolen our

[73] Below I address only those *Loving* prongs most relevant to my Decision.

intellectual property and data and, basically, defend yourself"), 219 ("I was held [in jail] on something called a fugitive from justice"), and 259 ("[t]he Whittemores said I manipulated a figure in order to cover up their misappropriation of federal funds"). The picture painted by these occurrences in Dr. Mikovits's professional history was too damaging to ignore. An expert's professional past is a relevant consideration when evaluating how much weight to give her testimony, since it reflects on her overall candor. *See, e.g.*, *Cox v. Sec'y of Health & Human Servs.*, 30 Fed. Cl. 136, 144 (1993) ("expert's criminal background[] raised issues of weight and not admissibility"); *Yalacki*, 2019 WL 1061429, at *12 n.18 (fact that expert-authored publication was later retracted impacted expert's credibility); *Weppler v. Sec'y of Health & Human Servs.*, No. 12-316V, 2014 WL 4057149, at *2 (Fed. Cl. Spec. Mstr. July 25, 2014) (physician's prior "convictions for false statements . . . [and] fraud cast serious doubt on his credibility").

Other special masters have disregarded expert opinions entirely for comparable improper conduct. *See, e.g.*, *Raymo v. Sec'y of Health & Human Servs.*, No. 11-0654V, 2014 WL 1092274, at *13-16 (Fed. Cl. Spec. Mstr. Feb. 25, 2014) (rejecting entire medical opinion as unreliable where expert credibility issues included a plagiarized report and a failure to reveal a medical license suspension). Based on what was disclosed about her past at hearing that undermined her professional competency on immunologic matters, coupled with her lack of specific expertise in studying or treating either of the conditions at question, I arguably could have simply determined that Dr. Mikovits's credibility was so damaged that her opinion was entitled to virtually no weight at all.[74] *See, e.g., Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1995) (concluding that it was reasonable for the special master to use *Daubert* to evaluate reliability of an expert's testimony); *Ryman v. Sec'y of Health & Human Servs.*, 65 Fed. Cl. 35, 40-41 (2005) (special masters perform gatekeeping function when determining "whether a particular petitioner's expert medical testimony supporting biological probability may be admitted or credited or otherwise relied upon").

However, despite such sound reasons to ignore her opinion, fairness to the Petitioner compelled me to evaluate Dr. Mikovits's opinion for its substance. Even then, her opinion was wanting. She offered a medical/scientific causation theory that was poorly-supported and utterly confusing (as Respondent's experts recognized) – a disorganized hodge-podge of scientific principles strung together without the persuasive connective "glue" that a competent and knowledgeable expert would provide. And her opinion relied heavily on ASIA as a mechanistic explanation for how the vaccines Petitioner received could have injured her - despite the fact that Petitioner clearly stated she would *not* be offering ASIA as a component of her case. *See* Pre-Hearing Reply (ECF No. 52) at 1 ("while Drs. Mikovits and Ruscetti reference ASIA, neither they nor Dr. Tornatore assert ASIA as their theory of causation"); Tr. at 6 ("the ASIA literature informs

[74] In addition, and as discussed earlier, Dr. Mikovits's expert reports were co-authored (and signed) by Dr. Ruscetti, although only Dr. Mikovits testified at hearing. Dr. Mikovits has been criticized in past Program cases for the practice of testifying alone regarding co-authored reports. *See, e.g.*, *Rogero v. Sec'y of Health & Human Servs.*, No. 11-770V, 2017 WL 4277580, at *23 (Fed. Cl. Spec. Mstr. Sept. 1, 2017), *mot. for review den'd*, slip op. (Fed. Cl. Jan. 11, 2018), *aff'd*, 748 F. App'x 996 (Fed. Cir. 2018).

the medical theory, but it's not the – it's not the basis for the medical theory"), 8, 294. Overall, such substantive deficiencies equaled, if not exceeded, the many professional lapses that would otherwise justify giving Dr. Mikovits's opinion little to no weight.

My comments on Dr. Mikovits largely do not apply to Dr. Tornatore – a well-credentialed expert with none of Dr. Mikovits's substantial professional lapses. He frequently offers fair, articulate opinions in Program cases, and has served as an expert in many cases I have adjudicated. But *in this case*, Dr. Tornatore was the wrong man for the job. His unquestionable expertise in treating CNS autoimmune illnesses like MS was not accompanied by comparable expertise in *specifically* treating or studying POTS or eczema, especially when compared to Drs. Gibbons and MacGinnitie. He thus could not leverage his personal expertise into a persuasive opinion on POTS or skin conditions, the interaction between the two, and/or the relationship of either to the HPV vaccine. He largely seems to have offered an opinion heavily reliant on just two independent items of literature (Kanduc and Voisin), neither of which were deemed important enough by Petitioner or her counsel to file in this case before the very eve of trial.

Overall, Petitioner's expert showing was almost facially inadequate in helping her meet her preponderant burden of proof – especially when judged against the far more reliable and scientifically persuasive showing made by Respondent's experts. Although (again – in the interests of fairness to the Petitioner), I review below the actual merits of the theories these experts presented, it is important to emphasize that Petitioner's experts did not *themselves* imbue her causation theory with evidentiary heft based on their own expertise – and in Dr. Mikovits's case greatly *undermined* whatever probative value the expert opinion she offered had on its own.

2. Petitioner's Causation Theory was Unreliable and Unpersuasive

Having reviewed each side's expert reports and medical literature, and considered the testimony at hearing, I find that Petitioner has not presented a plausible theory, supported by sufficient reliable evidence, that the HPV vaccine can cause, or significantly aggravate, either POTS or eczema.

An overarching deficiency in Petitioner's theory is the supposition that her primary, complained-of injuries – POTS and skin conditions variously characterized as eczema or urticarial lesions/hives – were more likely than not autoimmune in origin. Petitioner specifically proposed that a neuropathic form of POTS could be mediated by autoantibodies produced in response to the implicated vaccine, and thus via an adverse autoimmune process. Admittedly, there is literature support (such as Thieben, Gibbons, and Benarroch) for the idea that one *particular variant* of autonomic neuropathy producing POTS symptoms might be associated with a particular autoantibody, thereby suggesting autoimmunity as a plausible pathologic mechanism.

Dr. Gibbons (whose direct experience studying the etiology of POTS far outweighed that of Drs. Tornatore and Mikovits), however, persuasively established that the autoimmune-

mediated, neuropathic form of POTS is *very rare*, occurring only where an individual possesses a specific ganglionic autoantibody (which Ms. McKown was never demonstrated to possess). The literature filed with regard to the neuropathic variant, as Dr. Gibbons posited, best supports the conclusion that this POTS variant is far less common from those deemed non-neuropathic in etiology. And Drs. Tornatore and Mikovits in no way undercut Dr. Gibbons's testimony (corroborated by several items of literature) that the other POTS variants clearly are *not* autoimmune in etiology, such as hyperadrenergic POTS or POTS due to deconditioning, or that these are more common explanations for POTS (which, unlike the neuropathic form, are *more* likely to feature syncope of the sort Petitioner actually experienced). Gibbons at 1-10; Tr. at 353-54, 419-20.

The remaining scientific evidence offered by Petitioner suggesting POTS is typically autoimmune was thin, relying on case reports – a kind of evidence not given significant weight in Program cases. *See, e.g.*, *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011) ("[c]ase reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value"). And the evidence linking POTS to other forms of autoimmune disease (*see, e.g.*, Dahan, Cerpa-Cruz, Giannotta) was too inconclusive, or was rooted in accepting the abandoned ASIA component of Petitioner's causation theory. I thus do not find that POTS is more often than not autoimmune in origin – greatly reducing the likelihood that a vaccine could initiate an autoimmune process sufficient to cause it.

A secondary weakness of Petitioner's theory was Dr. Tornatore's unsuccessful linkage of POTS with eczema. He proposed that the two could interact via some kind of nervous system feedback loop, in which the same neuropathic injury could produce both the orthostatic symptoms of POTS as well as skin-related symptoms. But the primary literature discussing POTS says nothing about any association between POTS and any symptoms comparable to eczema or urticaria. *See, e.g.*, Thieben; Butts. To advance this argument in the face of what the medical community understands about POTS at present, Dr. Tornatore had to stretch certain items of literature well beyond their actual scope. Voisin, for example, was offered to support the contention that nerve damage (purportedly the result of an autoimmune cross-reaction) could not only be associated with POTS but with skin injuries as well. But, as noted earlier, Voisin discusses only allergic inflammation – it makes no mention of POTS (or the production of autoantibodies associated with neuropathic variant) or its pathogenesis, nor does it attempt to relate autoimmune disease to allergies more generally. At bottom, this element of Petitioner's theory over-relied on the fact that the nervous system *does* play some role in adverse skin processes like eczema or urticaria – even though, as Dr. MacGinnitie established, they cannot be assumed to be allergic in nature, and are not primarily autoimmune in character either.

Next, even if it is granted that the rare, neuropathic, autoimmune form of POTS could include symptoms consistent with skin rashes (while also featuring syncope -*despite* the fact, as established by Dr. Gibbons, that neuropathic POTS generally does not as it progresses), there

remain substantial deficiencies in Petitioner's theory that the HPV vaccine could trigger or exacerbate such symptoms via an autoimmune process. For a mechanism[75] by which this could occur, Dr. Tornatore mostly embraced molecular mimicry, relying heavily on Kanduc (an article that was only deemed important in the days before trial – and which Respondent reasonably questioned as unreliable simply on the basis of its source of publication). As discussed above, Kanduc examined the HPV16 polyprotein and recorded amino acid sequence similarities to the human proteome at the heptamer level, concluding that the proteome contains both heptapeptides and octapeptides (or enough sequence homology) with the vaccine sufficient to establish *potential* cross-reactive sequences. But Kanduc did *not* test the theoretical cross-reactivity between the sequences established, nor did it provide any meaningful evidence that the proposed homologous sequences were in any way pathogenic. And no other evidence was offered suggesting that any of the self homologous sequences are key to the process by which autoimmune neuropathic POTS occurs.

Thus, in advancing the contention that molecular mimicry constitutes a reliable scientific mechanism by which the HPV vaccine could cause or exacerbate POTS, all Petitioner's experts have done is observe that protein sequences contained in the HPV vaccine can be shown to possess some sequential and/or structural similarity with targets of where an autoimmune reaction resulting in neuropathic POTS is *speculated* to occur. They have not established that reliable science has demonstrated that *any* kind of external insult, whether viral or vaccine, has ever been associated with an autoimmune reaction resulting in POTS at the proposed homologous situs.

It is common for Program petitioners to propose molecular mimicry as part of a causation theory – and indeed in many cases it is a persuasive component (especially when accompanied with other corroborative evidence) that can help establish how a vaccine's antigenic presentation might induce an autoimmune pathologic process. But merely chanting the magic words "molecular mimicry" in a Vaccine Act case does not render a causation theory scientifically reliable, absent *additional evidence* specifically tying the mechanism to the injury and/or vaccine in question. *See Devonshire v. Sec'y of Health & Human Servs.*, No. 99-031V, 2006 WL 2970418, at *15 (Fed. Cl. Spec. Mstr. Sept. 2006), *aff'd*, 76 Fed. Cl. 452 (2007). For such reasons I have rejected the blanket reliance on molecular mimicry under comparable circumstances. *See, e.g., Johnson*, 2018 WL 2051760, at *26 (theory that HPV vaccine could cause POTS via molecular mimicry not found scientifically reliable).

Dr. Tornatore's alternative T cell degeneracy mechanism (which he largely seemed to abandon at hearing in favor of molecular mimicry) fares no better. Dr. Tornatore posited that T cells can recognize several different antigens (and mount an immune response against them) without having *any* homology to a specific vaccine components, but nevertheless result in the same

[75] As I have observed in other cases, petitioners need not offer a mechanism in attempting to establish a causation theory under the first *Althen* prong – but if they do, it is fair to evaluate their success in the effort from the standpoint of whether it is preponderantly established. *See, e.g.*, *K.L. v. Sec'y of Health & Human Servs.*, No. 12-312V, 2017 WL 1713110, at *14 (Fed. Cl. Spec. Mstr. Mar. 17, 2017), *mot. for review den'd*, 134 Fed. Cl. 579 (2017).

autoimmune process presumably ending in POTS. But, and as before, he could not establish that the HPV vaccine had been shown to have this capacity in any regard, whether in causing/exacerbating POTS or any other autoimmune condition. And as Dr. MacGinnite persuasively established, T cell degeneracy as a theory implies that *any* T cell in the body could be responsible for an autoimmune cross-reaction (and it would be impossible to determine the instigator) – an overbroad contention given how infrequently autoimmune diseases actually occur. Tr. at 491; *see also* Trost at 71.

The additional mechanistic theories offered by Drs. Mikovits and Ruscetti were similarly unpersuasive. The proposition that vaccination can induce the production of proinflammatory cytokines (IL-1 beta specifically), thereby causing inflammation sufficient to create a favorable environment for autoimmunity, was not preponderately established. Indeed, I have noted in other cases that this theory – which relies heavily on what vaccines are understood to do immunologically, but then attempts to convert that into describing a pathologic process – is scientifically and medically unreliable, absent proof relevant to the disease process or vaccine at issue. *See, e.g.*, *Godfrey v. Sec'y of Health & Human Servs.,* No. 10-565V, 2015 WL 10710961, at *10-14 (Fed. Cl. Spec. Mstr. Oct. 27, 2015) (insufficient reliable scientific evidence supported proposition that cytokine upregulation induced by HPV vaccine was pathogenic enough to cause juvenile ankylosing spondylitis), *mot. for review den'd*, slip op. (Fed. Cl. Apr. 29, 2016).

The fact that cytokine upregulation is promoted by vaccination – a medically reliable assertion standing alone – does not mean that this cytokine increase is definitionally *harmful*, especially given (as observed by Dr. MacGinnitie) that it is difficult to establish whether certain proinflammatory cytokines are instigators or merely mediators of a disease process begun in some other way. It certainly does not establish that the cytokine upregulation would be sufficiently chronic to cause harm over an extended period of time. And the ASIA theory (something Dr. Mikovits could not resist discussing, despite Petitioner's averment that it was not part of her case), which posits the aluminum vaccine adjuvant as contributing to the purported pathologic immune response, is especially suspect from a scientific standpoint, as has been observed repeatedly in the Vaccine Program. *See, e.g.*, *Morris v. Sec'y of Health & Human Servs.*, No. 12-415V, 2016 WL 3022141, at *12 (Fed. Cl. Spec. Mstr. Apr. 1, 2016) (discussing lack of reliability of ASIA theory); *Rowan v. Sec'y of Health & Human Servs.*, No. 10-272V, 2014 WL 7465661, at *16 (Fed. Cl. Spec. Mstr. Dec. 8, 2014), *mot. for review den'd*, 2015 WL 3562409 (Fed. Cl. May 18, 2015); *D'Angiolini v. Sec'y of Health & Human Servs.*, No. 99-578V, 2014 WL 1678145, at *60 (Fed. Cl. Spect. Mstr. Mar. 27, 2014), *mot. for review den'd,* 122 Fed. Cl. 86 (2015), *aff'd*, 645 F. App'x 1002 (Fed. Cir. 2016).[76]

Petitioner also was unable to provide a reliable causation theory that her eczema flares

[76] For this very reason, I have not permitted petitioners to assert claims at hearing relying on ASIA for their causation theory, given the well-established doubts about its scientific reliability. *See, e.g.*, *Johnson*, 2018 WL 2051760, at *7 n.11.

could be vaccine-caused and/or the product of an autoimmune process. A threshold category error Petitioner made was in attempting to place her skin-related symptoms into a framework of nerve stimulation also in the context of *allergic* inflammation. But, as Dr. MacGinnitie (the sole expert with sufficient credentials and expertise in the fields of allergy and immunology to opine intelligently on the subject) discussed, allergies are *not* thought to be associated with or caused by autoimmune disease or severe autonomic dysfunction (as Petitioner claims). Accordingly, Petitioner could not persuasively invoke the limited science she offered bearing on how nerves might interact with allergic responses (primarily Voisin) to also connect those responses to an autoimmune process.

Finally, Petitioner did not present sufficient reliable scientific or medical evidence associating the HPV vaccine with POTS or eczema/urticaria. Besides case reports, she offered a few articles like Brinth or Blitshteyn which, while facially seeming to suggest such an association, have built-in reliability problems – due to the selection bias at issue in the studied subjects, as pointed out by Dr. Gibbons, as well as their own lack of a reliable scientific basis. Tr. at 366-67, 381; Brinth at 2603, Blitshteyn at 135; *see also Johnson*, 2018 WL 2051760, at *24 (critiquing Brinth as essentially a case study evidencing only a "temporal correlation" between the HPV vaccine and POTS). Respondent's experts, by contrast, referenced several reliable and credible articles (some of which were trustworthy, large-scale epidemiologic studies[77]) like Chao[78], Butts, Grimaldi-Bensouda, Block, Scufka, and the EMA Report, all noting that the HPV vaccine is not properly associated either with POTS specifically or other known autoimmune conditions generally. The evidence against Petitioner's contentions regarding the HPV vaccine and POTS or eczema greatly outweighed her own evidence.

No doubt *none* of the above evidence, pro or con Petitioner's litigative position, establishes from a final scientific standpoint whether the HPV vaccine *could* cause POTS (and it is not my purview as special master in this case to make such determinations in the first place). It remains conceivable that scientific evidence may yet reliably establish an association. But the evidence

[77] Petitioners in Program cases, when confronted with strong epidemiologic evidence, are often quick to point out that they cannot be "required" to offer it as part of their evidentiary showing – and therefore (because even a good and trustworthy epidemiologic study cannot completely refute the possibility that an inherently rare event like a vaccine injury could occur) merely to consider it constitutes an unfair heightening of their evidentiary burden. *See, e.g., D'Toile v. Sec'y of Health & Human Servs.*, 132 Fed. Cl. 421, 430 (2017), *aff'd*, 726 F. App'x 809 (Fed. Cir. 2018). But although it is true that petitioners are not obligated to *offer* epidemiologic evidence to support their claim, it *can* be considered (especially when it exists and is especially relevant to the causal theory at issue) in evaluating the success of a Vaccine Act petitioner in meeting her evidentiary burden. Sound and reliable epidemiologic evidence relevant to a specific vaccine injury claim cannot be swept under the carpet with the argument that it is categorically unmentionable.

[78] I have in prior decisions discussed Chao in depth, noting its scientific reliability and persuasiveness. *See*, e.g., *Maciel v. Sec'y of Health & Human Servs.*, No. 15-362V, 2018 WL 6259230, at *27 (Fed. Cl. Spec. Mstr. Oct. 12, 2018), *mot. for review den'd*, slip op. No. 15-362V (Fed. Cl. Apr. 1, 2019); *Johnson*, 2018 WL 2051760, at *25; *Sullivan v. Sec'y of Health & Human Servs.*, No. 10-398V, 2015 WL 1404957, at *11-12 (Fed. Cl. Spec. Mstr. Feb. 13, 2015).

offered *in this case* simply does not meet Petitioner's preponderant burden that it is "more likely than not" the HPV vaccine could cause or exacerbate POTS or eczema.

C. *The Record Does Not Establish That Petitioner's POTS or Skin Symptoms Were Exacerbated by the HPV Vaccine (Althen Prong Two/Loving Prong 5)*

Petitioner's obligation under the second *Althen* prong/*Loving* prong five is to demonstrate a logical sequence of cause and effect connecting the particular facts of his case to her medical theory. *Sturdivant v. Sec'y of Health & Human Servs.*, No. 07-788V, 2016 WL 552529, at *18 (Fed. Cl. Spec. Mstr. Jan. 21, 2016) (discussing *Althen* prong two). But the record in this case does not contain preponderant evidence that the HPV vaccine likely exacerbated Petitioner's POTS and preexisting eczema via the proposed autoimmune process set forth in her theory. The medical record does not allow for the conclusion that Ms. McKown's injuries were the result of the autoimmune-mediated neuropathic POTS variant.

As Respondent's experts observed, there is a lack of testing results in the medical record suggesting that Petitioner was experiencing any of the hallmarks of an autoimmune disease, like inflammation. Rather ,the more common indicators of underlying systemic inflammation (including the CRP and ESR rates) remained relatively stable throughout the course of her illness. Dr. Tornatore posited that those readings could not wholly rule out the possibility that Petitioner was experiencing a neuropathic condition, but his argument seemed more relevant to the kinds of CNS-oriented autoimmune diseases he treats, featuring inflammation localized to the spine or brain, rather than what Petitioner alleges occurred here. Only one record (from July 2014 – hence long after the vaccinations in question) established that Petitioner ever experienced a slightly elevated ERS and ANA – but, as Dr. MacGinnite persuasively proposed, such slight elevations likely had no clinical significance (and were themselves barely positive). Tr. at 481. And even so, Ms. McKown's treaters consistently ruled out autoimmunity as a cause of her symptoms. *Id.*

There is also a paucity of testing results establishing the presence of the putative autoantibodies that would, under Petitioner's theory, corroborate that she was experiencing an autoimmune-oriented disease process, let alone neuropathic POTS. Indeed, none of her treaters (including the numerous specialists she saw at the Cleveland Clinic) even proposed to test for them (which they might have done had they harbored any suspicion that autoimmunity explained her symptoms). The fact that she did test positive for certain allegedly-relevant biomarkers (for example, her slightly elevated IgE levels to various environmental allergens) cannot be leveraged into a finding that she was experiencing neuropathic POTS, since (as Dr. Tornatore admitted during his direct testimony) elevated IgE is not specific to autoimmunity directly.

Moreover, the character of Ms. McKown's *actual* symptoms was inconsistent with her

allegation that she had suffered a neuropathic form of POTS mediated by an autoimmune process. Dr. Gibbons (the expert with the most direct experience treating POTS) emphasized that the rare neuropathic POTS variant discussed in the literature is typically accompanied by profound autonomic *damage* – something lacking in this record. Tr. at 358. Neuropathic POTS would also feature many more debilitating symptoms – none of which Petitioner experienced. At the same time, the syncopal episodes Petitioner experienced were (as Dr. Gibbons maintained persuasively) evidence of a *properly functioning* autonomic nervous system – not one damaged by an autoimmune neuropathy. Tr. at 323, 394, 421. And Petitioner's numerous other symptoms (including joint problems, pain, prolonged episodes of unconsciousness, and eczema) are not associated with any particular POTS subtype. Tr. at 346, 349, 352.[79] Dr. Tornatore's argument about the significance of Ms. McKown's episodes of flushing (or bluish coloring in the limbs) was especially strained, as none of the literature he relied upon persuasively established that limb discoloration is associated with the favored POTS variant (beyond a single reference in Voisin – an article not addressing POTS – to a 1901 study).

Treater support for a finding that Petitioner either experienced a neuropathic form of POTS, or that at least it had been made worse by vaccination, is similarly absent from the medical record. Multiple contemporaneous treaters, including her cardiologist Dr. Dooley, informed Petitioner of their view that the HPV vaccine was not associated with POTS. Ex. 6 at 25. Her dermatologist, Dr. Rice, also proved unwilling to confirm Petitioner's fears about vaccine association to her symptoms. *See* Ex. 4 at 16-21; Ex. 14 at 22. And treaters at the Cleveland Clinic, who performed a multi-specialty, comprehensive work-up based on Petitioner's various complaints, specifically refuted a connection between the HPV vaccine and skin rashes. Ex. 11 at 7. That work-up otherwise does not support the conclusion that Ms. McKown's symptoms had an autoimmune character or origin.

Admittedly, one treater – Dr. DeMio – reached a contrary conclusion. Ex. 14 at 25, 27. But (putting aside his questionable medical credentials to offer a reliable opinion on this subject)[80], Dr.

[79] The evidence is more inconclusive as to *what* POTS variant best explains Petitioner's symptoms. Dr. Gibbons opined that Petitioner's POTS was likely hypovolemic (or due to dehydration), or attributable to deconditioning related to her athletic activities but was ultimately uncertain. Tr. at 332-33, 355-56. To support this contention, he referenced the BUN to creatine ratio measurement (20:1) taken during Petitioner's initial hospitalization in April 2013, suggesting the existence of dehydration (with resolution of her symptoms following fluid intake corroborating this possibility). *Id.* at 333. In response, Dr. Tornatore reasonably posited that Petitioner otherwise exhibited no clinical signs or biomarkers for chronic dehydration over the course of her illness, and the BUN-creatine ratio levels were arguably less meaningful in this case than Respondent's experts argued. *Id.* at 559-60. I cannot on this record (especially in the absence of contemporaneous treater opinions as to the relevant POTS variant in question) determine if the evidence preponderates for or against Petitioner on this point – other than to conclude that, taken as a whole, the record does *not* preponderate in favor of a determination that she had the neuropathic, autoimmune form.

[80] Other special masters have observed that Dr. DeMio lacks the expertise to opine regarding the medical theories he has advanced. *See, e.g., Wyatt v. Sec'y of Health & Human Servs.*, No. 14-706V, 2018 WL 7017751, at *18-19, 21-22 (Fed. Cl. Spec. Mstr. Dec. 17, 2018) ("[o]nce again, Dr. DeMio has rendered an opinion in a case in which he lacks the underlying requisite medical expertise. Dr. DeMio has neither specialized training in either autoimmune or neurological disorders nor has he ever conducted research or written papers in either of these fields"), *mot. for review*

DeMio only saw Petitioner in September 2014 (roughly one year following her second dose of HPV), and his assessment seemed to turn more on the health course reported by Petitioner than his own contemporaneous examination. In addition, the testing for autoimmunity performed around this time was also negative. Ex. 9 at 7-12. I therefore give his opinion far less weight than the more numerous and more medically-trustworthy prior treaters who reached more evidentiarily-supported conclusions.

Ms. McKown's medical record *does* establish that she experienced a recurring skin condition, in the form of eczema flares post-vaccination. However, I do not find that she has established that these flares were attributable to the HPV or Hep A vaccines. As already noted, the medical record does not support the conclusion that she was experiencing inflammation or an autoimmune process in the relevant post-vaccination timeframe – and because, as discussed above, she did not establish a reliable theory that an allergic response like eczema is associated with autoimmunity in the first place, the presence of such evidence would not make it more likely vaccination caused her flares. Moreover, none of the dermatologists she saw (including a Cleveland Clinic specialist) associated her symptoms with either the vaccines she received specifically or an autoimmune process generally. Otherwise, I do not see on the basis of this medical record any connection between her skin symptom flares and POTS (beyond the fact that both were coincidentally occurring in the same general time period).

D. *Petitioner's Urticaria Was Not Vaccine-Caused*

Because Petitioner appears not to have experienced hives before vaccination, unlike her POTS claim she could conceivably establish that this set of symptoms (separate from her preexisting eczema) was directly vaccine-caused. However, she failed to do so, based on any of the three *Althen* prongs.

First, although Respondent's primary allergy expert, Dr. MacGinnitie, agreed that chronic urticarial lesions could be autoimmune in nature, Petitioner did not persuasively establish that either of the vaccines she received beginning in March 2013 could produce this particular outcome (for which she was first clearly diagnosed in 2014 many months later) – and if so, how. *See* Ex. 11 at 14; Ex. 1 at 76. Petitioner's experts lacked the experience and background necessary to offer a persuasive opinion on the subject of urticaria generally, and (for the same reasons discussed above) their immunologic theories were scientifically unreliable.

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den'd, slip op. No. 14-706V (Fed. Cl. June 5, 2019); *Holt v. Sec'y of Health & Human Servs.*, No. 05-136V, 2015 WL 4381588, at *16 (Fed. Cl. Spec. Mstr. June 24, 2015) ("[Dr. DeMio] is board certified in emergency medicine. He has no formal specialized training in . . . any of the several areas [pediatrics, immunology, neurology, or gastroenterology], in which he proffered opinions. His only publications involved chapters on arthritis, gout, inflammation, and nutrition in an integrative medicine textbook.").

Second, even if Petitioner had satisfied the "can cause" prong, the record does not suggest that she tested positive for any autoantibodies known to associated with the autoimmune form of urticaria discussed by Dr. MacGinnitie – and none of the allergists or dermatologic experts she did see ever proposed or concluded that her hives were vaccine-related, let alone autoimmune in nature. Indeed, the better evidence in the record (such as that from the summer of 2013, just after Petitioner was diagnosed with POTS and given medication for it) supports the conclusion that in certain cases any hives she experienced may have been associated with her medication. *See, e.g.*, Ex. 7 at 33. Finally, the timeframe in which Petitioner displayed such hives (beginning no earlier than three to four months after receipt of the first HPV dose) was not shown by Petitioner's experts to be medically reasonable, with her overall stuttering course not reasonably or credibly connected to some chronic autoimmune process that began with the first dose in March 2013.

E. *Other Althen/Loving Prongs*

Because my holding turns primarily on the preceding analysis of certain individual *Althen* or *Loving* prongs, I need not consider every single individual other prong. I do, however, make the following additional findings:

1. *Loving* Prong 3: Evidence of Worsening

In this case, it is easy to conclude based on the medical record and witness testimony that in the months and years after receiving the vaccines in question, Ms. McKown experienced more POTS-associated symptoms, as well as eczema flares, than she had pre-vaccination, and thus in a literal sense her condition was unquestionably "worse." Although it was difficult to assess the accuracy of Petitioner's claims regarding extended periods of time in which she was unconscious, I credit her averments that she experienced POTS-related symptoms, as well as bouts of eczema and hives, more frequently after receiving the two doses of HPV vaccine than before.

But determining that Petitioner's condition was comparatively worse post-vaccination only satisfies the second *Loving* prong, leaving the third unanswered: did her condition reflect an aggravation of what she otherwise would have expected to consider – as a person previously diagnosed with eczema, and as likely to have been suffering from POTS even before vaccination? *Locane*, 685 F.3d at 1381-82.

Considering only her POTS symptoms, and taking into account the literature filed in this case along with expert testimony from Dr. Gibbons (the sole expert with a specific focused background in POTS), I do not find that Petitioner's course was outside of what most individuals suffering from POTS will experience. The literature filed in this case establishes that non-neuropathic POTS can have a number of associated symptoms, including syncopal episodes (although, as Dr. Gibbons established, such episodes are counterfactually evidence of a properly

performing autonomic system). *See* Benarroch at 1215-16; Gibbons at 1, 5-7. It is also common for POTS to wax and wane over time, as a patient's course persists (as Dr. Gibbons posited). *See* Tr. at 352 (noting that it is not "unusual" for a POTS course to "fluctuat[e] in the frequency of symptoms"), 352-53 ("one of the characteristics of POTS is that after you've sort of developed the syndrome, things do get typically worse"), 395. Nothing in Petitioner's overall treatment history suggests the POTS symptoms she experienced were outside the norm, and none of her treaters proposed otherwise.

Petitioner's experts simply lacked the in-depth knowledge of POTS to opine persuasively to the contrary. Dr. Tornatore might have accurately described the "tempo" of Petitioner's symptoms as picking up post-vaccination, but that does not satisfy the third *Loving* prong. His testimony did not establish reliably that Petitioner's symptoms course went beyond what an autonomic or cardiologic expert would expect to see. Certainly nothing in the medical record suggests any treater with expertise in POTS thought Ms. McKown's symptoms were particularly alarming or unusual.

Regarding Petitioner's eczema flares, Dr. Tornatore described a worsening over a six-month period following Petitioner's initial doses of HPV and Hep A (beginning in June or July 2013), and later resulting in more significant flares in September of that year (after the second dose of HPV vaccine). Tr. at 151. But he acknowledged as well that the record evidenced skin-related symptoms that were "coming and going," and sometimes even normal. *Id.* at 146-48. Dr. MacGinnitie, who treats multiple patients with dermatologic conditions like eczema, agreed but concluded that Petitioner's case was nevertheless within the range of what is expected for an eczema patient. *Id.* at 486-69, 512. It is not enough for Petitioner to argue that she literally became "worse" in the days immediately after receipt of the last HPV dose – for that is another way of simply invoking the temporal relationship between vaccine and injury, a relationship well understood in the Program to have little evidentiary bearing when determining entitlement. *See, e.g.*, *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1341 (Fed. Cir. 2014) ("[a] temporal correlation alone is not enough to demonstrate causation"). Accordingly, the record (supplemented and interpreted with persuasive expert testimony from Dr. MacGinnitie) does not permit the conclusion that Petitioner's eczema flares were worse than what would otherwise have been expected.

2. *Althen* Prong Three: Timeframe

Petitioner proposes that aggravation of her POTS symptoms in late April 2013 (or four to five weeks following vaccination), after the yogurt shop incident, was a medically appropriate timeframe for a vaccine-induced injury to occur. In support, however, she relied almost exclusively on Schonberger – which studied the incident rate of onset of GBS following receipt of the flu vaccine -- an injury and vaccine combination that is distinguishable from that alleged herein, other

than the fact that it too involves nerve system-related harm. This, plus the broader deficiencies with Petitioner's theory (which did not otherwise establish that the HPV (or Hep A) vaccine doses could directly cause POTS and/or that Petitioner's POTS was the result of an autoimmune neuropathy), as well as the lack of evidence that in this timeframe Petitioner was experiencing any autoimmune or inflammatory process, prevents a finding that the timeframe was medically acceptable.

The same is true for flares of Petitioner's preexisting eczema. The records establish that her flares (a) occurred no earlier than July 2013, or over three months after the first HPV dose, (b) were associated by treaters with cessation of medicine intended to treat her POTS, (c) were never deemed autoimmune in nature by any competent treaters, and (d) were not persuasively established by Petitioner's experts to have any association with her coterminous POTS diagnosis. Petitioner's experts also did not credibly explain how an autoimmune process that could result in a POTS-related syncopal episode five weeks post-vaccination could then take another two-plus months to manifest in exacerbation of skin-related symptoms.

CONCLUSION

Petitioner has not carried her burden of proof, and therefore I must DENY entitlement in this case. In the absence of a timely-filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accord with this decision.[81]

IT IS SO ORDERED.

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

[81] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.