OFFICE OF SPECIAL MASTERS
No. 03-2211V
Filed: August 21, 2006
To Be Published

DAYNA LEIGH SCOTT	*	
	*	Entitlement; MMR vaccine;
Petitioner,	*	Differing diagnoses, APS, MS,
	*	and SLE; Failure to establish
v.	*	prima facie case; Onset outside
	*	window of proximate temporal
SECRETARY OF THE DEPARTMENT	*	connection
OF HEALTH AND HUMAN SERVICES,	*	
	*	
Respondent.	*	
	*	

William Pauzauskie, Topeka, KS, for petitioner.

Lisa Watts, Washington DC, for respondent.

DECISION[1]

VOWELL, Special Master

Dayna Leigh Scott[2] timely filed a petition for compensation under the National Vaccine Injury Compensation Act, 42 U.S.C. § 300aa-10 et. seq.[3] on September 23, 2003. Her petition was initially assigned to Special Master Richard Abell and was reassigned to me on February 13, 2006. At a causation hearing on February 28, 2006, I heard testimony from Doctors James Anderson, Alan Brenner, Thomas Leist, and Marcel Kinsbourne, as well as from petitioner and her husband, Jeffrey Scott. For the reasons set forth below, I conclude that petitioner is not

[1] Because I have designated this decision to be published, petitioner has fourteen (14) days within which to request redaction of any material "that includes medical files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire decision will be publicly available. 42 U.S.C. § 300aa12(d)(4)(B).

[2] Some of Mrs. Scott's medical records reflect her maiden name, "Johnson", and some utilize the name "Kimble" (her name prior to her current marriage to Jeffrey Scott). The petition in this case spells Mrs. Scott's first name as "Dayna" but various filings, including the court's electronic record, reflect the spelling as "Dana." The correct spelling is "Dayna."

[3] Hereinafter, for ease of citation, all "§" references to the Vaccine Injury Compensation Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2000 ed.).

entitled to compensation because she has failed to demonstrate by preponderant evidence that her medical condition was caused or significantly aggravated by a vaccine.

I. Nature of the Claimed Injury

Mrs. Scott alleged that a measles, mumps, and rubella ["MMR"] vaccination[4] she received on April 15, 2002 caused an immune reaction[5] that has been variously diagnosed as multiple sclerosis ["MS"],[6] antiphospholipid antibody syndrome ["APS"], systemic lupus erythematosus ["SLE"],[7] and vasculitis.[8] Encephalomyelitis[9] and acute disseminated encephalomyelitis[10] have also been mentioned as possible diagnoses. Subsequent filings and the testimony at the causation hearing establish that the MMR vaccination she received on April 15, 2002 forms the basis for Mrs. Scott's claim of a vaccine-induced or aggravated injury.

The hearing testimony and expert reports established that petitioner was proceeding on the theory that she suffers from vaccine-induced APS. This syndrome, also known as Hughes syndrome, is a disorder characterized by thrombosis, recurrent fetal loss, or thrombocytopenia,

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[4] The petition also makes reference to "Hepatitis shots" that petitioner received in 1994 and to "DPT vaccinations" without reference to the date of administration. Petition at p. 2.

[5] Specifically, Mrs. Scott claimed that she suffered "an immune reaction to immunization characterized by non-specific auto antibodies, including low levels of double-stranded DNA antibodies and also circulating anticoagulant." Petition at p. 2.

[6] Multiple sclerosis is "a progressive disease characterized by disseminated demyelination of nerve fibers of the brain and spinal cord." Early symptoms include abnormal sensations in the extremities or face, vertigo, visual disturbances, and muscle weakness. *Mosby's Medical Dictionary* at 1234 ["*Mosby's Medical Dictionary*"] (7th ed. 2006).

[7] Commonly known as "lupus," systemic lupus erythematosus is a chronic inflammatory disease. It is characterized by severe vasculitis, renal problems, and skin and nervous system lesions. Its cause is unknown, but both immune system disorders and viral infections have been suggested as possible causes, and lupus reactions to certain drugs have been noted. Diagnosis is based on objective physical examination and laboratory tests for antinuclear antibody in the cerebrospinal fluid and a positive lupus erythematosus cell reaction, as well as on subjective findings. *Mosby's Medical Dictionary* at 1813. Many lupus patients also have antiphospholipid antibodies. Transcript ["Tr."] at 10, 20-21.

[8] Vasculitis is an inflammation of the blood vessels caused by a systemic disease or an allergic reaction. *Mosby's Medical Dictionary* at 1942.

[9] Encephalomyelitis is "an inflammatory condition of the brain and spinal cord characterized by fever, headache, stiff neck, back pain, and vomiting." Seizures or decreased mental ability may result from severe inflammation. *Mosby's Medical Dictionary* at 640.

[10] Acute disseminated encephalomyelitis is a syndrome associated with viral infections, especially measles. Symptoms include headache, altered mental state, and seizures. *Dorland's Illustrated Medical Dictionary* at 610 (30th ed. 2003). *See also*, Tr. at 16; Pet. Ex. 20, p. 48.

and the presence of antiphospholipid antibodies[11] in the blood. It may manifest with symptoms that resemble MS. *See* Respondent's Exhibit ["Res. Ex."] E, J.W. IJdo, *et.al*., "Anti-phospholipid antibodies in patients with multiple sclerosis and MS-like illnesses: MS or APS?" 8 *Lupus* No.2, pp.109-110 (1999). APS may also present with symptoms resembling SLE. Tr. at 20-22. The International Classification of Disease codes place antiphospholipid and anticardiolipin antibodies under the same broad classification of "circulating anticoagulants." Tr. at 9. Anticardiolipin is one of several antiphospholipid antibodies. Tr. at 106.

Because the injury alleged is not one listed on the Vaccine Injury Table (42 C.F.R. § 100.3), Mrs. Scott has the burden of demonstrating by preponderant evidence that her injury was caused by a vaccination. Based on the record as a whole,[12] including the testimony taken at the causation hearing, and having carefully considered the briefs and additional articles filed post-hearing, I conclude that she has failed to establish a *prima facie* case that the MMR vaccination caused or significantly aggravated her condition.

II. Factual Findings: Medical History, Diagnoses, and Treatment

A. Medical History Prior to the April 2002 MMR Vaccination.

Prior to receiving the MMR vaccination on April 15, 2002, Mrs. Scott, then age 28, had been treated for a variety of medical conditions, including many upper respiratory infections. In the thirteen months preceding the MMR vaccination, she called or visited a health care provider on five occasions for upper respiratory infections, sinus infections, and complaints of cough, cold, or wheezing.[13] Pet. Ex. 2, pp. 45, 49 (two visits), 51, and 55. In addition, she was seen for a spider bite, and panic attacks. *Id*. at 45-46.

Of some significance to her case are an unexplained instance of paralysis at age 13, undocumented by contemporaneous medical records but referenced in a medical history[14] and

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[11] An antibody is an immunoglobulin produced in response to bacteria, viruses, or antigenic substances and is specific to a particular antigen. *Mosby's Medical Dictionary* at 114.

[12] *See* § 300aa–13(a): "Compensation shall be awarded...if the special master or court finds on the record as a whole..." *See also*, § 300aa–13(b)(1) (indicating that the court or special master shall consider the entire record in determining if petitioner is entitled to compensation).

[13] These were: an unknown day in April, 2001; October 26, 2001; November 17, 2001; January 18, 2002; and January 31, 2002. The medical visits for upper respiratory problems also continued after the vaccination. *See, e.g.*, Pet. Ex. 2, pp. 68-70, 74-75, 89.

[14] Mrs. Scott reported that she had been told that her paralysis of several days' duration was due to a "viral infection" but did not indicate what virus was implicated or suspected. Pet. Ex. 2, p. 59. No medical records concerning this incident were produced. While Dr. Leist, one of respondent's expert witnesses, speculated that the paralysis may have been due to Guillain-Barré Syndrome (Tr. at 163) or to transverse myelitis (Tr. at 173-74; Res. Ex. C, pp. 5-6), I did not rely in any way on his speculations.

several other problems noted in visits to her primary care provider, Dr. Joseph Sack, in the four years preceding the MMR vaccination.

Mrs. Scott saw Dr. Sack on November 2, 1998, complaining of left upper extremity "numbness" possibly associated with a motor vehicle accident on October 28, 1998. Pet. Ex. 2, p. 18. She had a weakly positive titer for mycoplasmal pneumonia on May 11, 2000, after presenting with symptoms that included left arm numbness and a feeling of lightheadedness. *Id*. at 31, 33. An echocardiograph on May 15, 2000 was significant for "[b]orderline increased septal wall thickness, suggestive of left ventricular hypertrophy."[15] *Id.* at 37. On September 27, 2001, she saw Dr. Sack with complaints of panic attacks, extreme fatigue, and stress. *Id.* at 46.

B. The MMR Vaccination and Subsequent Treatment (April to mid-May 2002).

On April 15, 2002, Mrs. Scott received an MMR vaccination required by her employer, Wesley Medical Center.[16] Pet. Ex. 5, p. 4. This was her second vaccination against measles, mumps, and rubella, albeit the first in a vaccination against all three viruses at once. She had received childhood vaccinations for mumps and rubella on September 3, 1974 and a measles vaccination on September 2, 1983. Pet. Ex. 6, p. 9.

Two days after the MMR vaccination, she experienced cough and congestion, which she reported to Dr. Sack on April 19, 2002.[17] Pet. Ex. 2, p. 56. Although Mrs. Scott testified that she reported to "employee health" prior to going to Dr. Sack's office (Tr. at 218), there is no record of a report to Wesley Occupational Health Services in the exhibits until November 4, 2002. Pet. Ex. 24, p. 6. In both reports, Mrs. Scott drew a connection to her MMR vaccination.[18] During her

[15] Echocardiography is an ultrasound procedure used to evaluate heart wall motion and possible vascular disease. *Mosby's Manual of Diagnostic and Laboratory Tests* ["*Mosby's Labs*"] at 824 (2d ed. 2002). Ventricular hypertrophy is the abnormal enlargement of the heart ventricles (chambers). *Mosby's Medical Dictionary* at 1951.

[16] Mrs. Scott was working as a licensed practical nurse at Wesley Medical Center when she received the MMR vaccination.

[17] The chart entry reflecting this visit begins with a notation that the dictation pertaining to this visit was lost and the chart entry was actually made on April 29, 2002. While there was no direct evidence about how the record of the visit was reconstructed, it clearly was not made by memory alone, as the chart entry contains details such as blood pressure, weight, and examination findings that would not likely be recalled in detail ten days later. Handwritten notes in the chart are fully consistent with this typed entry. Pet. Ex. 2, p. 56.

[18] In her testimony at the causation hearing, Mrs. Scott indicated that she was told by occupational health personnel that two days was too early to have a vaccine reaction. She also testified that Dr. Sack's office informed her that two days was not too early to experience a reaction and she was thus scheduled for an appointment. Tr. at 218. The contemporaneous medical records do not contain any indication that Dr. Sack considered her symptoms to be vaccine-related, but do indicate that Mrs. Scott thought they might be. The relationship between Mrs. Scott's cough and congestion and her recent MMR vaccination was reflected in a portion of the chart after "S:", a common medical abbreviation for subjective findings (the symptoms as described by the patient). Pet. Ex. 2, pp. 56-57. Mrs. Scott also linked the MMR vaccine and her respiratory problems in an October 31, 2002 telephone call to Dr. Sack.

visit to Dr. Sack on April 19, 2002, four days after the vaccination, she complained of cough, congestion, and fatigue, and noted that she had not felt well since having the MMR shot on Monday.[19] Doctor Sack diagnosed "Reactive airway disease." Pet. Ex. 2 at pp. 56-57. While Mrs. Scott testified at the hearing that Dr. Sack told her the vaccine could be responsible for her symptoms (Tr. at 220), the contemporaneous records do not reflect that Dr. Sack drew such a connection. Mrs. Scott returned on April 22, 2002, noting that her chest congestion was better, but that she was experiencing some dizziness.[20] She was diagnosed with improving reactive airway disease and a viral upper respiratory tract infection. Pet. Ex. 2, p. 57.

The first medical record containing any complaint of neurological symptoms after the MMR vaccination is dated May 14, 2002.[21] Pet. Ex. 2, p. 59. At this visit, Mrs. Scott provided a family history of MS. While Mrs. Scott testified that she mentioned her family history of MS to Dr. Sack at her April appointment with him (Tr. at 219), the medical records do not contain any reference to that history at either her April 19 or April 22, 2002 appointments. Based on all the evidence available to me, including Mrs. Scott's difficulties in consistently reporting the dates and nature of her symptoms throughout the many medical histories taken by various health care providers (discussed, *infra*), the quality of Dr. Sack's records otherwise which include recording relevant family history, *see, e.g.*, Pet. Ex. 2, p. 20 (recording a history of skin cancer prior to removal of several skin lesions) and p. 21 (recording a family history of pelvic and breast cancer when seen for elective hysterectomy scheduling), and the nature of the tests he ordered on May 14, 2002, I conclude that Mrs. Scott did not mention her mother's MS until the May 14, 2002 appointment, notwithstanding her hearing testimony.

At this May 14 visit, Mrs. Scott also complained of dizzy spells, problems with sensory changes, weakness in both hands and arms, and lightheadedness persisting for the previous five

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Pet. Ex. 2, p. 90. While the message form contains a date of "10-31" without a year in the upper left corner, the bottom right of the form contains a notation "Dr. Sack" followed by a date of 10/31/02. I therefore find that the phone message was left on October 31, 2002.

[19] Mrs. Scott seemed concerned on several other occasions about her work exposure to diseases. *See, e.g.*, Pet. Ex. 2, p. 67 (complaining of shortness of breath and noting that she worked at a hospital and was exposed to "everything") and Pet. Ex. 2, p. 75 (explaining that she had been exposed to MRSA [a drug-resistant bacteria] at work and was concerned that she might have caught it).

[20] While Dr. Anderson noted that dizziness could be a neurologic symptom, he also agreed that the reported dizziness could be due to her airway disease. Tr. at 65-66. Doctor Leist, a neurologist, testified that "dizziness" is an unspecific term. For dizziness to have neurological significance, it would have to be related to gait, balance, or coordination difficulties. Tr. at 149-150. Doctor Kinsbourne, petitioner's expert neurologist, agreed that the symptom of dizziness lacked significance. Tr. at 93.

[21] One entry located at Pet. Ex. 2, p. 59, apparently documenting Mrs. Scott's reasons for being seen by Dr. Sack, seems to reflect a date of May 14, 2001, although the quality of the copy and the handwriting make the year of entry difficult to discern. In view of the virtually identical symptoms and complaints in the later typed entry signed by Dr. Sack on the same page of Mrs. Scott's medical record, dated May 14, 2002, I find that the date of this visit was actually May 14, 2002.

days. She gave a history of being "paralyzed" for a few days at age 13 as the result of a virus. Upon examination, Dr. Sack noted decreased deep tendon reflexes in Mrs. Scott's upper extremities, although he did not grade or score the level of weakness. Doctor Sack ordered a series of laboratory tests and a head magnetic resonance imaging ("MRI").[22] Pet. Ex. 2, p. 59.

The MRI done on May 23, 2002 was clearly abnormal. It showed "focal areas of increased signal in the periventricular white matter bilaterally...in a patient of this age, demyelinating disease should certainly be considered." Pet. Ex. 2, p. 65. Mrs. Scott was referred to a neurologist, Dr. Janet Mullinix, for further diagnosis and treatment. *Id*. at 63, 65.

C. Initial Diagnosis of Multiple Sclerosis (June-October 2002).

The medical history Mrs. Scott provided to Dr. Mullinix on June 7, 2002 indicates that she began having neurological symptoms three to four weeks earlier, placing onset of her symptoms at the time of her May 14, 2002 visit to Dr. Sack or within a few days preceding that visit. Doctor Mullinix recorded Mrs. Scott's symptoms as awakening with a "tingly prickly sensation" in her right arm, feeling very lightheaded, having blurry vision, and experiencing a near "black out" at work. These symptoms persisted for about a week. She began to drop things with her right hand. The tingly and prickly sensation spread to her left side. She complained of stuttering and losing her train of thought in the previous three to four weeks. She noted that her legs felt "like jello" and that she dragged her feet. Pet. Ex. 17, pp. 4-5, 20-21. Doctor Mullinix found no weakness in Mrs. Scott's right arm upon physical examination. She noted that Mrs. Scott's tone, gait, and strength were all normal. Id. at 21. Doctor Mullinix ordered additional testing. A Brainstem Auditory Evoked Response was normal (*id*. at 11) and a Visual Evoked Potential ("VEP")[23] was abnormal. *Id.* at 10. Doctor Mullinix diagnosed Mrs. Scott with MS and recommended treatment with Rebif.[24] *Id.* at 6-7.

Over the next four months, Mrs. Scott made several visits to Dr. Sack, primarily for respiratory problems (Pet. Ex. 2, pp. 68-70, 75, 84), and one visit to an emergency room for chest

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[22] A brain MRI is frequently used to diagnose MS and other degenerative diseases. *Mosby's Labs* at 1067.

[23] The VEP is often referred to as a Visual Evoked Response ("VER") in the medical records. Evoked response tests are performed to assess the pathway between the sense organs and the brain cortex. The tests may locate conduction delays along this pathway indicative of damage or disease, even though the sense of hearing, sight, or touch otherwise appears to be normal. *Mosby's Labs* at 520-22. Doctor Brenner testified about a study concerning VER that would lend some support to a diagnosis of MS for Mrs. Scott. Tr. at 108-09, 118-19. *See* Res. Ex. L2, Daphna Paran, *et al.,* "Evoked Potential Studies in the Antiphospholipid Syndrome: Differential Diagnosis from Multiple Sclerosis," *Ann. Rheum. Dis*., published online 17 Aug 2005; doi:10.1136/ard.2005.040352 ["Paran article"]. As indicated on the copy of the article filed with the court, the web-based journal *Online First* "contains unedited articles in manuscript form that have been peer reviewed and accepted for publication but have not yet appeared in the paper journal...".

[24] "Rebif" is the trade name for interferon beta-1a. Pet. Ex. 17, pp. 21-23. Rebif is used to treat the relapsing-remitting form of MS. *Physician's Desk Reference* ["PDR"] at 3137 (58th ed. 2004).

pain and difficulty in breathing. *Id.* at 81, 84; Pet. Ex. 22, pp. 45-54. On October 4, 2002, she was seen by Dr. Gayle May[25] for weakness in her upper arms. Doctor May graded the weakness at 4/5 and noted no lower extremity weakness. Mrs. Scott returned to Dr. Sack six days later with respiratory problems, complaints of memory problems, difficulty in getting out of bed, and right sided weakness and numbness. Pet. Ex. 2, p. 89.

D. Subsequent Diagnoses and Treatment (November 2002-July 2004).

In November 2002, Mrs. Scott switched doctors, leaving Dr. Mullinix for Dr. Andrew Massey. Mrs. Scott told the resident who saw her at her first appointment at the Kansas University Clinic that she was displeased with the care given by Dr. Mullinix and did not believe Dr. Mullinix's diagnosis of MS. This resident referred Mrs. Scott to Dr. Massey. Pet. Ex. 2, p. 92. She first saw Dr. Massey on November 13, 2002. *Id*. at 94-95.

Doctor Massey's dictation from that visit reflects that Mrs. Scott provided a markedly different history of her recent symptoms than she had previously provided to Dr. Mullinix and Dr. Sack. She told Dr. Massey that two days after her MMR shot, she developed respiratory symptoms and a 102 degree fever, and two weeks later she awakened paralyzed on her right side.[26] Although Mrs. Scott thought this might have been a stroke, she did not seek medical attention until nearly six days later. She also complained that she was continuing to have daily episodes of right-sided weakness in the afternoon, persisting until she went to sleep. After she changed jobs and cut back her working hours, the symptoms ceased. Pet. Ex. 2, p. 95.

Although Dr. Massey assessed both Mrs. Scott's symptoms and the MRI findings as "not inconsistent" with MS, he believed further testing was warranted, noting a possible "post-vaccinational encephalomyelitis." Pet. Ex. 2, p. 96. I conclude that this notation by Dr. Massey merely constituted a possible explanation of the juxtaposition of the vaccine and Mrs. Scott's symptoms, as she described them. As no subsequent medical record or testimony reflects a diagnosis of encephalomyelitis, I conclude that any diagnosis of encephalomyelitis was subsequently abandoned. I find no evidence that Mrs. Scott actually suffered from encephalomyelitis, as the contemporaneous records do not reflect the symptoms upon which this possible diagnosis was based.

The laboratory tests found an elevated partial thromboplastin time ("PTT").[27] Doctor

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[25] Although the report is signed by Dr. Sarah Johnson, the write-up reflects that the examination was performed by Dr. May, a resident.

[26] The underlined symptoms were not referenced in Mrs. Scott's April 2002 visits to Dr. Sack nor were they referenced in Dr. Mullinix's records for her June 7, 2002 visit.

[27] A PTT is performed to assess blood coagulation. *Mosby's Labs* at 350-53. As Dr. Anderson noted during his testimony, the PTT was only transitorily elevated. In most cases of APS, an elevated PTT remains elevated. Tr. at. 11; 72-73.

Massey therefore ordered additional blood tests for anticardiolipin antibodies, lupus anticoagulant, and anti-thrombin 3 levels. He also performed a lumbar puncture.[28] Pet. Ex. 2, p. 97. Because he continued Mrs. Scott on Rebif, pending receipt of the new laboratory test results (*id.*), I conclude that Dr. Massey still considered MS the most likely diagnosis as of November 13, 2002.

Doctor Massey reviewed the blood tests on November 20, and the cerebrospinal fluid tests on December 9, 2002. Pet. Ex. 2, p. 97. He saw Mrs. Scott on December 9, 2002 and noted with regard to the blood tests: "Laboratory studies revealed an anticardiolipin antibody, but it was IgM 42 MPL U/m while IgG and IgA were negative."[29] Pet. Ex. 2, p. 98. The analysis of her cerebrospinal fluid provided "evidence of an immune-mediated disorder." Doctor Massey did not make a definitive diagnosis, commenting:

> [T]his may represent relapsing, remitting multiple sclerosis, but with the findings of an anticardiolipin antibody and coagulation defect (which may be related?) The [sic] possibility of multiple ischemic TIAs[30] or strokes should be considered, though thrombotic episodes are usually seen with the IgG anticardiolipin antibody, the prolonged PTT and presence of an IgM anticardiolipin antibody raises the specter of antiphospholipid antibody syndrome, which might be treated differently.

Pet. Ex. 2, p. 98. Doctor Massey continued to prescribe Rebif for Mrs. Scott and sent her to Dr. Michael Cannon for a hematology consultation. *Id.*

The hematology consultation with Dr. Cannon led to a rheumatology consultation with Dr.

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[28] A lumbar puncture involves placing a needle in the subarachnoid space of the spinal column to measure pressure and to obtain cerebrospinal fluid for laboratory examination. The presence of blood or bacteria and the amount of glucose or protein present in the spinal fluid may assist in diagnosis of autoimmune and demyelinating disorders and many other diseases. *Mosby's Labs* at 605.

[29] Anticardiolipin antibodies, like other antibodies, are produced from B lymphocytes. Tr. at 100-01. Lymphocytes are white blood cells that play a major role in the body's response to infection. They exist in two forms, B cells and T cells. When a B cell is exposed to a specific antigen, it becomes activated and produces two additional types of cells. Upon exposure to a specific antigen, T cells divide rapidly and produce more T cells that are reactive to that antigen. Some of these T cells attack the foreign antigen and are called "killer" T cells. *Mosby's Medical Dictionary* at 1127. IgM antibodies are the initial type or "class" of antibody produced in response to the activation of a B cell by a viral or bacterial infection. IgM antibodies are relatively short-lived. As the body's response to the infection progresses, in what is called a "class switch," the IgM antibodies produced convert to IgG, IgA, and IgE antibodies, in response to the types of cytokines encountered. Tr. at 100-103. Cytokines are proteins produced by lymphocytes. *Mosby's Medical Dictionary* at 506. Anticardiolipin and antiphospholipid antibodies are not suggestive of any particular infection. Tr. at 73-74.

[30] "TIA" is a common abbreviation for "transient ischemic attack," which is "an episode of cerebrovascular insufficiency, usually associated with partial occlusion of a cerebral artery." *Mosby's Medical Dictionary* at 1880.

Anderson, who testified at the causation hearing as one of Mrs. Scott's expert witnesses.[31] Doctor Massey summed up the findings of Dr. Anderson and Dr. Cannon in a February 17, 2003 letter to Dr. Sack:

> Because her stroke-like symptoms and MRI findings could be due to a coagulopathy or vasculopathy associated with the anticardiolipin antibody syndrome associated with lupus, and as the diagnosis of multiple sclerosis is a diagnosis of exclusion, the information that we are able to accumulate to date suggests to me that the treatment course outlined by Dr. Anderson may be superior to continuing beta interferon-1a [Rebif] for a diagnosis of "multiple sclerosis". I would caution her as well as all of us that without a pathognomonic clinical marker [32] the diagnosis of "anticardiolipin antibody syndrome associated with lupus" may not be the right diagnosis, but at this point in time it seems to be the best diagnosis."

Pet. Ex. 2, p. 100.

Doctor Anderson's initial assessment of Mrs. Scott appears at Pet. Ex. 2, p. 91. His evaluation did not reference the MMR vaccination or any cause for Mrs. Scott's condition. He diagnosed her with antiphospholipid antibody syndrome. As he testified at the causation hearing, this syndrome encompasses a broad group of illnesses that are characterized by the presence of either anticardiolipin antibodies or lupus anticoagulant factors in the blood, plus clinical symptoms consistent with the diagnosis. Such clinical symptoms include migraine headaches, deep venous thrombosis, and recurrent miscarriages. About half the people with APS also have lupus. Tr. at 9-10.

Mrs. Scott was hospitalized at the Via Christi Regional Medical Center on April 8, 2003, after a three-day history of headache and left-sided "numbness and tingling," according to the discharge summary dictated by Dr. Sack. Pet. Ex. 20, pp. 9-10. Dr. Massey was the consultant who evaluated Mrs. Scott during her stay. He noted the difficulty in diagnosing her:

> Though she had radiologic, serologic, and chemical evidence to support a diagnosis of secondary antiphospholipid antibody syndrome, perhaps associated with lupus (?), there is also evidence to suggest disseminated sclerosis. However, she has no objective physical or neurologic deficits on

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[31] Doctor Anderson's curriculum vitae may be found in the Supplementation to Petitioner's Prehearing Memorandum. He is board-certified in internal medicine and rheumatology, has a private clinical rheumatology practice, and is a clinical associate professor in internal medicine at the University of Kansas School of Medicine. *Id;* Tr. at 8. I accepted him as an expert in the field of rheumatology. Tr. at 9.

[32] "Pathognomonic" clinical marker refers to a sign or symptom specific to a particular disease. *See Mosby's Medical Dictionary* at 1410. In this context, Dr. Massey is noting that there is no single definitive test or symptom for anticardiolipin antibody syndrome.

> today's examination to support the laboratory tests. In addition, the marked frequency of these spells, their association with stress, and the failure of one of these episodes to evolve into a fixed neurologic deficit would suggest a more benign disorder as well.

Pet. Ex. 20, p. 27. (emphasis added) Doctor Massey's comments are significant in that APS is not diagnosed based on the presence of anticardiolipin antibodies in the blood alone. As Dr. Anderson testified, asymptomatic circulating anticoagulants are common in the normal population, with about 2% of women having elevated anticardiolipin antibodies. Tr. at 17-18; 73-74. The Paran article[33] (Res. Ex. L2) also indicates that antiphospholipid antibodies are found at a low frequency in the normal population; at a higher frequency in persons with autoimmune diseases, especially lupus; and in 8-32% of MS patients. *Id.* at 3. Pregnancy loss, thrombosis, or neurological disturbances are the symptoms required to meet the classification criteria for APS.[34] In the absence of these symptoms, circulating anticoagulants are not clinically significant. Tr. at 18.

The brain MRI conducted during this hospitalization was not significantly different from the earlier MRI and the Magnetic Resonance Angiography found no evidence of any aneurysm. Pet. Ex. 20, p. 52. The spinal MRI did not demonstrate any demyelinating disease, although it showed some degenerative disc changes. *Id.* at 46. An EEG on April 25, 2003, was normal. *Id.* at 3.

Mrs. Scott saw Dr. Massey again on July 14, 2004, after a third MRI, which did not reveal any new abnormalities. Doctor Massey did not make a specific diagnosis; he assessed her as suffering from "[m]ultiple episodes of transient hemiparesis[35] without fixed neurologic deficit, etiology unknown. Again, this may relate to a vasculopathy associated with anti-phospholipid antibody syndrome, and/or systemic lupus erythematosus." He emphasized that she should be examined again "as shortly after the onset of the symptoms as possible" in order to make a specific diagnosis. Pet. Ex. 33, p. 2. Dr. Massey's concern that she be seen while experiencing the neurological symptoms may be explained in part from the visit preceding this July 2004 visit. On June 28, 2004, Dr. Massey had noted that Mrs. Scott had reportedly experienced two

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[33] Paran article, *supra*, n.23.

[34] The international criteria established for diagnosis of APS requires either pregnancy loss or thrombosis plus the presence of the relevant antibodies on two blood tests six weeks apart, for diagnosis. *See* Res. Ex. F, Wendell A. Wilson, *et al.*, "International Consensus Statement on Preliminary Classification Criteria for Definite Antiphospholipid Syndrome", 42 *Arthritis & Rheumatism* no.7, pp. 1309-1310 (July 1999). Mrs. Scott had neither pregnancy loss or thrombosis. Doctor Anderson explained that neurological symptoms coupled with antibody titers are commonly considered as clinical evidence of APS, even in the absence of fetal loss or thrombosis. *See* Tr. at 10.

[35] "Hemiparesis" means muscular weakness on one side of the body. *Mosby's Medical Dictionary* at 866.

incidents of hemiparesis, but that she had not sought medical attention for either.[36] Pet. Ex. 33, p. 3.

E. Dr. Anderson's Treatment and Opinions on Causation (May 2003 - February 2006).

On May 19, 2003, approximately six weeks after Mrs. Scott's hospitalization at Via Christi hospital, Dr. Anderson opined that there was a reasonable medical probability that Mrs. Scott "has had an immune reaction to immunization." Doctor Anderson prefaced this opinion with the observation that he had "visited with her again at length about the onset of her symptoms after immunization, the symptoms that she had, and reviewed all of her previous and recent laboratory." Pet. Ex. 1, p. 1. Unfortunately, Dr. Anderson did not set forth what Mrs. Scott told him about the time of onset and the nature of her symptoms,[37] nor did he discuss the basis for his opinion that the unnamed vaccination caused the disease process at work in Mrs. Scott.

Doctor Anderson had been less positive about the causal connection three months earlier when he opined: "Lastly, there still remains the underlying question of whether immune system activation is related to exposure to vaccination, whether she has idiopathic[38] systemic lupus erythematosus, and I think we are simply going to have to monitor her progress." Pet. Ex. 1, p. 11.

In an August 2003 letter to petitioner's attorney, Dr. Anderson notes something at odds with the medical records–a second immunization. His letter states: "She was immunized as part of a routine job requirement because of her work as a nurse. After the initial immunization, she developed respiratory problems, and subsequently after a follow-up immunization, she developed hemiparesis."[39] Pet. Ex. 1a, p.1 (emphasis added). Although Mrs. Scott had childhood immunizations against measles, mumps, and rubella, her medical records reflect only one MMR vaccination for a job requirement, in April 2002, after which she sought medical attention for respiratory problems. Pet. Ex. 5, p. 4. Doctor Anderson characterized what happened to Mrs. Scott as "a typical postimmunization response of activation of her immune system. Unfortunately, I think this involved the presence of specific autoantibodies and the presence of anticardiolipin antibodies and this probably led to significant neurologic problems, including

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[36] One of the incidents involved a sudden onset of right-arm weakness, light-headedness, confusion, and inability to remain erect. After lying down on the porch for several hours, she went to bed, only to awaken confused. She reported that her children wanted her to go to the hospital, but that she refused to go.

[37] Doctor Anderson did not have all the prior medical records at the time he rendered this opinion. Tr. at 61. He reviewed her medical records within a few weeks of the hearing, but did not review all of Dr. Sack's records and had not seen Dr. Mullinix's records as of the date of the hearing. Tr. at 62-64, 66-67.

[38] Defined as "without a known cause." *Mosby's Medical Dictionary* at 943.

[39] Although Dr. Anderson did not testify about two MMR immunization reactions at trial, this statement suggests that he viewed the case as a challenge-rechallenge scenario. Mrs. Scott's case does not present a challenge-rechallenge pattern because there is no evidence that she suffered any ill effects from her 1974 immunization for mumps and rubella or her 1983 immunization for measles.

hemiparesis and abnormal neurological symptoms." Pet. Ex. 1a, p. 1. He did not explain how the immune system activation by the vaccination could or did cause her to develop anticardiolipin antibodies, nor, beyond citing hemiparesis, did he identify the "significant" neurological symptoms to which he referred. While he referenced Mrs. Scott's neurologist, that neurologist (Dr. Massey) had been unable to find any fixed neurologic defects during Mrs. Scott's April 2003 hospitalization. Ex. 33, pp. 2-3. Doctor Anderson concluded his letter to Mrs. Scott's attorney: "It is my opinion that this woman was immunized and developed complications directly related to her immunization." Pet. Ex. 1a, p. 1.

Doctor Anderson explained his earlier opinion regarding the link between immunization and circulating anticoagulants in a record dated June 24, 2004. He noted that there were reports of measles, mumps, and smallpox infections causing circulating anticoagulants. He reasoned that immunization with these agents might well cause a similar immune response. He opined that "[s]he does not have lupus." Pet. Ex. 29, p. 2.

However, in November 2004, Dr. Anderson seemed less sure that the medical literature supported his belief that the MMR vaccination caused Mrs. Scott to develop anticardiolipin antibodies. His opinion reads:

> It is my opinion that the syndrome of problems for Dayna Scott began after her MMR vaccination suggesting at least a temporal relationship. I think this is anticardiolipin antibody syndrome. It is not a specific disorder that is commonly characterized as a reaction to the MMR. I do think, however, that the pathophysiology is entirely consistent. There certainly are reports in the literature of antiphospholipid antibodies being produced by vaccinations. There are reports in the literature of anticardiolipin antibodies being produced by the infection to the agents being immunized against. I think it makes clear sense, but in this young woman, immunization with proteins utilized to provide an antibody response caused a cross-reactive antiphospholipid antibody response. It actually surprised me a little bit that we were unable to find specific literature related to this, but I suspect it has not been routinely tested.

Pet. Ex. 35, p. 1. In the same letter, Dr. Anderson stated:

> I think that because of it's [sic] temporal relationship to her immunization, the documented production of anticardiolipin antibodies, and the support in the literature of immunizations and infections causing anticardiolipin and antiphospholipid antibodies, that in all likelihood, this woman was immunized for MMR, developed antiphospholipid or anticardiolipin antibodies, developed neurologic symptoms that were similar to multiple sclerosis based on the anticardiolipin antibodies, and I would strongly discourage

her from being exposed to MMR in the future.

Id. at 2. In correspondence with petitioner's counsel in response to this letter, Dr. Anderson indicated that the literature he had previously provided (filed as Pet. Exs. 22 and 37) were all the articles to which he referred. Additional articles were filed post-hearing as Pet. Ex. 65. Doctor Anderson briefly discussed the medical literature, including several of the articles filed post-hearing, at Tr. 29-49.

During the hearing, Dr. Anderson attributed the initial connection between Mrs. Scott's abnormal antibodies and the MMR vaccination to two of her previous doctors, but did not explicitly testify that any other of Mrs. Scott's doctors also believed that the MMR vaccine caused her symptoms. Tr. at 15. He did not identify which two doctors he meant, but Dr. Massey's earlier (and apparently abandoned) suggestion of a possible post-vaccinal encephalomyelitis[40] and Pet. Ex. 22, p. 1 (a letter from Dr. Anderson to petitioner's counsel) suggest Dr. Massey was one of the two. Doctor Massey, however, stopped short of opining that the MMR vaccination caused Mrs. Scott's condition, later calling it "etiology unknown" (Pet. Ex. 33, p. 2), and I was unable to find any further reference in his medical records to a connection between the vaccination and her condition. Doctor Calvin Olmstead, a neurologist Mrs. Scott saw in 2005, recorded the MMR vaccination in the history Mrs. Scott provided to him (while expressing some skepticism about her account). Doctor Olmstead did not opine that the vaccination was causal. Pet. Ex. 55, pp. 4-5.

Doctor Anderson also testified that he came to his conclusion on causation after reviewing the literature and discussing Mrs. Scott's case with Dr. Cannon. Tr. at 52. He did not elaborate on what Dr. Cannon may have said, but Dr. Cannon's records do not reflect any opinion that the MMR vaccination caused Mrs. Scott's illness.

By August 2004, Dr. Sack had apparently adopted Dr. Anderson's view of the cause of Mrs. Scott's "[h]istory of left-sided weakness," calling it "secondary to MMR reaction." Pet. Ex. 31, p. 7 (emphasis added). He did not characterize her illness as MS, APS, or make any particular diagnosis. Although Dr. Sack noted a history of "left-side" weakness, Mrs. Scott's initial report to him concerned bilateral upper extremity tingling and weakness. Pet. Ex. 2, p. 59. Her initial report to Dr. Mullinix reflected a right-sided onset, with symptoms later spreading to the left. Pet. Ex. 17, pp. 4-5.

F. Treatment and Diagnoses by Dr. Lynch and Dr. Olmstead (April 2005).

Mrs. Scott was admitted to Wesley Medical Center on April 2, 2005 for severe headaches, where she saw Dr. Olmstead because Dr. Massey was unavailable. Pet. Ex. 55, p. 2. In an April 5, 2005 letter to Dr. Massey, Dr. Olmstead recounted the history provided by Mrs. Scott as

[40] Doctor Massey's encephalomyelitis suggestion may well have been based on Mrs. Scott's medical history of fever two days post-vaccination and six days of paralysis a week or two after the vaccination. As indicated in Part IIG, below, I did not find this history to be credible.

"approximately three weeks after having an MMR injection she had a stroke. She had completely recovered from this stroke. She was then subsequently diagnosed as having antiphospholipid antibody syndrome and...an abnormal MRI." He recorded the basis for this admission as a two-week history of intensifying paresthesias[41] affecting all four limbs, coupled with a persistent headache. Doctor Olmstead immediately considered MS the probable diagnosis, a conclusion he believed was buttressed by a brain MRI showing active lesions that enhanced with gadolinium. He also ordered a VEP, which showed highly unusual results. He commented that he had never seen "P100 latencies that prolonged." He discussed her case with Dr. Anderson, who recommended repeating tests for anticardiolipin antibodies and lupus anticoagulant. Pet. Ex. 55, pp. 2-3. Doctor Olmstead concluded his letter with the following observation: "I further understand that there is litigation pending, with the allegation that the MMR injection caused a stroke. I do not have the benefit of the full story, but based on the information available and based on the fact that Dayna's biological mother also has MS, my contention is that Dayna never did have a stroke and probably does not have antiphospholipid antibody syndrome, and that her symptoms are explicable on the basis of relapsing/remitting MS." *Id.* at 3.

Doctor Olmstead's consultation report from the same hospitalization appears equally skeptical about the history of Mrs. Scott's illness and its association with a vaccination. He characterized her report as "an interesting story." He summarized the medical history provided by Mrs. Scott as receiving an MMR vaccination, followed three weeks later by a stroke that affected her left side. Pet. Ex. 55, p. 4 (emphasis added).

Mrs. Scott was referred to Dr. Lynch, another neurologist, who saw her at the University of Kansas Hospital Neurology Clinic in September 2005. Doctor Lynch recounted a history of an MMR vaccine in 2002, followed by difficulty breathing and left-sided weakness and dysarthria[42] occurring a "week or two after her injection" with a left-sided numbness that never resolved. Pet. Ex. 57, p. 2. Doctor Lynch observed a malar rash[43] that Mrs. Scott indicated was intermittent. *Id*. at 3.

Doctor Lynch concluded that Mrs. Scott suffered from "vasculitis secondary to lupus or other collagen vascular disease," adding that her central nervous system abnormalities would suggest an inflammatory process and not a demyelinating one. Pet. Ex. 57, p. 3. The medical reports and records from Dr. Lynch were the last medical records filed.

G. Resolving the Conflicts in the Evidence.

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[41] "Paresthesias" refers to sensations such as numbness, tingling, or "pins and needles." *Mosby's Medical Dictionary* at 1402.

[42] Dysarthria is difficult, poorly articulated speech. *Mosby's Medical Dictionary* at 602.

[43] A malar rash refers to a rash on the cheek or cheekbones. A 'butterfly" rash across the cheekbones and nose is a symptom of lupus. *Mosby's Medical Dictionary* at 1813.

Mrs. Scott's account of the timing and severity of her post-vaccinal symptoms changed often between April 2002 and September 2005. I find the earlier (and relatively contemporaneous) medical records to be the most accurate reflection of what she experienced and when. The records of April 19 and 22, 2002, from Dr. Sack do not reflect any fever or complaint of fever. These two visits, at four and seven days post-vaccination, contain no mention of paralysis or muscle weakness. At the May 14, 2002 visit, Dr. Sack first recorded complaints of weakness and sensory changes post vaccination, including dizziness over the previous five days, but made no reference to paralysis. Pet. Ex. 2, pp. 56-57, 59. The time frame "five days" would suggest an onset of May 9, 2002 for these symptoms, or 24 days after the vaccination.

Although petitioner's husband, Jeff Scott, testified about an onset of neurological symptoms approximately one week after the vaccination (Tr. at 211) and Mrs. Scott testified about an onset of neurological symptoms two to three weeks after she saw Dr. Sack for respiratory complaints (Tr. at 217-18), I credit the contemporaneous medical records for time of onset of the neurological symptoms. While I did not discount the testimony of Mr. and Mrs. Scott entirely, Mrs. Scott is simply not an accurate historian. Her accounts regarding timing and severity of the first post-vaccination neurological symptoms differ throughout the medical records. Her affidavit and her hearing testimony likewise differ from the contemporaneous records.

In resolving these conflicts, I find that the onset of post-vaccination neurological symptoms began on or after May 9, 2002. I also find that Mrs. Scott experienced symptoms in 1998, 2000, and 2001, prior to her vaccination, similar to those about which she complained after the vaccination. Pet. Ex. 2, pp. 18, 31, 46. While the 1998 symptoms of left-arm numbness may be attributable to the traffic accident a few weeks earlier, the 2000 symptoms of arm numbness and 2001 symptoms of general fatigue do not appear related to this accident.

In addition to the discrepancies in the onset of Mrs. Scott's symptoms, there are numerous discrepancies in the medical records regarding the nature of those symptoms. I find that Mrs. Scott experienced only respiratory problems and fatigue in the week following her vaccination, symptoms remarkably similar to those she had experienced in the preceding thirteen months. The relatively mild complaints of weakness, sensory changes, dizziness, and lightheadedness she provided to Dr. Sack on May 14, 2002, form the most contemporaneous account of what Mrs. Scott actually experienced on or about May 9, 2002, and I adopt these recorded symptoms as the facts of this case.

Three weeks later, Mrs. Scott elaborated on those symptoms in her medical history to Dr. Mullinix, describing tingling in her right arm, blurry vision, and a "near blackout" at work, in addition to dropping things with her right hand. She additionally described a spread of the tingling to her left side, legs that felt "like jello," dragging her feet, and stuttering and losing her train of thought. I accept Mrs. Scott's account of these symptoms, but find that the additional symptoms first occurred, more likely than not, between the May 14, 2002 visit to Dr. Sack and this June 7, 2002 visit to Dr. Mullinix. These additional symptoms are so strongly suggestive of

MS or other neurological problems[44] that Dr. Sack, who recorded a family history of MS and ordered tests diagnostic of MS, would scarcely have failed to record them.

I find no evidence in the contemporaneous records that Mrs. Scott experienced fever in the week after her vaccination, initial left-sided weakness after her vaccination,[45] or "paralysis" or a "stroke" in the two or three weeks post-vaccination. I do not accept as factual any medical history provided by Mrs. Scott that includes these symptoms in the period between April 12, 2002 and June 7, 2002. I reject Mrs. Scott's affidavit and hearing testimony to this effect.

In evaluating Mrs. Scott's testimony, I relied upon her somewhat hesitant demeanor on the witness stand, as well as her husband's testimony about her memory problems and several references to subjective memory problems in the medical records. In discounting Mrs. Scott's testimony and affidavit concerning the onset of paralysis within the three weeks post-vaccination, I also considered it highly unlikely that a patient with a history of numerous doctor visits for relatively minor complaints would not seek immediate medical attention for a sudden attack of paralysis or possible stroke.

I note that two of Mrs. Scott's treating physicians appear to share my skepticism about her ability to recount accurately her symptoms. *See, e.g.*, the consultation report by Dr. Massey, in which he recounts Mrs. Scott complaining that her left arm is "mostly useless." Doctor Massey then described Mrs. Scott holding her left arm by her side, but observed that when she was distracted, she moved her left arm normally with no "incoordination." He further noted that she seemed to walk with a left limp, but when asked to hop on one leg, she chose to begin hopping on the left leg with no difficulty. Pet. Ex. 20, p. 27. During his testimony, Dr. Anderson also acknowledged that several of Mrs. Scott's treating experts had some doubts about whether she was accurately reporting her symptoms. Tr. at 85. Her primary care physician, Dr. Sack, commented upon her discharge from Via Christi Regional Medical Center in April 2003: "I rec. care in labeling her condition right now. Objective testing for clarification will help (indecipherable) treatment." Pet. Ex. 20, p. 15 (emphasis added). Doctor Olmstead characterized her medical history as "an interesting story" and rejected her account of suffering a stroke three weeks post-vaccination. Ex. 48, pp. 27-28.

Having resolved the factual discrepancies regarding the onset and nature of Mrs. Scott's symptoms, I now turn to the issue of causation.

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[44] Early symptoms of MS include paresthesias, muscle weakness, vertigo, and visual disturbances. Later symptoms include ataxia (an abnormal or staggering gait). *Mosby's Medical Dictionary* at 1234. Doctor Brenner intimated during his testimony that, based on the symptoms Dr. Sack recorded, any reasonable physician would have suspected MS and would have ordered the tests that Dr. Sack in fact ordered. Tr. at 140-41.

[45] The affected arm or side of the body Mrs. Scott has identified in her onset history has varied from her initial complaint of bilateral weakness in May 2002, to right-sided weakness in June 2002, to left-sided weakness in November 2002 (Dr. Massey) through April 2005 (Dr. Olmstead).

III. Causation

A. Legal Standards in Actual Causation Cases.

To be eligible for compensation under the Vaccine Program, a petitioner must either demonstrate a "Table" injury, to which a statutory presumption of causation attaches, or prove by a preponderance of the evidence that a vaccine listed on the Vaccine Table caused or significantly aggravated an injury. *Althen v. Sec'y, HHS*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Grant v. Sec'y, HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). None of the diagnoses for Mrs. Scott's condition is listed on the Vaccine Table; therefore, Mrs. Scott must demonstrate that the MMR vaccination she received on April 14, 2002 caused or significantly aggravated her condition or injury.

In the case of an "off-Table" injury, a petitioner must "show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen,* 418 F.3d at 1278. See also, *Hines v. Sec'y, HHS*, 940 F.2 1518, 1525 (Fed. Cir. 1991). Circumstantial evidence and medical opinions may be sufficient to satisfy the second *Althen* factor. *Capizzano v. Sec'y, HHS*, 440 F.3d 1317, 1325 (Fed. Cir. 2006).

Petitioner need not show identification and proof of specific biological mechanisms, as "the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body." *Althen*, 418 F.3d at 1280. The petitioner need not show that the vaccination was the sole cause or even the predominant cause of the injury or condition; showing that the vaccination was a "substantial factor" in causing the condition and was a "but for" cause is sufficient for recovery. *Shyface v. Sec'y, HHS*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). *See also, Pafford v. Sec'y, HHS,* 451 F.3d 1352, 1355 (Fed. Cir. 2006). Petitioners may not be required to show "epidemiologic studies, rechallenge, the presence of pathologic markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect... ." *Capizzano*, 440 F.3d at 1325. Causation is determined on a case by case basis, with "no hard and fast *per se* scientific or medical rules." *Knudsen v. Sec'y*, *HHS* 35 F.3d 543, 548 (Fed. Cir. 1994).

When petitioners establish a *prima facie* case for compensation, the burden shifts to respondent to establish, "also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine." *Whitecotton v. Sec'y, HHS*, 17 F.3d 374, 376 (Fed Cir. 1994), *rev'd and remanded sub nom*. *Shalala v. Whitecotton*, 514 U.S. 268 (1995). Close calls regarding causation must be resolved in favor of the petitioner. *Althen*, 418 F.3d at 1280. *But see, Knudsen*, 35 F.3d at 550 (when evidence is in equipoise, the party with the burden of proof failed to meet that burden).

B. Constituents of a *Prima Facie* Case.

In Vaccine Act litigation, the Act itself establishes both the constituents of a *prima facie* case and rebuttable presumptions regarding causation. In a Table case, the presumption is in favor of causation, provided petitioner establishes that a covered injury occurred within the relevant time frame. *See* § 300aa–14. In an "off-Table" case, the petitioner has the burden of establishing causation. *See* § 300aa–13; *Grant,* 956 F.2d at 1147.

When a petitioner alleges an "off-Table" injury, eligibility for compensation–the *prima facie* case–is established when the petitioner demonstrates, by a preponderance of the evidence, that: (1) petitioner received a vaccine set forth on the Vaccine Injury Table; (2) she received the vaccine in the United States; (3) she sustained or had significantly aggravated an illness, disease, disability, or condition caused by the vaccine; and (4) the condition has persisted for more than six months.[46] Vaccine litigation rarely concerns whether the vaccine appears on the Table, the situs for administration, or whether the symptoms have persisted for the requisite time. Currently, vaccine litigation focuses most often on the issue of whether the injury alleged was caused by the vaccine.

Althen speaks to this third element of the *prima facie* case, causation. Establishing eligibility for compensation–the *prima facie* case–does not end the causation determination, because the statute provides a second requirement: "that there is not a preponderance of the evidence that the illness, disability, injury, condition, or death described in the petition is due to factors unrelated to the administration of the vaccine described in the petition." Whether this is a requirement placed on petitioners–to prove the absence of causes other than the vaccine–or a means by which respondent may rebut a *prima facie* case was the subject of some controversy in the early years of the program, but it is now clear that this is the respondent's burden. *See, e.g., Shalala*, 514 U.S. at 270-71. In this case involving a Vaccine Table injury, the U.S. Supreme Court indicated that the Vaccine Act implicitly places the burden to prove an alternate cause on the respondent. *See also Wagner v. Sec'y, HHS*, 37 Fed. Cl. 134 (1997) (petitioner could not be required to show absence of an explanation other than the vaccine for her condition). Once petitioner establishes a *prima facie* case, eligibility for compensation has been established, unless respondent shows by a preponderance of the evidence that something other than the vaccine caused or significantly aggravated petitioner's injury, disability, disease or condition. *Althen* 418 F.3d at 1278; *Knudsen*, 35 F.3d at 547.

C. Timing and Evidence in Establishing the *Prima Facie* Case.

Determining when the petitioner has met the burden of establishing the *prima facie* case is important because, at that point, the burden shifts to respondent to establish by a preponderance of

[46] Section 300aa–13(a)(1)(A). This section provides that petitioner must demonstrate by a preponderance of the evidence the matters required in the petition by section 300aa–11(c)(1)..." Section 300aa–11(c)(1) contains the four factors listed above, along with others not relevant in this case.

the evidence the existence of causation by a "factor unrelated to the administration of the vaccine."[47] Precisely when this burden shift occurs in the processing of a Vaccine Act case is not specified in the Act.

One might argue that once the petition alleges all the statutorily required elements and some evidence establishes the existence of each element, the burden then shifts to the respondent to prove the "factor unrelated." The plain language of the statute, however, suggests otherwise. In two subsections of § 300aa–13, the statute directs the court or special master to consider the complete record in determining whether the petitioner is eligible for compensation.[48] This necessarily requires consideration of matters in addition to those offered by petitioner in support of her case. In § 300aa-13(b), under the heading "Matters to be considered" the statute lists "relevant medical and scientific evidence," "any diagnosis, conclusion, medical judgment, or autopsy report," and "the results of any diagnostic or evaluative test." See also, *Ryman v. Sec'y, HHS*, 65 Fed. Cl. 35, 40 (special master performs gatekeeping function when he "determines whether expert testimony may be admitted or credited or otherwise relied upon.")

If a petitioner files an affidavit stating that he experienced onset of relevant symptoms within a certain time frame after vaccination and his medical expert opines that the occurrence of these symptoms within this time frame conclusively establishes a vaccine reaction, is respondent limited to proving an alternative cause for the resultant disease? The statute itself suggests otherwise, as it indicates that special masters are not bound by any particular "diagnosis, conclusion, judgment, test result, report, or summary" and in determining the weight to be afforded to these matters, "shall consider the entire record...." § 300aa–13(b)(1). To decide whether petitioner has established a *prima facie* case based only on petitioner's evidence would run contrary to the plain language of the statute. Respondent may challenge the factual underpinnings of a causation opinion, the opinion itself, or both before the special master determines whether a *prima facie* case has been established. Special masters weigh the evidence found in the medical records (*see, e.g., Ryman*, 65 Fed. Cl. at 40-41); consider evidence of bias or prejudice on the part of a witness, affiant, or expert (*see, e.g., Baker v. Sec'y, HHS*, 2003 U.S. Claims LEXIS 290, No. 99-653V, 2003 WL 22416622 (Fed. Cl. Spec. Mstr. Sept. 26, 2003)); weigh opposing medical opinions and the relative qualifications of experts (*see, e.g., Epstein v. Sec'y, HHS*, 35 Fed. Cl. 467, 477 (1996) and *Lankford v. Sec'y, HHS*, 37 Fed. Cl. 723, 726-27 (1997)) ; examine medical literature, studies, reports, and tests submitted by both sides (*see, e.g., Sharpnack, v. Sec'y, HHS*, 27 Fed. Cl. 457 (1993), *aff'd* 17 F.3d 1442 (Fed. Cir. 1994)); as well as considering a myriad of other factors in determining the facts of the case and the mixed questions of law and fact that arise in causation determinations. Special masters decide questions of credibility, plausibility, reliability, and ultimately determine to which side the balance of the

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[47] Section 300aa–13(a)(1)(B).

[48] Section 300aa-13(a)(1) provides in pertinent part: "Compensation shall be awarded under the Program to a petitioner if the special master or court finds on the record as a whole..." Subsection (b)(1) provides in pertinent part: "In evaluating the weight to be afforded to any such diagnosis, conclusion, judgment, test result, report, or summary, the special master or court shall consider the entire record..." (emphasis added).

evidence is tipped. *See, e.g., Burns v. Sec'y, HHS*, 3 F.3d 415, 417 (Fed. Cir. 1993) (credibility determinations uniquely within the special master's purview). *See also, Pafford*, 451 F.3d at 1359 ("Notably, this court accords great deference to a Special Master's determination on the probative value of evidence and the credibility of witnesses.").

If the special master concludes that petitioner's evidence of causation is lacking based on the record as a whole, the burden never shifts to respondent to demonstrate the "factor unrelated" as an alternative cause for petitioner's injury. *Bradley v. Sec'y, HHS,* 991 F.2d 1570, 1575 (Fed. Cir. 1993) (when petitioner has failed to demonstrate causation by a preponderance, alternative theories of causation need not be addressed) and *Johnson v. Sec'y, HHS*, 33 Fed. Cl. 712, 722 (1995) *aff'd,* 99 F.3d 1160 (Fed. Cir. 1996) (even in idiopathic disease claims, the special master may conclude petitioner has failed to establish a *prima facie* case).[49] If petitioner fails to establish one or more of the *Althen* factors, petitioner has failed to establish a *prima facie* case, as she has failed to establish causation. By challenging any of *Althen's* three causation factors, through cross-examination, introduction of medical literature, contrary testimony of well-qualified experts, or some other method, respondent may stymie petitioner's efforts to establish a *prima facie* case.

D. Mrs. Scott's Diagnoses.

Even a cursory examination of this record would reveal significantly different diagnoses of Mrs. Scott's condition among her treating physicians. Doctor Anderson is convinced that she has APS.[50] He has focused his treatment (Coumadin[51] therapy) on this condition. Doctor Mullinix diagnosed MS. Pet. Ex.17, pp. 6-7. Some years later, Dr. Olmstead similarly diagnosed relapsing/remitting MS. Pet. Ex. 55, pp. 2-3. Doctor Massey initially thought she had MS, but later adopted "anticardiolipin antibody syndrome associated with lupus" as the most likely diagnosis. Pet. Ex. 2, p. 100. Doctor Lynch opined that she has vasculitis secondary to lupus or some other collagen vascular disease. Pet. Ex. 57, p. 3.

The differing diagnoses can be explained in part by the similarities among the symptoms of these diseases and the lack of a pathognomonic marker for any of them. Antiphospholipid

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[49] If the respondent were limited to presenting the matters set forth in § 300aa-13(a)(1)(B)–proving by a preponderance of the evidence that the petitioner's condition is due to a factor unrelated to the vaccine–any petitioner with a disease for which medical science has not yet discovered a cause would be at a distinct advantage in Vaccine Act litigation. Section 300aa–13(a)(1)(B) indicates that respondent may not rely upon "idiopathic, unexplained, unknown, hypothetical, or undocumentable" causes as a "factor unrelated."

[50] During cross-examination, Dr. Anderson, citing the difficulty of making a lupus diagnosis, acknowledged that Mrs. Scott might actually have lupus. He explained that his clinical impression was that she did not have lupus at present. Tr. at 78-80. He also indicated that some of her laboratory tests supported the lupus diagnosis (the high level of double-stranded DNA), while others (the lack of Smith antibodies) did not. At least one of her laboratory tests was inconsistent with an APS diagnosis (the elevated PPT that later corrected). Tr. at 11-14. His basis for the APS diagnosis is largely Mrs. Scott's clinical presentation. Tr. at 79-80.

[51] Coumadin is the trade name for warfarin sodium, an anticoagulant. *PDR* at 1048.

antibodies are frequently found in patients with SLE, with some studies suggesting that 30-50% of those diagnosed with SLE have antiphospholipid antibodies in their blood. *See* Res. Ex. L1, Cuadrado, *et.al,* "Can Neurologic Manifestations of Hughes (Antiphospholipid) Syndrome be Distinguished from Multiple Sclerosis?", 79 *Medicine* No.1, pp.57-68 (2000). ["Cuadrado article"] This article indicates that about 30% of SLE patients have antiphospholipid antibodies, while Ex. 2, p. 2 of Supplementation of Exhibits to Petitioner's Prehearing Memorandum ("Pet. Sup. Mem."), a fact sheet on APS for health care professionals found at http://neuroland.com/cvd/aps.htm, indicates that up to 50% of SLE patients have these antibodies. Doctor Anderson testified that about half of SLE patients have these antibodies. Tr. at 10.

Primary APS is characterized as the presence of the antibodies and certain clinical features (most often thrombosis and fetal loss), but without an underlying systemic disease. Secondary APS is characterized by the presence of the antibodies along with systemic or autoimmune disease, most commonly SLE. Catastrophic APS is a life-threatening version of the syndrome in which at least three organ systems are affected by thrombosis, leading to damage or destruction of the organs. Res. Ex. H, Jaime Labarca, *et al.*, "Antiphospholipid Syndrome Associated with Cytomegalovirus Infection: Case Report and Review", *24 Clin. Infect. Dis.* No. 2, pp. 197-200 (1997) ("Labarca article").

These differing diagnoses are not fatal to Mrs. Scott's causation-in-fact case, as the label for the illness is not crucial in a non-Table case. *See, e.g., Kelley v. Sec'y, HHS*, 68 Fed. Cl. 84, 100 (Fed. Cl. 2005). However, on at least one level, the label for her disease may be significant, as Dr. Kinsbourne, one of petitioner's expert witnesses, stated that he was aware of no evidence linking SLE to vaccines (Pet. Ex. 65. p. 2), although his trial testimony indicated he may now have a different opinion about a link between SLE and vaccines. Tr. at 201. As petitioner's experts testified only about a theory of causation regarding APS, I have evaluated the evidence with regard to that theory and the APS diagnosis alone.

What is clear from the record is that Mrs. Scott has some central nervous system damage, as established by the VER and the brain MRI. Mrs. Scott also has a "circulating anticoagulant." What portion of her symptoms is related to these defects and what portion may be attributed to stress or other factors is not the present concern; the issue is whether the MMR vaccination is the cause of or a substantial factor in the central nervous system damage and anticoagulation problems.

E. Applying the *Althen* Factors to Determine Causation.

Althen's three prongs govern my inquiry into causation in the instant case. Considering those factors, I conclude that Mrs. Scott has failed to meet her burden to show causation. In this case, the causation call is not close, for Mrs. Scott failed to establish two of the three *Althen* factors. As petitioner failed to make a *prima facie* case, the burden never shifted to respondent to demonstrate "factors unrelated." Consideration of the *Althen* factors in reverse order is most efficient, for it is the third element–the proximate temporal relationship between the vaccination

and the disease–in which the failure of Mrs. Scott's proof is the most clear and obvious.

(1) Proximate Temporal Relationship[52]

Althen requires Mrs. Scott to demonstrate a "proximate temporal relationship," between her central nervous system or coagulation defects, not merely that the symptoms occurred post-vaccination. The timing of onset was discussed at length in Part II, above, because the temporal connection between the MMR vaccination and the onset of neurological symptoms figured significantly in the expert opinions of both Dr. Anderson and Dr. Kinsbourne. Doctor Anderson testified that the basis for his opinion that the MMR vaccination caused Mrs. Scott's APS was that "temporally she received the MMR in an appropriate time frame before she developed symptoms." Tr. at 25. I asked him to clarify what was an appropriate time frame, and he responded twice that it would take "at least five to fifteen days" to produce anticardiolipin antibodies. Tr. at 25-26. His testimony linked the production of anticardiolipin antibodies to the symptoms Mrs. Scott presented, testifying that she "developed symptoms seven or eight days after [the vaccine], and she started seeking medical advice a couple weeks later, and the laboratory studies were done weeks or months later, and actually even years later, she still has IgM anticardiolipin."[53] Tr. at 54-55. He later modified this testimony about when she developed symptoms to "within a week or two after the vaccine." Tr. at 63. Doctor Kinsbourne, petitioner's other expert witness, initially testified to onset of Mrs. Scott's neurological symptoms "about a week after the MMR vaccination." Tr. at 189. Doctor Kinsbourne later acknowledged that the medical records did not support this time frame. Tr. at 197.

As the Court of Federal Claims has noted, a doctor's "conclusions...are only as good as the reasons and evidence that support them." *Davis v. Sec'y, HHS*, 20 Cl. Ct. 168, 173 (1990). For the reasons stated at length earlier in this opinion, I do not find Mrs. Scott's testimony (or her after-the-fact reports to health care providers) that she experienced numbness, tingling, or

[52] In Petitioner's Final Argument ["Final Argument"], filed on May 8, 2006, counsel for petitioner suggested that I consider testimony in an entirely unrelated case for the proposition that symptoms of an MMR reaction could arise outside the temporal relationship relied upon by his own experts in this case. Final Argument, p. 7-8. I decline to do so. Petitioner's experts were clear in the time frames they found appropriate in this case. The fact that those time frames run counter to the onset evidence adduced here does not justify consideration of another expert's opinion about onset of another disease in another petitioner. Petitioner's counsel also engaged in a gratuitous attack on counsel for respondent, suggesting that the "tone, tenor and tenacity of the Government's criticisms of Petitioner's records, facts, and opinions" were unwarranted and different from those he had experienced in previous vaccine litigation. *Id*. at 8-9. I observed no inappropriate or unprofessional actions by respondent's counsel, either at the hearing or in written submissions to the court.

[53] The connection between vaccine causation and the presence of the antibodies years later is not clear in this testimony, and is even more muddled by an earlier opinion by Dr. Anderson. In a letter dated July 9, 2004, Dr. Anderson predicted that if Mrs. Scott's condition were related to her immunization, "the further she gets away from immunization, the more she should improve." Pet. Ex. 27, p. 1. Whether Dr. Anderson is referring to her coagulation state or the symptoms Mrs. Scott might experience is likewise unclear from this statement, but as he believes her symptoms are caused by the circulating anticoagulant, the distinction may be purely academic.

paralysis within two weeks of her MMR vaccination to be credible. I emphasize that I do not believe that Mrs. Scott is deliberately or knowingly lying in order to enhance her petition for damages; I believe that she has compressed or conflated the later appearance (three and one-half to four weeks post-vaccination) of mild neurological symptoms with her initial worry that her respiratory problems might be related to the vaccination.[54] Likewise, the mild neurological problems she reported to Dr. Sack on May 14, 2002, have enhanced over the intervening years. What role Mrs. Scott's reported memory difficulties have played in her inconsistent reports of symptoms is difficult to pinpoint, given that her health care providers have generally noted both short-and-long term memory to be intact, but it is clear that the severity of her symptoms, the side of her body affected at onset, and her recounting of the diagnoses have all changed markedly over her various medical histories. My reluctance to accept the later reports as more accurate than the relatively contemporaneous reports is buttressed by a similar reluctance of Mrs. Scott's treating physicians to accept her accounts of symptoms at face value.

I also relied on Dr. Sack's record-keeping as a whole, finding his clear and concise recording of symptoms, physical findings, relevant family history, reasoning for his diagnosis, and treatment decisions to be far better than those I have previously observed in records by primary care providers. While the absence of a reference to specific symptoms in a medical record in April 2002 does not conclusively establish that there were no symptoms during that time frame,[55] the general good quality of Dr. Sack's records, coupled with his recording such symptoms in May 2002, leads me to the conclusion that Mrs. Scott did not have the unrecorded symptoms at her April visits and did not report neurological symptoms post-vaccination until the May 14, 2002 visit. Because I have found that Mrs. Scott's mild neurological symptoms began, at the very earliest, twenty-four days post-vaccination, they were outside the window of "proximate temporal" connection established by her own expert. *Cf. Pafford*, 451 F.3d at 1358 (if "symptoms normally first occur ten days after inoculation but petitioner's symptoms first occur several weeks after inoculation, then it is doubtful that the vaccination is to blame.").

Special masters frequently accord more weight to contemporaneously recorded medical symptoms than those recounted in later medical histories, in affidavits, or in trial testimony. "It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." *Murphy v. Sec'y, HHS*, 23 Cl. Ct. 726, 733 (1991), *aff'd*,

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[54] Both expert testimony and medical literature establish that the MMR vaccination is not associated with respiratory problems. See Res. Ex. A, Report of Dr. Brenner, p. 8; Res. Ex. J, Martti Virtanen, *et al.*, "Day to Day Reactogenicity and the Healthy Vaccinee Effect of Measles-Mumps-Rubella Vaccination," *106 Pediatrics* No.5 (2000); and Res. Ex. K, Tom Jefferson, *et al.*, "Unintended events following immunization with MMR: a systematic review," 21 *Vaccine* 3954-60 (2003). Given Mrs. Scott's past reactive airway disease and history of bronchitis, pleurisy, asthma, and sinus infections, even if respiratory problems were medically linked to the MMR vaccine, I could not conclude by a preponderance of the evidence that her medical complaints on April 19 and 22, 2002 were caused in fact by the vaccination.

[55] *See, e.g., Murphy v. Sec'y, HHS*, 23 Cl. Ct. 726, 733 (1991) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance.").

968 F.2d 1226 (Fed. Cir. 1992), *cert. denied*, 506 U.S. 974 (1992). *See also*, *Cucuras v. Sec'y, HHS*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). Memories are generally better the closer in time to the occurrence and the motivation for accurate explication of symptoms is more immediate. *Reusser v. Sec'y, HHS*, 28 Fed. Cl. 516, 523 (1993).

Doctor Anderson's opinion about the temporal relationship is, therefore, flawed because it is based on his incorrect appreciation of when the symptoms arose. When an expert's opinion is based upon facts not established by the record, a fact-finder may reject the expert's opinion. *Bradley*, 991 F.2d at 1574.

Expert witnesses who are also treating doctors can be extremely persuasive witnesses, because they know the patients, their symptoms, and have followed the progress of their disease. They are most persuasive when their opinions about the cause of the disease dictate their treatment. In the instant case, however, the Coumadin therapy for the circulating anticoagulant is not dependent on the cause, or, apparently, even the label for the disease. In spite of his disagreement with Dr. Anderson about the cause of Mrs. Scott's condition, Dr. Brenner has no quarrel with his treatment for her. *See, e.g.*, Tr. 113-14.

Although a treating doctor's opinions may form the basis for finding vaccinal causation (*see Capizzano*, 440 F.3d at 1323), the instant case illustrates one of the disadvantages of using a treating doctor as an expert. In forming his opinion, Dr. Anderson did not view the entire record, and based his causation opinion on his erroneous assumption that onset of neurological symptoms occurred within a week or two of vaccination and that all of the symptoms Mrs. Scott described actually occurred. Doctor Brenner, respondent's expert, reviewed the entire record of Mrs. Scott's treatment prior to rendering his opinion, and thus noted the discrepancies in the accounts of symptoms and when they arose.

(2) The Medical Theory: Molecular Mimicry

Petitioner's treating expert, Dr. Anderson, relied on the theory of "molecular mimicry" to explain how the MMR vaccination triggered an autoimmune response in Mrs. Scott. Although he did not explain, either in his testimony or in his written opinions, what molecular mimicry is, the theory is discussed in *Principles of Neurology*.[56] Stated briefly, this theory postulates that in the immunization process, in certain genetically susceptible persons, something goes awry. Individual lymphocytes (B or T cells) commonly recognize (cross-react with) several different antigens. In most people, the vaccine causes those B or T cells that recognize the antigen in the vaccine to produce antibodies to that antigen. However, if the virus in the vaccination shares an antigen with something else in the body (a self-antigen), an immunization that activates these B or T cells to fight the virus also activates these lymphocytes to attack the self-antigen. For example, according to the theory, in central nervous system disorders such as MS, the vaccine causes the body's immune system to attack the myelin sheath surrounding the nerve cell in what is called an

[56] Maurice Victor & Allan Ropper, *Principles of Neurology* at 959 (McGraw-Hill, 7th ed. 2001).

autoimmune reaction. *Id.* at 959. *See also*, Lauren Sompayrac, *How the Immune System Works* at p.101 (2d ed. 2003). Because antiphospholipid and anticardiolipin antibodies are often found in individuals who have viral, bacterial, or other infections, an autoimmune reaction is a suspected cause of their production. Tr. at 14-15.

An article by Dr. Ronald Asherson, filed as part of Pet. Ex. 22 at p. 3,[57] briefly discusses the molecular mimicry theory with regard to APS and summarizes two animal studies providing support for the theory. The first involved mice immunized with tetanus toxoid, hemophilus influenza, and Neisseria gonorrhea. After immunization, the mice developed clinical symptoms consistent with APS. *Id.* at 5. The second study involved mice who received a synthetic peptide similar to cytomegalovirus. The mice developed antiphospholipid antibodies, but not APS. *Id.* at 6.

Considering the testimony and medical references, I conclude that Dr. Anderson's theory provided a sufficient basis to establish the first prong of the *Althen* test, a biologically plausible mechanism.

(3) Logical Sequence of Cause and Effect between the MMR Vaccine and Injury

Assuming, *arguendo*, that Mrs. Scott's symptoms arose within an appropriate window of time, and accepting the molecular mimicry theory as biologically plausible, I nevertheless conclude that petitioner has failed to establish the second prong of *Althen*–the logical sequence of cause and effect between vaccination and injury–by a preponderance of the evidence. In coming to this factual conclusion, I carefully considered the medical records and the testimony of all four expert witnesses, as well as the medical articles and studies to which they referred. I found Dr. Brenner's[58] testimony, in particular, to be the most persuasive. Not only was he eminently qualified to render the opinions he expressed, his testimony was based on objective medical tests and consistent with the medical records.

Reduced to their essence, Dr. Anderson's written opinions and testimony are based on the following reasoning: (1) Viruses and vaccines can cause APS; (2) Mrs. Scott had a vaccination; (3) thereafter, she developed neurological symptoms and blood tests that were consistent with APS; therefore, (4) the vaccine was the cause of her APS. *See*, Tr. at 57; Pet. Ex. 35, pp. 1-2; Pet. Ex. 29, p. 2. In order to test the strength or validity of Dr. Anderson's conclusion, it is necessary

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[57] Asherson, *et al.*, "Infections, Antiphospholipid Antibodies, and Antiphospholipid Syndromes" (found at www.rheuma21st.com) ["Asherson article"].

[58] Doctor Brenner's curriculum vitae is at Res. Ex. B. He is board-certified in rheumatology and has an active clinical practice. In addition, he serves as a consulting rheumatologist and immunologist at two Massachusetts hospitals and as a consultant to the Centers for Disease Control. While he has testified for respondent in approximately 20-30 vaccine hearings, he has also rendered opinions in favor of vaccine causation and has been part of a team investigating adverse events associated with anthrax vaccinations. Tr. at 97, 120. I accepted him as an expert in rheumatology. *Id.* at 99.

to examine his underlying factual assumptions. While the fact of the vaccination is not in question, there are ample questions about the leap between "some vaccinations can cause APS" to "this vaccine did cause APS" in Mrs. Scott.

The causation issue is further complicated by the fact that having antiphospholipid antibodies does not equate to having the antiphospholipid antibody syndrome. Some agents may trigger the production of antibodies without also triggering the disease. The two studies of mice summarized in the Asherson article at Pet. Ex. 22, pp. 5-6, illustrate this, with one set of mice developing antibodies only, while those exposed to tetanus, gonorrhea, and hemophilus influenza developed the syndrome as well. *See also*, Pet. Ex. 72, Z. Habibghahahi, "Anticardiolipin Antibody in Patients with Multiple sclerosis," an article published by the Lupus Foundation of America, date unknown, at 3 ("Antiphospholipid antibody, but not the syndrome, can be induced by drugs or infection.").

The causation issue is also muddled by the evidence that, while specific vaccines may trigger antiphospholipid antibodies and one vaccine (rabies) may trigger APS, there is a dearth of evidence that vaccines in general, as opposed to specific vaccines, can trigger either. Finally, the issue of persistent anticardiolipin or antiphospholipid antibodies and their relationship to viral infections bears on the issue of causation in this case.

Examining Dr. Anderson's starting point, there are several questions that must be answered. First, do viruses trigger APS? If so, are measles, mumps, and rubella viruses among those implicated in APS? If so, can the attenuated measles, mumps, or rubella viruses present in the MMR vaccine work similarly? And, even if all of these questions are answered affirmatively, is there sufficient reliable information in this record from which to conclude that the MMR vaccination was in fact responsible for Mrs. Scott's APS?

(a) Viruses and Vaccines as Causal Factors in APS

The medical literature submitted by both petitioner and respondent establishes to a reasonable degree of medical certainty that certain antiphospholipid antibodies are strongly related to infections, including viral infections. *See, e.g.*, Res. Ex. G, Imed Uthman and Azzudin Gharavi, "Viral Infections and Antiphospholipid Antibodies," 31 *Semin. Arthritis Rheum* No. 4, p. 256 (2002); Pet. Ex. 22, p.3 (Asherson article). Viral infections, including those caused by HIV, Hepatitis C, varicella zoster, Epstein-Barr, adenovirus,[59] cytomegalovirus, and parvovirus B viruses have been linked to the development of antiphospholipid antibodies, and in some cases, to APS as well. *See* Res. Ex. H, pp. 197-200 (Labarca article). Bacterial infections and some drugs

[59] Adenoviruses are pathogens associated with respiratory illnesses. Tr. at 205-06. There is ample evidence that Mrs. Scott had frequent respiratory infections.

can apparently trigger the presence of circulating anticoagulants in the blood stream.[60] No literature submitted, however, links persistent anticardiolipins of the type present in Mrs. Scott to viral infections. Tr. at 118.

(b) Are Measles, Mumps, or Rubella Viruses Associated with APS?

The fact that Virus A can cause Disease Z does not mean that Virus B also causes Disease Z. At best, it is weakly circumstantial evidence that Virus B might act similarly, and it may be no evidence at all that a vaccination for Virus B can cause Disease Z. Even accepting the molecular mimicry theory, different viruses are likely to present different antigens. While one may mimic a self-antigen, others may not. While there is medical literature linking the appearance of anticardiolipin antibodies, and in particular, the development of IgM antibodies, to the influenza and rabies vaccines (Tr. at 83; Pet. Ex. 37, Solnick, "Influenza Virus Vaccination Clinically Safe for Patients with Systemic Lupus," Vaccination News Homepage, 11/01/02)[61] and evidence of a possible link to the hepatitis B recombinant vaccine,[62] there is no evidence suggesting that the MMR vaccine is linked to the appearance of anticardiolipin or antiphospholipid antibodies, let alone to APS. An article for health care professionals found at Ex. 2, p. 4, of Pet. Sup. Mem., lists the percentages of people with various diseases who concurrently have antiphospholipid antibodies. There is a question mark next to the entry that includes measles and mumps, suggesting that no link between the two diseases and antiphospholipid antibodies has been established. Vaccination with an attenuated or killed Virus A may, indeed, cause Disease Z, but that is extremely weak evidence that a vaccination for a different disease also causes Disease Z.

Within the medical literature submitted by the parties, I found reference to older articles linking wild mumps and rubella viruses to the appearance of antiphospholipid antibodies. *See, e.g.,* Res. Ex. H, p. 198 endnote 12 (Labarca article). The article referenced in the endnote was published in 1986. Doctor Brenner's testimony discussed this 1986 study, pointing out that the

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[60] Bacterial infections and mycoplasmal pneumonia have also been associated with the development of antiphospholipid antibodies (Pet. Ex. 22, p. 12 (Asherson article), as have various drugs, including the oral contraceptives Mrs. Scott took prior to 2002. *See*, Ex. 2, p. 4 to Supplementation of Exhibits to Petitioner's Prehearing Memorandum, a fact sheet on APS for health care professionals found at http://neuroland.com/cvd/aps.htm. Mrs. Scott had evidence of a mycoplasmal infection in 2000. Pet. Ex. 2, p. 33.

[61] This brief report notes the increase, after influenza vaccination, of a variety of autoantibodies in SLE patients. The autoantibodies included IgM anticardiolipin antibodies in three of the 24 patients studied. The report does not directly state whether the IgM antibodies were persistent, but suggests that they were not. It notes that the appearance of the variety of autoantibodies found lacked "clinical significance," meaning that they were not associated with any disease process. During his testimony (Tr. at 31), Dr. Anderson indicated that he relied on this report to show that vaccinations can produce persistent IgM anticardiolipin antibodies. The abstract does not support that proposition.

[62] *See* Ex. 4 to Supplementation of Exhibits to Petitioner's Prehearing Memorandum ["Pet. Sup. Mem."], Porobić, *et.al*., "Antiphospholipid antibodies following vaccination with recombinant hepatitis B vaccine " 142 *Clinical & Experimental Immunology* No. 2, p. 377 (Nov. 2005).

study was done at a time when the term "anticardiolipin" did not necessarily mean what it does today. It is therefore extremely weak evidence that wild mumps or rubella infections could cause IgM anticardiolipin antibodies, and even weaker evidence that the attenuated virus in the vaccine could do so. Tr. at 107. Doctor Brenner also noted that in a case study involving a mumps infection, the antibody involved was IgA. The only other reference to mumps or rubella and antiphospholipid or anticardiolipin antibodies was a passing reference in the Asherson article, Pet. Ex. 22, p. 6, indicating that elevated antiphospholipid antibodies have been seen in mumps and rubella infections. The bulk of the article deals with APS in HIV infections. Without the opportunity to examine the articles or studies that form the basis for this conclusory statement, I do not find this to be strong evidence that two of the attenuated viruses present in the MMR vaccine can cause the development of antiphospholipid antibodies, much less APS.[63] Neither party submitted any medical literature linking the MMR vaccine to APS.

I am mindful that the Federal Circuit has held that petitioners may not be required, as a condition precedent to recovery, to produce medical literature linking a vaccine to their disease. *Althen,* 418 F.3d at 1281. However, when medical literature is submitted as evidence linking the syndrome to factors other than the vaccine in question, the lack of any literature suggesting a causal connection between the vaccine and the syndrome is some circumstantial evidence that the other causal factors are more likely than the vaccination to have played a role in the development of the syndrome. *Cf. Pafford* , 451 F.3d. at 1358 (noting that in the presence of other potentially causative agents, petitioner have a difficult burden in proving "but-for" causation by the vaccine). The Supreme Court has also cautioned against automatically rejecting novel scientific theories, while noting:

> [S]ubmission to the scrutiny of the scientific community is a component of "good science," in part because it increases the likelihood that substantive flaws in methodology will be detected.

The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a particular technique or methodology on which an opinion is premised.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U. S. 593-94 (1993).

(c) Linkage between Vaccination and Mrs. Scott's Antibodies and APS.

Doctor Anderson testified that only the rabies vaccine had been associated in the medical literature with the development of APS, as opposed to the mere presence of anticardiolipin or antiphospholipid antibodies. The other vaccines (influenza, oral polio, and hepatitis B) referenced in the medical literature were associated with the production of these antibodies, but not with APS–the syndrome from which Mrs. Scott suffers. Tr. at 82-84.

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[63] Doctor Anderson apparently read the article to stand for the same proposition, as he acknowledged that he had been unable to find a link between MMR vaccine and APS in the medical literature. Tr. at 82.

More telling, however, on the issue of causation in this case is the fact that viral-or vaccine-associated APS most frequently presents with significant differences from the type of antibodies present in Mrs. Scott. As Dr. Brenner explained,[64] the immunology of a viral infection roughly mirrors that of the immune system's reaction to a vaccine. Tr. at 100. Upon first exposure to a virus, the immune system makes IgM antibodies. After several weeks, the immune system begins switching from the production of IgM antibodies to IgG antibodies. Within three months, the IgM antibodies to this particular agent are no longer produced.[65] Tr. at 100-101. While the IgG antibodies may also decline in numbers, once the person is exposed to the same viral agent (through infection or vaccination), IgG antibodies are produced, but IgM antibodies are not. This is called an anamnestic response, and is exactly the response a vaccination is designed to trigger, should the vaccinated person be exposed to the targeted disease. Even though antibodies to the disease may not be measurable some months after vaccination, the T cell lymphocytic memory cells "remember" the first vaccinal exposure, and begin to manufacture the appropriate form of IgG after the second exposure. Tr. at 101-102.

According to Dr. Brenner, this immunological principle has been demonstrated in two reports of APS triggered by cytomegalovirus.[66] In explaining Res. Exs. H and I, Dr. Brenner testified that early in the course of their infections, the patients developed IgM antibodies. Later, the patients developed IgG antibodies. In these two cases, the antibodies disappeared within six months to a year. Doctor Brenner testified that this is the normal course in virus-induced APS. Tr. at 102-103.

He further explained that, because Mrs. Scott had previously received vaccinations against measles, mumps, and rubella (the vaccinations in 1974 and 1983), the second vaccination against all three viruses in April 2002, would trigger an IgG, not an IgM response. Thus, whatever was causing her to produce IgM antibodies, the MMR vaccination could not be responsible. Tr. at 100-02, 112, 115.

Doctor Anderson agreed that in people with normal immune systems, the initial antibodies produced would be IgM, followed by IgG antibodies. Tr. at 38-39. In Mrs. Scott's case, given the presence of IgM antibodies post-vaccination, he concluded that she had simply been one of the small minority of people who fail to respond to vaccinations. Tr. at 25. The April 2002 vaccination had, therefore, produced the initial IgM response. Tr. at 25-26. He did not explain why she would respond to the second vaccination, but fail to respond to the first two. Doctor Anderson also addressed the issue of the persistent IgM antibodies, analogizing to patients with rheumatoid arthritis. In that disease, the IgM antibodies specific to rheumatoid factor are

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[64] Doctor Anderson provided similar testimony. Tr. at 38-39.

[65] The IgM anticardiolipin antibodies present in Mrs. Scott are what their name implies–antibodies to anticardiolipin–not antibodies to the measles, mumps, or rubella viruses.

[66] *See* Res. Ex. H. (Labarca article), and Res. Ex. I, I Uthman, *et al.*, "Hughes syndrome associated with cytomegalovirus infection," 8 *Lupus* no. 9, pp. 775-777 (1999) ["Uthman casenote"].

persistent and never convert to IgG. Tr. at 38-39.

Doctor Brenner characterized this analogy as "apples and oranges" (Tr. at 115), as the rheumatoid factor antibodies are specific to the disease and not a post-infectious immune response. Nor, he testified, are the IgM antibodies present in Mrs. Scott related to IgM rheumatoid antibodies. *Id.* Because IgM antibodies are produced in response to an inciting event (i.e., a bacterial or viral infection), persistent IgM antibodies would imply the continuing presence of the inciting agent. Tr. at 103, 116. In viral infections associated with anticardiolipin antibody production, the anticardiolipin antibodies disappear within a year. Tr. at 118.

Mrs. Scott's medical situation differed from the IgM followed by IgG model. She consistently presented with IgM antibodies, and never developed IgG antibodies. Additionally, her antiphospholipid IgM antibodies were persistent, remaining for the entire period between the initial blood tests in 2003 and the hearing.

As Dr. Brenner explained at the hearing, anticardiolipin antibodies may be further subdivided into those that are post-infectious, that is, triggered by a viral, bacterial, or other infection, and those that are autoimmune, based on another blood factor called beta2glycoprotein. Pathologic or autoimmune anticardiolipins are anti-beta2 cardiolipin 1 dependent. Most viral infections are associated with independent anti-beta2 cardiolipin 1. Tr. at 106-07. Patients with chronic Hepatitis C with thalassemia are an exception, but the antibodies in those patients are not associated with thrombotic events. See Res. Ex. G, p. 258 (Uthman article). Leprosy and parvovirus infections may also be exceptions to the general rule that infections do not ordinarily produce beta2glycoprotein 1 dependent antibodies. *See* Pet. Ex. 22, p. 5 (Asherson article).

Doctor Leist's[67] testimony also countered Dr. Anderson's opinion on causation. He noted that there was no cutaneous evidence of a heightened response to the MMR vaccine and noted that the neurological symptoms about which Mrs. Scott complained after her MMR vaccination were not markedly different from those she displayed prior to the vaccination. Tr. at 148-49. Doctor Leist agreed with Dr. Brenner that the IgM antibodies persistent in Mrs. Scott made it unlikely that the MMR vaccination was causal. He explained that a persistent IgM response would mean that there had to be an ongoing replication of the causal virus in Mrs. Scott, and there was no evidence in her symptoms that any of the attenuated MMR viruses from the vaccination remained in her body. Tr. at 159-60. Additionally, Dr. Brenner noted that Mrs. Scott had not gotten better on Coumadin therapy, whereas most patients with APS improve with that therapy. Tr. at 133.

Doctor Lynch's opinion that Mrs. Scott suffered from inflammation also suggests that the causative agent for the IgM antibodies was something other than the vaccine. *See* Pet. Ex. 57, p. 3

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[67] Doctor Leist's curriculum vitae is at Res. Ex. D. He is a board-certified neurologist engaged in an active clinical neuroimmunology practice at Thomas Jefferson University where he also heads the MS center. He has published a number of articles dealing with the immune system (*id.*) and spends about 35% of his time in research. Tr. at 144. I accepted him as an expert in neurology. *Id.* at 145.

(urging treatment for inflammation). Doctor Anderson acknowledged Dr. Lynch's suggestion, but disagreed with it. Tr. at 51. Doctor Anderson did acknowledge that factors other than the vaccination could be responsible for the IgM antibodies. Tr. at 78.

(d) Timing of Antiphospholipid Antibodies' Development.

Doctor Anderson acknowledged that a previous history of neurological symptoms, such as the lightheadedness, numbness, fatigue and stress that occurred in 2000, long before the MMR vaccination, were similar to the symptoms she presented in May 2002. Tr. at 55-56. Because no other tests were done at that time, he was unwilling to opine if these symptoms were earlier evidence of APS in Mrs. Scott. *Id*. While he acknowledged that Mrs. Scott's treating physician diagnosed her as having a viral URI shortly after immunization and also acknowledged that viral infections could trigger the antibodies, Dr. Anderson was unwilling to rely on the treating physician's diagnosis of a viral infection. Tr. 57-58. When asked whether it was more likely the MMR vaccine, rather than the URI, that triggered Mrs. Scott's APS, he responded, "I'm not sure. It's hard to prove when people have the virus. We know she got the immunization."[68] Tr. at 59-60. He thereafter questioned the treating physician's diagnosis. *Id*. On cross-examination, Dr. Anderson indicated that a viral infection or a mycoplasma infection could potentially account for the IgM antibodies in November 2002, some seven months after the MMR vaccination, and acknowledged that she had a serologic test that was weakly positive for mycoplasma in 2000. However, he thought the presence of persistent IgM antibodies in 2002 as the result of a viral or mycoplasmal infection in 2000 to be unlikely. Tr. at 74-75.

I found this explanation regarding the persistent IgM antibodies and infection to be internally inconsistent with his testimony that an MMR vaccination in 2002 could cause persistent antibodies three years later. While persistent antibodies are unusual in and of themselves, Dr. Anderson did not offer any cogent explanation for what would make vaccine-induced anticardiolipin or antiphospholipid antibodies persistent in Mrs. Scott, while an infection with other agents well-associated with the development of such antibodies would not.

In contrast, I found Dr. Brenner's explanations and contrary opinion quite cogent and persuasive, as he relied upon proven data regarding the immune response to viral infection. As outlined above, Dr. Brenner testified that the nature of the antibodies produced and their persistency demonstrated the unlikelihood that the MMR vaccination triggered the development of APS in Mrs. Scott. Doctor Brenner also provided testimony and medical studies that strongly suggested that the antiphospholipid and anticardiolipin antibodies in Mrs. Scott likely predated

[68] Doctor Anderson also testified that the MMR vaccination could have caused the URI itself. Based on the two studies found in Res. Exs. J and K, *supra*, n. 52, and Dr. Brenner's opinion interpreting them (Res. Ex. A, p. 8), I do not accept Dr. Anderson's conclusion. While some vaccines may cause respiratory symptoms, the weight of the evidence is that the MMR vaccine does not. In the prospective blind trial covered in Res. Ex. J, in which twins received either an MMR vaccine or a placebo, respiratory symptoms were found more often in the twin receiving the placebo than in the twin receiving the MMR vaccine. In the meta-analysis detailed in Res. Ex. K, the authors likewise concluded that respiratory symptoms were not causally associated with the MMR vaccine.

her vaccination.

Two articles, Res. Exs. N and O,[69] cited by Dr. Brenner during his testimony were based on the same study of autoantibodies and SLE. They provide persuasive evidence for Dr. Brenner's position that Mrs. Scott likely had antiphospholipid and anticardiolipin antibodies prior to the May 2002 neurologic symptoms. The study examined 130 individuals with a confirmed diagnosis of SLE who also had available at least one stored blood sample drawn prior to the appearance of clinical symptoms. These individuals were matched with similar control subjects who did not have a diagnosis of SLE, but who also had similar blood samples available for study. The Arbuckle article (Res. Ex. N) noted that over 90% of patients who were positive for certain autoantibodies had a positive test long before clinical manifestations of disease. *Id.* at 1531. Antiphospholipid antibodies in the individuals with SLE appeared in the mid-range, two to three years before the onset of clinical symptoms, including clotting events, and their diagnosis. *Id.* The McLain article (Res. Ex. O) focused on the antiphospholipid antibodies in particular. Of the 130 SLE patients in the study, 24 were positive for IgM or IgG anticardiolipin antibodies prior to SLE diagnosis, with a mean of three years prior to diagnosis. Only four patients (3%) developed anticardiolipin antibodies after SLE diagnosis. The McLain article also concluded that anticardiolipin antibodies tended to precede clotting events by several years. *Id.* at 1231. While this study cannot conclusively establish that Mrs. Scott had antiphospholipid antibodies present prior to her MMR vaccination, it is strong circumstantial evidence for that proposition,[70] casting further doubt on Dr. Anderson's opinion on causation and the temporal relationship between vaccine and disease. Additional circumstantial evidence of a pre-vaccination onset for the APS is found in the echocardiogram done on May 15, 2000. Pet. Ex. 2, p. 37. Doctor Brenner testified that the left ventricular hypertrophy seen on that test is often seen in antiphospholipid syndrome. Tr. at 110.

E. Significant Aggravation–Vaccines as a Contributing Factor

If Mrs. Scott had antiphospholipid antibodies before the MMR vaccination, petitioner may still prevail if she can establish that the MMR vaccine, if not the cause of her APS, significantly aggravated it. Doctor Kinsbourne testified that if Mrs. Scott had an "antiphospholipid reaction at some level before the vaccination or even for a period of time before the vaccination," the MMR shot "precipitated a subclinical or latent disorder into the current condition, causing Ms. Scott the

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[69] Res. Ex. N, Melissa Arbuckle, *et al.*, "Development of Autoantibodies before the Clinical Onset of Systemic Lupus Erythematosus," 349 *New England Journal of Medicine*,1526-33 (2003) ["Arbuckle article"]; Res. Ex. O, Micah McLain, *et al.,* "The Prevalence, Onset, and Clinical Significance of Antiphospholipid Antibodies Prior to Diagnosis of Systemic Lupus Erythematosus," 50 *Arthritis & Rheumatism*, No. 4, 1226-32, (Apr. 2004) ["McLain article"].

[70] I reiterate that I am not diagnosing Mrs. Scott's condition as SLE or anything else. There do not appear to be any differences between the antiphospholipid antibodies present in APS and the antiphospholipid antibodies present in SLE; given the similarity of symptoms and the difficulty in distinguishing between the two diagnoses, studies of antiphospholipid antibodies in one disease would logically translate to the other.

symptoms and disabilities that have been amply discussed." Tr. at 190. Doctor Kinsbourne followed this quoted testimony with the comment that MMR was linked "by a temporal interval which would fall within the medically reasonable range." *Id.* I do not accept his conclusion, as it appears based on the same temporal association that I find to be contrary to the weight of the evidence in this case. Additionally, the sequence of cause and effect for an argument of significant aggravation suffers from the same logical defects discussed earlier. And, as Dr. Brenner noted, in studies done on people with immune disease, there is little demonstration of abnormal responsiveness to vaccination. Doctor Brenner therefore concluded that vaccines are unlikely to precipitate a subclinical problem into an established disease in a person. Tr. at 123-24.

I also note that Dr. Kinsbourne was not a particularly strong witness.[71] When questioned about the basis for his written opinions in this case, he deferred to Dr. Anderson. Tr. at 199-201. A fair reading of Pet. Ex. 63, p. 2, an email message Dr. Kinsbourne wrote to petitioner's counsel, suggests that he relied on Dr. Anderson to establish causation. His hearing testimony was similarly reliant. Tr. at 199. That email message also suggests that his testimony was carefully shaded to favor only those diseases for which vaccine causation might be established.[72] I found Dr. Kinsbourne combative and confrontational, in answering both my questions and those of respondent's counsel, and only when firmly pressed would he admit to an obvious misinterpretation of the medical records.

The weight of the evidence thus leads me to conclude that petitioner has failed to demonstrate that the MMR vaccination precipitated a subclinical APS into a clinically diagnosable syndrome.

F. Conclusions on Causation Claim.

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[71] I am not disparaging Dr. Kinsbourne's credentials as a physician. His curriculum vitae (Pet. Ex. 66) is most impressive, encompassing 38 pages. I accepted him as an expert in neurology. As a pediatric neurologist, however, his research, writing, and clinical efforts have focused on fields other than the causes of autoimmune diseases. He does not currently have an active clinical practice. Tr. at 198.

[72] Dr. Kinsbourne wrote: "On the positive side, the diagnosis of MS is now completely out of the question. However, I don't believe there is any literature which supports the view that MMR can cause systemic lupus" on December 2, 2005. He went on to comment: "I have not been able to find evidence in the literature that APS causes peripheral nerve palsies, and therefore, I incline to the lupus diagnosis." Pet. Ex. 63, p. 2. This statement suggests that he selected his diagnosis on the basis of whether that diagnosis was consistent with vaccine causation. At trial, when asked if he still held the opinion about literature not supporting a connection between MMR and lupus, he commented that "it isn't anymore, but as that's not my diagnosis, I haven't pursued it further." Tr. at 201-202. Medical opinions that attempt to conform the diagnosis to diseases for which vaccine causation can be established are far less reliable than those that form a diagnosis based on symptoms and a search for a cause. *Cf. Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 43 F.3d 1311, 1317 (9th Cir. 1995) (decision on remand from 509 U.S. 579 (1993)).

When the evidence is in conflict, the trier of fact must make credibility assessments. The Vaccine Act itself contemplates that a special master will weigh and evaluate opinions, testimony, and exhibits in determining whether causation exists. In determining the weight to be given to the testimony of the experts and the medical articles and studies, I found Dr. Anderson's theories to be less than persuasive. He relied upon factors not established in the medical records, discounted the treating neurologists' failure to find a fixed neurological defect in Mrs. Scott in making his diagnosis, and discounted the findings of Mrs. Scott's family physician regarding the cause of her initial, post-vaccination symptoms.

I thus conclude that petitioner has failed to demonstrate causation by preponderant evidence. She has not shown a proximate temporal relationship between vaccination and onset of relevant symptoms and has failed to establish a logical sequence of cause and effect between vaccination and her disease. Her experts' opinions were significantly undercut by their reliance on a temporal relationship that did not exist, as well as by the criticisms of those opinions by respondent's experts. Although Dr. Anderson had a biologically plausible theory regarding causation, the evidence failed to establish the other two *Althen* factors, and thus failed to establish causation.

IV. CONCLUSION

Petitioner has not demonstrated by a preponderance of the evidence that her condition was either caused in fact or significantly aggravated by the MMR vaccination she received on April 14, 2002. She has thus failed to establish a *prima facie* case for compensation and the petition for compensation is therefore DENIED. In the absence of a motion for review filed pursuant to RCFC, Appendix B, the clerk is directed to enter judgment accordingly.

IT IS SO ORDERED.

Denise K. Vowell
Special Master