In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 08-601V

Filed: July 30, 2012

LAKEYSHA ISAAC,	)	
	)	TO BE PUBLISHED
Petitioner,	)	
	)	
v.	)	Entitlement; Guillain-Barré
	)	Syndrome; molecular mimicry;
SECRETARY OF	)	tetanus-diphtheria vaccine
HEALTH AND HUMAN SERVICES,	)	
	)	
Respondent.	)	
	)	

Ronald C. Homer, Conway, Homer & Chin-Caplan, P.C., Boston, MA, for Petitioner; Lisa A. Watts, United States Dep't of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT [1]

LORD, Special Master.

I. INTRODUCTION AND SUMMARY

Petitioner LaKeysha Isaac ("Petitioner") alleges that she suffered Guillain-Barré Syndrome ("GBS") as a result of the tetanus-diphtheria ("Td") vaccine she received on September 13, 2005. Pet'r's Post-Hr'g Br. at 1, ECF No. 39.[2] On August 26, 2008, Petitioner filed a Petition seeking compensation under the National Vaccine Injury Compensation Program (the "Program"), 42 U.S.C. § 300aa-10 et seq. (2006).[3] On

[1] In accordance with Vaccine Rule 18(b), Petitioner has 14 days to file a proper motion seeking redaction of medical or other information that satisfies the criteria in 42 U.S.C. § 300aa-12(d)(4)(B). Redactions ordered by the Special Master, if any, will appear in the document as posted on the United States Court of Federal Claims' website.

[2] GBS is "rapidly progressive ascending motor neuron paralysis of unknown etiology, frequently seen after an enteric or respiratory infection." Dorland's Illustrated Medical Dictionary 1832 (32nd ed. 2012).

[3] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 et seq. (2006). Hereinafter, individual section references will be to 42 U.S.C. § 300aa of the Vaccine Act.

February 11, 2009, Respondent (the "Secretary") filed a report pursuant to Vaccine Rule 4(c) maintaining that compensation was inappropriate and that the Petition should be dismissed. Resp't's Rule 4(c) Rep. at 2, ECF No. 16.

A hearing was held on July 27, 2010. Following the hearing, the parties, at my request, attempted for a period of time to negotiate a resolution of this case, but they were unsuccessful. See Joint Status Rep. at 1, Apr. 19, 2012, ECF No. 52. The case is now ripe for decision.

Petitioner has not alleged a "Table" injury. See 42 C.F.R. § 100.3 (2011). "An off-Table petitioner, who does not benefit from a presumption of causation, must specify [her] vaccine-related injury and shoulder the burden of proof on causation." Broekelschen v. Sec'y of Dep't of Health & Human Servs., 610 F.3d 1339, 1346 (Fed. Cir. 2010). Therefore, to establish causation-in-fact, Petitioner must show by a preponderance of the evidence that but for vaccination she would not have been injured, and that vaccination was a substantial factor in bringing about the injury. Cedillo v. Sec'y of Dep't of Health & Human Servs., 617 F.3d 1328, 1338 (Fed. Cir. 2010). Petitioner's burden is to show 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 v. Sec'y of Dep't of Health & Human Servs., 418 F.3d 1274,1278 (Fed. Cir. 2005).

Petitioner has not presented preponderant evidence in support of a theory that the Td vaccine can cause GBS. Petitioner's expert relied on a theory of molecular mimicry supported by a report of challenge/rechallenge to assert that Td vaccine could cause GBS. The challenge/rechallenge evidence was based on a 33-year-old article concerning one individual who appeared to suffer repeated episodes of a demyelinating neurological disorder (at the time believed to be GBS) following tetanus vaccination. Petitioner's expert noted at hearing that the Institute of Medicine ("IOM") had cited this report as evidence of a causative link between Td vaccination and GBS.

The IOM no longer holds that there is evidence showing a link between tetanus vaccine and GBS. Rather, the IOM is neutral on this subject. Although Petitioner's expert at hearing laid great emphasis on the IOM's previous endorsement of a link between Td vaccination and GBS, he did not update or supplement his theory in response to the latest information from the IOM, even when offered the opportunity to do so.

On balance, the gap between what is known and what is hypothesized by Petitioner's expert is too large to constitute a reliable theory – it is mere speculation. Petitioner's expert hypothesized causation with respect to a vaccine and a medical condition that have no known association. He did so by asserting a possible analogy

between the vaccine and an unrelated bacterium that actually has not been shown to produce the form of GBS from which Petitioner suffers. On this record, there is insufficient reliable evidence to support Petitioner's theory of causation, even as a possibility, under Prong 1of the Althen test. See Moberly ex rel. Moberly v. Sec'y of Dep't of Health & Human Servs., 592 F.3d 1315, 1324 (Fed. Cir. 2010) (finding that a special master is entitled to some indicia of reliability).

Without preponderant evidence of a theory of possible vaccine causation, there cannot be a finding of a logical sequence of cause and effect based on that theory. See Moberly, 592 F.3d at 1326. Nevertheless, I review the evidence under Prong 2 of the Althen test for the sake of judicial economy and to provide additional context for my ultimate conclusion. The evidence on Prong 2 does not preponderate in Petitioner's favor. No treating physician actually endorsed vaccine causation and no evidence of vaccine causation appeared elsewhere in the medical record.[4] Petitioner's expert opinion on Prong 2 was not reliable, for a variety of reasons, and his analysis of the medical record was rebutted effectively by Respondent's expert. Because Petitioner failed to establish by preponderant evidence the requirements necessary to establish entitlement to compensation, the Petition is dismissed.[5]

II. BACKGROUND

A. Petitioner's Allegations

Petitioner alleged that she received tetanus-diphtheria and hepatitis A ("Hep A") vaccinations on September 15, 2005, and as a result suffered "a neurological demyelinating injury," GBS. Amended Pet. at 1, ECF No. 13. In the course of the hearing, Petitioner effectively abandoned the allegation of causation by the Hep A vaccine. See Tr. at 51-52; Pet'r's Post-Hr'g Br. at 1.[6]

Petitioner stated that she was in relatively good health before her vaccinations. "Shortly after" receiving them, she developed pain in her arms and back and weakness in her right leg. Amended Pet. at 2 (quoting Pet'r's Ex. 16 at 1). The pain persisted but improved in about a week. Two weeks after the immunizations, Petitioner noticed tingling in her extremities. Amended Pet. at 2 (citing Pet'r's Ex. 16 at 2).

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[4] Petitioner points to one notation in the medical history by Dr. Michael Winkelmann, a physical rehabilitation specialist. For the reasons discussed at length below, after careful consideration of Dr. Winkelmann's notation, I do not find that it constitutes a treating physician's opinion favoring causation.

[5] The Secretary conceded Prong 3 of the Althen test. See Tr. at 6-7.

[6] Dr. Tornatore did not opine that the Hep A vaccination caused Petitioner's GBS. See Pet'r's Ex. 20 at 8. Accordingly, Petitioner's claim of injury due to Hep A vaccination is unsupported and is hereby dismissed. 42 U.S.C. §§ 300aa-13(a)(1) ("The special master or court may not make such a finding based on the claims of a petition alone, unsubstantiated by medical records or by medical opinion."). In this opinion, I omit references to Respondent's evidence refuting the allegation of causation due to Hep A vaccination.

Petitioner presented to the emergency room at Mississippi Baptist Medical Center on September 28, 2005, with chest pressure, nausea, vomiting, and numbness. Amended Pet. at 3 (citing Pet'r's Ex. 4 at 51). No diagnosis was recorded at that time, and she was discharged two days later. Pet'r's Ex. 3 at 81. Petitioner stated that at the time of discharge, she was unable to stand. She continued to have difficulty ambulating at home and fell trying to reach the bathroom. Amended Pet. at 3-4 (citing Pet'r's Ex. 16 at 3).

Petitioner presented to the emergency room at St. Dominic Hospital early in the morning on October 1, 2005. Her chief complaint was numbness of the feet and weakness of the legs. Amended Pet. at 4 (citing Pet'r's Ex. 4 at 51). A nerve conduction study on October 3, 2005, revealed findings "most consistent with acute inflammatory demyelinating polyneuropathy." Amended Pet. at 5 (quoting Pet'r's Ex. 4 at 54). She was given IVIG therapy.[7] On October 3, 2005, one of Petitioner's treating neurologists, Dr. Alan Moore, indicated that her "AIDP is likely related to gastrointestinal illness." Pet'r's Ex. 4 at 54. Over the course of the following days, Petitioner began to improve and by October 11, 2005, she was participating in physical therapy. Amended Pet. at 7 (citing Pet'r's Ex. 4 at 34).

Petitioner was discharged from St. Dominic on October 13, 2005, with diagnoses of GBS, Bell's palsy, and hypertension. Amended Pet. at 7 (citing Pet'r's Ex. 4 at 48). She was admitted to Methodist Rehabilitation Center where she was treated by Dr. Michael Winklemann, a physiatrist. Amended Pet. at 8.[8] Dr. Winklemann noted Petitioner's history of GBS, "It was felt that immunization series had been the trigger for the development of Guillain-Barré . . . the culprit at this point in time, is felt to be the immunization. . . ." Amended Pet. at 8 (citing Pet'r's Ex. 6 at 3). Dr. Art Leis, an electromyographer, noted as part of Petitioner's history that she "[h]ad vaccination for [TD] and hepatitis [A] about 2 weeks before onset of altered sensation . . . ." Amended Pet. at 8 (citing Pet'r's Ex. 6 at 7).[9]

Petitioner continued to receive occupational and physical therapy, and she eventually returned to work. Amended Pet. at 9-11 (citing Pet'r's Ex. 16 at 5-6). She stated that she continues to experience symptoms. Amended Pet. at 11 (citing Pet'r's Ex. 16 at 6).

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[7] IV means "intravenous," Dorland's at 967, and IG means "immunoglobulin," id. at 913.

[8] Physiatry is "the branch of medicine that deals with the prevention, diagnosis, and treatment of disease or injury, and the rehabilitation from resultant impairments and disabilities, using physical agents such as light, heat, cold, water, electricity, therapeutic exercise, and mechanical apparatus, and sometimes pharmaceutical agents." Dorland's at 1443.

[9] Electromyography is "an electrodiagnostic technique for recording the extracellular activity (action potentials and evoked potentials) of skeletal muscles at rest, during voluntary contractions, and during electrical stimulation." Dorland's at 602.

B. GBS and Molecular Mimicry

GBS is a rare peripheral nerve neuropathy "characterized by acute flaccid paralysis and areflexia," believed to be caused by microbial organisms triggering an immunological response to autoantigens in peripheral nerves. Pet'r's Ex. 20, Tab B at 2; Tr. at 96 ("GBS is rare in the general population."). Two forms of the disorder have been recognized: acute motor axonal neuropathy ("AMAN") and acute inflammatory demyelinating polyneuropathy ("AIDP"). See Pet'r's Ex. C at 3. AMAN "is frequently seen in eastern Asian countries such as China and Japan." Id. AIDP is the form that "usually" occurs in Western countries. Id. AIDP occurs when the damage is to the myelin in nerve cells, not the axons. See Tr. at 42-44.[10] It is undisputed that Petitioner's diagnosis was AIDP.

Following the mass swine flu inoculation campaign in 1976, there were reports of a spike in cases of GBS. Pet'r's Ex. 20, Tab. C at 2. No medical literature reporting on other vaccinations has indicated a spike in cases of GBS. Tr. at 105 (citing Resp't's Ex. G). Nevertheless, the possibility of a connection between vaccines and GBS continues to be explored. See Resp't's Ex. G.

One theory proposing a link between vaccination and GBS is based on the biological concept of molecular mimicry. Molecular mimicry is "sequence and/or conformational homology between an exogenous agent and self-antigen." Ct. Ex. 2 at 1.[11] Such homology may cause cells in the immune system originally directed against the exogenous agent to react also against the self-antigen, leading to tissue damage and autoimmune disease. Id.

Proving that molecular mimicry occurs in the human body is difficult. Id. According to the IOM, there are many agents with homologous structures, most of which are not associated with autoimmune phenomena or autoimmune diseases. Id. The presence of cross-reactive antibodies and T-cells, taken in isolation, is not sufficient proof of molecular mimicry as the mechanism that induces autoimmune disease because cross-reactions are relatively common and are not generally pathogenic. Id.

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[10] Myelin is "the substance of the cell membrane of Schwann cells that coils to form the sheath . . . it has a high proportion of lipid to protein and serves as an electrical insulator." Dorland's at 1218. An axon is "the process of a neuron by which impulses travel away from the cell body; at the terminal arborization of the axon, the impulses are transmitted to other nerve cells or to effector organs. The larger axons are surrounded by a myelin sheath." Id. at 186-87.

[11] Homology is "the quality of being homologous; the morphological identity of corresponding parts; structural similarity due to descent from a common form." Dorland's at 868. Sequence is "the order of arrangement of residues or constituents in a biological polymer . . . . Id. at 1696. Conformation is "the particular shape of an entity." Id. at 403.

Notably, molecular mimicry has been used to explain the induction of a form of GBS by a bacterium, *C. jejuni*,[12] due to the homology between lipo-oligosaccharide of *C. jejuni* and GM1, a ganglioside found on the surface of nerve cells. Pet'r's Ex. 20, Tab B at 2.[13] The relationship between gangliosides and structures on the outer core of *C. jejuni* was confirmed experimentally in animals. Pet'r's Ex. 20, Tab B at 5-7. *C. jejuni* infection was reported to have induced the form of GBS known as AMAN. It has been noted that approximately one-quarter of GBS cases are preceded by *C. jejuni* infection. Pet'r's Ex. 20, Tab. B at 3. In such cases, GBS is a consequence of the immune response to *C. jejuni* rather than the direct effect of that microorganism. Id.

The literature on *C. jejuni* and GBS appears to provide reliable evidence that molecular mimicry could explain the cause of at least some autoimmune disease, including the axonal form of GBS. Additional literature submitted by Petitioner discusses other examples of molecular mimicry triggering autoimmune disorders. See Pet'r's Ex. 20, Tab A.

The fact that molecular mimicry exists as a biological phenomenon does not automatically mean that vaccines can cause autoimmune disease by that process. Applicable law instructs that special masters need not accept unsupported theories of vaccine causation without some indicia of reliability, and that a temporal association is not sufficient in itself. I accept the theory of molecular mimicry in some cases and reject it in others, depending on the particular vaccine, the injury, the reliability of the expert testimony supporting and opposing causation, and the weight of the other evidence in the record. See infra note 33.

Indicia of reliability may vary from case to case. As we know, there are no hard and fast rules. In a case in which influenza vaccine was alleged to have caused GBS by a process of molecular mimicry, there would be at least some indication (from the swine flu experience) that influenza vaccine can cause GBS. Here, by contrast, there is no "historical" link between tetanus vaccine and GBS. It is a farther stretch to establish that tetanus vaccine may cause GBS by the process of molecular mimicry. It would take more evidence than it would in a case of flu vaccination to tip the balance toward a finding of possible vaccine causation.

On this record, I find no reliable evidence linking tetanus vaccination and molecular mimicry. The medical literature submitted by Petitioner does not provide support for the proposition that tetanus vaccination can cause autoimmune disease by a

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[12] *C. jejuni* is a species of Campylobacter "that is a common cause of enteric campylobacteriosis in humans . . . ." Dorland's at 275.

[13] The term ganglioside refers to "[a]ny of a group of galactose-containing cerebrosides found in the surface membranes of nerve cells." The American Heritage Dictionary of the English Language 723 (4th ed. 2006).

process of molecular mimicry.[14] Moreover, the IOM concluded recently that the scientific evidence it reviewed did not confirm molecular mimicry as a "mechanism leading to the development" of post-vaccination injury. Ct. Ex. 2 at 3.

Petitioner's success in claiming a vaccine injury caused by molecular mimicry between the tetanus vaccine and GBS thus depends on the expert testimony of Dr. Carlo Tornatore. If I were to find that Dr. Tornatore's testimony supplied preponderant, reliable evidence, I would hold that Petitioner had satisfied Prong 1, notwithstanding the lack of supporting evidence elsewhere in the record. See Moberly, 592 F.3d at 1325-26 ("Assessments as to the reliability of expert testimony often turn on credibility determinations, particularly in cases such as this one where there is little supporting evidence for the expert's opinion.") (citations omitted). Unfortunately, I do not find Petitioner's expert's testimony reliable, for the reasons explained in the Discussion below.

C. Petitioner's Expert

1. Dr. Carlo Tornatore's Report

Dr. Tornatore is a neurologist at Georgetown University Hospital in Washington, D.C., and director of the Multiple Sclerosis Center there. Pet'r's Ex. 21 at 4. He trains residents and interns in neurology. Tr. at 11-12. Dr. Tornatore is a member of several neurological organizations, including the American Neurological Association. Tr. at 19.

Dr. Tornatore noted that Petitioner was vaccinated on September 15, 2005, and within two to three weeks developed symptoms that ultimately were diagnosed as GBS. Pet'r's Ex. 20 at 2. He opined that the tetanus vaccination resulted in Petitioner's "inflammatory neuropathy." Id. at 8. Dr. Tornatore noted that Petitioner had "chest pain, nausea and vomiting" but no symptoms "to suggest a systemic viral or bacterial illness." Id. "Indeed, rather than diarrhea, she was noted to be constipated during this period." Id. He stated that Petitioner's blood tests were suggestive of a diffuse inflammatory cause rather than an infectious agent, noting that "her IgA was elevated, her rheumatoid factor was borderline high and that her ANA revealed atypical cytoplasmic staining, all of which speak to a more diffuse activation of the immune system." Id.[15]

Dr. Tornatore described molecular mimicry as a mechanism by which vaccines trigger autoimmune responses that may lead to inflammatory demyelinating polyneuropathies. He cited swine flu and tetanus as two such vaccines, noting a 1979 article on the swine flu vaccination campaign and the 1978 Pollard and Selby report on

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[14] There may be additional support for the theory of molecular mimicry in the medical literature somewhere, but it is not in the record before me. It is worth repeating, again, that I do not require medical literature to satisfy Prong 1. An expert's opinion on the theory of molecular mimicry may preponderate in the context of the entire record in that case, whether or not it is supported by medical literature.

[15] IgA refers to immunoglobulin A. Dorland's at 913.

a single case of GBS. Id. "Indeed," his report continued, "the Institute of Medicine has recognized that tetanus vaccination can be a cause of Guillain-Barré based on the article by Pollard and Selby." Id.[16]

Dr. Tornatore stated that molecular mimicry is the process that resulted in Petitioner's illness. "[T]he vaccinations resulted in an immune response that then targeted not only the vaccine but antigens on the peripheral nerves/myelin resulting in an inflammatory demyelinating polyneuropathy as manifested clinically by sensory and motor signs and symptoms." Id. at 8-9.

Dr. Tornatore also opined that the temporal relationship of the vaccination and the onset of symptoms was appropriate, based on the experience with swine flu vaccine in the 1970s. Id. at 9.

2. Dr. Tornatore's Testimony

Dr. Tornatore explained the process of demyelination that results in GBS. Tr. at 22-24. He described the concept of molecular mimicry, as well as several articles discussing that phenomenon. Tr. at 24-26.

Dr. Tornatore explicitly linked the Pollard and Selby article to the theory of molecular mimicry "although they [Pollard and Selby] did not speak to molecular mimicry," but rather to the idea "that somebody who was rechallenged with the same protein developed the same problem on three different occasions, and so that that antigenic stimulus was recognized as the inciting factor for causing" GBS. Tr. at 26-27.[17] Dr. Tornatore opined that Pollard and Selby's article "offers specificity because in particular with vaccines, a vaccine is basically just a protein for the most part depending on the type . . . what you're doing is you're introducing foreign antigens into the body." Tr. at 27. Dr. Tornatore described the significance of Pollard and Selby's rechallenge findings:

> And the expectation is that the body will fight it off, and rarely it gets it wrong and not only does it clear that protein but then the white blood cells will attack native proteins and cause in very rare cases things like Guillain-Barr[é], and perhaps other immune problems.
>
> What the Selby and Pollard article shows is the specificity because you can say, how do you know if it was the vaccine versus maybe something coincident with it? And so the rechallenge was the key, and so the same symptoms recur with the same antigenic stimulus on three

[16] J.D. Pollard & G. Selby, Relapsing Neuropathy Due to Tetanus Toxoid, 37 J. Neurol. Sci. 113 (1978).

[17] As discussed below, Pollard and Selby, in their article discussed by Dr. Tornatore, apparently rejected molecular mimicry as a possible explanation for the case of challenge/rechallenge they reported. They stated, "Theories of cross-reactivity or shared antigens clearly provide unsatisfactory explanations for this event considering the relative rarity of such breakdowns in immunological tolerance and the great variety of antigens which can evoke this response." Pet'r's Ex. 20, Tab D at 11.

> different occasions. And so that specificity was seen as evidence that, yes it's a rare event, but rare if you can reproduce it is indeed true.

Tr. at 27-28.

Dr. Tornatore testified that Petitioner's GBS occurred within an appropriate time frame, based on reports of GBS induced by swine flu vaccination in 1976-77. Tr. at 28-29, 53. He described as significant records indicating that Petitioner felt pain and stiffness in her arm and back and weakness in her leg shortly after receiving the vaccination. Tr. at 30-31. This indicated to Dr. Tornatore that she suffered "an allergic type reaction to the vaccination." Tr. at 31, 69.[18] "In fact, subsequently as we'll see there's other fingerprints that point to her immune system being turned on by presumably the vaccine. So there's a little piece of historical information that says right away something happened, and she had a kind of something happen during the initial vaccination." Tr. at 31.

Dr. Tornatore noted that two weeks after the vaccination, on September 28, 2005, Petitioner presented to the ER at Mississippi Baptist Medical Center with nausea, vomiting, chest tenderness, and numbness of the fingertips and toes. Tr. at 31-32. Although he had difficulty making out all of the information in the medical records, Dr. Tornatore stated that "the important thing is the only thing that they treated her with was protonix . . . which is a proton pump inhibitor, to decrease the amount of acid in the stomach because they thought this could be reflux . . . ." Tr. at 33. He reviewed the results of Petitioner's blood tests and opined that they did not indicate the presence of infection. Tr. at 34-35. He noted the absence of symptoms that would indicate a gastrointestinal infection. Tr. at 36-37.

Dr. Tornatore noted that Petitioner's symptoms accelerated and that she had some lower back pain. "You can get low back pain with Guillain-Barr[é], also." Tr. at 38. He also "threw out" the thought that Petitioner's constipation was caused by damage to her autonomic nervous system, due to GBS. Tr. at 39. Dr. Tornatore emphasized that the absence of diarrhea indicated that Petitioner did not have a gastrointestinal infection. Tr. at 37-39.

Nerve conduction studies on October 3 confirmed that Petitioner had an acute inflammatory demyelinating polyneuropathy. Tr. at 39-41. This indicated a viral infection or vaccine reaction, not infection with *C. jejuni,* according to Dr. Tornatore. Tr. at 42-44.

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[18] On cross-examination, Dr. Tornatore also pointed to some evidence of "autonomic insufficiency," which he testified was inconsistent with infection, Tr. at 64-65, but he did not actually rely on this evidence in expressing his opinion. Tr. at 64 ("[A]nd again, I didn't want to go down this, but she has evidence in the record of some degree of autonomic instability . . . ."). He also elaborated on the pain and weakness Petitioner reported to a nurse "shortly after" her vaccination. Tr. at 67. He testified that "that is a hallmark" of GBS, but why "it kind of waxed and waned is a little unclear." Tr. at 68.

Because Petitioner's symptoms were not consistent with any of the known pathogens associated with GBS, Dr. Tornatore concluded that "based on that, you know, we can say, well that really leaves us with only one other possible culprit, and then that would be the tetanus vaccination." Tr. at 45-46. He stated that further testing showed IgA elevation, atypical antibodies, borderline elevation of the rheumatoid factor, and elevated ANA immunofluorescence. Tr. at 46-47. "[T]hese are not things you see following a routine infection," Dr. Tornatore stated. Tr. at 47. He repeated that Petitioner did not have any symptoms consistent with the most likely viral agents that could cause GBS. Tr. at 62-63. Dr. Tornatore stated that testing for other possible causative agents was not conducted in Petitioner's case because she did not have symptoms that usually accompany infection by those agents. Tr. at 47-48. "The only thing she did have was a vaccination two weeks prior, a tetanus vaccination which we know rarely can cause" GBS. Tr. at 63.

Dr. Tornatore testified that Petitioner's treating physician, Dr. Michael Winkelmann, felt that immunization was the culprit for her GBS, and ruled out West Nile virus. Tr. at 49-50. Other notations in her history similarly indicated vaccine causation, according to Dr. Tornatore. See Tr. at 50.

Asked whether he had an opinion as to the cause of Petitioner's GBS, Dr. Tornatore responded, "I think given the Pollard and Selby article, I think the tetanus toxoid is the most likely culprit, if I had to pick." Tr. at 51. He explained the rarity of such vaccine reactions by stating that:

> [I]t's kind of the perfect storm of somebody who perhaps had the genetic background such that their immune system is perhaps more prone to overactivity, that they, in this case she, I don't know for a fact but I think she probably had a tetanus, she probably had a DPT when she was a child, and so she had been previously exposed to that antigen so she was reexposed a second time.
>
> You know, could it be that the combination of the vaccinations in aggregate may have overstimulated her immune system?

Tr. at 52.

Dr. Tornatore then described procedures in which scientists try to induce allergic reactions in animals to develop experimental models of GBS, or experimental models of MS. Tr. at 52-53. "And so, you know, could multiple vaccinations at one time in the susceptible individual be the right thing? Hard to know. You know, it's probably again a confluence of different things that happened." Tr. at 53.

On cross-examination, Dr. Tornatore admitted that the medical record contains a notation by a treating physician, Dr. Moore, that Petitioner's "AIDP is likely related to gastrointestinal illness," but testified that he discounted that notation and disagreed with the conclusion. Tr. at 42, 55-56, 77. See Pet'r's Ex. 4 at 54. Because Petitioner improved with anti-reflux medication and did not have diarrhea or an elevated white blood cell count, Dr. Tornatore felt that gastrointestinal illness was not the cause of

Petitioner's GBS, an opinion with which one of her treating physicians, Dr. Winkelmann, agreed, according to Dr. Tornatore. Tr. at 57-58.

Dr. Tornatore recognized that the source of Dr. Winkelmann's statement indicating that the "immunization series had been the trigger for the development" of the GBS did not appear in the medical records. Tr. at 58-60. He speculated that Dr. Winkelmann came to this conclusion after he had reviewed Petitioner's records up to that point in time. Id. at 60.

Dr. Tornatore stated that molecular mimicry explained how the vaccine could have caused GBS. Tr. at 70. He said it was not necessary to know which component of the vaccine shared homology with the host antigens that theoretically caused GBS, "Because you have the Pollard and Selby article where you had the positive rechallenge." Id.

Dr. Tornatore's testimony in this respect is essential to my decision in this case. He stated:

> And so all you need to know is that it's the vaccine itself. The individual components it would be nice to know what those are specifically. However, that is for this case, that specificity is not required. The only specificity is that there was a challenge and positive rechallenge case, the Pollard and Selby case, that the Institute of Medicine said, you don't need to know the absolute molecular component but it's teaching us that if something happens one time and you challenge a person a second time with that same entity, that entity is the ca[u]se.

Tr. at 70-71.

Dr. Tornatore testified that the absence of epidemiological studies showing an association between tetanus vaccine and GBS was not significant, because "epidemiologic studies are not meant to identify rare events." Id. at 72-73. He indicated that there may have been no subsequent case reports in the more than three decades since the Pollard and Selby article was published only because "it's already been reported." Id. at 73.

3. Petitioner's Medical Literature

Petitioner's Exhibit 18 is an excerpt from the 1994 IOM report on adverse events associated with childhood vaccines. Pet'r's Ex. 18, Institute of Medicine, Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality, 47 (National Academy Press, 1994). The exhibit reproduces one page of the IOM report, which discusses GBS. Pet'r's Ex. 18 at 7. The excerpt reads, "ADEM and GBS can occur after the administration of either live attenuated or killed vaccines (in the case of vaccinia virus and the swine influenza vaccines, respectively)." Id.[19]

[19] ADEM, acute disseminated encephalomyelitis, is "an acute or subacute encephalomyelitis or myelitis characterized by perivascular lymphocyte and mononuclear cell infiltration and demyelination . . . . It is

Petitioner's Exhibit 20, Tab A is a 1991 article by Lahesmaa et al., reporting on molecular mimicry between microbial agents. Pet'r's Ex. 20, Tab A, R. Lahesmaa et al., Molecular mimickry between HLA B27 and *Yersinia, Salmonella, Shigella* and *Klebsiella* within the same region of HLA α_1-helix, 86 Clin. Exp. Immunol. 399 (1991). The article reports two examples of molecular mimicry at the amino acid level "between arthritis triggering microbes and HLA B27," a molecule found in "organisms known to trigger reactive arthritis." Pet'r's Ex. 20, Tab A at 5-6.

Petitioner's Exhibit 20, Tab B is a 2006 article by Komagamine and Yuki, reporting on "the first verification of the causative mechanism of molecular mimicry in an autoimmune disease." Pet'r's Ex. 20, Tab B at 2, Tomoko Komagamine & Nobuhiro Yuki, Ganglioside Mimicry as a Cause of Guillain-Barré Syndrome, 5 CNS & Neurological Disorders – Drug Targets 391 (2006). The article reports that "[t]wo-thirds of GBS patients experience some form of infection before GBS onset," including gastrointestinal infections. Pet'r's Ex. 20, Tab B at 3. The article focuses on *C. jejuni*, a bacterium, as an antecedent pathogen in GBS. Id. at 2-3. Twenty-six percent of GBS patients showed evidence of recent *C. jejuni* infections as compared with one to two percent of controls. Id. at 3. "The median interval from the onset of diarrhea to the onset of neuropathic symptoms was 9 days," which was evidence, the authors stated, "that GBS is a consequence of an immune response to *C. jejuni* rather than the direct effect" of the bacterium or its toxins. Id.

The authors discuss molecular mimicry between microbes and certain autoantigens, specifically GM1 ganglioside, which is located in the nerve cell membrane. Id. at 5-7. The relationship between gangliosides and structures on the outer cores of *C. jejuni* was confirmed experimentally in animals. Id. at 7-8. The ability of anti-ganglioside antibodies to cause nerve damage has been studied in animals and in vitro. Id. at 8-9.

Petitioner's Exhibit 20, Tab C is an article by Schonberger et al., reporting on the relationship between the 1976 influenza vaccination program and GBS. Pet'r's Ex. 20, Tab C, Lawrence B. Schonberger et al., Guillain-Barre Syndrome following Vaccination in the National Influenza Immunization Program United States, 1976-1977, 110 Am. J. Epidemiol. 105 (1979). The authors maintained, "Epidemiologic evidence indicated that many cases of GBS were related to vaccination." Pet'r's Ex. 20, Tab C at 2. The authors hypothesized a causal relationship based on higher rates of reported GBS in the weeks following vaccination. Id. at 17.

The article noted the suggestion that "many different antecedent events, including vaccination, might trigger GBS." Id. at 19. It also described a link between killed rabies vaccine produced in nervous tissue and allergic neuritis, "which has many features in common with GBS." Id.

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believed to be a manifestation of an autoimmune attack on the myelin of the central nervous system." Dorland's at 613.

Petitioner's Exhibit 20, Tab D is an article by Pollard and Selby discussing a case of relapsing neuropathy following tetanus vaccination. Pet'r's Ex. 20, Tab D, J.D. Pollard & G. Selby, Relapsing Neuropathy Due to Tetanus Toxoid, 37 J. Neurol. Sci. 113 (1978). This article reported on a "unique case history" involving a 42-year-old patient who suffered three episodes of a demyelinating neuropathy, "each of which followed an injection of tetanus toxoid." Pet'r's Ex. 20, Tab D at 2. The authors asserted, "There is little doubt that the three clinical episodes of demyelinating neuropathy resulted from the administration of tetanus toxoid." Id. at 6; see id. at 12. They stated that similar "pathogenetic mechanisms" are involved in the demyelination seen in the post-inoculation case "as in the more usual post-infective variety." Id. at 7. Pollard and Selby also stated, "Theories of cross-reactivity or shared antigens clearly provide unsatisfactory explanations for this event considering the relative rarity of such breakdowns in immunological tolerance and the great variety of antigens which can evoke this response." Pet'r's Ex. 20, Tab D at 11.

Petitioner's Exhibit 26 is an excerpt from the most recent report of the IOM concerning the adverse effects of vaccines. Pet'r's Ex. 26, Institute of Medicine, Adverse Events of Vaccines: Evidence and Causality, ix-x, 5 (The National Academies Press, Prepublication ed. 2011). The exhibit contains the preface to the report, which discusses the overall project of evaluating the safety of vaccines and the difficulty of assessing possible causation. Pet'r's Ex. 26 at 3. The report states, "The committee particularly counsels readers not to interpret a conclusion of inadequate data to accept or reject causation as evidence either that causation is either present or absent. Inadequate data to accept or reject causation means just that—inadequate." Id. at 4.

D. Respondent's Expert

1. Dr. Thomas Paul Leist's Report

Dr. Leist is an expert in adult neurology. Tr. at 88. He is an assistant professor of neurology at Thomas Jefferson University in Philadelphia and chief of the division of neuroimmunology. Resp't's Ex. B at 1. He sees patients with autoimmune diseases that affect the central nervous system and conducts research to develop medications for patients with diseases such as multiple sclerosis. Tr. at 85-87. Dr. Leist reviewed Petitioner's medical history, including her Hep A and tetanus vaccinations on September 15, 2005. Resp't's Ex. A at 1-6. He noted Petitioner's symptoms when she presented to Baptist Health System on September 28, 2005 – chest pressure, headache, nausea and emesis, as well as numbness and tingling in the fingers and toes. Id. at 2. On October 1, 2005, when Petitioner presented to the emergency room at St. Dominic-Jackson Memorial Hospital, she reported "a numb sensation in feet and that they won't hold her up.'" Id. (quoting Pet'r's Ex. 4 at 52). Dr. Leist noted that Petitioner reported "that she had experienced headache, stiff neck, nausea and vomiting 2 weeks before which resolved after one week." Resp't's Ex. A at 2. The return of these symptoms led to Petitioner's admission to St. Dominic Hospital. Id. The medical record noted Petitioner was in pain and was constipated. Id. at 2-3. The

differential diagnosis was of a myopathy, GBS, multiple sclerosis, or spine disease. Id. at 3. Dr. Leist reviewed the results of laboratory and other testing conducted at St. Dominic. Id.

Dr. Leist reviewed the notes of a consultation on October 3, 2005, recording that "the numbness had started in feet and fingers about 6 days and had later ascended." Id. Again, the presence of nausea and vomiting was noted without bladder or bowel problems. According to this consult, "the acute inflammatory demyelinating polyneuropathy was likely related to a gastrointestinal illness . . . ." Id. at 4 (citing Pet'r's Ex. 4 at 54). Dr. Leist described Petitioner's additional symptoms as her illness progressed, as well as her subsequent treatment. Resp't's Ex. A at 4-6. This included Petitioner's session with Dr. Winklemann on November 2, 2006. Id. at 6.

Dr. Leist opined that the presence of "nausea and vomiting is compatible with an acquired gastrointestinal illness including a gastrointestinal infection." Id. at 7. He noted that Petitioner had an elevated glucose level, which "can be observed during infections particularly in individuals with impaired glucose control." Id. Dr. Leist noted that Petitioner had impaired glucose control and had been diagnosed with pre-diabetes in 2007. Id. "Sensory complaints can occur as an early manifestation of evolving glucose homeostasis," he stated. Id.[20]

Based on the medical records, Dr. Leist stated that Petitioner developed "subacute onset of weakness that progressed over the next few days" following "a gastrointestinal illness on or about on September 27, 2005." Id. at 9. He noted that Dr. Moore, the neurology consultant who evaluated Petitioner at St. Dominic-Jackson, commented that she had a form of GBS likely caused by a gastrointestinal illness. Id.

Dr. Leist referred to studies that "have shown a high incidence of respiratory infection, gastroenteritis, and febrile illness in GBS patients as compared with the controls." Id. He opined that *C. jejuni* "stands out as it is the pathogen most frequently identified in association with Guillain Barre Syndrome." Id. Dr. Leist noted that Petitioner had some but not all of the symptoms of *C. jejuni* infection, and that laboratory testing for that agent was not performed. Id.

Dr. Leist discussed the literature regarding a possible association between tetanus toxoid and vaccine injury. Id. at 9-10. He noted in particular that the Pollard and Selby report on the case of one individual who developed GBS on three separate occasions over a 13-year period following tetanus toxoid "is viewed as convincing at least for this one individual." Id. at 10. He explained that the same individual "subsequently experienced multiple recurrences of demyelinating polyneuropathy, most occurring following acute viral infections." Id. (citing Ct. Ex. 3 at 2-3). Dr. Leist noted

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[20] Glucose is "an aldohexose . . . found as a free monosaccharide in fruits and other plants and in the normal blood of all animals." Dorland's at 789. Homeostasis is "a tendency to stability in the normal body states (internal environment) of the organism. It is achieved by a system of control mechanisms activated by negative feedback." Id. at 867.

two other cases evaluated in the 1994 IOM report. Resp't's Ex. A at 10. In addition, he wrote that larger epidemiologic studies carried out after the 1994 IOM report have not demonstrated an association between tetanus vaccine and GBS. Id.

Dr. Leist opined that Petitioner suffered a gastrointestinal illness caused by a pathogen "such as C. jejuni." Id. He stated that the "immune activation" noted by Dr. Tornatore in his report, as well as Petitioner's gastrointestinal symptoms, more likely than not were caused by infection. Id. at 10-11.

2. Dr. Leist's Testimony

Dr. Leist agreed with the diagnosis of GBS. Tr. at 89. He opined that, given Petitioner's symptoms of stiffness and aching followed by a gastrointestinal disturbance around September 27, 2005, an intervening infection was the more likely cause of her GBS, diagnosed on October 3, 2005. Id. at 89-93. He testified that the laboratory findings were consistent with infection. Id. at 93-96.

Dr. Leist questioned the significance of the Pollard and Selby case report on the possible association between Td vaccines and GBS. Tr. at 98.[21] He stated that follow-up publications concerning the same individual described by Pollard and Selby revealed that the subject "went on to have additional episodes of worsening of his by then chronic peripheral nerve disease . . . during episodes when he had diagnosed viral infections." Id. According to Dr. Leist, the subsequent literature concerning this same individual indicated worsening of the underlying peripheral nerve disease "independent of tetanus." Tr. at 99. Pairing that information with the epidemiological data, which do not reveal significant rates of injury despite "a relatively high frequency" of tetanus inoculation in the general population, Tr. at 99; see Resp't's Ex. G; Resp't's Ex. H; Tr. 109, Dr. Leist concluded that "there has not been a clear association between tetanus vaccine, tetanus containing vaccines, and GBS," Tr. at 99. He expressed the view that the IOM would reevaluate the evidence presented in the Pollard and Selby article in future publications. Id. (Dr. Leist's prediction in this respect proved correct).

Dr. Leist recognized molecular mimicry as a mechanism that can cause GBS in the presence of certain pathogens, in particular, the bacterium *C. jejuni*. Tr. at 107-08. He stated that there are no scientific studies linking tetanus vaccine with the theory of molecular mimicry, and noted that the association between tetanus vaccination and GBS was based on the Pollard and Selby article. Tr. at 109.

On cross-examination, Dr. Leist conceded that molecular mimicry is a method by which autoimmunity can occur. Tr. at 124. He testified that "how the individual pathogen will do it, that's something that is too general a question." Tr. at 124-25.

On re-direct, Dr. Leist was asked to elaborate on the significance of the gastrointestinal symptoms that Petitioner experienced around the time she became ill

[21] At the time of hearing, the latest IOM report on vaccine injuries had not yet been released.

with GBS. Tr. at 130-31; see Tr. 127. In particular, Dr. Leist was asked about Petitioner's symptoms as compared to the symptoms of persons infected with *C. jejuni*. Tr. at 130-31. He offered *C. jejuni* infection as an example of an agent that can produce illness by a process of molecular mimicry. Tr. at 132-33.

On re-cross examination Dr. Leist agreed that epidemiological studies "cannot look at unique events," but do show "the general potentiality of an agent to cause something." Tr. at 134. In his view, the "low residual risk" of injury from tetanus vaccination makes it "even more important" in the individual case to eliminate other possible causative agents. Tr. at 135.

3. Respondent's Medical Literature

Respondent's Exhibit C is an article discussing whether *C. jejuni* infection causes AIDP. Resp't's Ex. C, S. Kuwabara et al., Does Campylobacter jejuni infection elicit "demyelinating" Guillain-Barré syndrome?, 63 Neurology 529 (2004). The authors concluded that "slowing of motor nerve conduction in *C. jejuni*-positive patients is not caused by segmental demyelination, and therefore, *C. jejuni* infection does not cause typical AIDP." Resp't's Ex. C at 6.

Respondent's Exhibit F explored the possibility of classifying subgroups of GBS based on the type of illness that preceded the GBS. Resp't's Ex. F, M. Koga et al., Antecedent symptoms in Guillain-Barré syndrome: an important indicator for clinical and serological subgroups, 103 Acta Neurol. Scand. 278 (2001). The authors noted that two-thirds of GBS patients have histories of antecedent infectious illness. Resp't's Ex. F at 3. The study compared the symptoms of GBS sufferers based on infectious serology. Id. at 6. The study "confirmed there is a significant association between serological evidence of recent *C. jejuni* infection and antecedent diarrhea and abdominal pain." Id. at 7-8.

Respondent's Exhibit G reviewed adverse events following administration of three vaccines (not Hep A or tetanus). Resp't's Ex. G, Tetsuo Nakayama & Kazumasa Onoda, Vaccine adverse events reported in post-marketing study of the Kitasato Institute from 1994 to 2004, 25 Vaccine 570 (2005). The authors commented, "A long-term discussion has been carried out about the causal relationship between vaccines and miscellaneous demyelinating neurologic diseases, such as ADEM, Guillain-Barré syndrome, and multiple sclerosis. " Resp't's Ex. G at 6. They noted that "possible mechanisms have been proposed," including "molecular mimicry between vaccine antigen and myelin protein." Id. at 7.

Respondent's Exhibit H is a study of the risk of GBS from vaccines containing tetanus toxoid. Resp't's Ex. H, Jessica Tuttle et al., The Risk of Guillain-Barré Syndrome after Tetanus-Toxoid–Containing Vaccines in Adults and Children in the United States, 87 Am. J. Public Health 2045 (1997). This article reports on data from a 1991 study conducted by the Centers for Disease Control. Resp't's Ex. H at 2. The study found no enhanced risk of GBS within six weeks following administration of

tetanus-toxoid–containing vaccine. Id. at 4. The authors noted the conclusion of the IOM in its 1991 report and stated that it "was based on limited data, primarily a single case report of an individual with apparently unusual susceptibility to Guillain-Barré syndrome." Id. at 5. The authors maintained that within their data set using "two large, active surveillance studies . . . the number of cases of Guillain-Barré syndrome . . . is less than the number expected by chance alone." Id.[22]

E. The Institute of Medicine

The legislation establishing the Vaccine Program, P.L. 99-660, charged the IOM with the task of reviewing the medical and scientific literature regarding risks associated with vaccines. National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 101 Stat. 1330-221, as amended by Pub. L. 100-203 codified as amended, 42 U.S.C. §§ 300aa-10 et seq. (2006); see Terran ex rel. Terran v. Sec'y of Dep't of Health & Human Servs., 195 F.3d 1302, 1313, 1315 (Fed. Cir. 1999) (noting that Congress "directed the Secretary to request that the Institute of Medicine of the National Academy of Sciences conduct studies exploring the link between childhood vaccines with certain illnesses"). In accordance with its statutory mandate, the IOM periodically issues reports on adverse events following vaccination. The views of the IOM on the issue of whether there may be a causal relationship between tetanus vaccine and GBS have evolved over time. That is particularly significant for this case because of Dr. Tornatore's emphatic reliance on the IOM's 1994 Report to support his theory of tetanus vaccine causation.

1. 1994 IOM Report[23]

In its 1994 Report, Ct. Ex. 3, Institute of Medicine, Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality, 88-89 (National Academy Press, 1994), the IOM stated:

> There is a biologic plausibility for a causal relation between vaccines and demyelinating disorders. The literature describing a possible association between GBS and tetanus toxoid, DT, or Td consists of case reports. The most convincing case in the literature is that reported by Pollard and Selby (1978), who described a 42-year-old man who

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[22] The 2011 IOM report stated that the Tuttle study was not considered in the IOM's weight of epidemiological evidence because it "provided data from passive surveillance systems and lacked unvaccinated comparison populations." Ct. Ex. 1 at 1.

[23] Both experts discussed the portion of the 1994 IOM report referring to the IOM's findings on a causal relationship between vaccinations containing tetanus toxoid and GBS. See Resp't's Ex. A at 10 (Dr. Leist's reference to this portion of the 1994 IOM report in his expert report); Tr. at 71, 73 (Dr. Tornatore's testimony regarding the 1994 IOM report's causal connection of tetanus toxoid and GBS); Tr. at 98-100, 125-126 (Dr. Leist's testimony regarding the 1994 IOM report's causal connection of tetanus toxoid and GBS). This portion of the 1994 IOM report was not admitted into the record by either party. On July 27, 2012, the Court entered the excerpt, pages 88-89 of the 1994 IOM report, to complete the record. See Order, July 27, 2012, ECF No. 57.

> developed GBS on three separate occasions (over a 13-year period) following receipt of tetanus toxoid. The relation between tetanus toxoid and GBS is convincing at least for that one individual, even though this man has subsequently experienced multiple recurrences of demyelinating polyneuropathy, most following acute viral illnesses. . . .

Ct. Ex. 3 at 2-3.

The 1994 IOM report concluded, "The evidence favors a causal relation between tetanus toxoid and GBS. If the evidence favors a causal relation between tetanus toxoid and GBS, then in the committee's judgment the evidence favors a causal relation between vaccines containing tetanus toxoid (DT and Td) and GBS." Id. at 3.

2. 2011 IOM Report

a. Molecular Mimicry

The 2011 IOM report included a discussion of molecular mimicry as a possible mechanism of vaccine injury causation. Ct. Ex. 2. The IOM found little clinical, diagnostic, or experimental "evidence . . . that could be consistent with the hypothesis of molecular mimicry in rare and selected case reports." Id. at 3. The IOM concluded, "Based on the literature reviewed, molecular mimicry was not confirmed to be a mechanism leading to the development of the adverse events post-vaccination." Id.

b. The Pollard and Selby Case Report

In the 2011 IOM report, the Pollard and Selby case report was not discussed at all in the section concerning vaccines containing tetanus toxoid and GBS. Ct. Ex. 1 at 1-2. Gone as well was any statement indicating a causal association between tetanus toxoid and GBS. The IOM's 2011 "Causality Conclusion" concerning tetanus vaccines and GBS was, "The evidence is inadequate to accept or reject a causal relationship between diphtheria toxoid-, tetanus toxoid-, or acellular pertussis-containing vaccines and GBS." Id. at 2.

While no longer in the section on GBS, the Pollard and Selby case report was discussed in the section on tetanus-containing vaccines and Chronic Inflammatory Disseminated Polyneuropathy ("CIDP"). Id. [24] The IOM stated:

> Pollard and Selby (1978) appear to present evidence of vaccine rechallenge leading to symptoms of peripheral neuropathy in a patient, subsequently diagnosed with a spontaneously relapsing remitting

[24] CIDP is "an acquired peripheral neuropathy of presumed autoimmune etiology, which presents either as a chronically progressive or relapsing-remitting disorder." Angelika F. Hahn et al., Chronic Inflammatory Demyelinating Polyradiculoneuropathy, in Peripheral Neuropathy, 2221, 2221 (Peter J. Dyck & P.K. Thomas eds., 4th ed. 2005). "Because of its progressive course, CIDP has been set apart from the self-limited acute inflammatory demyelinating polyneuropathy (AIDP) or 'Guillain-Barré syndrome' (GBS)." Id.

> neuropathy, who developed symptoms in association with acute viral infections; however, the authors did not rule out other possible causes and did not provide evidence beyond a temporal relationship with vaccine administration. The spontaneous development of peripheral neuropathy makes it difficult to conclude that the tetanus toxoid vaccines were the causative agent.

Id. at 3.

The IOM, in its "Causality Conclusion" regarding tetanus toxoid and CIDP, concluded, "The evidence is inadequate to accept or reject a causal relationship between diphtheria toxoid-, tetanus toxoid-, or acellular pertussis-containing vaccine and CIDP." Id. at 4.

c. Parties' Responses to the 2011 Report

On May 24, 2012, the parties were invited to comment on and file responses to Court Exhibit 2, the excerpt from the 2011 IOM report on the theory of molecular mimicry. See Order, ECF No. 53.

In response, Petitioner submitted a brief arguing that reliance on the IOM was inappropriate because the standards for scientific proof are higher than the standards for evidence needed to prevail in the Vaccine Program. Petitioner claimed that the IOM requires "direct evidence to prove the existence of molecular mimicry as a mechanism of vaccine-induced injury." Petitioner's Response to the Court's May 24, 2012 Order at 2, ECF No. 55. Petitioner stressed that molecular mimicry is a "well-established mechanism in selected animal models" that has yet to be "convincingly proven" as relevant to human autoimmune disease. Id. (quoting Ct. Ex. 2 at 2). Petitioner claimed that evidence sufficient for the IOM could only be found through testing "at the very time mimicry occurred," which does not happen in clinical practice. Petitioner's Response to the Court's May 24, 2012 Order at 4-5. Petitioner differentiated between the "direct evidence" she claims the IOM requires and circumstantial evidence, which can be deemed reliable within the Vaccine Program. Id. at 5. Further, Petitioner contrasted the preponderant evidence standard of vaccine causation with what she characterized as the "medical certainty" standard required by the IOM to prove causation. Id. at 7. [25]

[25] This may be an appropriate point to note that Petitioner and her expert laid great emphasis on the conclusion from the 1994 IOM report of a causal link between Td and GBS. See Pet'r's Ex. 18; Tr. at 70-73. Now that the IOM has withdrawn its previous endorsement of a causal link, Petitioner urges that the IOM's information should not be considered significant. See Pet'r's Response to the Court's September 23, 2011 Order, ECF No. 45. I understand and appreciate the distinction between the proof that is required to establish entitlement to compensation in the Vaccine Program and the greater proof that may be required to satisfy the scientific community of vaccine injury causation, and I do not place undue reliance on the IOM's causality conclusion (which is a non-conclusion, in any event). But I do rely on the IOM's evaluation of the scientific significance of the Pollard and Selby report. That evaluation was undertaken and communicated to the public by a committee of scientific experts assigned by law to evaluate scientific evidence. See Terran, 195 F.3d at 1313, 1315 (noting that Congress "directed the Secretary to request that the Institute of Medicine of the National Academy of Sciences conduct studies exploring the link between childhood vaccines with certain illnesses"). The IOM's latest evaluation of the

Petitioner also filed Exhibit 27, which consists of two additional excerpts from the 2011 IOM report. See Pet'r's Ex. 27. The first excerpt discussed increased susceptibility in individuals who experience adverse reactions to vaccines. Id. at 3-4. Petitioner in this case did not present any evidence of a predisposition that made her particularly vulnerable to adverse reaction. Petitioner's second excerpt reiterated that individuals with immune function abnormalities respond differently to infection and vaccination compared with healthy individuals. Id. at 5. Petitioner's second excerpt quotes the IOM's view that "[i]t is possible to look for molecular mimicry as a possible cause of vaccine antigen and self-antigen . . ." injury, though it still would need confirmation in humans even if it became established in an animal model. Id. at 6.[26]

In the Secretary's response, Respondent maintained that Court Exhibit 2 strengthened her position that Petitioner has not submitted preponderant evidence in support of vaccine causation. See Respondent's Response to the Court's May 24, 2012 Order at 3, ECF No. 54. Respondent argued that Petitioner's molecular mimicry argument is particularly weak in this case because Dr. Tornatore "relied wholly upon the Pollard & Selby Report," id. at 2, and "conceded that he could not establish the essential elements necessary to implicate molecular mimicry as a contributing factor," id. at 3. Respondent concluded that the IOM excerpt "fully support[s] respondent's expert's testimony in this case, undermine[s] petitioner's reliance on the Pollard & Selby case report from 30+ years ago, and show[s] that molecular mimicry has not been established (by clinical, diagnostic, or experimental evidence) as a mechanism by which Td vaccine *specifically* causes GBS." Id.

III. DISCUSSION

A. Petitioner's Burden of Proof

Petitioners seeking to establish causation-in-fact must show by a preponderance of the evidence that but for vaccination they would not have been injured, and that vaccination was a substantial factor in bringing about the injury. Cedillo, 617 F.3d at 1338; Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352 (Fed. Cir. 1999).[27] Proof of actual causation must be supported by a sound and reliable "medical or scientific explanation that pertains specifically to the petitioner's case, although the

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significance of the Pollard and Selby report contradicts Dr. Tornatore's testimony at hearing. The argument that special masters should not rely on the opinion of the IOM concerning the significance of particular scientific evidence is unpersuasive.

[26] I agree that the standard of proof to demonstrate causation in the scientific community requires more and different evidence than is required in the Vaccine Program. That is why I do not simply adopt the findings of the IOM but give due consideration to the IOM in reaching my own conclusion, based on the evidence in the record as a whole.

[27] Petitioner has not alleged a "Table" injury. See Pet'r's Post-Hr'g Br. at 11, ECF No. 39; 42 C.F.R. § 100.3.

explanation need only be 'legally probable, not medically or scientifically certain.'" Moberly, 592 F.3d at 1322 (quoting Knudsen by Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994)); see also Grant v. Sec'y of Health & Human Servs. 956 F.2d 1144, 1148 (Fed. Cir.1992) (finding a medical theory must support actual causal connection).

Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." Knudsen, 35 F.3d at 548. A petitioner may use circumstantial evidence to prove the case, and "close calls" regarding causation must be resolved in favor of the petitioner. Althen, 418 F.3d at 1280. Petitioner's burden is to show 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." Id. at 1278. If Petitioner succeeds in establishing the case-in-chief, the burden then shifts to Respondent to prove alternative causation by a preponderance of the evidence. Id. If Petitioner fails to establish the case-in-chief, the burden does not shift. Doe 11 v. Sec'y of Health & Human Servs., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); see Cedillo, 617 F.3d at 1335 (citing Walther v. Sec'y of Dep't of Health & Human Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007)).[28]

B. Althen Prong 1[29]

In evaluating whether a petitioner has presented sufficient evidence of a medical theory, "the special master is entitled to require some indicia of reliability to support the assertion of the expert witness." Cedillo, 617 F.3d at 1339 n.3 (quoting Moberly, 592 F.3d at 1324). A special master is not required to rely on a speculative opinion that "is connected to existing data only by the ipse dixit of the expert." Snyder ex rel. Snyder v. Sec'y of Dep't of Health & Human Servs., 88 Fed. Cl. 706, 743 (2009) (quoting Gen. Elec. Co. v. Joiner, 522 U.S. 136, 146 (1997)).

Assessing the reliability of an expert opinion in Vaccine Act cases can be challenging because often there is little supporting evidence. See Althen, 418 F.3d at 1280 (noting that the "field [is] bereft of complete and direct proof of how vaccines affect the human body"). Most expert opinions extrapolate from existing data and knowledge. The weight to be given to an expert's opinion is based in part on the size of the gap between the science and the opinion proffered. Cedillo, 617 F.3d at 1339 (citing Joiner,

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[28] The Federal Circuit discussed the shifting burdens of proof in some detail in de Bazan. see de Bazan v. Sec'y of Dep't of Health & Human Servs., 539 F.3d 1347, 1353-54 (Fed. Cir. 2009). In this case, Petitioner has not presented sufficient evidence to shift the burden of proof to the Secretary to establish causation by an alternative factor. See Doe 11, 601 F.3d at 1358.

[29] In Respondent's Post Hearing Brief, the Secretary indicated that "Respondent concedes that Prong 1 of Althen is not at issue in this case. Resp't's Post-Hr'g Br. at 6, ECF No. 41. Based on the Secretary's conduct of the case, this appears to be a typographical error. Respondent does concede an appropriate time frame for vaccination injury, under Prong 3. See Tr. at 6-7, 91.

522 U.S. at 146). Evidence should be viewed under the preponderance standard as it is understood in civil courts, "not through the lens of the laboratorian." Andreu ex rel. Andreu v. Sec'y of Dep't of Health & Human Servs., 569 F.3d 1367, 1380 (Fed. Cir. 2009).

Under Althen Prong 1, a petitioner must present a medical theory that the vaccine could cause the injury complained of. Although the theory of causation need not be corroborated by medical literature or epidemiological evidence, the theory must be sound, reliable, and reputable – in other words, the theory need not be scientifically certain, but it must have a scientific basis. See Knudsen, 35 F.3d at 548 (finding actual causation "must be supported by a sound and reliable medical or scientific explanation").

1. The Special Master's Role

The issue under Prong 1 is whether there is preponderant evidence of a theory that vaccination could have caused the injury. In Moberly, the Federal Circuit reiterated that the standard of proof in a vaccine case is the traditional tort standard of preponderance of the evidence; otherwise stated, the matter to be proved must be shown to be more likely than not. Moberly, 592 F.3d at 1322 n.2. In the past, confusion has arisen as to the correct application of the standard of proof under Prong 1 of the Althen test, which asks whether it has been shown that there is a theory of possible vaccine causation. See Pafford v. Sec'y of Dep't of Health & Human Servs., 451 F.3d 1352, 1355-56 (Fed. Cir. 2006). The confusion arises when the matter to be established is conflated with the amount of evidence that needs to be shown to establish it. Properly understood, what is required under Prong 1 is preponderant evidence of possible vaccine causation. As discussed above, this requires a showing of a reliable theory of causation.

The standard is easier to apply than to articulate. On this record, it has been shown to be not very likely that molecular mimicry could cause GBS from a tetanus vaccine. Boiled down to the essentials, the only evidence favoring the theory is the ipse dixit of Petitioner's expert. Against the theory, among other factors, are the IOM's conclusion, Pollard and Selby's statement, and Dr. Leist's opinion. It is clear that the evidence preponderates against Petitioner, even though under Prong 1 she need only show preponderant evidence of a theory of possible causation. Could the tetanus vaccine have caused Petitioner's GBS by the process of molecular mimicry? On this record, there is not preponderant evidence showing that it could.

As noted elsewhere herein, in deciding these issues, a special master does not resolve vigorous medical controversies, but merely decides which side has presented more and better evidence in the case at bar, considering all the facts in the particular record. Whether the special master actually is persuaded that the vaccine could cause the injury is not the issue. Doe 93 v. Sec'y of Dep't of Health & Human Servs., 98 Fed. Cl. 553, 566-67 (2011). The special master is called upon to weigh the evidence, not to weigh in on the science. Thus, "[t]he *sole* issues for the special master are . . . whether

it has been shown by a preponderance of the evidence that a vaccine caused . . . injury." Knudsen, 35 F.3d at 549; see Hodges v. Sec'y of Dep't of Health & Human Servs., 9 F.3d 958, 961 (Fed. Cir. 1993) ("Congress assigned to a group of specialists, the Special Masters . . . , the unenviable job of sorting through these painful cases and, based upon their accumulated expertise in the field, judging the merits of the individual claims."). The appropriate inquiry is whether, as a legal not a medical matter, a possible theory of vaccine injury has been demonstrated by preponderant evidence.

Significant consequences flow from the fine but real distinction between deciding whether a theory is persuasive versus deciding whether the evidence supporting it is preponderant, relative to all the other evidence of record in the particular case at bar. The distinction may seem evanescent, but it arises from the unique setting of the Vaccine Program, in which we start and end at the same point: this is a field bereft of knowledge. See Althen, 418 F.3d at 1280. In most cases, there are no definitive answers to the question of vaccine causation. The answers will come in the future, as medical science progresses. In the meantime, in the Vaccine Program, special masters cannot decide whether a theory of vaccine causation actually is likely to be correct; they can decide only whether preponderant, reliable evidence favors a finding that there is a theory under which vaccine causation was possible in the particular case.

It follows that each case must be considered on the record in that case. See Knudsen, 35 F.3d at 548 (finding that "[c]ausation in fact under the Vaccine Act is based on the circumstances of the particular case"); Campbell ex rel. Campbell v. Sec'y of Dep't of Health & Human Servs., 69 Fed. Cl. 775, 784 (Fed. Cl. 2006) (indicating that a "'cookie cutter'" approach to resolving causation issues "remains the antithesis of the individualized determinations required by the Vaccine Program"). This is reflected in the well-established doctrine that special masters' decisions – even their own past decisions – are not precedential. See Hanlon v. Sec'y of Dep't of Health & Human Servs., 40 Fed. Cl. 625, 630 (1998) (finding decisions are not binding on special masters except in the same case), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); see also Stone v. Sec'y of Dep't of Health & Human Servs., 676 F.3d 1373, (Fed. Cir. 2012) ("[T]he special master is entitled to consider the record as a whole in determining causation."). A special master also is not compelled to accept the same theory of causation in different cases. Rickett v. Sec'y of Dep't of Health & Human Servs., No. 2011-5038, 468 Fed.Appx. 952, at *959 (Fed. Cir. 2011) ("A special master's acceptance of a theory in one case does not require him or her to accept the theory in subsequent cases involving similar facts or the same vaccine. Rather, a different evidentiary record can lead to different outcomes." (citations omitted).[30]

[30] I am aware that in some earlier cases, GBS has been attributed to tetanus vaccination. See, e.g., Althen. For all the reasons stated above, the decisions in other cases are not binding in this one. In addition, as science advances, decisions in the Program must respond accordingly.

2. The Expert's Role

Because the special master's role is to make a legal determination as to the preponderance of the evidence, not a medical determination as to the validity of the proposed causation theory, the role of the medical expert in the Vaccine Program is critical. In the vast majority of cases, it is the medical expert who must present preponderant evidence of a theory of possible vaccine causation. But the special master may require more than just the ipse dixit of the expert. "[T]he special master is entitled to require some indicia of reliability to support the assertion of the expert witness." Cedillo, 617 F.3d at 1339 n.3 (quoting Moberly, 592 F.3d at 1324). See Porter v. Sec'y of Dep't of Health & Human Servs., 663 F.3d 1242, 1250-51 (Fed. Cir. 2011); Snyder, 88 Fed. Cl. at 743 (quoting Joiner, 522 U.S. at 146).

"[S]pecial masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act." Porter, 663 F.3d at 1250. What is meant by credibility in the Vaccine Program is not coextensive with the concept of credibility as applied to fact witnesses in a conventional trial setting.[31] In the Vaccine Program, the concept is closer to reliability than believability. See Moberly, 592 F.3d at 1325-26.

Determining the credibility of expert witnesses involves evaluating the expert's testimony for consistency, clarity, and logical reasoning. See Moberly, F.3d at 1325 ("[T]he factfinder must decide the reliability, consistency, and probative value of the scientific evidence, with the guidance of scientific opinion.") (quoting Hodges, 9 F.3d at 967). If medical literature has been submitted by the petitioner or the respondent, the expert's testimony is examined in light of the available peer-reviewed literature. Also pertinent is whether the expert's testimony is informed by the medical record in the particular case at bar, and responds appropriately to the expert testimony presented by the opposing party. These are examples of the kinds of credibility factors that indicate whether an expert has presented reliable evidence of possible vaccine causation.

3. The Evidence in This Case

To sum up the evidence adduced (and not adduced) on Prong 1: Petitioner alleges that a Td vaccination caused her to contract GBS. None of the medical literature submitted by Petitioner and her expert discusses molecular mimicry as a possible theory of causation linking Td vaccination and GBS.[32] Upon careful review,

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[31] "In general, when two expert witnesses, both highly qualified, dispute an issue of medical fact with supporting and contradictory evidence, it is immaterial whether one witness makes a better appearance on the stand." Broekelschen, 618 F.3d at 1349.

[32] Petitioner's expert's theory seemed to be that any vaccine can cause any demyelinating disorder. Accordingly, Petitioner submitted evidence of data derived from the 1970s swine flu vaccination campaign to show that that flu vaccination can cause GBS. See Pet'r's Ex. 20, Tab C. I find no reliable evidence in this material supporting a broad hypothesis causally linking any and all vaccinations with any and every demyelinating disorder.

none of the medical records support molecular mimicry or any other theory of possible vaccine causation. Petitioner's case therefore rests on the reliability of her expert's conclusion that molecular mimicry explains how a Td vaccination could cause GBS.

Respondent disputed the existence of a reliable theory of possible vaccine causation. The Secretary's expert relied on epidemiological evidence to cast doubt on the existence of a cause and effect relationship between tetanus vaccination and GBS. See Resp't's Ex. H. The Secretary's expert also questioned the report of challenge/rechallenge in the Pollard and Selby study. The Secretary's expert correctly predicted at hearing that the IOM would reverse its previous endorsement of a relationship between tetanus vaccination and GBS. Tr. at 99. The Secretary's expert disputed that any of the information in this medical record pointed to vaccine injury as opposed to an infectious process, which is known to trigger many cases of GBS.

Petitioner relied on literature showing that one form of GBS, the axonal form, may be caused by a bacterial infection, through the process of molecular mimicry. The Secretary submitted literature indicating that the demyelination characteristic of the particular form of GBS suffered by Petitioner, however, was not caused by that bacterial infection. See Resp't's Ex. C; Tr. at 43. Respondent's Exhibit C supported the Secretary's expert's testimony that the existence of a bacterium that causes one form of GBS by the process of molecular mimicry does not mean that a tetanus vaccination could have caused Petitioner's GBS by the same process. See Tr. at 107-08. As Pollard and Selby stated, "Theories of cross-reactivity or shared antigens clearly provide unsatisfactory explanations . . . considering the relative rarity of such breakdowns in immunological tolerance and the great variety of antigens which can evoke this response." Pet'r's Ex. 20, Tab D at 11; see also Ct. Ex. 2 at 1.

If reliable, Dr. Tornatore's testimony alone theoretically would be sufficient to satisfy Prong 1 – notwithstanding the absence of supporting medical literature, or evidence in the medical record, or a specific biological mechanism explaining how molecular mimicry could result in AIDP because of a tetanus vaccination.[33] Dr. Tornatore's testimony in this case was not reliable. The reasons for reaching this conclusion are discussed below, with no intent to disparage the expert, but rather to elucidate the basis for my decision, so that it can be understood in the first instance and effectively reviewed if appealed.

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[33] I have in the past relied on little more than the testimony of an expert to award compensation to a petitioner based on the theory of molecular mimicry. See, e.g., Kennedy v. Sec'y of Dep't of Health & Human Servs., No. 09-474, 2012 WL 1929801 (Ct. Cl. Spec. Mstr. May 8, 2012); Myer v. Sec'y of Dep't of Health & Human Servs., No. 06-148, 2011 WL 3664358 (Ct. Cl. Spec. Mstr. July 28, 2011); Daily v. Sec'y of Dep't of Health & Human Servs., No. 07-173, 2011 WL 2174535 (Ct. Cl. Spec. Mstr. May 11, 2011). In each instance, the expert testimony provided was reliable in itself and preponderant in the context of the evidence in the record as a whole.

a. Misplaced Reliance on Case Report – Pollard and Selby

In support of his theory, Dr. Tornatore's report cited the article published in 1978 by Pollard and Selby. See Pet'r's Ex. 20, Tab D. At hearing, Dr. Tornatore described the Pollard and Selby report as a case of GBS caused by tetanus vaccination and indicated that the IOM recognized it as such. See Tr. at 70-71.

The 1994 IOM report did not in any way identify molecular mimicry as the theoretical basis to explain the Pollard and Selby report. Further, the Pollard and Selby article cited by Dr. Tornatore, discussing exactly the phenomenon he posits here – "why tetanus toxoid antigen should result in cellular hypersensitivity to myelin," Pet'r's Ex. 20, Tab D at 11, expressly stated, "Theories of cross-reactivity or shared antigens clearly provide unsatisfactory explanations for this event considering the relative rarity of such breakdowns in immunological tolerance and the great variety of antigens which can evoke this response." Id. Thus, Pollard and Selby apparently rejected molecular mimicry as the cause of the vaccine reaction reported in their article.[34]

Moreover, the Pollard and Selby report no longer is regarded by the IOM as evidence of vaccine causation. As Dr. Leist noted, the individual on whom Pollard and Selby reported was viewed at the time as exhibiting a reaction to tetanus vaccination on three separate occasions over a 13-year period. Resp't's Ex. A at 9-10. The IOM recognized, even in 1994, that this same individual "subsequently experienced multiple recurrences of demyelinating polyneuropathy, most occurring following acute viral infections." Id. at 10 (citing Ct. Ex. 3 at 2-3). The 1994 IOM report nevertheless concluded, "The evidence favors a causal relation between tetanus toxoid and GBS." Ct. Ex. 3 at 3.

As described above, after the hearing in this case, the IOM published a new report changing its views on the Pollard and Selby article in several significant respects. (1) The IOM no longer characterized the individual reported on as suffering from GBS, but from a recurring/relapsing disorder called CIDP. Ct. Ex. 1 at 1-2. (2) The IOM characterized the evidence concerning a causal relationship between tetanus vaccine and CIDP or GBS as "inadequate to accept or reject." Ct. Ex. 1 at 2, 4. (3) The IOM pointed out that the individual reported on by Pollard and Selby was "subsequently diagnosed with a spontaneously relapsing remitting neuropathy and experienced episodes in association with acute viral infections." Id. at 2-3. In other words, this individual's disorder was not linked with "specificity" to vaccination; his relapsing/remitting neuropathies had only "a temporal relationship with vaccine administration." Id. at 3. The 2011 IOM report thus negated the earlier findings that were based on the Pollard and Selby report, such that Dr. Tornatore's reliance on the IOM to buttress the theory of vaccine injury due to tetanus vaccination must, based on the 2011 IOM Report, be viewed as inappropriate.

[34] As noted above, Dr. Tornatore's assertion that the Pollard and Selby article did not speak to molecular mimicry is in error. See Tr. at 26. It addressed the theory in order to reject it.

Dr. Tornatore did not update his testimony when offered the opportunity to do so. By order dated September 23, 2011, the pertinent portion of the 2011 IOM report was entered into the record, without objection, and the parties were invited to comment on the new information. Order, ECF No. 43. Dr. Tornatore offered no supplementation of his report or his testimony, notwithstanding that the new IOM report indicated that his reliance on Pollard and Selby to bolster his theory of causation in this case was misplaced. See Porter, 663 F.3d at 1252 (involving an expert who failed to articulate any reason for disagreeing with the IOM report). [35] Respondent's expert's report and testimony, on the other hand, were consistent with the 2011 IOM report. In fact, Dr. Leist correctly predicted that the IOM would retract its previous endorsement of a relationship between tetanus vaccination and GBS. Tr. at 99.

b. Absence of Focus on Molecular Mimicry as a Theory of Vaccine Injury

Dr. Tornatore's testimony did not adequately address the pertinent issue, which is the putative causal relationship between tetanus vaccination and GBS by a process of molecular mimicry. Dr. Tornatore explained how molecular mimicry might cause GBS. Tr. at 22-23. He described the process of molecular mimicry, noting that the concept that a foreign molecule can mimic a protein that is found in the host "is an old concept, it's over 300 years old." Tr. at 24. But he did not link tetanus or Td vaccination to GBS by the process of molecular mimicry.

In questioning Dr. Tornatore at hearing, Petitioner's counsel asserted that Dr. Tornatore had been "describing the fact that the vaccination could mimic the foreign protein in your body and then attack's one's own tissue." Tr. at 24. On the contrary, Dr. Tornatore did not actually describe the "fact" of vaccine causation by a process of molecular mimicry. Instead, he switched to the paradigm of challenge/rechallenge, based on the Pollard and Selby article. Tr. at 26-27, 70-71.

Challenge/rechallenge in the Vaccine Program generally pertains to Prong 2, as it may furnish evidence that there was a logical chain of cause and effect linking vaccination to injury. In this case, Dr. Tornatore testified that the case of challenge/rechallenge reported by Pollard and Selby supported his theory of molecular mimicry pertaining to Prong 1. See Tr. at 70-72. I agree in general that evidence that an injury did happen (even to someone other than the vaccinee) makes it more likely that it could happen. This is consistent with the principle established by the Federal

[35] In her response to the order inviting comment from the parties on the 2011 IOM report, Petitioner submitted a legal memorandum arguing that the IOM requires more scientific evidence than needs to be shown in a vaccine case. I agree with Petitioner that the IOM's standards of scientific proof are not required in the Vaccine Program. As stated above, somewhere between what the IOM would require to establish molecular mimicry as a theory of vaccine injury causation, and what we have in this case, which is the bare speculation of an expert, there is scope for a special master to decide whether there is sufficient evidence to preponderate under Prong 1 of Althen. In this case, there is no preponderant evidence.

Circuit that evidence adduced under one prong of the Althen test may be applied to satisfy the other prongs. See Capizzano v. Sec'y of Dep't of Health & Human Servs., 440 F.3d 1317, 1325-26 (Fed. Cir. 2006). The problem, as discussed herein, is that it no longer appears that the Pollard and Selby case report represents a case of challenge/rechallenge.

Meanwhile, Respondent's expert provided science-based evidence countering Dr. Tornatore's theory of causation. Dr. Leist pointed out that the medical literature Dr. Tornatore submitted involved a cross-reaction based on a particular component of an organism known to cause GBS. See Tr. at 107-08. Evidence that *C. jejuni* causes GBS by molecular mimicry does not constitute preponderant evidence that molecular mimicry is a possible explanation for vaccine causation. Further, AIDP, the form of GBS at issue in this case, has been found not to be caused by cross-reaction with *C. jejuni*. Resp't's Ex. C at 6.

In these circumstances, Dr. Tornatore needed to present rebuttal evidence to show that, contrary to Dr. Leist's testimony, the evidence supported possible vaccine causation by the process of molecular mimicry. Dr. Tornatore's presentation failed in this respect. Instead, as noted above, he shifted ground and relied on the notion of challenge/rechallenge. See Tr. at 26-27, 70-71.

c. Misplaced Reliance on an Isolated, Ambiguous Statement by a Physiatrist

In the course of his testimony, Dr. Tornatore was asked by Petitioner's counsel whether he had noted that any of Petitioner's treating doctors associated her vaccination with her GBS. Tr. at 48-49. In response, Dr. Tornatore pointed to the notes of Dr. Michael Winkelmann, a physical rehabilitation specialist at Methodist Rehabilitation Center, where Petitioner was sent for physical rehabilitation after being discharged from the hospital. Pet'r's Ex. 6. Dr. Winkelmann's note stated, under History of Present Illness, "It was felt that immunization series had been the trigger for the development of the Guillain-Barré." Pet'r's Ex. 6 at 3; see Tr. at 49. Referring to this single entry in Dr. Winkelmann's notes, Dr. Tornatore testified, "In fact I think that's important." Tr. at 50; see Pet'r's Ex. 20 at 5. Dr. Tornatore's reliance on the statement by Dr. Winklemann is unwarranted, as discussed below, and further weakens the reliability of his expert testimony.[36]

Dr. Winkelmann is not a neurologist or an immunologist. Dr. Winkelmann is a specialist in the branch of medicine that deals with rehabilitation "using physical agents such as light, heat, cold, water, electricity, therapeutic exercise, and mechanical apparatus, and sometimes pharmaceutical agents." Dorland's Illustrated Medical

[36] Normally, the review of treating physicians' statements would occur under the analysis of Althen Prong 2. I discuss Dr. Winkelmann's statement here because it relates to Dr. Tornatore's reliability as an expert, which is a critical issue under all prongs of the Althen test. I would not have devoted so much attention to Dr. Winkelmann's statement but for the fact that Dr. Tornatore deemed it "important." See Tr. at 50.

Dictionary 1443 (32nd ed. 2012); see Pet'r's Ex. 6 at 5 ("She is admitted now for rehab."). It seems odd that Dr. Tornatore would consider Dr. Winkelmann's opinion of vaccine causation "important," given Dr. Winkelmann's area of specialization.

Similarly, Dr. Winkelmann treated Petitioner only after this "important" note concerning the cause of Petitioner's condition was created. Tr. at 50 ("So her treating physician, and somebody who subsequently took care of her, also felt that the vaccination was the culprit.") (emphasis added); see also Pet'r's Ex. 6 at 5 ("She was under the care of Dr. Thiel at St. Dominic Hospital. She is admitted now for rehab."). Dr. Winklemann was not Petitioner's treating physician when she fell ill. It would appear that at the time he made the statement on which Dr. Tornatore places importance, which was the time of Petitioner's admission to the rehab facility, Dr. Winkelmann had not yet had a treating relationship with the Petitioner. See Tr. at 103 (Dr. Leist testifying that Dr. Winkelmann was not present at the onset of GBS and opines as to causation later only "upon transfer," with no memorialized communications between Dr. Winkelmann and Petitioner's initial treating physicians).

Dr. Tornatore explained that he credited Dr. Winkelmann's statement because, unlike the physicians who did not identify vaccination as causative, or who identified an infectious cause, "Dr. Winkelman[n] has the retrospectoscope where he can look back, he knows everything that happened." Tr. at 79. Dr. Tornatore assumed that "obviously he [Dr. Winkelmann] must have gone through the previous records and come up with something." Tr. at 79-80. This assumption is not based upon any evidence of record.[37]

For all the reasons discussed in this section, I find that Dr. Tornatore's evidence is not sufficient to preponderate under Althen Prong 1. The theory that Td vaccination could cause GBS by molecular mimicry was effectively challenged by Respondent's expert, without meaningful response. Seemingly unable to explain the basis for the theory of molecular mimicry as it might pertain to Td vaccination and GBS, Dr. Tornatore relied instead on the concept of challenge/rechallenge. That phenomenon, with respect to Td vaccination and GBS, no longer is accepted by scientific experts as a link between Td and GBS, as Dr. Leist indicated at hearing, and as was shortly thereafter confirmed by the IOM. Others factors undermined Dr. Tornatore's reliability as an expert, as discussed above – inconsistencies in his presentation, undue reliance on ambiguous evidence, and erroneous statements concerning the article by Pollard and Selby. In sum, Petitioner has not shown by preponderant evidence that Td vaccination could have caused her GBS by the process of molecular mimicry. I proceed

[37] Dr. Tornatore appeared in this respect to be more of an advocate than an objective expert. Expert witnesses are not charged with serving their clients' interests but are employed to "give their unbiased opinion in order to assist the trier of fact in understanding the relevant evidence." Stencel v. Fairchild Corp., 174 F. Supp. 2d 1080, 1085-86 (C.D. Cal. 2001); see Borgognone v. Trump Plaza, No. 88-CV-6139 (ILG), 2000 WL 341135, at * 4 (E.D.N.Y. 2000) (noting the expert's obligation under Rule 702 to assist the trier of fact and eschew partisan advocacy on behalf of the plaintiff's cause). To the extent that Dr. Tornatore's opinion appears to be that of a partisan rather than an objective expert, his opinion carries less weight.

to the second prong of the Althen test for the sake of judicial economy and to provide additional context for my decision.

C. Althen Prong 2

The second prong of Althen requires a petitioner to prove "a logical sequence of cause and effect showing that the vaccination was the reason for the injury." Andreu, 569 F.3d at 1374 (quoting Althen, 418 F.3d at 1278). The sequence of cause and effect must be "'logical' and legally probable, not medically or scientifically certain." Knudsen, 35 F.3d at 548-49. A petitioner is not required to show "epidemiologic studies, rechallenge, the presence of pathological 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. Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second Althen factor. Id. at 1325-26; see Andreu, 569 F.3d at 1375-77. Further, evidence used to satisfy one prong of the Althen test may overlap to satisfy another prong. Capizzano, 440 F.3d at 1326.

1. Treating Physician Evidence

"[T]reating physicians are likely to be in the best position to determine whether 'a logical sequence of cause and effect shows[s] that the vaccination was the reason for the injury.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280); see Andreu, 569 F.3d at 1375-76. The testimony of treating physicians concerning vaccine injury therefore is afforded extra weight when balancing the evidence. See Andreu, 569 F.3d at 1375-76. A special master may find that a petitioner has established causation based on a treating physician's opinion that a vaccination was causally linked to the vaccinee's injury, if the special master finds the opinion to be both reliable and persuasive. Moberly, 592 F.3d at 1323; Andreu, 569 F.3d at 1375-76. The opinions of treating physicians are not conclusive of the issue. See 42 U.S.C. §300aa-13(b)(1) ("Any such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court."); Broekelschen, 618 F.3d at 1346-49 (affirming special master's finding that Petitioner's injury was not transverse myelitis, notwithstanding the diagnosis of some treating physicians); Cedillo, 617 F.3d at 1348.

In Moberly, the Federal Circuit ruled that the treating physicians' opinions were insufficient to establish causation. The Federal Circuit upheld the lower courts' findings that none of the vaccinee's treating physicians offered a reliable statement that the vaccine caused the injury. The Moberly court contrasted Andreu, stating that "there was direct testimony from Andreu's treating physicians stating 'unequivocally' that the [vaccination] caused his seizures," whereas in this case "there was no treating physician evidence that supported the claim of causation." Moberly, 592 F.3d at 1324-25. Instead, the notations in the "medical records regarding the temporal proximity of the [vaccination] to the seizures were all speculative." Id. at 1323.

In Broekelschen, the medical records contained conflicting diagnoses. The Federal Circuit upheld the special master's finding that "certain evidence, such as the medical records and doctors' notes, were not as persuasive as other evidence because the treating doctors were 'not consistent in their diagnoses.'" Broekelschen, 618 F.3d at 1347 (quoting Broekelschen v. Sec'y of Dep't of Health & Human Servs., No. 07-137V, 2009 WL 440624, at *43 (Fed. Cl. Spec. Mstr. Feb. 4, 2009)). The Federal Circuit also upheld the special master's finding that a detailed discharge summary by one doctor was more persuasive than notes by other doctors that did "'not provide any reasoning for their statements.'" Broekelschen, 618 F.3d at 1347 (quoting Broekelschen, 2009 WL 440624, at *11).[38]

In light of recent Circuit decisions, it appears that the weight to be afforded the opinion of a treating physician varies according to the nature of the views expressed and the facts of the particular case. Consistent with the Vaccine Act, such opinions are not dispositive. See 42 U.S.C. §§ 300aa-13(a)(1) (requiring an award based "on the record as a whole"); 13(b)(2) (stating that conclusions in the medical record as to vaccine causation "shall not be binding on the special master or court"). Opinions may be more or less persuasive, depending on the circumstances of the case. A variety of circumstances bear on a special master's decision with respect to treating physicians' opinions.

Among the many circumstances that might be weighed: clarity and context of the treating physician's opinion; nature and duration of the physician's relationship with the vaccinee; the physician's specialty and level of expertise; and the consistency of the treating physician's opinion with the medical record. Also pertinent, according to the Federal Circuit, is the strength of petitioner's showing on the other Althen prongs. For example, where a petitioner produces evidence sufficient to meet the requirements of prongs 1 and 3, a special master may rely solely on the testimony of a treating physician to establish prong 2. See Moberly, 592 F.3d at 1324-25. See also Andreu, 569 F.3d at 1377 n.4 (petitioners presented evidence from a well-qualified medical expert in support of Prong 1).

For the reasons discussed below, I am not persuaded by the notations of treating physicians in this medical record that there is a logical sequence of cause and effect between Petitioner's vaccination and her medical complaints.

The medical record discloses very little by way of treating physician opinions concerning whether vaccination was the cause of Petitioner's GBS. One of Petitioner's

[38] But see Campbell v. Sec'y of Dep't of Health & Human Servs., 97 Fed. Cl. 650, 667 (2011) ("Any expectation that treating physicians will record the precise biological theories behind their belief that a patient's condition was caused by a particular trigger is discordant with the reality of medical treatment. Doctors are and must be concerned with treating patients, not with articulating the precise biological theories upon which they base their diagnoses.").

treating neurologists, Dr. Moore, was the only doctor to note explicitly his opinion: the cause was likely a gastrointestinal infection. Pet'r's Ex. 4 at 54. Dr. Winkelmann, a physiatrist who treated Petitioner during the rehabilitation phase of her illness, noted only by history that it was "felt" that the cause was vaccination. Pet'r's Ex. 6 at 3.

The source of the information noted by Dr. Winkelmann under the "history of present illness" is unclear. Pet'r's Ex. 6 at 3. Since the "feeling" that the vaccine had triggered Petitioner's GBS was not noted in any of the medical records created during Petitioner's hospitalizations, see Pet'r's Ex. 4; Pet'r's Ex. 25, it is not likely that this was the "feeling" of any of Petitioner's treating physicians. See Pet'r's Ex. 6 at 3. There was no reason for Dr. Winkelmann to note his own opinion as to causation when Petitioner was admitted to his care for rehabilitation, making it less likely that he would have done so.

Further, the statement, "It was felt that immunization series had been the trigger for the development of the Guillain-Barré," is equivocal on the face of it. Pet'r's Ex. 6 at 3. The way the sentence is written again indicates that it may or may not be a statement by Dr. Winkelmann of his own belief, but rather a record of someone else's belief. The statement also by its terms records only that there was a feeling that vaccine might have triggered the GBS. The statement implies that this was simply a "feeling," not a medical finding.

Other treating personnel noted at various points that Petitioner had received a vaccination a couple of weeks before the onset of her illness, but none stated that vaccination had caused it. A treating physician's recognition of a temporal relationship does not advance the analysis of causation, as Dr. Tornatore agreed. See Tr. 50 (Dr. Tornatore discounting the probative value of notations that Petitioner "had a vaccine two weeks prior [to onset]").

Upon careful consideration of the record, I find that none of the notations, other than Dr. Moore's single statement, even constitutes a treating physician "opinion." In contrast to cases in which the record reveals extensive analysis of the causation issue, it appears in this case that once the diagnosis of GBS was made there simply was very little medical attention paid by treating personnel to the cause of Petitioner's illness.

2. Dr. Tornatore's Evidence

On this record, Petitioner's case on Prong 2 depends, as before, on Dr. Tornatore's testimony. Dr. Tornatore testified that, two weeks after vaccination, Petitioner's GBS symptoms were recorded. Tr. at 31-32. She was afebrile and was treated for acid reflux. Tr. at 33-34. Her condition improved. Tr. at 34. According to Dr. Tornatore, her blood tests did not indicate the presence of a virus. Tr. at 35-36. She had nausea and vomiting, but no diarrhea. Tr. at 36-37. On that basis, Dr. Tornatore ruled out a gastrointestinal viral syndrome. Tr. at 37.

Petitioner then developed neurological symptoms. Tr. at 38. She had lower back pain. Dr. Tornatore testified that low back pain can be a symptom of GBS. Id. Dr. Tornatore stated that Petitioner's reflux symptoms also could have been caused by GBS. Tr. at 39. He described nerve conduction studies and concluded that the results in Petitioner's case indicated that she suffered a type of GBS that is "very typical after one's had a viral infection or after a vaccination." Tr. at 42.

Dr. Tornatore testified that Petitioner's symptoms were not consistent with known infections that might have caused her GBS. See Tr. at 45-46. Dr. Tornatore testified that Petitioner's GBS occurred within an appropriate time frame, based on reports of GBS induced by swine flu vaccination in 1976-77. Tr. at 28-29, 53. He described as significant records indicating that Petitioner felt pain and stiffness in her arm and back, and weakness in her leg shortly after receiving the vaccination. Tr. at 30-31. This indicated to Dr. Tornatore that she suffered "an allergic type reaction to the vaccination." Tr. at 31 ("she had a kind of something happen during the initial vaccination"); see also Tr. at 66 ("[T]o me that [problems right after vaccination] is a very, very important piece of information that something unusual happened to her.").

On cross-examination, Dr. Tornatore also pointed to some evidence of "autonomic instability," which he testified was inconsistent with infection, Tr. at 64-65, but he did not actually rely on this evidence in expressing his opinion. Tr. at 64 ("I didn't want to go down this . . . ."). He also elaborated on the pain and weakness Petitioner reported to a nurse "shortly after" her vaccination. Tr. at 67. He testified that "that is a hallmark" of GBS, but why "it kind of waxed and waned is a little unclear." Tr. at 68.

There were significant weaknesses in Dr. Tornatore's testimony. Dr. Tornatore's opinion that Petitioner's report of a stiff neck and sickness at the time of vaccination was symptomatic of GBS, Tr. at 67-68, appears to be at variance with his testimony that these symptoms were an immediate allergic reaction to the vaccine. Tr. at 31, 69. An allergic reaction is not the same condition as GBS. If there is some medical relationship between these two conditions, Dr. Tornatore's testimony did not elucidate it.

Dr. Tornatore testified that "presumably" "a kind of something happened" after the vaccination, but he could not support the presumption with evidence. Tr. at 31. While a temporal association may be useful in the clinical setting to develop a course of treatment, temporal proximity alone is not a reliable indication of vaccine injury. See Grant, 956 F.2d at 1148 ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Neither does the absence of other identified causes necessarily implicate vaccines. "[T[he absence of alternative causes for a condition does not alone suffice to ascribe causation to the vaccine." Lampe v. Sec'y of Dep't of Health & Human Servs., 219 F.3d 1357, 1361 (Fed. Cir. 2000) (citing Grant, 956 F.2d at 1149). His "presumption" that "something happened" as a result of Petitioner's vaccination is not entitled to significant weight. [39]

[39] In general, the Federal Circuit has held that a proximate temporal association alone is not sufficient to show a causal connection between the vaccination and the injury. See Grant, 956 F.2d at 1148. In

Dr. Tornatore distinguished the type of GBS suffered by Petitioner from the type of GBS caused by *C. jejuni*. Tr. at 42-43. *C. jejuni*, a bacterium, causes axonal degeneration. Tr. at 42. Petitioner, although she had some axonal effects, principally suffered demyelination, which is not typical of *C. jejuni* infection. Id. Dr. Tornatore called this distinction "very important." Id.

Dr. Tornatore's own testimony ruling out *C. jejuni* infection still left two possible causes of Petitioner's GBS– virus and vaccine. Ruling out a bacterial infection did not logically make vaccine causation more likely than viral infection. Dr. Tornatore's testimony on this point was confused and confusing. Tr. at 42-46. He appeared in part to be responding to Dr. Leist, but Dr. Leist did not assert that *C. jejuni* was the cause of Petitioner's GBS, only that the cause probably was an infectious agent like *C. jejuni*. Resp't's Ex. A at 10-11. See also Resp't's Ex. C at 3 (concluding that *C. jejuni* does not appear to cause the demyelinating form of GBS).

Dr. Tornatore discounted entirely the notation by Dr. Moore, who performed Petitioner's EMG, that her "AIDP is likely related to gastrointestinal illness." Tr. at 42, 56-57. Dr. Tornatore stated: "Now they're assuming that there was a gastrointestinal illness, but they just took [that] based on the history that they got from her at the top, they were not the treating physicians." Tr. at 42-43; see also Tr. at 77 ("He [Dr. Moore] did an EMG, he spent maybe an hour with her, and so he is not the person that should be offering the definitive reason for her diagnosis."). When Dr. Moore's clear statement that the likely cause of this case of GBS was a gastrointestinal illness is compared to the ambiguous notation by Dr. Winkelmann ("It was felt that …."), on which Dr. Tornatore relied, the inconsistency of Dr. Tornatore's testimony is apparent. He rejected Dr. Moore's clear notation but placed great importance on Dr. Winkelmann's more ambiguous notation. In context, neither notation offers persuasive evidence as to vaccine causation.

In addition, the experts disagreed on their interpretation of the medical record. In the course of his testimony, Dr. Tornatore pointed to several factors that, in his opinion, indicated vaccine causation. "IGA elevation is a sign that there is some activation of the immune system," he stated. Tr. at 46. Petitioner had antinuclear antibody ("ANA")

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addition, the absence of other identified causes does not necessarily implicate vaccines. See Lampe, 219 F.3d at 1361 (citing Grant, 956 F.2d at 1149). In Capizzano, the Federal Circuit stated that the "fact that these physicians' diagnoses may have relied in part on the temporal proximity of [the petitioner's] injuries to the administration of the vaccine is not disqualifying." Capizzano, 440 F.3d at 1326. This statement from Capizzano was in turn relied upon by the Federal Circuit in Andreu, which translated the statement in Capizzano to mean, "A treating physician may rely on the close temporal proximity between a vaccine and an injury in concluding that there is a logical sequence of cause and effect between the vaccine and the injury." Andreu, 569 F.3d at 1377. In context, it appears that treating physician statements based on temporal proximity and the absence of evidence of another cause must be considered; such statements do not satisfy Prong 2 of Althen absent preponderant evidence on Prongs 1 and 3. See Andreu, 569 F.3d at 1375 ("If a claimant satisfies the first and third prongs of the Althen standard, the second prong can be met through medical opinion testimony.") (citing Capizzano, 440 F.3d at 1326).

immunofluoresence. Tr. at 47. In addition, he pointed to "another antibody test," in which "the titer was negative, [but] the pattern was atypical." Tr. at 46. Petitioner's rheumatoid factor was also "borderline elevated." Tr. at 46-47. Further, Petitioner showed elevated "capa [sic] light chains." Tr. at 47. This, according to Dr. Tornatore, "really speaks to an unusual type of immune activation," "vaccine as opposed to antecedent infection." Id.[40]

Dr. Leist disputed the factors on which Dr. Tornatore relied to diagnose a vaccine injury. Dr. Leist contradicted Dr. Tornatore's testimony concerning an initial vaccine reaction. He noted that the reported reaction "of feeling achiness" was remote from the initial site of the vaccination, see Tr. at 91, and that "there is no indication that [Petitioner] had an immediate anaphylactoid reaction at the time of vaccine administration." Tr. at 91-92.

Dr. Leist also challenged Dr. Tornatore's opinion that vaccination was more likely than infection as the cause of Petitioner's GBS. He noted that "there was very little infectious work up in this patient." Tr. at 92. Dr. Leist disputed Dr. Tornatore's reliance on Petitioner's normal white blood cell count, stating that a viral infection might be present without being manifested by an increase in white cells. Tr. at 93. He stated that the mild elevations in Petitioner's ESR (erythrocyte sedimentation rate), rheumatoid factor and IGA levels were too low to be significant. Tr. at 93-95. None of the laboratory findings ruled out infection as a cause of Petitioner's illness, according to Dr. Leist. Tr. at 95. He stated that the time frame in which Petitioner experienced her first symptoms was consistent with a gastrointestinal infection as the cause of Petitioner's GBS. Tr. at 97-98. Dr. Leist contended that Petitioner's gastrointestinal symptoms were more significant than acid reflux, "So it wasn't just a matter of taking an over-the-counter medication and eliminating that particular problem." Tr. at 101. He testified that abdominal pain is a symptom in some patients who have an infection and subsequently develop GBS. Tr. at 106.

In addition to disagreeing with Dr. Tornatore's analysis of the medical records, Dr. Leist pointed to peer-reviewed epidemiological studies indicating no association between GBS and vaccines containing tetanus toxoid. Tr. at 105. He testified reasonably that while such studies do not rule out rare events, they make it all the more important to examine such cases carefully before concluding that vaccines caused the injuries. See Tr. at 115.

On this record, in which I find scant medical record evidence one way or the other, no meaningful treating physician opinions, and two qualified experts who disagree concerning the evidence, I rely for my decision on the testimony of the expert I

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[40] Kappa light chains are found in immunoglobulin molecules. Dorland's at 335. These factors are not mentioned in either expert report. See Pet'r's Ex. 20 at 8-9; Resp't's Ex. A at 7-11. Without further explanation, I cannot give significant weight to the elevated kappa light chains. If elevation of kappa light chains is "really" significant, as Dr. Tornatore stated, one would have expected some mention of them in his expert report.

find more reliable. Dr. Tornatore's reliability was diminished by apparent contradictions, gaps, vagueness, and illogic. Dr. Leist's testimony was clearer, more consistent, more logical, and more closely tied to the facts of this case. For all the reasons explained above, on the record as a whole, I find insufficient evidence to satisfy Petitioner's burden on Prong 2 of the Althen test.

D. Althen Prong 3

The Secretary conceded Prong 3 of the Althen test. Tr. at 6-7. Respondent's expert also testified that the Prong 3 time frame was appropriate. See Tr. at 91 ("[S]he had the administration of the vaccine . . . she then presented with GI symptoms within a time frame consistent for an association with Guillain-Barré Syndrome."). The timing of the onset of Petitioner's illness in relation to vaccination therefore is deemed appropriate. This factor alone does not determine entitlement to compensation. See Grant, 956 F.2d at 1148.

IV. CONCLUSION

Petitioner has not presented preponderant evidence to support her claim under the Vaccine Act that the Td vaccination was the cause of her injuries. Accordingly, she is not entitled to compensation under the Vaccine Act, and her Petition must be **DISMISSED**. In the absence of a timely motion for review filed pursuant to Vaccine Rule 23, the Clerk is directed to enter judgment according to this decision.

IT IS SO ORDERED.

s/ Dee Lord
Dee Lord
Special Master