OFFICE OF SPECIAL MASTERS

No. 03-0620V

(Filed: February 7, 2006)

ADELA QUINTANA DE BAZAN Petitioner, v. SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, Respondent.	*	UNPUBLISHED

Peter G. Lomhoff, Esq., Oakland, California, for Petitioner.
Heather L. Pearlman, Esq., U.S. Department of Justice, Washington, D.C., for Respondent.

ENTITLEMENT DECISION[1]

ABELL, Special Master:

Petitioner alleges that receipt of a tetanus-diphtheria ("Td") vaccination caused-in-fact her acute disseminated encephalomyelitis ("ADEM") eleven hours post-vaccination.

In fine, Petitioner has not demonstrated by preponderant evidence a prima facie element of her claim – that the onset of her ADEM occurred within a medically appropriate time frame were it triggered by the vaccination in question. Accordingly, this petition is denied.

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[1] This document constitutes my final "decision" in this case, pursuant to 42 U.S.C. § 300aa-12(d)(3)(A). Unless a motion for review of this decision is filed within 30 days, the Clerk of this Court shall enter judgment in accord with this decision.

Petitioner is reminded that, pursuant to 42 U.S.C. § 300aa-12(d)(4) and Vaccine Rule 18(b), a petitioner has 14 days from the date of this decision within which to request redaction "of any information furnished by that party (1) that is trade secret or commercial or financial information and is privileged or confidential, or (2) that are medical files and similar files the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, "the entire decision" may be made available to the public per the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002).

FACTS

On 18 March 2005, this Court issued a preliminary ruling on certain factual issues in dispute. That ruling is incorporated herein by reference. In pertinent part, the Court made the following findings:

> In brief, Mrs. Bazan received a Td vaccination sometime before 11:00 a.m. the morning of 19 April 2000. By 9:00 p.m. she began experiencing certain symptoms and a general decline in health that can be associated with ADEM which progressed in intensity over the next several days finally causing her to seek emergency medical attention on 2 May 2000.
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> . . . Both parties, however, agree that ADEM is the correct diagnosis. And they agree that the onset of symptoms began roughly 11 hours after the vaccination in question. Therefore, the only real questions left in this case are (1) can Td cause ADEM and (2) did this particular vaccination cause-in-fact Petitioner's ADEM. These questions will require the testimony of medical experts.

In the Onset Ruling, it was noted that, during the medical visit when the vaccine was administered, Mrs. Bazan presented with a sore throat, swelling in the left of her neck, and nasal discharge. Petitioner's Exhibit ("Pet. Ex.") 16 at 56. At that time, the Court made "no finding as to what the foregoing facts signify, if anything, but merely notes their inclusion in the medical records."

On further consideration, the Court cannot say whether the symptoms present at the 19 April 2000 health care visit were indicative of an underlying viral or bacterial infection. Such is certainly suggested by the medical records; however, the Court cannot draw such a conclusion by a preponderance of the evidence. In fact, the Court takes particular note of a letter from the treating physician who writes, "I feel confident in stating that she did not appear to have any viral or bacterial infection when I evaluated her." Pet. Ex. 37. Had the treating physician noted a bacterial or viral infection, presumably Mrs. Bazan would have been treated accordingly. Instead, her physician attributed these symptoms to and treated them as allergies or rhinitis. Is it possible that her symptoms were caused by an underlying, precipitant but mild (or asymptomatic) viral or bacterial infection? Certainly. The Court is particularly suspicious given Mrs. Bazan's history of colds and other sinus issues. See, Onset Hearing Transcript at 77-78. All things considered, however, the Court cannot say there is preponderant evidence of a viral or bacterial infection in the medical records.

ENTITLEMENT HEARING

On 13 May 2005, the Court convened a hearing for the purpose of taking evidence from the key expert witnesses in this case. Petitioner called Dr. Susan Hansen. Respondent called Dr. Subramaniam Sriram.

Dr. Hansen is Mrs. Bazan's treating neurologist. Board certified in neurology, electrodiagnostic medicine, and clinical neurophysiology, Dr. Hansen is an adjunct and associate

professor at Stanford University Hospital, which includes an appointment at Lucile Packard Children's Hospital. In her full-time clinical practice, Dr. Hansen rarely sees patients with ADEM - approximately one every five years for a total of perhaps five cases. Transcript ("Tr.") at 13.

Dr. Sriram is the director of the multiple sclerosis clinic at Vanderbilt Medical Center in Nashville, Tennessee. In that capacity, Dr. Sriram conducts clinical research concerning treatments for immunological diseases affecting the central nervous system and basic science research involving animal models. He is board certified in internal medicine and neurology and is widely published in his field. In consultation at the local children's hospital, Dr. Sriram sees roughly 5 to 6 cases of ADEM each year. Tr. at 71.

ACUTE DISSEMINATED ENCEPHALOMYELITIS OR ADEM

ADEM is a disorder of the central nervous system ("CNS") wherein the material that sheathes and protects the nerves (the myelin) is inadvertently destroyed by one's immune system. It is typically preceded – or triggered – by an infectious illness such as a virus, more rarely by a bacterial infection, or even more rarely – as Petitioner would argue here – by a vaccination. However, it can occur spontaneously. Pet. Ex. 22. And, as indicated by Dr. Sriram, in 30 to 50 percent of ADEM cases the etiology is unknown. Tr. at 60. Moreover, it is possible for ADEM to be triggered by an underlying or asymptomatic infection as there is no correlation between the severity of the antecedent infection and the severity of the demyelinating disease. Id.

ADEM is a diffuse affront to the central nervous system, which includes the brain, brain stem, optic nerves and spinal cord. It is therefore distinguished from a more specific violation such as optic neuritis wherein the myelin surrounding the optic nerves is destroyed oftentimes resulting in blindness. Similarly, ADEM is distinguishable from other conditions such as brachial neuritis, polyradiculopathy, and Guillane-Barré Syndrom ("GBS") wherein the peripheral nervous system ("PNS"), located in the arms and legs, is targeted.

Actually, there is some contention between the parties as to the correlation between conditions affecting the CNS versus those affecting the PNS. According to The Merck Manual of Diagnosis and Therapy, "Myelin formed by the oligodendroglia in the CNS differs chemically and immunologically from that formed by Schwann cells peripherally, but both types have the same function: to promote transmission of a neural impulse along an axon." Sec. 14, Ch. 180, filed as Pet. Ex. 22, available at http://www.merck.com/pubs/mmanual/section14/chapter180/180a.htm. The Merck Manual further indicates that injuries which affect the CNS and those affecting the PNS are presumed to have the same immunopathogenesis. However, it is unclear whether any conclusion can be drawn from that statement or whether it is merely to say that both are immune mediated. On that point, the Institute of Medicine explicitly distinguishes demyelinating diseases of the CNS from those of the PNS. Tr. at 19. Moreover, according to Dr. Sriram:

> Although there is myelin in both the central and the peripheral nervous system, the myelin is structurally different between the two parts of the nervous system. They don't have all the same proteins. They don't have all the same type of

membrane structure either.

However, both the peripheral and the central nervous system are targets of an immune response, and you can get a demyelinating disease of the peripheral nervous system or you can get a demyelinating disease of the central nervous system.

Just because you have an inciting antigen that causes peripheral nervous system demyelinating disease, that does not necessarily extend the assumption that that can also cause central nervous system demyelinating disease. In fact, just the opposite.

We have very rarely seen this kind of an overlap between events -- immunizations that cause both central and peripheral nervous system demyelinating disease, so the flu vaccine that causes Guillain-Barré does not cause ADEM as we know it.

Tr. at 53 (emphasis added).

The exact cause of ADEM is as yet unknown. However, every indication points to some sort of immunological trigger, like a viral infection, communicated to the lymphatic system which responds by creating lymphocytes to combat the perceived threat. Those lymphocytes then divide producing "chemical mediators" called cytokines which are anathema to myelin. As these lymphocytes divide, they traverse the circulatory system and may pass through the protective blood brain barrier. Once on the other side of the blood brain barrier the lymphocytes continue to divide and the cytokines produced damage the myelin sheaths protecting the nervous system.

According to Dr. Sriram:

[F]rom the time that you immunize someone to the time you get enough lymphocytes within the body sufficient enough to traverse the circulatory system into the brain will take a minimum of five to seven days, and then, having traversed to the brain, then it goes through a second period of amplification in the brain wherein the disease manifests itself.

So, if we look at an animal model, normally, after you inject the animal with a particular antigen, for example, myelin proteins, the disease onset is anywhere between 10 to 14 days. So that is sort of the timeframe it takes for the immune system to be stimulated, activated, and for the cells to proliferate to produce an immunological disease.

So, if you look at the timeframe, if you want to extend the timeframe to human disease, you'll have to therefore -- since the immunological kinetics in the mouse is very similar to that in humans, for example, the time taken to produce an antibody response, the time taken to form an immune T-cell response, are similar in humans as well as in rodents.

So, given the fact that these kinetics are fairly -- are parallel, it therefore becomes reasonable to assume that these immunological timeframes that you're seeing in the animals are likely to be similar to that in humans as well; that is, that if you immunize a human being with a myelin protein, for whatever reason, it has happened in the past, we have done that before, that it takes about 10 to 14 days for

the ADEM disease to manifest itself.

Granted, it may be a little shorter, it may be a little later, but that is approximately the timeframe because that is the approximate time it takes to educate, activate, and proliferate these key lymphocytes which are the essential mediators of the disease.

Tr. at 41-42.

On the issue of timing, in 1994, the Institute of Medicine ("IOM") declared:

> There is plausibility for a causal relation between vaccines and demyelinating disorders. The reports in the literature that describe a possible association between demyelinating diseases of the CNS (ADEM, transverse myelitis, and optic neuritis) are case reports. There are at least two case reports in the literature for each of the above mentioned demyelinating diseases of the CNS. The case reports describe the demyelinating disease that occurs within the biologically plausible latency period of 5 days to 6 weeks.

Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality at 85, filed as Pet. Ex. 34 (emphasis added). However, the IOM report goes on to state that there is not enough evidence to distinguish the frequency of these case reports from the expected background rate, meaning there is no evidence that the documented occurrences of a demyelinating disease following immunization are more than mere coincidence. Therefore, the IOM concludes, "The evidence is inadequate to accept or reject a causal relationship between tetanus toxoid, DT, or Td and demyelinating diseases of the CNS (ADEM, transverse myelitis, and optic neuritis)." Id. at 86.

Certain special masters have previously ruled on Td derived ADEM petitions.[2] In a case analogous to the one at bar, a special master found that a Td vaccination caused-in-fact ADEM which manifested roughly two weeks post-vaccination. Johnson v. Secretary of HHS, No. 99-219V, 2000 WL 1141582 (Fed. Cl. Spec. Mstr. July 27, 2000).[3] It was held, "Where an immunological process requires a certain number of days or weeks to manifest itself (as it does here) and the challenge and effect are so linked temporally, that process is sufficient legally to support an expert opinion of causation." That special master found it particularly relevant (1) that the treating physicians believed the vaccination triggered the injury, (2) that their belief found support in the medical literature, and (3) "the whole sequence of immunological challenge and illness occurred within the proper temporal framework to allow for the demyelination process."

Legal Standard

[2] Though decisions by this Office and by the Court of Federal Claims pertaining to other petitions are not binding on the case at bar, the analysis contained therein is often quite illuminating.

[3] Dr. Sriram also testified on behalf of Respondent in the Johnson case.

A petitioner may prevail under the National Childhood Vaccine Injury Act of 1986 (Vaccine Act or Act)[4] in one of two ways. First, if it is shown that an injury recognized by the Vaccine Injury Table, 42 C.F.R. § 100.3, occurred within the prescribed time frame, a petitioner is afforded a presumption of causation. § 11(c)(1)(C)(I). Second, if a petitioner's injury is not a "Table Injury," she may prove, by preponderant evidence, that the vaccination caused-in-fact the injury alleged. §§ 11(c)(1)(C)(ii)(I) and (II). In either event, this Court cannot find in favor of a petitioner based on her claims alone. At the very least, the claims must be substantiated "by medical records or by medical opinion." § 13(a)(1). In this particular case, Petitioner is not alleging a Table Injury. Therefore, she must prove her case through the traditional tort method of causation-in-fact.

As with any area of the law, the legal standard concerning causation-in-fact is the subject of an ongoing dialogue among the various courts and between bench and bar.[5] It is generally agreed that causation-in-fact requires "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury." Grant v. Secretary of HHS, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A reputable medical or scientific explanation must support this logical sequence of cause and effect. Id. at 1148; Strother v. Secretary of HHS, 21 Cl. Ct. 365, 370 (1990), aff'd, 950 F.2d 731 (Fed. Cir. 1991). Temporal association of the onset of the injury with the vaccination is not sufficient to establish causation-in-fact. Grant, 956 F.2d at 1148; Strother, 21 Cl. Ct. at 369. Additionally, showing an absence of an alternative cause of injury does not meet petitioner's affirmative duty to show causation. Grant, 956 F.2d at 1149. The Federal Circuit recently restated the causation-in-fact standard as follows:

> [Petitioners'] burden is to show by preponderant evidence that the vaccination brought about [the] 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. Secretary of HHS, 418 F.3d 1274, 1278 (Fed. Cir. 2005). It is clear, however, that Althen did not come to destroy precedent but to clarify, "[R]equiring that the claimant provide proof of medical plausibility, a medically-acceptable temporal relationship between the vaccination and the onset of the alleged injury, and the elimination of other causes-is merely a recitation of this court's well-established precedent." Id. at 1281.

Discussion

The question narrowly tailored in this case is whether Td can cause ADEM with onset 11 hours post-vaccination.

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[4] The statutory provisions governing the Vaccine Act are found in 42 U.S.C. §§300aa-10 *et seq*. (West 1991 & Supp. 1997). Hereinafter, reference will be to the relevant subsection of 42 U.S.C.A. §300aa.

[5] It is anticipated that this dialogue will be furthered when the Federal Circuit hears Pafford v. Secretary of HHS, No. 01-165V, 2004 WL 1717359 (Fed. Cl. Spec. Mstr. July 16, 2004), aff'd 64 Fed.Cl. 19 (2005). Oral argument is presently scheduled for 10 February 2006.

Petitioner's medical expert, Dr. Hansen, avers that it can and bases her opinion on various case reports. At the hearing, she testified, "There are a number of cases, first of all, that show ADEM has developed after tetanus vaccinations . . . -- there are also a number of cases that clearly document a very brief period of time between the vaccination and the development of neurological symptoms." Tr. at 8.

It is axiomatic to say that one must be cautious when attempting to infer causation based on case reports. As has been recognized, of all the evidence proffered in support of causation, case reports provide the least support. Put another way, "It is difficult, however, to infer causality from individual case reports. The reported cases may simply represent coincidental temporal association with vaccination." Vaccinations and Risk of Central Nervous System Demyelinating Diseases in Adults, ARCHIVES OF NEUROLOGY, Vol. 60, Apr. 2003, filed as R. Ex. H.

That being said, case reports certainly can be probative concerning the question of causation, particularly in instances such as this where an occurrence is so rare that physicians would be quite likely to report any case that involved the onset of ADEM within close temporal proximity of a vaccination. Moreover, the IOM relied on case reports in considering the question of whether Td can cause demyelinating disease in the CNS. Pet. Ex. 34 at 85. Based on those case reports, the IOM indicates that "[t]here is biological plausibility for a causal relationship between vaccines and demyelinating disorders." Id. Likewise, the IOM also relied in part on case reports in determining that Td can cause GBS in the peripheral nervous system. R. Ex. I.

According to Dr. Hansen, "The medical literature clearly shows that ADEM and other similar demyelinatng disease sometimes develops within about eleven hours or less following a vaccination that causes the disease." P. Ex. 71 at 7 (emphasis added). During the hearing, however, it became evident that the case reports upon which Dr. Hansen relies are anything but clear. As Dr. Hansen admitted on cross, they are "open to interpretation." Tr. at 32. A careful review of the medical reports and literature in question raises some concerns as to the reliability of Dr. Hansen's "interpretation."

Concerning the question at bar, whether ADEM can develop within 11 hours, Dr. Hansen points to an editorial published in a 2001 edition of NEUROLOGY, entitled ADEM: Distinct disease or part of the MS spectrum? by doctors Hans-Peter Hartung and Robert I. Grossman. This editorial includes a chart summarizing the findings of three studies on ADEM published in that same edition. The editorial indicates that, in those three studies, the "disease evolution" of ADEM occurred within a range of 1-42 days, 0-31 days, and 0-14 days respectively. Hansen apparently interprets "disease evolution" as the timing from the triggering event to the onset of ADEM. However, a careful examination of the underlying studies does not support her reading.

The 1-42 day range comes from a study by Hynson, et al., entitled Clinical and neuroradiological features of acute disseminated encephalopyelitis in children. Pet. Ex. 21. That study states, "Neurological symptoms developed over 1 to 42 days (mean 4.2 days). The child who developed symptoms over 42 days had become withdrawn and unsteady 7 days after a febrile illness, with a gradual increase in ataxia and irritability from that time." Hence, in that child's case, the

onset of ADEM was preceded by a febrile illness seven days earlier while the development of neurological symptoms associated with ADEM stretched out over 42 days thereafter. Hence, a cogent reading of this study appears to indicate that the 1 to 42 day range refers to the development of the neurological symptoms post onset rather than the timing from trigger to onset as Dr. Hansen suggests.

The 0-31 day range derives from a study by Dale, et al., entitled Acute disseminated encephalopyelitis, multiphasic disseminated encephalomyelitis and multiple sclerosis in children. Pet. Ex. 29. The Dale study indicates that "the mean latency between predemyelinating illness and the onset of neurological signs was 13.0 days (range 2-31 days)." Id. at 2410. However, "Neurological presentation varied from an acute explosive onset, with a maximum neurological deficit attained within 1 day, to more indolent progression with maximum deficit at 31 days (mean 7.1 days)." Id. at 2411. Hence, the 0-31 range referred to in the editorial cited by Dr. Hansen refers not to the latency period between the triggering event and the onset of ADEM but to the time it takes for ADEM to become fulminate once it first manifests.

Finally, the 0-14 range comes from a study by Schwarz, et al., entitled Acute disseminated encephalomyelitis: A follow-up study of 40 adult patients. Pet. Ex. 26. A table in that study indicates that "Duration of symptoms before admission" had a range of 0-14 (mean 4). Nowhere in this article is it indicated that ADEM developed within hours of a triggering event. Rather, when that latency period is discussed, it is mentioned that "One patient, who later progressed to MS, experienced the first symptoms a few days after active immunization against diphtheria and tetanus." Id. at 1315 (emphasis added). Hence, "Duration of symptoms before admission" more likely than not refers to the time from onset of ADEM to hospitalization.

Dr. Hansen also refers to an article published by Respondent's counsel, Dr. Sriram which states that the shortest latency between a rash (related to rubella) and neurological symptoms was 24 hours; moreover, "encephalitic syndromes have the shortest latency." R at Ex C at 344. Respondent explained that the 24 hour period mentioned in that article is timed not from the triggering event, the introduction of rubella, but from the manifestation of the symptomotology of that infection, the rash. With that study, it is unclear when the trigger, rubella, was first introduced versus when the rash developed. However, in the case at bar we know that the vaccination was given 11 hours prior to the first sign or symptom of ADEM. Therefore, assuming *arguendo* that Petitioner's interpretation is correct, still the latency period is 24 hours and not 11 hours.

Failing to provide any instance of ADEM developing within 11 hours as is her supposition, Dr. Hansen goes on to state, "There are a few cases that are reported within a few hours. It's not as common, but there are cases in which demyelinating -- both peripheral and central infections have been -- or reactions have been reported within a few hours." Tr. at 25. When pressed for examples, Dr. Hansen points to injuries involving the PNS, Pet. Ex. 56, or to injuries catalogued in the 1970s which might possibly have involved the CNS. Tr. at 29.

Dr. Hansen fails to adequately explain why cases involving the PNS, which is located in the

arms and legs, can be properly analogized to those involving the CNS including the brain, brain stem, spinal cord and optic nerves. As previously discussed, there are indications in the literature that demyelinating diseases in the PNS and CNS have a similar immunopathogenesis. But, as Dr. Sriram cogently explained, just because both disease processes are believed to be immune mediated is not to say that the mechanisms involved, and therefore the time frames involved, are at all similar. If Dr. Hansen is correct, that PNS and CNS are substantially similar, one would expect to see anecdotal cases indicating the manifestation of a CNS injury within hours of vaccination as with the case reports involving PNS. Instances of ADEM are rare; yet, the Court finds the lack of even one single, directly analogous case or anecdote quite probative.[6] While Petitioner is not required under the Vaccine Act to demonstrate the exact biological mechanism whereby a vaccination is alleged to have caused a particular injury,[7] the lack of a cogent explanation concerning the expert's reliance on the analogy between PNS and CNS demyelinating diseases goes to the weight, or reliability, of her testimony.

According to the Federal Circuit in Althen, the Vaccine Act does not require that petitioners submit medical literature linking a particular vaccination with the injury alleged. By analogy, petitioners are not necessarily required to submit the opinion of a medical expert. Instead, a petitioner's claim may be substantiated "by medical records or by medical opinion." § 13(a)(1) (emphasis added). Where a petitioner can demonstrate a prima facie case (a plausible medical theory, logical sequence of cause and effect, and appropriate temporal proximity) based solely on the medical records – for instance, in cases involving rechallenge – testimony from a medical expert might be superfluous. But in this particular case, Petitioner is offering both the testimony of Dr. Hansen and numerous medical articles in order to prove a prima facie element of her claim – the question of a medically appropriate temporal proximity. As in any such instance, the evidence presented by Petitioner should be based on reliable medical or scientific evidence. Althen, 418 F.3d at 1278; Grant, 956 F.2d at 1148. Moreover, the onus is on this Court to evaluate the reliability of the evidence presented in keeping with the "gatekeeping" function required by Daubert v. Merrow Dow Pharm., Inc., 509 U.S. 579, 597 (1993). See Terran v. Secretary of HHS, 195 F.3d 1302, 1316 (Fed. Cir. 1999) (holding that it is appropriate for a special master to utilize the factors enunciated

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[6] It is also of interest, if this case is truly *sui generis* as Petitioner's expert seems to indicate, that a report of Petitioner's case was not published in a medical journal for the edification of the medical and scientific world; at least the Court is not aware of any such publication.

[7] According to the Federal Circuit in Knudsen v. Secretary of HHS, 35 F.3d 543, 549 (1994):

> Furthermore, to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program. The Vaccine Act does not contemplate full blown tort litigation in the Court of Federal Claims. The Vaccine Act established a federal "compensation program" under which awards are to be "made to vaccine-injured persons quickly, easily, and with certainty and generosity." House Report 99-908, supra, at 3, 1986 U.S.C.C.A.N. at 6344.
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> The Court of Federal Claims is therefore not to be seen as a vehicle for ascertaining precisely how and why DTP and other vaccines sometimes destroy the health and lives of certain children while safely immunizing most others.

in Daubert when addressing issues of medical causation).

In fine, no reliable medical or scientific evidence was proffered by Petitioner which would indicate that the onset of ADEM within 11 hours of a triggering event is considered a medically appropriate time frame. Dr. Hansen failed to produce one anecdotal record much less scientific literature on point. Instead, Petitioner's argument rests on analogy to case reports involving PNS without adequately explaining why those reports have any bearing on the case at bar. While Petitioner does provide some evidence of similarity between the two, by weight such does not rise to the level of preponderant evidence. For the foregoing reasons, though she is the treating neurologist and obviously defends her conclusions with strong conviction, on the question of timing, the Court finds Dr. Hansen's testimony to be less reliable than that of Respondent's expert, Dr. Sriram. In the end, Dr. Hansen's asseverations remain too conjectural or speculative.

That is not to say that the Court agrees with the entirety of Dr. Sriram's testimony. Finding that the timing of onset was not medically appropriate, the Court reaches no conclusion as to whether Td *can* cause ADEM. However, as per the decision in Johnson, it is not difficult to imagine that a similar scenario, where no alternative cause was identified and where the ADEM developed within a medically appropriate time frame, might result in an entirely different outcome under the current causation-in-fact analysis provided by the Federal Circuit in Althen.

Conclusion

Is it possible that the Td vaccination at issue caused Petitioner's ADEM? Anything is possible. But the Court cannot say it is more likely than not particularly given the lack of a medically reasonable temporal relationship between the vaccination and the onset of Petitioner's demyelinating disease.

Petitioner has not proved by preponderant evidence that her ADEM was caused-in-fact by the Td vaccination administered approximately eleven hours before onset. Therefore, no alternative remains but to **DENY** this petition. In the absence of a motion for review filed pursuant to RCFC, Appendix B, the clerk is directed to enter judgment accordingly.

IT IS SO ORDERED.

Richard B. Abell
Special Master