UNITED STATES COURT OF FEDERAL CLAIMS

OFFICE OF SPECIAL MASTERS

No. 97-0449V

(Filed: November 16, 2000)

* *		
BRENDA SCOTT-SHEPPARD, deceased, by her husband MURRVIN SHEPPARD,	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	
* *		

Ronald C. Homer, Esq., Boston Massachusetts, for the Petitioner,

David Terzian, Esq., United States Department of Justice, Washington, D.C., for Respondent.

ENTITLEMENT DECISION

I. Issue

The issue before this Court is whether Brenda Scott-Sheppard's Tentanus Toxoid ("TT") vaccination caused her demyelinating disease and resulting death. In this case, the Court ruefully decides against Petitioner's claim finding that despite biological plausibility, the facts do not support Petitioner's claims by a preponderance.

II. Facts

On April 27, 1995, Brenda woke up with numbness in her legs. At 6:41 a.m., her husband took her to an emergency room in Gainesville, Florida, where her condition began to deteriorate further. P's Ex. 8 at 4. (It was her third visit to the emergency room in three weeks.) The nurse recorded Brenda's own

words: "I've been feeling weak. N &V,[1] not getting any better." *Id.* The nurse also recorded that Brenda said she couldn't feel her feet. *Id.* Brenda began experiencing numbness in her back and legs, diffuse weakness, abdominal distention, and diarrhea. P's Ex. 8 at 4. A lumber puncture revealed an abnormal inflammation of the central nervous system. Tr. at 28. Despite various treatments such as steroids, there was no remarkable improvement in Brenda's neurologic condition. Over the next few months, her condition worsened to the point that she required full respiratory ventilation. *See, e.g.*, P's Ex.s 2, 19-59, 232-260. She was evaluated by a neurologist who noted that Brenda was "locked-in from progressive upper cord-brainstem disease presumed to be MS . . . appearance atypical." P's Ex. 2 at 891. Eventually, Brenda progressed to a virtually motionless state. P's Ex. 2 at 885-86. Except for her eyes and eyelids, Brenda was paralyzed. *Id.*

Eventually discharged home to be cared for by family members, Brenda was a quadriplegic with bilateral optic neuritis; she required chronic ventilator support through a tracheotomy; and, a Foley catheter for constant urinary drainage. P's Posthearing Brief at 8. Finally, Brenda tragically passed away in January of 1999, the consequences of respiratory failure.

Brenda's significant hospitalization on 27 April 1995, her tragic decline over the next few months, and her eventual death are clear events without a clear origin. Though Brenda's health was generally excellent prior to the onset of her condition, three probative events preceded her visit to the emergency room on 27 April 1995. As recorded by her husband in his affidavit, the first event happened, "[a]round April 17, 1995, Brenda told me she wasn't feeling well. She didn't seem ill, but when she didn't sleep well that night, I took her to the emergency room . . . [Brenda] was seen by Dr. Sally Samples who told me she suspected Brenda had an eating disorder." Third, an evaluation by Dr. Samples occurred on 19 April 1995. Brenda was admitted overnight to Alachua General hospital for rehydration and investigations. According to records, no abnormalities were identified and she felt better after rehydration. Finally, on 20 April 1995 during Brenda's appointment with Dr. Samples, she received what has been alleged as the cause or significant aggravation of her injuries, a TT vaccination.

III. Expert Medical Testimony

Petitioner's Expert, Dr. Derek Smith, M.D.[2]

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[1] Presumably, "N&V" is a medical abbreviation for nausea and vomiting. *See* NEIL M. DAVIS, MEDICAL ABBREVIATIONS: 8600 CONVENIENCES AT THE EXPENSE OF COMMUNICATIONS AND SAFETY, 6TH EDITION 121 (1993).

[2] Dr. Smith is a board-certified neurologist who practices neurology at Massachusetts General Hospital and Brigham and Women's Hospital in Boston Massachusetts. Tr. at 8. In addition, he is a Clinical Instructor in Neurology at Harvard Medical School. Tr. at 9. A practicing physician, Dr. Smith treats patients with multiple sclerosis and other demyelinating disorders. Tr. at 9. At present, Dr. Smith conducts research in the area of demyelinating disorders, especially with respect to the central nervous system, with grants from the National Multiple Sclerosis Society and the National Institute of Health. Tr. at 10. His neuroimmunology research is conducted at the Brigham & Women's Hospital in Boston, Massachusetts, a research laboratory that is as large or larger than the neuroimmunology laboratory housed at the National Institute of Health. Tr. at 12. He has published

Dr. Smith began his testimony by focusing the timing of his expert opinion: Brenda's demyelinating disorder did not pre-exist the TT vaccination. In fact, there was no evidence to suppose that the disorder existed at all prior to the TT vaccination. Tr. at 14. Ergo, though one could not be absolutely sure of the presence of the disorder (short of an autopsy), it was probable that the disorder did not pre-exist the vaccination.

Dr. Smith opined that Brenda "had the rapid onset of a fulminate central nervous system inflammatory process at some point between 4/25/95 and 4/28/95." P's Ex. 21 at 3. During this time period, Dr. Smith observed, on April 27, 1995, Brenda was readmitted to the hospital. A neurologic consult obtained on April 28, 1995, "notes the onset of left lower extremity weakness and bilateral lower extremity numbness over 3 to 4 days." *Id.* at 2, P's Ex. 15 at 19. In his view, Brenda suffered a neurologic injury (as opposed to an infectious process) as evidenced "by clinical examination, by spinal fluid analysis, and by radiographic analysis." *Id.* at 3. In all respects, Dr. Smith reasoned that Brenda's history was "consistent with an immune mediated injury of the central nervous system." *Id.*

According to Dr. Smith, the tetanus toxin, formally known as tetanospasmin, is elaborated by the bacteria clostridium tetani. Tr. at 16. And since the tetanus toxin is the mechanism that causes the tetanus disease (Tr. at 16), "the key to preventing tetanus is actually to prevent the elaboration of [the] tetanus toxin." Tr. at 17. Through delivery of a vaccine, a small amount of tetanus toxin is introduced "so that an immune response could be created" but without actually creating the tetanus disease. P's Posthearing Brief at 10 (citing to Tr. at 15-17).

By way of comparison, the science of preventing small pox was successful because of the use of a similar virus, cow pox. This "cross-reactivity" between the two similar diseases allowed for a presumably safer and right immune response to counter small pox. Tr. at 15-16. The process of creating the right immune response is similar to the process of the tetanus vaccine, although there are no other "cross-reactive" bacteria or viruses capable of producing an effective toxin against tetanus. Tr. at 16.

The importance of understanding how the tetanus toxin directs an immune response against the body was key to understanding what took place in Brenda's case. After introduction into the body, "the tetanus toxin is first taken up by peripheral nerves. It is then transported by the peripheral nerves in what's called retrograde transport within the neuron and eventually crosses a synapse and is transported into the central nervous system." Tr. at 17. In a tetanus vaccination,

> a small amount of the tetanus toxoid is being taken up by peripheral nerves. . . . [A]t the same time that this is occurring, there is an immune response against this tetanus toxoid that is designed to occur. That's the point of giving the vaccination. So it is known that the tetanus toxoid can cause what's called Guillain-Barre syndrome. And I believe that the

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several articles with respect to the immunology of central nervous system demyelinating disorders and a book chapter with respect to the epidemiology of multiple sclerosis. Tr. at 10.

> way that occurs is that the immune response which is directed against the tetanus toxoid is then directed against the nerve when the tetanus toxoid is taken up by the peripheral nerve.

Tr. at 17-18.

In addition to the tetanus toxoid being taken up by the peripheral nerve, Dr. Smith noted that it is also taken up by the central nerve. This is so not because of tetanus passing the blood brain barrier; rather, it is because of direct synoptic connections–described as "retrograde transport"–between the peripheral nervous system and the central nervous system. Tr. at 21-22. And this is how the disease tetanus is known to occur. Tr. at 21.

Taking this model to its next logical step, Dr. Smith analogized, "[I]f this is the mechanism for how tetanus causes Guillain-Barre syndrome, [a peripheral nervous system demyelination] and tetanus is taken into the central nervous system–which we know it is–then it is equally plausible that this is how it would cause central nervous system demyelination." In Dr. Smith's view, this would biologically explain how Brenda's own immune response to the tetanus toxoid could be directed toward her central nervous system.[3] One reason for this biological explanation lay with the fact that tetanus, "by its nature, targets the nervous system and is taken up by the nervous system. . . . [In Brenda's case, the] tetanus was taken up in the peripheral nervous system, transported to the central nervous system through this retrograde transport which we know occurs, and thereby, the immune response against the tetanus toxoid was also directed also into the central nervous system." Tr. at 52. To support this conclusion, Dr. Smith explained in detail, this theory he felt indicated by evidence in the medical records.

First, when she presented initially, Barbara had a viral illness and evidence of gastroenteritis. She had nausea, abdominal discomfort, anorexia, and she complained of fever. Tr. at 23. What was unusual was that a CBC revealed that Brenda's microphages had migratory behavior clearly indicating that she had activation of her immune system. Tr. at 22-23. "Microphages are cells [whose] primary function is to travel through the body and take up proteins in the situation of an infection, and then, present those to the immune system so that an immune response can be generated." Tr. at 24. Because Brenda was already in a hyperimmune state, the crux of Dr. Smith's opinion was that she would get a much more pronounced response to the vaccination than would normally be the case in that situation. Tr. at 24-25. The reason for this is that where the microphages were already acting in an efficient manner, and since the tetanus vaccination was designed to elicit an immune response to begin with, naturally the response would be stronger. Tr. at 24-25. According to Dr. Smith, "[W]e know that people with infection can have a much stronger immune response to a vaccination. And . . . for that reason, the Department of Health and Human

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[3] Dr. Smith did not necessarily disagree with Brenda's death certificate listing the cause of death as multiple sclerosis (MS) since MS "is something of an umbrella diagnosis for a number of central nervous system demyelinating disorders, and she clearly had a central nervous system demyelinating disorder that was chronic. . . ." Tr. at 44.

Services recommend[s] that any patient with a moderate fever or, specifically, a gastrointestinal problem not be immunized." Tr. at 22-23.[4]

Secondary to the first point, an "infection within the gastrointestinal system would actually dampen the normal suppressive function gastrointestinal immune system," Dr. Smith concluded that Brenda's symptomology reflected not simply a decreased ability to elicit a normal gastrointestinal response, but an inability to have an suppressive effect at all. Tr. at 26. In fact, "the suppressive effect[5] of it has been shut off" as compared to a healthy individual. Tr. 27. Because of this fact and the symptomology indicated, Dr. Smith reasoned that it was logical to conclude that the immune response to the tetanus vaccination in Brenda's case would have been much stronger.

Dr. Smith's second point concerned the chronic destruction of Brenda's central nervous system and myelin sheaths by her own immune system. Specifically, he addressed a scientific justification for why some individuals progress to a chronic course. First, the reason a vaccination is designed to induce an immune response is because the immune system has a memory. Tr. at 33. Dr. Smith explained that this is so that "the next time there's an exposure, the immune response can happen in a much more rapid fashion." Tr. at 33.

Second, another mechanism by which an acute immune response can become a chronic immune response is the phenomenon described as epitope spreading. This phenomenon, important in the case of vaccinations as shall be seen, serves to describe how an immune response initially directed against one protein may become directed against another protein in close proximity. Tr. at 34. The epitope is a small part of a protein; it is a sequence of amino acids. *Id.* The antigen presenting cell (a macrophage) is a component of the immune system. Tr. at 35. When the APC breaks down a suspect protein into the small epitopes, it then presents them on a separate molecule, a major histo compatibility (MHC)[6] molecule so that a T cell will recognize the proper response to that protein. Tr. at 34. The T cell is the memory component of the immune system and serves future responses. Once the response occurs, the immune system will continue to respond as long as it perceives–through the antigen presenting cells--that the protein that it recognizes is present and associated with an infection. Tr. at 36.

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[4]And, of some probative value, the Court notes that the guidelines published by the CDC and Respondent HHS fail to reference any specific reason for such a recommendation. Ergo, it is reasonable to infer that Respondent is unsure whether to administer a vaccination during such a weakened state since the vaccinee might either have her condition exacerbated or she might react directly to the vaccine itself.

[5] The Court accepts *arguendo* and Respondent's expert does not contradict, the fact that the suppressive effect of the gastrointestinal system is well accepted in medicine. The "phenomenon called oral tolerance that's been recognized for a long time which is that if the gastrointestinal system is exposed to a particular antigen or protein, then the immune response after that point in time to that particular protein or antigen will be less than it would be otherwise." Tr. at 27.

[6]This molecule, known as major histo-compatibility complex, is the protein on the antigen presenting cell that actually presents the epitope. *See* Tr. at 34-35.

For instance, in a tetanus vaccination, the antigen presenting cells will take up the tetanus toxoid, process its protein sequence, and then, in conjunction with the MHC molecule, present the epitopes of the toxoid protein to a T cell. The T cell will be able to recognize a part of the tetanus toxoid and this is how the immune system will remember or recognize the tetanus toxoid in the future. Tr. at 35. This process is "self-limiting" in the sense that the normal response does not extend to attacking the body's own cells. Tr. at 32, 39.

While it was thought at one time that T-cell response was highly specific to an antigen epitope, Dr. Smith testified that recent studies within the past 10 years demonstrated two mechanisms thought to explain why a T cell might attack its own system. Tr. at 38-39. First, a T cell response against an epitope of one protein might be directed against a very different epitope from a different protein. And this is known as "degeneracy". Tr. at 38. Second, "epitope spreading" occurs when a primary epitope

> induces an immune response, a local immune response. And the T cells then elaborate . . . [hormone-like] . . cytokines into the local environment where the immune response is occurring. . . . They recruit other immune system components to the location, and the destruction that occurs as a result of this will release tissue or proteins from the local tissue . . . that [protein from the local tissue] can be taken up again by antigen presenting cells, and presented to other T cells that are in the local environment. And this is how an immune response against one protein can become directed against multiple proteins.

Tr. at 39-40. If there is a strong initial immune response, and it has sufficient momentum, Dr. Smith postulated that it could lead to epitope spreading, which in turn, could lead to demyelination and chronic immune response. Tr. at 40.

In Brenda's case, Dr. Smith focused his discourse on two conclusions. He thought it clear and definite that epitope spreading had occurred because of the tetanus toxoid and this explained Brenda's demyelinating condition. He also opined that the severity of her immune response was a secondary effect, *based on medical probablity,* of the tetanus toxoid. Tr. 42-43. Dr. Smith also viewed Respondent's expert's opinion--that Brenda suffered from Devic's Disease[7]–as consistent with his own opinion that Brenda was suffering a form of MS. In other words, because Devic's Disease was a form of MS, either or both are sequelae to a tetanus toxoid. Tr. at 49.

Finally, what this Court perceives as a critical distinction is how Dr. Smith could opine on Brenda's tetanus vaccination as the intiation of her demyelination disease: "In somebody who has already had the diagnosis established, it's known that the occurrence of a viral illness increases a risk of an attack of the

[7] Dr. Smith felt that Brenda's symptomology reflected MS but was also compatible with Devic's disease. Tr. At 48. However, for Dr. Smith, there "was a question of cerebellar lesion on her MRI scan that would not normally be considered part of Devic's disease . . . [and,] she developed a movement disorder . . . of her left upper extremity." Tr. at 50. The movement disorder would not be consistent with a Devic's disease diagnosis.

disease. But . . . in somebody who has no history of demyelinating illness, a viral illness is not thought to be a significant risk factor for developing the disease." Tr. at 65. In Brenda's case, "one would not think that because she had a viral illness, there was the cause of her demyelinating disease." *Id.* Moreover, the virus would not have followed the route of the tetanus toxoid for two reasons: first, it was in Brenda's gastrointestinal system, and second, unlike the tetanus toxoid, the virus wasn't targeting her nervous system.

Respondent's Expert, Dr. Kottil W. Rammohan, M.D.[8]

Dr. Rammohan, Respondent's expert, admitted that of the medical abstracts he had reviewed, he had never seen a case that came close to matching Brenda's condition. Tr. at 76. However, he concluded that Brenda died of classical Devic's[9] disease, "a variant of multiple sclerosis in the sense that it's a demyelinating disorder of the nervous system, but it's a very different disease than the standard MS . . . ." Tr. at 77. What lead him to this conclusion were several facts in the record that he immediately melded with his theory of why Brenda's condition took the course that it did.[10]

"A patient with Devic's disease will develop . . . optic neuritis that eventually leads to blindness, as it did in Mrs. Scott-Sheppard. She was almost blind by the time she died." Tr. at 77.
"And it also causes demyelination, which is not just simple demyelination." Tr. at 77. In his opinion, this distinguishing factor was important to diagnosing Brenda's condition accurately for the

> standard demyelination that occurs in multiple sclerosis is where the axons are relatively spared, and the myelin, which is around the axon, the insulation, is stripped. But what happens in Devic's disease is that an actually destructive lesion of the spinal cord occurs. Virtually, the whole spinal cord falls apart. It's not simply just the myelin falling apart. And when that happens, what you find is an irreversible destruction that there is no recovery from, unlike multiple sclerosis, where you can get an attack and some partial or complete recovery. Patients with Devic's, true Devic's disease do not recover simply because it's just a devastating disease, and the entire spinal cord is damaged, and the disease

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[8] Dr. Rammohan is a neurologist who is board certified in neurology, internal medicine, physical medicine, rehabilitation, and psychiatry. He received his medical training from the Madras Medical College, University of Madras, Madras, India. He completed six years of clinical training evenly divided between internal medicine and neurology. Of academic note, he is a Vice Chairman and Associate Professor in the Department of Neurology at Ohio State University and he is the Director of both the Neuroimmunology Laboratory and the Multiple Sclerosis Center for that institution. Dr. Rammohan has authored or co-authored over 28 articles and numerous abstracts in his field. Presently, his practice of seeing over 3000 patients with another physician means that his time is spent in *clinical practice*. He testified that 99% of his practice is MS. *See* Tr. at 72.

[9] Dr. Smith felt Devic's disease an acceptable "designation" for Brenda's condition since it tended to describe a majority of her condition. Tr. at 49.

[10] Because of Dr. Rammohan's coherent explanation, the Court herein quotes extensively from his testimony rather than attempting to paraphrase his opinion.

progresses fairly relentlessly and, in this case, it marches right up the spinal cord all the way into the brain stem, and she became . . . locked in, which basically means that she was still awake and alert, but completely unable to [respond] because she was totally paralyzed. And at that point, she also needed the support of [a] ventilator.

Now, one of the things in her [case] that distinguished this from the standard multiple sclerosis, [was] first the course, and secondly, patients with multiple sclerosis have many, many lesions on the brain [as revealed] by magnetic resonance imaging of the brain. Patients with Devic's typically do not have any lesions in the brain, or a few lesions in the brain. And if you look at [Brenda's] . . . MRI reports . . . she had just a couple of lesions in the brain. She had nothing like what one would see with multiple sclerosis.

In multiple sclerosis, you can see 40 or 50 placks. She had none. She had one tiny one in the cerebellum which was thought to be, in a later interpretation, an artifact. There was maybe a couple of spots in the basal ganglia. But if you put up her MRI of the brain, and asked without any history, most people, one look at it and say, 'oh, that looks like typical MS.' It was not.

And then, if you look at the spinal fluid that she had, that was again in keeping with what one would see with Devic's disease. Since it's a destructive disease of the spinal cord and optic chiasm, what you find in the spinal fluid is an extremely inflammatory picture. What you see in the spinal fluid in MS is about zero to five cells. The cell count doesn't go up. But in Devic's, cell count is in the hundreds. And her white count in the spinal fluid was 150, or somewhere in that range.

The protein in the spinal fluid never goes up in multiple sclerosis. It hardly goes up because it is really not a major problem. The igg in the spinal fluid goes up, but not the protein. In her spinal fluid, protein was elevated.

Tr. at 77-79. When asked for the etiology of Devic's, Dr. Rammohan replied, " Like MS, we don't know. We don't know what causes MS. We don't know what causes Devic's disease. Even though we consider it in the umbrella, like Dr. Smith said, of demyelinating diseases of the nervous system, everybody recognizes that if you have a patient with Devic's disease, it's a whole different ball game compared to what you would do with regards to an MS patient. It's an aggressive disease that's lethal." Tr. at 79-80.

Apart from Dr. Rammohan's testimony on what condition Brenda died from, and more to the point, what caused that condition, he also disagreed with Dr. Smith's theory. While he acknowledged that Dr. Smith's theory was possible, he thought it unlikely. The Court does not find it probative to discuss the differences in the two opposing theories since, as shall be seen, the decision does not turn on deciding which theory is more credible.

Returning to the heart of what this Court sees as the disagreement between the experts, Dr. Rammohan found troubling, that part of Dr. Smith's opinion that tetanus toxoid and tetanus toxin are essentially the same thing. For Dr. Rammohan, who categorically disagreed, the two are quite different, to wit:

> if somebody recovered from tetanus, they have no immunity against tetanus. They have to be immunized to get immunity to the tetanus. So you cannot give small amounts of toxin to produce immunity. What you have to do is to modify the toxin, and that modification process is what converts a toxin to a toxoid.
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> And basically, what is done is you take the toxin and you treat it with formaldehyde. And when you treat it with formaldehyde, it cross-links all three chains. So when all three chains are cross-linked, you can no longer attach this toxin to the cell or internalize the light chain into the cell.
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> So that diagram that you had here of how the toxin is taken up by the nervous system simply does not happen with a toxoid. It cannot happen with a toxoid. In fact, every batch of toxoid that is produced is tested by injecting mice to make sure they don't get tetanus because if you have one molecule of the toxin attaching to the nervous system and going into the nervous system, that toxin is a bad batch. It will cause death because the amount of toxin you need to cause death is so small that one has to be careful that the toxoid is devoid of toxin. [Where Dr. Smith testified that] the toxoid attaches to the nervous system does not occur. It cannot occur. It should not occur. If it happens, that's not acceptable.
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> Now, one of the things that is known about tetanus toxoid is it's probably the best antigen that we have. And if you, in fact, look at the search that I did when we compared, you know, tetanus toxoid and multiple sclerosis, there were 14 articles. So I thought, oh, boy, there must be 14 cases reported. But if you look at those papers, what you find is that they were using tetanus toxoid as a housekeeping protein to check how effective the immune response in the patients were. In other words, it was just a marker to see if the patient's blood cells are responding properly.
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> So tetanus toxoid by itself does not cause multiple sclerosis, and tetanus toxoid has nothing to with entering the nervous system. It shouldn't. . . . Not even one molecule of the toxoid is picked by the nervous system, peripheral or central nervous system. The toxoid cannot and should not be picked up by the nervous system.

Tr. at 80-82.

The short of the matter is that Dr. Rammohan's opinion was that the whole premise of Dr. Smith's

hypothesis is completely wrong, based on the fact that tetanus toxoid is never even taken up by the central nervous system. Even if it were, the central nervous system and peripheral nervous system are "not similar from an immunological standpoint" (Tr. at 91) despite the fact that Devic's disease (which attacks the central nervous system) and diseases like Guillare Barre Syndrome (which attacks the peripheral nervous system) are both demyelinating diseases. *See, e.g.*, Tr. at 124. In addition, when Brenda received the tetanus toxoid, the fact that she had a gastrointestinal problem was irrelevant in the sense that it would not "augment or retard the immune response that she would have gone through." Tr. at 83.[11]

And finally, Dr. Rammohan testified that, "There's absolutely clear evidence that Scott-Sheppard went through something that took her to the emergency room a week or 10 days *before* she got the tetanus toxoid. My speculation would be that if there is a link of something to what subsequently followed, that

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[11] The Court notes an apparent contradiction at this point that was somewhat reconciled by Dr. Rammohan,

> Now, it's possible that if you're in the midst of an infection and if you're immunized, that's probably not a good thing to do because, you know, you may modify the immune system because there is something else happening that's already revved up the immune system, and that's probably the recommendation of the Institute of Medicine saying if somebody already is suffering from a fever and an infection, don't immunize at that time. That's not a good thing to do because you're adding insult to injury by bringing in something that can also make the person not well.
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> But I'm not aware of anything that suggests that if you have a viral infection, and if you give a vaccine, that the vaccine immunization is enhanced or retarded. I don't know that there are studies that I have seen that suggested something like that.
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> . . . .
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> You can go through an acute infection with measles, and it can, during the recovery process when the immune system is fighting the measles virus, you can see damage occurring in the nervous system. And that is called acute disseminated encephalomyelitis, which can be a violent disease. It's usually a monophasic illness, meaning it occurs one time. It doesn't keep recurring again and again.
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> And that is well accepted that viruses have antigens that mimic the central nervous system. Dr. Smith talked about molecular mimicry. That's a good example. If you look at measles virus, there's a protein in measles virus called myelin -- actually, matrix protein. Matrix protein of measles virus shares homology to the myelin basic protein that Dr. Smith was talking about.
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> So if you responded as you normally would when you get an infection of measles or the vaccine with measles, you could theoretically have a situation where in the process of responding to that particular protein of the virus, you generate cells that are autoreactive, meaning since the brain has myelin basic protein, and since there is homology between myelin basic protein and metric protein, one could theoretically have the situation.
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> Having said that, I must also point out that, as much homology as there is, that *actual occurrence of this problem is rare.* Even though viruses share homology to proteins in the brain, this is not something that we see every day. It's a rare event after an infection.

Tr. at 89 (evidence added).

illness might well have been the beginning of it all. In other words, when she went to the emergency room, that might have been the beginning of her neurological problem because there are many things in her [case] that suggests that." Tr. at 88 (emphasis added.) This was so because during Brenda's ER visit prior to her immunization, she used the word "weak" to describe her own condition. *Id.* But even if that were not the beginning of Brenda's problems–that is, assuming her condition began after the suspect vaccination–Dr. Rammohan queried whether "the virus that Dr. Smith talked about that she went through have caused all of the problems that she subsequently went through." *Id.*[12]

IV. Program Standard and Analysis

This Court interprets the Vaccine Act by looking to the plain language of the statute. In order to find a true interpretation, this may require either a strict, moderate, or broad construction depending on the text, context, and plain meaning of the words used. The usual canons of interpretation apply. So, to label a requisite approach as "strict" or "broad" is to miss the mark and err in interpretation.

In the case at bar, Petitioner has not alleged a Table injury under the plain terms of the statute. Therefore, he is required to prove causation-in-fact under the alternative method provided by the Vaccine Act. *See* 42 U.S.C. § 300aa–(b)(1)(B) (1995). This requires proof by a preponderance of the evidence that Brenda's tetanus vaccination was the cause of her injuries.

The "preponderance of the evidence" standard is the traditional standard of proof in civil and administrative proceedings. *Steadman v. SEC*, 450 U.S. 91, 101 n. 21 (1981). In *Steadman*, the Supreme Court noted that "evidence . . . of a poor quality--irrelevant, immaterial, unreliable and nonprobative--and of insufficient quantity--[is] less than a preponderance." *Steadman,* 450 U.S. at 102. As Justice Harland has explained it, a preponderance of the evidence standard "requires the trier of fact 'to believe that the *existence* of a fact is *more probable* than its nonexistence before [the trier of fact] may find in favor of the party who has the *burden to persuade* the [judge] of the fact's existence.' "*In re Winship*, 397 U.S. 358, 371 (1970) (Harlan, J., concurring) (*quoting* F. JAMES, CIVIL PROCEDURE 250-51 (1965)). The preponderance of the evidence standard has also been explained as "the greater weight of the evidence, evidence which is more convincing than the evidence which is offered in opposition to it." *Hale v. Department of Transp., F.A.A.*, 772 F.2d 882, 885 (Fed.Cir.1985). Accordingly, as a corollary proposition, it follows that if the "evidence appears to be equally balanced, or if it cannot be said upon which side it weighs heavier, then plaintiff has not met his or her burden of proof." *Smith v. United States*, 557 F.Supp. 42, 51 (W.D.Ark.1982), *aff'd*, 726 F.2d 428 (8th Cir.1984).

In this case, the Court finds that Petitioner has not met his burden of production or persuasion. At the outset, the Court notes that this is the second such "cause-in-fact" case to allege as sequela to a Tetanus

[12] "[T]he trigger might have been a viral infection, and it's very well-known that Guillain-Barre syndrome can be caused by viruses. Bacterias can do that, campylobacter. It's a bacteria. Campylobacter causes GBS. It's well-known. It's quite accepted." Tr. at 90.

vaccination, a central nervous system demyelinating illness or a significant aggravation of an existing central nervous system demyelinating illness.

As a matter of clarification, it may be helpful to state what the Court is not deciding here. First, the Court is not making a decision based on the validity of Petitioner's expert medical theory. As shall be seen, it is simply not necessary to the decision. For even if Petitioner's theory were valid, *arguendo*, this Court finds that the reasoning or methodology employed by Dr. Smith does not properly apply or fit the instant facts. His opinion may fit another set of circumstances but that case is not before the Court. This is not based on the Court's expertise; rather, it is that the Court finds Respondent's expert's opinion more convincing *in the context* of its relationship to the facts.

This brings the Court to the fact among a core of facts which made the difference–the apparent illness that began prior to vaccination. As in any petition for compensation, a petitioner must show that she received a Table vaccine, that she alleges an injury which could be expected to result from that vaccination, that the injury occurred temporally within a medically acceptable process, and that the medical theory is capable of accounting for *all* of the relevant facts, by a preponderance of the evidence.

In order to meet his burden, Petitioner relies in part, on a recent case decided by this Court, *Helen Rogers v. HHS*.[13] To the extent that *Rogers* presents a similar theory by Petitioner's medical expert, Dr. Smith, the Court can and does accept such a possibility. However, the facts in that case are completely inapposite to the instant facts. In *Rogers*, the special master articulated a rational connection between the facts found and the decision she made. But to restate, the Court does not find that connection here. In contradistinction to the instant plaintiff, the *Rogers* petitioner met her burden of going forward with this core set of facts. Indeed, Helen Rogers's health was "unremarkable with the exception of an incident of vertigo that occurred . . . three years prior to her tetanus toxoid inoculation and an extended bronchial infection about three months before the inoculation."[14] Moreover, the plaintiff in *Rogers* supplied medical records wherein the clinical observations of the treating physicians reveals that they believed that Mrs. Rogers injuries were due to her TT vaccination. Unlike Brenda, The Rogers plaintiff did not exhibit a viral-type event prior to vaccine administration. (This is not to say that Brenda had a virus but in the words of her own expert, a viral type illness that could just as likely have caused her injuries.)

Per contra, in the case *sub judice*, none of Brenda's treating physicians (at the relevant times in question) stated that they suspected a causal relationship between her tetanus toxoid shot and her demyelinating disease. In contrast to *Rogers*, the instant petitioner reported to the emergency room on 17 April 1995, approximately 10 days *prior* to the onset of her symptoms and 3 days *prior* to her TT vaccination. It is that time frame that Petitioner must account for in her theory. This Court finds that she did not because the facts of her case cannot be separated from the temporal proximity of the viral-type illness.

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[13] No. 94-0089V, 2000 WL _____(Fed. Cl. Spec. Mstr., Decision reissued Sept. 1, 2000).

[14] *Id.*

Dr. Smith admitted that it was possible that Brenda had a viral illness before her vaccination. Dr. Rammahon posited that this viral illness could be the likely cause. This is a critical and distinguishing fact.

According to her husband, "Around April 17, 1995, Brenda told me she wasn't feeling well. She didn't seem ill, but when she didn't sleep well that night, I took her to the emergency room at North Florida Regional the following day. She was seen by Dr. Sally Samples who told me she suspected Brenda had an eating disorder. Dr. Samples gave Brenda an appointment for April 20, 1995 at her office and recommended she get some counseling for her eating disorder. Dr. Samples ordered some tests and they all came back negative. . . . Brenda *still didn't feel well that day* so I insisted she get another opinion and took her to Alachua General Hospital." (Emphasis added.)

Presumably, Brenda's husband is describing a condition that had persisted from April 17, 1995 up to the day of the TT vaccination, April 20, 1995. The totality of those days refers to a condition that, from Brenda's own impressions, reveal a more likely conclusion that her demyelinating condition began and pre-existed her TT vaccination. That her TT vaccination might have significantly aggravated her condition causing an acute autoimmune response is quite possible. Indeed, it is a perplexing question but a "possibility" is not the standard. To say that Brenda was sick and then became worse does not *ipso facto* make for significant aggravation. Rather, without a fact to indicate that the TT vaccination was a substantial factor in Brenda's injuries, then it is near impossible in the instant case to discern what caused or substantially aggravated her condition or made her life significantly worse. Ergo, the legal burden does not shift to Respondent. Nothing in the records indicates that Brenda's condition was significantly aggravated because of the vaccine as opposed to the expected course of Devic's disease or some type of viral illness, or that the viral illness itself was aggravated.[15] Dr. Rammohan's conclusion that Brenda suffered from Devic's disease, a condition that also presents with an acute demyelinating illness is just as probable. Dr. Smith opined that the TT vaccine worked in conjunction with the TT vaccine to cause her illness. Petitioner's Closing Argument at 30. With those explanations and the events occurring around or after April 17, 1995, the Court is presented with two opposing theories that, in the Court's mind, seem equally credible, but with facts that militate against Petitioner's theory.

The Court finds that the facts in this case, in contrast to *Rogers*, are not of the quality or weight that could give rise to a favorable inference for Petitioner. Looking at the totality of the record, the possibility that Brenda could have had an acute hyperimmune response to the vaccination is very real. In fact, it may very well have happened. And in a case where two experts opine from the ends of the earth, their antipodal theories standing in equipoise before the Court, a special master is forced to choose one over the other. Without factual differences, such a decision is necessarily the embodiment of subjectivity. In other words, on what basis does the special master choose in such a situation? In this case, as the Court has noted, Respondent's expert married the facts far more cogently than Petitioner's expert. The Court found

[15] The Court notes that Dr. Smith didn't think the viral illness could have caused Brenda's condition, whereas Dr. Rammohan affirmatively testified that it could. Because of the number of patients Dr. Rammohan treats in this area, the Court is constrained to his opinion.

Respondent's expert's testimony far more persuasive and probative in accounting for the facts. But Respondent's proffer of an expert's opinion is of no great moment for, even assuming the validity of Petitioner's theory, the Court finds that Petitioner's medical expert has not carried the burden required in this case. It may very well in another case, but here, the preponderance standard simply has not been met.

V. Conclusion

If Respondent had not presented any medical expert to contradict Petitioner's expert, this Court would still be compelled to deny compensation. Petitioner's burden has not been met and it is unnecessary to discuss the onus of persuasion where there is not a sufficiency of facts in the record to meet Petitioner's theory. To restate, Brenda Scott-Sheppard must be denied entitlement for two reasons, either of which independently impel the Court to deny entitlement. First, assuming her theory plausible, the facts do not marry with that theory as explained in the two days of weakness that existed prior to shot. And even if Petitioner's theory could account for all her facts, the Court is more persuaded by Dr. Rammahon who seemed to have a better grasp of the events that appeared to have precipitated Brenda's injury.

It is, perhaps, apropos to point out that in any decision rendered by this Court concerning the issue of causation, conclusions of a special master are not scientific conclusions and are not substitutes for medical science. This Court is bound to a statute that requires weighing of evidence under a preponderance standard. It delivers public policy for the case presented. Ruefully, the Court is aware that Mr. Sheppard has suffered and will continue to suffer from the tragic events that lead up to his wife's eventual death. And if this Court had the power to compensate him, it would have done so in two paragraphs. But in this case, it is not possible to determine by a preponderance, the cause of Brenda's condition.

For the reasons stated above, this Court finds that Petitioner was unable to establish that Brenda's death was associated or derived from a vaccine-related injury. In the absence of a motion for review filed pursuant to RCFC, Appendix J, the clerk is directed to enter judgment accordingly.

IT IS SO ORDERED.

Richard B. Abell
Special Master