OFFICE OF SPECIAL MASTERS

No. 94-0089

(Filed: June 6, 2000)
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HELEN D. ROGERS	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Ron Homer, Boston, Massachusetts, for Petitioner.

R. Lynne Harris, United States Department of Justice, Washington, DC, for Respondent.

DECISION

French, Special Master.

This case arises under 42 U.S.C. §300aa-1 et seq., the National Vaccine Injury Compensation Act of 1986.[1] Petitioner filed her petition on February 15, 1994, claiming that as the result of a Tetanus Toxoid vaccine (hereinafter sometimes TT) administered on February 15, 1991, she sustained a post-vaccinal myelitis, an autoimmune demyelinating disease with permanent neurological sequelae. Respondent defends by arguing that her condition is consistent with Multiple Sclerosis, (hereinafter MS), a factor unrelated to the

[1] 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(modified as amended at 42 U.S.C.A. §§ 300aa-1 through - 34 (West 1999 & Supp. 1998)). References shall be to the relevant subsection of 42 U.S.C.A. § 300aa.

vaccine.[2]

Procedural Background

A hearing was held in Washington, D.C. by telephone conference call on August 21, 1997. Inasmuch as the facts in this case were not contested, the hearing was confined to the testimony of medical experts. Petitioner presented the testimony of Alan Scott Levin, M.D., J.D. Respondent presented the testimony of Barry G. W. Arnason, M.D. As will be explained hereafter, a supplemental hearing would be required.

On September 17, 1999, the court issued a decision denying Petitioner's claim. Petitioner filed a motion for reconsideration, and after consideration of Petitioner's arguments and a careful review of Respondent's response in opposition, the court was convinced that the court should permit presentation of further evidence to consider additional evidence and argument relative to research findings that might possibly change the outcome of this case. An Order to Vacate the court's decision of September 17, 1999 was filed on October 12, 1999 This decision is being issued based on additional expert testimony that has been provided on the key issue in this case, that is, whether the vaccine in question (TT) is capable of causing MS or MS-like symptoms.

A supplemental hearing was held by telephone conference call on January 27, 2000. Dr. Derrick Smith testified for the Petitioner; Dr. Roland Martin testified on behalf of the Respondent, and Dr. Barry Arnason, for Respondent, filed supplemental commentary. The additional evidence convinces the court that a preponderance of the evidence permits a finding in favor of the Petitioner.

FINDINGS OF FACT

The following facts describe the claimant's medical history and clinical course and are so found. Helen Rogers was born on August 29, 1954. The history of her general

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[2] MS is an inflammatory myelopathy, and is considered the most common of a number of immune-mediated demyelinating diseases of humans. Immunologic Disorders In Infants & Children, E. Richard Stiem, at 904 (4th ed. 1996) "Although the pathogenesis of such disorders is unknown, it is thought that they may result when toxins, medications, or viruses trigger an autoimmune response. The nature of the immune response and the particular targets attacked produce distinctive patterns of immunopathology and associated clinical findings." Id. at 890. Petitioner's expert explained that MS is not considered a disease entity but a syndrome, or constellation of symptoms that define a disease process--"a pigeon hole into which we place patients and the clinical outcome of the disease process is unique to the patient." Transcript of proceedings of August 21, 1997 (hereinafter Tr. I) at 28, 29, 64.

health prior to her February 15, 1991 Tetanus Toxoid immunization is unremarkable with the exception of an incident of vertigo that occurred about three years prior to her tetanus toxoid inoculation and an extended bronchial infection about three months before the inoculation.

On February 15, 1991, Ms. Rogers received a Tetanus Toxoid vaccination as part of a pre-employment physical examination. The next day, she noticed that she had cold feet and had to wear thermal socks to keep her feet warm. Two days later, on February 18, 1991, Petitioner began a new job. She began to notice numbness and tingling in her feet, the left foot more than the right, and that she appeared to move with less agility than usual. Petitioner had been active in athletics, and at the time of her Tetanus Toxoid inoculation, she was serving as basketball coach for her daughter's team. Petitioner's Exhibit (hereinafter P. Ex.) 20 at 1. During March and April, her feet continued to tingle. Her ankles and calves began to swell and she had nocturnal cramps in both legs. Her job required her to stand on a concrete floor for long periods of time, and she believed her leg and foot problems were related to the new job. Her condition, however, continued to worsen, the tingling and numbness radiating up through both legs but greater in the left leg. She delayed seeing a doctor because she did not want to take time off so soon after beginning a new job.

On April 16, 1991, Ms. Rogers was examined by Dr. James Matthews, a specialist in internal medicine. He diagnosed probable neuritis or sciatica in the lower extremities and treated her with Voltaren without any apparent success. P. Ex. 6 at 2; P. Ex. 18 at 14. At her second visit, on May 1, 1991, Dr. Matthews began to suspect the presence of a neuropathy and referred her to Dr. Reuben Richardson, a neurologist at the Alabama Neurological Clinic. P. Ex. 11 at 1; P. Ex. 18 at 18-20. Dr. Reuben Richardson saw her the next day. According to Dr. Richardson's medical record of May 2, 1991, he suspected that Petitioner was suffering from a possible reaction to the Tetanus Toxoid, but he wanted to rule out Multiple Sclerosis (MS). P. Ex. 7 at 97. Ms. Rogers continued to see Dr. Richardson on the following dates: May 16, May 20, June 27, and July 19, 1991. According to his medical notes of July 19, 1991, Dr. Richardson had come to the conclusion that she was suffering from an autoimmune demyelinating disorder (hereinafter, sometimes AIDM), and that "it certainly was MS-like." P. Ex. 7 at 11; P. Ex. 18 at 31.

On August 19, 1991, Petitioner was examined by Dr. John Whitaker, a neurologist and Professor and Chairman of the Department of Neurology, at the University of Alabama, Birmingham School of Medicine. He was of the opinion that Petitioner was suffering from some form of post-vaccinal myelitis[3] (P. Ex. 2 at 2) and that her condition was probably a demyelinating process secondary to her tetanus injection. P.Ex. 7 at 14; P. Ex. 6 at 11; P. Ex. 18 at 38. By February 1992, Dr. Richardson had reached a similar

[3] Myelitis is defined as "inflammation of the spinal cord." Dorland's Illustrated Medical Dictionary, at 1086 (27th ed. 1988).

conclusion--that she had sustained a post-vaccinal "myelopathy."[4] P. Ex. 7 at 18.

Ms. Rogers was treated with a variety of medications, but after an initial improvement, she worsened. By November of 1991, she was in a wheelchair, suffered ascending paralysis, paresthesias, incontinence of the bowel, and by 1992, optic neuritis. P. Ex. 5 at 75. At present, she is wheelchair-bound and dependent on her family for all her personal care and hygiene. P. Ex. 20 at 2.

STATUTORY REQUIREMENTS

Petitioners may establish causation in one of two ways.[5] First, Petitioner may demonstrate what is commonly referred to as a Table case. The Vaccine Table lists vaccines covered by the Act and certain injuries and conditions that may result from the vaccines. § 14. If the court finds that a person received a vaccine listed on the Table and suffered the onset or significant aggravation of an injury listed on the Table, within the prescribed time period, the Petitioner is entitled to a presumption that the vaccine caused the injury. §13 (a)(1)(A). The Petitioner must then show that the injury for which Petitioner seeks compensation is a sequela of that Table injury. §14(a)(I)(e). Respondent may rebut the presumption of causation by presenting evidence that the injury or condition was due to factors unrelated to the administration of the vaccine. §13(A)(1)(b). Petitioner in this case cannot establish an on-Table case for the reason that the claimed injury is not listed in the Table of covered injuries.

Even though Petitioner is unable to establish an on-Table case, she may establish causation by proving that the vaccine actually caused the alleged injury. Actual causation requires proof of a "logical sequence of cause and effect showing that the vaccine was the reason for the injury." Strother v. Secretary of HHS, 21 Cl Ct. 356, 370 (1990), aff'd without opinion, 950 F.2d 731 (Fed. Cir. 1991). The mere temporal relationship between a vaccination and the injury, and the absence of other apparent etiologies for the injury, are patently insufficient to prove actual causation. Wagner v. Secretary of HHS, No. 90-1109V, 1992 WL 144668, at *3 (Cl. Ct. Spec. Mstr. June 8, 1992). Rather, Petitioner must show a medical or scientific theory causally connecting the vaccination and the injury. Strother, 21 Cl. Ct. at 370 (citing Hasler v. United States, 718 F.2d 202, 205-06 (6th Cir.

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[4] A Myelopathy is a general term denoting functional disturbances and or pathological changes in the spinal cord. The term is often used to designate a nonspecific lesion in contrast to inflammatory lesions (myelitis). Dorland's Illustrated Medical Dictionary, at 1088 (27th ed., 1988).

[5] Petitioner must prove her case by a preponderance of the evidence which requires that the trier of fact "believe that the existence of a fact is more probable than its nonexistence." In re Winship, 397 U.S. 358, 372-73 (1970) (Harlan, J., concurring), quoting F. James, Civil Procedure 250-51 (1965). Mere conjecture or speculation will not establish a probability. Snowbank Enter. v. United States, 6 Cl.Ct. 476, 486 (1984).

1983)).

"[E]vidence in the form of scientific studies or expert medical testimony is necessary to demonstrate causation" for a Petitioner seeking to prove causation in fact. H.R. Rep. No. 990908, 99th Cong. 2d Sess., pt. 1 at 15 (Sept. 26, 1986), reprinted in 1986 U.S. Code Cong. and Admin. News 8344, 6356. Because Petitioner's claim rests on a hypothesis not yet accepted universally by the medical community, the court will address recent guidelines by which one may evaluate scientific evidence and expert medical testimony. In this regard, the Supreme Court decision in Daubert v. Merrell Dow Pharmaceuticals, Inc., 113 S.Ct. 2786 (1993), is instructive. Although Daubert dealt with the admissibility of scientific evidence, and in this case the court is assessing the scientific validity of evidence already presented, Daubert is helpful in providing an analytical framework for evaluating reliability of expert testimony.[6] The court in Daubert wrote:

> [I]n order to qualify as 'scientific knowledge,' an inference or assertion must be derived by the scientific method. Proposed testimony must be supported by appropriate validation--i.e., 'good grounds,' based on what is known. In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability.

Id. at 2795. A key criterion of scientific reliability is whether a theory has been tested and subjected to peer review and publication. Id. at 2796-97. While acknowledging that publication is not the sine qua non of admissibility, the Court found that the submission of a novel scientific theory to the scrutiny of publication is a component of "good science" and the fact of publication is a relevant, though not dispositive, consideration. Id. at 2797. Finally, the Court noted that, while not a precondition, the general acceptance of a scientific theory within the scientific community can have a bearing on the question of assessing reliability while a theory that has attracted only "minimal support" may be viewed with skepticism. Id.

Federal courts have disagreed over whether the testimony as to causation presented by a treating physician must satisfy the Daubert test. The 3rd Circuit, joined by the 4th Circuit, holds that a physician's testimony is admissible under the Daubert test even if it is not supported scientific studies. The court held that suggested alternative causes of an illness, once addressed by the expert physician, go not to admissibility, but to the weight of the testimony. The court in Heller v. Shaw Industries Inc. , 167 F.3d at 152 noted that Daubert's factors are flexible and need not be applied in every situation. See, e.g. In re Breast Implant Litig., 11 F. Supp. 2d 1217 (D. Colo.1998) The 5th Circuit in Moore v.

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[6] In Daubert, the Supreme Court held that Federal Rule of Evidence 702 is binding on federal courts with respect to establishing the admissibility of scientific evidence. Daubert, 113 S.Ct. at 1795. The Federal Rules of Evidence are not binding on this tribunal. The Vaccine Act requires either scientific studies or expert medical testimony to demonstrate causation in fact.

Ashland Chemical, Inc., 151 F.3d 269,279 (5th Cir. 1998) held otherwise. (dissent) (en banc). A stinging dissent in Moore warned however that it made no sense to lock the gate on such causation evidence that has been derived through [valid] principles of clinical medicine, [for example, differential diagnosis -- a common tool used by clinicians -- a method for determining diagnosis and treatment of patients.] Moore, Id. 151 F. 3d at 290. *See* National Law Journal, Monday, May 29, 2000 at B19. The Moore dissent is consistent with the requirements of the Vaccine Act. Evidence may be in the form of scientific studies or expert medical testimony to demonstrate causation in fact.

Because Helen Rogers' injury is not listed on the Vaccine Injury Table her claim that Tetanus Toxoid caused her present condition must be analyzed under the causation in fact rubric. This analysis in turn devolves to a two part inquiry: Can the vaccine cause such condition, and if the answer is affirmative, did the vaccine cause that condition in this case. See Guy v. Secretary of HHS, No. 92-779F, 1995 WL 103348 (Fed. Cl. Spec. Mstr. Feb. 21, 1995) (two-step causation in fact analysis used); Alberding v. Secretary of HHS, No. 90-3177V, 1994 WL 110736 (Fed. Cl. Spec. Mstr. March 18, 1994) (two-step causation in fact analysis used). The evidence will be analyzed according to these guidelines.

PETITIONER'S EVIDENCE

Dr. Alan S. Levin testifying for Petitioner:

Dr. Alan S. Levin is a Certified Diplomate of the American Board of Allergy and Immunology. He is a Fellow of several medical societies including the Colleges and Societies of Emergency Physicians, American Pathologists, and Clinical Pathologists. He testified that he is current in research and literature in the field of multiple sclerosis. Dr. Levin is of the opinion that the February 15, 1991 injection of Tetanus Toxoid caused or substantially contributed to the autoimmune demyelinating process from which Ms. Rogers now suffers. Transcript of August 21, 1997 proceedings (hereinafter Tr. I) at 13. He questions Respondent's allegation that Petitioner suffers from MS. He believes that her signs and symptoms were atypical for an MS diagnosis (P. Ex. 17 at 2), noting that her treating physicians could not decide whether it was MS or "an MS-like" disorder, and a diagnosis of MS was never made.[7] He points out that all of her treating neurologists, Drs. Richardson, Matthews, and Whittaker, believed that her condition was related to the Tetanus Toxoid inoculation. Dr. Levin argues, however, that it does not matter whether her condition is diagnosed as MS or whether it is a similar condition that falls under the umbrella of demyelinating autoimmune disease because the basic pathophysiology in such disorders, he argues, is the same. Tr. I at 28.

[7] According to Dr. Levin, some neurologists would diagnose Petitioner's condition as MS, and some would not. Tr. at 27. In his opinion, the presentation, laboratory profile, the anti-myelin antibody test, and somatosensory potentials were inconsistent with a classical MS diagnosis in this case. P. Ex. 17 at 1; Tr. I at 32.

According to Dr. Levin, the majority of the medical community concedes that Tetanus Toxoid can cause or trigger autoimmune demyelinating disorders in general. The Vaccine Safety Committee of the Institute of Medicine (hereinafter IOM) has accepted the plausibility of an association between certain vaccines and demyelinating disorders, based on case reports in medical literature. The 1994 Report of the IOM provides a detailed discussion of historic and scientific evidence leading the IOM to include in its 1994 Report the following statement :

> [I]t is biologically plausible that injection of an inactivated virus, bacterium, or live or attenuated virus might induce an autoimmune response in the susceptible host, either by deregulation of immune response, by nonspecific activation of T-cells directed against myelin proteins, or by autoimmunity triggered by sequence similarities such as those of myelin. The latter mechanism might evoke a response to a self-antigen (molecular mimicry).

1994 IOM Report (citation omitted).[8]

More specifically, autoimmune demyelinating disorders have been reported in association with Tetanus Toxoid. Based on case reports, the IOM has concluded that there is a probability that a causal relation exists between the Tetanus Toxoid and two specific demyelinating disorders, namely, Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), also known as Guillain-Barre' syndrome (GBS), and Brachial Neuritis. Id. at 84, 86, 89, 90-91,and 94. These two disorders are classified as peripheral nervous system demyelinating diseases.[9]

The following quotation from an article published in 1980 confirms that for several years, the medical community has suspected that an association exists between Tetanus Toxoid and peripheral nerve demyelinating processes:

> The literature on peripheral neurological [sequelae] following tetanus toxoid vaccination is reviewed. There were 9 cases of Guillain-Barre' syndrome . . . 8 cases of brachial plexus neuropathy, 9 cases of cranial nerve involvement, and 6 cases with more or less isolated peripheral nerve lesions. In regard to the report of Pollard and Selby in 1978 there is evidence that

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[8] Adverse Events Associated with Childhood Vaccines, "Evidence Bearing on Causality" Vaccine Safety Committee, Division of Health Promotion and Disease Prevention, Institute of Medicine, National Academy Press, Washington, D.C. 1994 (hereinafter 1994 IOM Report) at 36, 48; See also at 83-84, 88.

[9] Inflammatory myelopathies are divided into two categories of neuroimmunologic disorders: Conditions Primarily Affecting the Peripheral Nervous System, and Conditions Primarily Affecting the Central Nervous System. E. Richard Stiem, Immunologic Disorders in Infants & Children, at 890 (4th ed. 1996).

> such peripheral nerve disease is due to tetanus vaccination if the latency between vaccination and the side-effects does not exceed the incubation period of 4 to 21 days; in rare cases, however, short intervals of several hours and long intervals of 29 days or more have been observed.

Guillain-Barre Syndrome ([GBS])nach Tetanus-Schutz-Impfung, Oversicht und Fallmitteilung, (Guillain-Barre' Syndrome Following Tetanus Toxoid Administration), George Thieme Verlag Stuttgart, New York, akt. Neurol. 7 (1980) 195-200. Respondent's Exhibit (hereinafter R. Ex.) D at 195.

By virtue of the relatedness of the conditions and their pathogenesis, Dr. Levin analogizes MS, a chronic demyelinating disease of the central nervous system, with GBS/AIDP, a demyelinating disease of the peripheral nerves, reasoning that if Tetanus Toxoid is implicated in GBS/AIDP, it is reasonable to believe that it can cause or trigger MS. He believes that if an individual has the genetic propensity to develop MS, and if that individual, after stimulation with the Tetanus Toxoid, develops T-cell receptors which are associated with MS,[10] and if there are no other confounding causal agents, then it can be reasoned that, more likely than not, the tetanus agent caused the disease process in that individual. Tr. I at 93.

In support of his theory, Dr. Levin submitted a medical article, published in 1993, that, according to Dr. Levin, implicates Tetanus Toxoid as a trigger of MS. The article reports a study involving discordant versus concordant monozygotic (genetically identical) and dizygotic twins sets. Laboratory experiments demonstrated that stimulation of T-cell receptors with Tetanus Toxoid resulted in replicating reactions consistent with MS. Both control and concordant twin sets showed "skewed T-cell receptor repertoire" consistent with MS, leading Dr. Levin to conclude that the Tetanus Toxoid antigen could contribute to the pathogenesis of MS and other T-cell-mediated diseases. The article reports:

> These changes in the repertoire therefore reflect a general skewing that becomes visible not only after stimulation with a suspected target antigen, but also [following] an unrelated foreign antigen like tetanus toxoid. . . . The observations made for MS twins might therefore generally apply to discordant identical twins suffering from other T-cell-mediated autoimmune diseases . . . . Further investigation of monozygotic twins affected with autoimmune diseases other than MS are needed to determine whether these findings are specific for MS or are a more general phenomenon associated with auto-immunity.

Utz-Biddison, H. McFarland, D. McFarlin, Loriage, & Martin, "Skewed T-cell Receptor

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[10] T-cells are lymphocytes (cells) that are the body's "immunologically competent cells and their precursors" that help to suppress the effects of antigens. Dorland's Illustrated Medical Dictionary, at 342,963 (27th ed. 1988).

Repertoire in Genetically Identical Twins Correlates with Multiple Sclerosis," Letters to Nature, (hereinafter Nature or Utz-Biddison study) Vol. 364, 15 July 1993, at 243, 245, 246; P. Ex. 29 at 243-46; R. Ex. A at 243-46.

Dr. Levin explains the article as follows:

> DR. LEVIN: Basically, all it says is that if you take the cells from these people, take lymphocytes out of these people and put them in a test tube, and you evoke an immune response with myelin basic protein, you are going to see the development of T-cell receptors that are associated with multiple sclerosis. And then if you take the cells and give them Tetanus Toxoid, you will get the same type of receptors on these cells.
>
> THE COURT: Then that means that the receptors themselves are identical to the ones that cause MS?
>
> THE WITNESS: Right.

Tr. I at 26.

Dr. Levin's position is that Tetanus Toxoid is capable of triggering autoimmune demyelinating processes such as MS and that it did so in this case. Shortly after Ms. Rogers received the vaccination, she started to experience symptoms consistent with the development of the demyelinating disease process. Her lower extremities were feeling cold within 24 hours and she developed "numbness three days later." Tr. I at 31. Dr. Levin testified that although this period of time is short, it is not an unreasonable time frame for implying a causal connection between the vaccination and the triggering of the disease process. He cites again, in support, the medical article by George Thieme (cited previously) published in Neurology relating to onset of GBS following Tetanus Toxoid because he considers it relevant in this particular case:

> [GBS] may be considered causally associated to tetanus vaccination "if the latency between vaccination and the side-effects does not exceed the incubation period of 4 to 21 days; in rare cases, however, short intervals of several hours . . . have been observed."

George Thieme, "Guillain-Barre' Syndrome Following Tetanus Toxoid Administration," Neurol. 7, R. Ex. D at 195; cited supra; Tr. I at 59. (Emphasis supplied.)

Dr. Levin adds that laboratory tests administered shortly after Helen Rogers' vaccination confirmed the presence of autoimmune demyelinating (ADEM) process. Tr. I at 32. ADEM and MS are both demyelinating diseases of the central nervous system. GBS (Guillain Barre' or AIDP) differs from ADEM and MS in that GBS is a demyelinating disease of the peripheral nerves. Dr. Levin's position is that they are pathologically similar and can be analogized.

RESPONDENT'S EVIDENCE

Testimony of Dr. Barry G. W. Arnason for Respondent:

Dr. Arnason is board certified in the field of Psychiatry and Neurology and in the field of Immunology. He is also an expert in the combined field of study known as neuro-immunology. He is a member of national and international medical societies, and is an expert in the field of Multiple Sclerosis. Dr. Arnason testified that he is ninety-five percent certain that the disease from which Ms. Rogers suffers is Multiple Slerosis and that MS has never been associated with Tetanus Toxoid as an antecedent trigger. Tr. I at 24, 77, 78, 80. Dr. Arnason quoted briefly from a section on demyelinating disease he himself wrote for a medical textbook he believes to be "the most widely used medical textbook in the world":

> When signs pointing to damage of white matter [myelin] tracts in optic nerves, brain stem and spinal cord are present together and more than one attack is known to have occurred, a diagnosis of multiple sclerosis can be made with greater than 95 percent certainty.[11]

Harrison, Textbook on Internal Medicine, 1991; Tr. I at 74.

Dr. Arnason explains that multiple sclerosis is a disease in which the myelin of the central nervous system is damaged by the body's immune response against its own tissues. Myelin is the insulation around the nerve fibers, and when the insulation is lost, the ability of nerves to conduct impulses is compromised. That condition manifests itself in symptoms which can be varied and can involve many different parts of the nervous system, which is one reason this disease is called "multiple" sclerosis. A second diagnostic criterion is the involvement of multiple parts of the nervous system, on multiple occasions. Tr. I at 69.

Certain clinical evidence convinces Dr. Arnason that Ms. Rogers' disorder had already manifested itself prior to the tetanus shot although it was not recognized as a

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[11] These alleged signs and symptoms of MS are well documented in the medical records of Petitioner's clinical course, with the exception of a prior attack. Dr. John Whitaker observed scattered lesions on images of cranial MRI and CSF alterations and a high intensity signal on the on the dorsal portion of the cervical cord. P. Ex. 2 at 54, 58. On August 25, 1992, Dr. Emil Wright (ophthalmologist) identified "retrobulbar optic neuritis." Letter of Emil F. Wright, Jr., M.D., dated September 2, 1992. P. Ex. 5 at 69, 75. Dr. Whitaker described Petitioner's condition as "relapsing demyelinating disease whether confined to the cervical cord or disseminated in the presence of multiple sclerosis." P. Ex. 28 at 3. Dr. Arnason will argue that his view of the evidence suggests that Petitioner probably had a prior attack as well.

manifestation at the time. Nearly three years prior to the February 15, 1991 tetanus shot, Ms. Rogers had an episode of vertigo of such impact that she herself noted it many months later when she filled out a form. Dr. Arnason testified that vertigo is a common initial manifestation of multiple sclerosis. Although it is not possible to be certain that the vertigo episode was the initial manifestation of multiple sclerosis, other manifestations lead Dr. Arnason to believe it was probably the first clinical indication of Petitioner's disease.[12] Tr. I at 71-72.

Dr. Arnason's opinion is reinforced by the fact that Petitioner's first MRI scan following the tetanus vaccination revealed spots showing abnormalities in the myelin of the brain that were not active at the time of the scan. "In other words, not only were [the lesions] inactive, they were in the wrong place for the symptoms that she was complaining of at that time."[13] Tr. I at 105-106. Conversely, a subsequent scan of the neck revealed an active lesion consistent with the symptoms Ms. Rogers was demonstrating at the time. Even if one could not be sure that the vertigo was the initial manifestation of the disease, it is clear from the MRI scan, according to Dr. Arnason, that there had been earlier manifestations of MS, some of which may have been subclinical.[14]

In addition to evidence of vertigo and old lesions in the myelin of the brain, a spinal fluid examination showed abnormalities of the oligoclonal bands, abnormal protein bands of immunoglobulin which, Dr. Arnason claims, are seen in 90 percent of MS patients. Tr. I at 72. Dr. Arnason testified that such abnormality is not diagnostic in and of itself, but is "evocative" of MS. Ms. Rogers demonstrated also an elevated total IgG level and "actual synthesis . . . [or in other words] production of immunoglobulin within the nervous system," indicating that the process had been ongoing for some time. Tr. I at 72. He considers the IgG [immunoglobulin] finding also evocative of MS. Tr. I at 73. The inflammatory nature of the myelopathy, according to Dr. Arnason, is highly unusual in other forms of

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[12] Dr. Levin challenges as mere speculation any conclusion proposed by Respondent that Ms. Rogers' prior episode of vertigo was MS-related. But even if the episode of vertigo was a prodromal symptom of the disease, the Tetanus Toxoid, he argues, simply exacerbated the latent disease process rather than "triggered" the initial event. Because the actual presence of MS was never established prior to vaccination, he believes it more likely that the shot must have evoked the disease process. Tr. I at 33, 62.

[13] Dr. Arnason testified that "MS plaque stays active for usually from two to six or seven weeks" so that the lesions would have to have been active at least seven weeks prior to the tetanus shot. Tr. I at 105.

[14] MS is usually triggered by something. Many attacks, however, occur without any evident antecedent [and(some may be "subclinical")]. Infections and other causes are possible triggers. See generally, Harrison's Principles of Internal Medicine, 12th ed., Vol. 2, Part Thirteen, Neurologic Disorders," Chapter 356, "Demyelinating Disease," at 2030.

myelopathy but is characteristic of MS. Tr. I at 75.

Dr. Arnason rejects emphatically Dr. Levin's reliance on analogizing the various manifestations of demyelinating diseases. Dr. Arnason argues that demyelinating disorders cannot be analogized because they differ in significant ways that prevent their being simply lumped together for purposes of ascribing causation. MS is a disorder of the central nervous system; GBS is a disorder of the peripheral nerves. The myelin of the central nervous system, he states, is quite distinct from peripheral nerve myelin although he acknowledges that underlying mechanisms may be similar at the level of the cell types involved. Tr. I at 76. Dr. Arnason rejects the claim that pathophysiological similarity of process extends to etiology. In other words, it does not necessarily follow that a trigger in one disease indicates that it would trigger another form of the disease process. Dr. Arnason did not explain further but insists that no scientific evidence exists that implicates an association between Tetanus Toxoid and MS. Tr. I at 88. Dr. Arnason, at the hearing, did not address the Utz-Biddison study of the effect of Tetanus Toxoid on MS as reported in the Nature article discussed supra. Nor did he address its conclusions. The court subsequently requested that he address the article. In response, Dr. Arnason submitted an eight-page supplemental report challenging the article's conclusions and denying the study's usefulness in this case as will be discussed hereafter. Dr. Arnason considers the onset of symptoms following Petitioner's inoculation to be a coincidence, only, and the natural progression of her disease.

At the close of the August 21, 1997 hearing of medical experts held in Washington DC, the court ordered closing arguments to be submitted in the form of post-hearing briefs. Briefs were filed on October 24, 1997 and November 17, 1997 respectively. The court, however, was not satisfied with the evidence or the arguments, and requested further information concerning MS and its causes. The record was held open for an extended period due to three problems: The complicated nature of the subject matter; delay in response from the medical experts; and further delay caused by two different substitutions of attorney for Respondent. The case would not be ripe for decision for many months thereafter.

The Supplemental Evidence:

In response to the court's request for supplemental information relating to the causes of MS, Respondent submitted the commentary of Dr. Barry Arnason, about the medical article which he failed to address at hearing. ("Skewed T-cell receptor repertoire in genetically identical twins correlates with multiple sclerosis,")[15] As described earlier, the Utz-Biddison study published in Nature, is considered by Petitioner's experts to be strongly supportive of Ms. Rogers' claim. Dr. Arnason's rebuttal testimony states that although the cause of MS is not known, both genetic and environmental factors are thought to be

[15] Ursula Utz et al., (Utz-Biddison Study) published in Nature, 364:243-247, 1993. R. Ex. F.

involved in development of this disease. A genetic component does not mean that MS will in fact develop--something in addition to the genes determines whether MS will develop. "The 'something in addition to' is thought to be an environmental factor, but the nature of this factor or factors is completely unknown." R. Ex F at 1-2. Dr. Arnason continues: MBP (myelin basic protein) is a normal component of human myelin, and is a recognized candidate as a trigger based on observations in animal experiments. Experimental animals immunized with MBP develop an MS-like disease by bringing on an inappropriately excessive proliferation of T-cell responses (cloning) that attacks and destroys the brain's myelin. Signs and symptoms of MS then follow.

Dr. Arnason introduces his paper by cautioning that the paper is "highly technical even for a trained immunologist." R. Ex F at 1. The court, indeed, found the technical matter quite difficult, but it is clear that Dr. Arnason is insisting that the Utz-Biddison study does not establish causation. His reasons are not clear. On the one hand, he admits that prior work identifies a response by MS patients to Tetanus Toxoid (Id. at 6). Yet he argues that an in vitro experiment, in which cells are subjected to prolonged stimulation with Tetanus Toxoid over several days, "cannot be taken as indicative of T-cell behavior in the living body in response to a single stimulation or vaccination." Id. Dr. Arnason submitted a brief statement that he solicited from Dr. Roland Martin. R. Ex G. Dr. Martin is one of the Utz-Biddison researchers. Dr. Arnason considers the Martin statement supportive of his (Dr. Arnason's) position that Tetanus Toxoid could not in any way be related to multiple sclerosis. The court construes Dr. Martin's statement only marginally supportive of Dr. Arnason's view as will be explained hereafter. Dr. Arnason submitted three other medical articles, R. Exs. H, I, and J. He failed to discuss the new exhibits, and at least one of the three articles appears to support Petitioner's position rather than his own. Dr. Arnason's supplemental evidence, as a whole, was curiously unhelpful to Respondent's position.

For Petitioner, Dr. Levin submitted a post-hearing supplemental report, identifying what he considers flaws and misinterpretations in Dr. Arnason's rebuttal analysis. P. Ex 34. He reaffirms his opinion that the Utz-Biddison paper clearly shows that TT "most definitely [can] cause the expression of the precise T-Cell receptors which cause Multiple Sclerosis in the appropriately susceptible host." P. Ex. 34 at 1. Dr. Levin's report was consistent with the current medical literature filed in this case, and was helpful to the court.[16]

[16] Dr. Levin testified that his wife is also an immunologist and internist and works extensively on sectors associated with immune demyelinating diseases including MS. Tr.I at 9. Dr. Levin theorized that his own expertise "goes more to the basic immunogenetics and the basic immunology of the disease process, and Dr. Arnason is more the clinical expert and that of clinical medicine." Tr. I at 12 . (The term "clinical," is defined as "pertaining to or founded on actual observation and treatment of patients, as distinguished from theoretical or basic sciences." Dorland's Illustrated Medical Dictionary, at 345 (27th ed. 1988)).

Petitioner filed also the post-hearing statement of Dr. Derek R. Smith, MD, in support of her claim. P. Ex 32. Dr. Smith is a neurologist associated with Brigham and Women's Hospital, Massachusetts General Hospital, and Harvard Medical School in Boston, Massachusetts. He is a member of a medical group, "Multiple Sclerosis Clinical and Research Unit," in Boston, Massachusetts. Dr. Smith agrees with Dr. Levin based on recent research that he believes implicates Tetanus Toxoid as an antigen capable of triggering an MS-like response in humans. Dr. Smith criticized Dr. Arnason's rebuttal, which he believes is flawed, and considers the Utz-Biddison study more supportive of Petitioner's claim than Dr. Arnason is willing to admit. He is convinced that recent research supports the view that autoimmune demyelinating diseases in general can be related to a number of antigens, that in all probability the Tetanus Toxoid is one such antigen; and that the Tetanus Toxoid, more likely than not, caused or exacerbated Helen Roger's condition. The published report of the Utz-Biddison study, he argues, along with the results of medical research with animal models, support such a conclusion.

Petitioner's Rebuttal Evidence

Following the court's withdrawal of its original decision (vacated on October 12, 1999), both parties petitioned the court to allow additional oral testimony. Petitioner requested, and the court allowed, the appearance of Dr. Derek Smith to testify in person to supplement and explain his written statement. Dr. Smith is board certified in adult neurology and has authored a number of papers most notably in the area of neuroimmunology and multiple sclerosis. He sees multiple sclerosis patients almost exclusively. He has done laboratory research on T-cell responses in MS patients and conducted clinical trials incorporating some of the results.

Dr. Smith is of the opinion that prior to vaccination, Petitioner had a benign, subclinical form of Multiple sclerosis that rendered her susceptible to and less resistent to an aberrant response to the vaccine. Transcript of proceedings of January 27, 2000 at 19-20 (hereinafter Tr. III at–) In other words, having that condition, Ms. Rogers was predisposed to having an abnormal reaction to the TT antigen: He cites published evidence that T cells can respond to a large variety of antigens under certain circumstances. Tr. III at 25-26. He states: "It's clear from more recent studies that . . . a tetanus toxoid peptide antigen may in fact activate cells that respond against these different [antigen] proteins, including myelin basic protein, and that a tetanus vaccination could activate an immune response that would develop into a full-blown disease especially in a presensitized host. He believes that is what happened in fact, in this patient: "She went from having a benign MS course to a very progressive course." Dr. Smith testified that Dr. Martin's research itself would support this theory. Id. at 26 (citations omitted). "Interestingly and importantly, these findings apply to the T-cell response against myelin basic protein, but also to foreign recall antigen, namely, the tetanus toxoid, having been already sensitized by a prior administration of the TT vaccine." Dr. Martin's own early research, according to Dr. Smith, suggests a causative role of such foreign peptide antigens as TT that might shape the T-cell repertoire in MS patients. Id. at 27. Dr. Smith believes that this early (1997) research suggests the likelihood that TT had a role in

exacerbating an MS condition. Id.[17] Dr. Smith insists that scientific evidence indicates that this response can occur. He adds that Ms. Rogers clinical history demonstrates that it, in fact, did. Id. at 34. Her history, he argues, speaks for itself. The time frame of her initial response was appropriate. She deteriorated over the course of the next weeks and months, and her clinical course demonstrated evidence of an ongoing inflammatory response in her central nervous system. Id. at 36.

Dr. Smith recognizes that a majority of physicians may not yet support his theory, but he believes that a significant minority would, in fact, perceive this as realistic. "It is a judgment call, in any individual case," but he notes that the physicians involved in Ms. Rogers' care and treatment had the instinct that the hypothesis of causation that Dr. Smith proposes was likely. Id. at 43. Dr. Smith testified that he has had discussions with other physicians who treat MS patients who feel that they have seen this same scenario. Tr. I at 44, 46,47. Other of his colleagues are of the same opinion, and also believe that although such reactions are not common, a similar TT reaction can occur. Tr. III at 50-51, 56. He insists that the Nature article, in particular, "did certainly widen the realm of possibility with respect to tetanus toxoid as a potential trigger."

Testimony of Dr. Roland M.G. Martin:

Respondent called on Dr. Martin, one of the authors of the Nature article, as a rebuttal witness. Dr. Martin submitted his written statement explaining the purpose of the Utz-Biddison research study and supplemented his statement of intent in oral testimony at the supplemental hearing. He makes it very clear that the research which Petitioner's experts believe support their positions, was intended to present a concept, or theory, a conceptual framework or possible mechanism of cause and effect, and was not intended as proof of a causal relationship between TT and MS. He insists that such theory has not been studied yet, and as far as he is concerned, there is no knowledge that links "very clearly" a vaccination with an autoimmune disease. That knowledge, he states, is simply not yet available. For example, no epidemiological studies have been published. He does not believe that it is not possible: "I think it is correct to say that it is within the realm of possibility but there is currently no proof." Tr. III at 70-73, 76. The following colloquy clarifies Dr. Martin's position:

> THE COURT: [I]s this correct then, that this is a theoretical mechanism of injury?
>
> THE WITNESS: That is correct, yes.
>
> THE COURT: . . . But you are saying that you cannot say with any degree of certainty that that is the mechanism of injury.

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[17] Dr. Smith describes the mechanism of injury as "degeneracy," a term being used in more recent research that replaces the former concept of "cross-reactivity" or "molecular mimicry," a term used by Dr. Arnason. Id. at 31-32.

> THE WITNESS: That is correct. We are examining those questions now and plan the experiments, but they have not been examined yet. And we think that we have a conceptual frame by which we can follow those lines. . . .
>
> THE WITNESS: I would agree with Dr. Smith, as I said previously, that there is the possibility that something like that occurs. That's what the frame supports. But there is no scientific evidence yet that shows that it in fact occurs. . . . It's an unlikely trigger but, again, and here I agree with Dr. Smith, it's not completely excludable . . . but . . . it does not prove. No.

Tr. III at 86-87, 92.

It is not surprising that as a research scientist, Dr. Martin requires and holds to a very high level of proof required by laboratory scientists, that is, a level of 95% certainty For example, Dr. Martin's states that no knowledge exists to implicate a vaccination with an autoimmune disease. Dr. Martin misstates or overlooks evidence that such conclusions, to the contrary, have been published in relation to at least two autoimmune diseases. Published case studies have led the Institute of Medicine to suggest a relationship to other autoimmune diseases including the plausibility of a link to MS. The Vaccine Act does not require proof at the level of scientific standards nor does the Act require the existence of epidemiological studies. The Act requires the same level of proof as in traditional tort litigation, a simple preponderance.

DISCUSSION

As an initial matter, a secure diagnosis of Multiple Sclerosis is not critical to the outcome of this case. Dr. Arnason believes Ms. Rogers meets the criteria for MS, and his impressive credentials as a clinician carry considerable weight, but doubt remains. When Ms. Rogers was first seen, her treating physicians were unsure about a diagnosis. Dr. Whitaker was leaning towards MS as a diagnosis, but no conclusive diagnosis was ever established. P. Ex. 2 at 59. He concluded that her condition was "relapsing demyelinating disease whether confined to the cervical cord or disseminated in the presence of multiple sclerosis." P. Ex. 28 at 3. Dr. Levin believes her tests and clinical course, in many respects, were inconsistent with a classical MS diagnosis. P. Ex. 17 at 1; Tr. I at 32. The court is inclined to accept Dr. Levin's explanation that "some neurologists would diagnose her condition as MS and others would not." Tr. I at 27. But nonetheless, her condition falls clearly within the parameters of an autoimmune demyelinating disorder whether it was MS or whether it was "MS-like." Based on the presence of inactive lesions observed on the first MRI following her inoculation and on other test results described in Dr. Arnason's testimony, the court agrees that her condition probably predated her Tetanus Toxoid inoculation, whatever the diagnosis, MS or "MS-like." Tr. I at 27. That finding does not rule out a finding that her latent disorder was exacerbated by the inoculation.

Evidence that Tetanus Toxoid can cause certain autoimmune demyelinating

disorders is not questioned. Based on a review of the evidence filed in this case, the court concludes that, contrary to Respondent's arguments, one cannot simply dismiss out of hand the possibility that frank MS or MS-like symptoms could be triggered by the Tetanus Toxoid. The court was unconvinced by Dr. Arnason's arguments to the contrary. At hearing, Dr. Arnason displayed a cavalier and condescending attitude when questionsed about this issue. He relied on his past achievements, addressed Petitioner's evidence lightly in passing, and appeared less knowledgeable of recent research data referenced by his opponents. None of the three articles he cited in support of his position was current. The most recent on which he relies was an article published in 1993, and the court found it irrelevant to the specific issue. The other two articles were published in 1984 and 1976 respectively--certainly not up to date. The court respectfully disagrees with Dr. Arnason's view that Dr. Roland Martin rejects the possibility of a causal link. In contrast to Dr. Arnason's own blanket rejection of any such possibility that Tetanus Toxoid could be a contributory factor, Dr. Martin acknowledges that it is plausible. Dr. Martin insists simply that the Utz-Biddison study should not, by scientific standards, be viewed as drawing final conclusions one way or the other. As one would expect, Dr. Martin, in an abundance of scientific caution, believes that his research suggests the the wisdom of further research.

Dr. Arnason's insistence that no existing evidence implicates Tetanus Toxoid is simply not supported by the evidence. In sum, Petitioner's experts were more persuasive, their testimony was better supported and led the court the a finding that thought-provoking evidence of a link between TT and MS or MS-like demyelinating disorders, indeed exists. The evidence favoring Petitioner's claim can be summarized as follows: First, the following appears in the 1994 report of the Institute of Medicine (IOM):

> The establishment of a relation between acute central and peripheral nervous system demyelinating disease and infections and vaccines has opened the question of a possible relation to chronic demyelinating disease, specifically, multiple sclerosis. . . . [I]t would be feasible that vaccines also might precipitate an exacerbation either in a patient who was predisposed to develop the disease or in a patient with already established disease. However there is no clear-cut causal relation between any virus or vaccine and multiple sclerosis.[18]

In other words, according to the IOM, a causal link between Tetanus Toxoid and MS is plausible. That position is consistent with the expert opinion in this case. The IOM is an authoritative source to which the court ascribes considerable weight.

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[18] Institute of Medicine. Stratton KR, Howe CJ, Johnston RB, eds. Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality. 36, Washington, DC: National Academy Press, 1994.

Second, the treating physicians ascribed Petitioner's symptoms to an immune system reaction to the Tetanus Toxoid. The opinions of treating physicians, working in the trenches, take a practical view of clinical implications, and this court gives them considerable weight.[19] Third, Drs. Levin and Smith for Petitioner and Dr. Martin for Respondent, have convinced the court that the present status of medical science does not negate the proposed theory of causation, and, that some medical literature, in fact, tends to support it. Moreover, treating physicians use differential diagnosis as a method of applying their expertise in every day practise of clinical medicine to determine what caused their patient's illness and how to treat it. In this case, the treating physicians concluded that Helen Rogers had an MS-like response to the Tetanus.

Fourth, the following list of considerations, according to Petitioner's experts, support that theory: 1) Increased MBP reactive T-cells have been found in MS patients (as compared to normal controls); (P.Ex. 32 at 2); 2) TT is used in MS research to invoke an immune response similar to that invoked by MBP; (Tr. at 21,22, 24); 3) TT is used to induce demyelination when injected into animals (P.Ex. 32 at 2-3); 4) TT is used as a control antigen in studies of myelin-antigen-specific T-cells as in the Utz-Biddison study (although, as Dr. Martin points out, invitro research required more than one exposure to the antigen in order to produce the aberrant response. The court notes that if Ms Rogers had not had a prior TT shot, the evidence of a causal link, therefore, would have been less secure.) P. Ex. 32 at 2-3, 6 5.

In assessing the relative weight to be ascribed to the evidence in an off-Table case, the court is bound by legal requirements for proof that exceed mere "possible" cause. Petitioner is required to prove not plausibility, but probability. The court concludes, as the IOM concluded in its 1994 report, that proof of the basic premise as it relates to MS in general, has not yet been established by the high confidence level required by laboratorian. standards. Prior to the supplemental hearing of January 27, 2000, the court gave greater weight to that factor and concluded that the evidence did

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[19] In this matter, the holdings of the 3rd and 4th circuits as discussed on page five of this decision appear to be relevant. The 3rd Circuit found that a differential diagnosis is a physician's "tool of the trade"and that even in the absence of scientific research or supporting studies, when a doctor has 'good grounds' for his or her conclusions, that testimony is admissible . . . and if used to testify to a novel conclusion, is not alone sufficient grounds to exclude the testimony. When addressed by the expert physician, to repeat the discussion addressed earlier, the suggested alternative causes of an illness go to the weight of the testimony. Heller, 167 F.3d at 149. The dissent in Black v. Food Lion Inc., 171 F.3d argued that a differential diagnosis should be admissible if it is scientifically valid in the medical community even if it is not supported by studies. Moore, 151 F. 3d at 290. This court concludes that not only is such evidence admissible, based on the evidence here, it represents a rational viewpoint and relevant expert opinion evidence that carries considerable weight in Petitioner's favor in this particular case.

not constitute a preponderance in Petitioner's favor. Having considered all the evidence now available, the court finds otherwise. Decisions in vaccine cases need not be based on scientific or laboratorian standards of proof, but on a preponderance.

Dr. Levin agrees that the Nature article alone "does not prove that TT caused Petitioner's MS." No single study, he continues, "can prove causation in a specific patient." P. Ex. 34 at 1. The study can suggest only "the biologic plausibility" of a vaccine-related cause, Id. But he believes that Petitioner's history, clinical course, and laboratory findings are sufficient to establish the likelihood of causation in this specific case. Id. The court agrees. In several ways, Petitioner's evidence meets the guidelines for good science set forth by the Daubert court: Case reports and valid studies have been published and subjected to peer review that suggest the plausibility of a causal link between the disease and Tetanus Toxoid. That factor confers upon the theory a measure of respectability. The court has heard positive and persuasive medical opinion from an impressive group of experts with excellent credentials who appear to be on the leading edge in autoimmune demyelinating disease research and literature; and Ms. Rogers' treating clinicians believed her condition was triggered by the inoculation. The court cannot hold that science has proved that TT causes MS in general, but the court is persuaded that the preponderance lies with the Petitioner in this case. The evidence presented suggests that it is highly likely that the TT exacerbated significantly Ms. Roger's subclinical MS-like demyelinating disease and is primarily responsible for her present condition. The theory of a link may not be accepted at the present time by a widespread majority of the scientific community, but it is supported by a significant minority, (P. Ex. A at 246) and the evidence here tips the scales in favor of Petitioner's claim, if by only a small margin. [20]

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[20] The court is constrained to address briefly Dr. Levin's theory that similarities among demyelinating disorders suggest similar pathologies (etiologies)-- that what causes one such disorder is probably capable of causing demyelination in another. One cannot deny that the hypothesis is attractive and offers a measure of logic, but lacking any objective support, the court is unable to ascribe to it any reliable weight. Dr. Arnason insists that demyelinating disorders cannot be analogized, and that pathophysiological similarity of process does not "necessarily" extend to etiology. Tr. I at 76. The court notes, however, that his use of the words "not necessarily" gives pause as to its plausibility, moreover, Dr. Arnason acknowledes that underlying mechanisms "may be similar at the level of cell types involved." Id. at 76. Nonetheless, in formulating its decision in Rogers, this court neither relies upon it nor takes any position one way or the other. In Trojanowicz v. Secretary of HHS, 95-215V (Ct. Fed. Cl. Spec. Mstr. July 1, 1998,)(Reissued for publication Oct. 16, 1998) WL774338 *aff'd* 43 Fed. Cl. 469 (Mar. 24, 1999) Chief Special Master,Golkiewicz, addressing a similar argument, found it impermissible to extrapolate from similarities in pathogenesis to a conclusion of shared causative agents. The two cases, Rogers and Trojanowicz differ. Most importantly, the Trojanowicz court found that the medical literature filed in that particular case was equivocal, and the expert opinion

(continued...)

CONCLUSIONS

The court concludes that Petitioner is entitled to compensation for her vaccine-related injury. The parties are directed to enter into discussions relative to the damages portion of this case.

IT IS SO ORDERED.

E. LaVon French
Special Master

[20](...continued)
evidence, though "undoubtedly well-intentioned," (Id. at 9, fn.7) was inadequate and unpersuasive: "In fact, if anything, the [medical] articles appear to undercut substantially [the medical expert's] working premise." Id. at 8. The two cases can be distinguished, and the undersigned does not disagree with the Trojanowicz holding. The evidence favoring Petitioner's theory of causation in that case was simply not apparent. Conversely, in Rogers, a considerable amount of scientific, expert opinion evidence, and testimony, (in fact, a preponderance of such evidence as set forth herein), was presented. The sufficiency of that evidence was the distinguishing issue; and supported a finding, in this specific case, in favor of the Petitioner's causation claim.