OFFICE OF SPECIAL MASTERS

No. 02-223V

Filed: March 17, 2005

RYAN KELLEY,	*	
	*	
	*	
Petitioner,	*	
v.	*	TO BE PUBLISHED
	*	
SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES,	*	
	*	
	*	
Respondent.	*	
	*	

Ronald C. Homer with whom was Sylvia Chin-Caplan, Conway, Homer & Chin-Caplan, P.C., Boston, Massachusetts, for Petitioner.

Heather L. Pearlman, United States Department of Justice, Washington, D.C., for Respondent.

DECISION[1]

GOLKIEWICZ, Chief Special Master

I. PROCEDURAL BACKGROUND

On March 21, 2002, petitioner, Douglas Kelley, filed a petition pursuant to the National Vaccine Injury Compensation Program[2] (hereinafter referred to as "the Program"), on behalf of

[1]The parties are reminded of their right, pursuant to Vaccine Rule 18, to object to the public disclosure of any information contained herein within 14 days of the filing this decision. The parties should note that this decision was only slightly modified from the original version, published on October 12, 2004, and subsequently withdrawn on October 22, 2004, pursuant to Petitioner's Motion for Reconsideration. The only substantive change is the discussion of the supplemental hearing, infra, at 19.

[2] The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, (continued...)

his minor son, Ryan Kelley ("Ryan").[3] Petitioner alleged that Ryan suffered neurologic injuries, including Guillain-Barré Syndrome ("GBS") and chronic inflammatory demyelinating polyneuropathy ("CIDP") as a result of a tetanus toxoid ("TT") vaccine Ryan received on March 22, 1999. Petition ("Pet.") at 1. On November 5, 2002, petitioner filed an "Amended Petition for Vaccine Compensation," alleging that as a result of receiving the March 22, 1999 TT vaccination, Ryan suffered GBS, also referred to as Acute Inflammatory Polyneuropathy. Amended Petition ("Amend. Pet.") at 1. On February 3, 2003, respondent filed a Rule 4 Report contesting the sufficiency of the evidence and recommending that compensation be denied. Respondent's Report ("R. Report") at 9.

To resolve outstanding factual questions and elicit expert testimony, a hearing was held on March 30, 2004. Petitioner presented Carlo Tornatore, M.D.,[4] and respondent presented Vinay Chaudhry, M.D.,[5] as their respective expert witnesses.

Following the hearing, the undersigned requested petitioner's counsel to file an updated *curriculum vitae* for Dr. Tornatore and a copy of the article referenced by Dr. Tornatore at the hearing. See Order, filed Mar. 31, 2004. In addition, the undersigned asked the parties to attempt to contact Dr. Pollard regarding the issue of whether the patient discussed in the Pollard & Selby study, "Relapsing Neuropathy Due to Tetanus Toxoid," was diagnosed with CIDP or relapsing GBS. Id. During a status conference held on April 29, 2004, the parties indicated that they were unsuccessful in contacting him.

On May 14, 2004, the parties were ordered to file simultaneously their post-hearing briefs by June 18, 2004 and any reply briefs by July 2, 2004. Order, filed May 14, 2004. On June 18 and June 24, 2004 respondent and petitioner filed their respective post-hearing briefs. Respondent's Post-Hearing Brief ("R. Brief"), filed June 18, 2004; Petitioner's Post-Hearing Brief ("P. Brief"), filed June 24, 2004. On July 2, 2004, respondent and petitioner filed responses to each other's post-hearing briefs. Response to Petitioner's Post-Hearing Brief ("R. Resp."), filed Jul. 2, 2004; Petitioner's Reply to Respondent's Post-Hearing Brief ("P. Reply"), filed Jul. 2, 2004.

On September 24, 2004, a decision denying entitlement was issued in this case. On

---

[2](...continued)
42 U.S.C.A. §§ 300aa-10 et seq. (West 1991 & Supp. 2002) ("Vaccine Act" or the "Act"). Hereinafter, individual section references will be to 42 U.S.C.A. § 300aa of the Vaccine Act.

[3]On December 4, 2003, the court granted petitioner's counsel's motion to amend the caption in this case as Ryan had reached the age of majority. Accordingly, as the current caption reflects, Ryan Kelley is now the sole petitioner in this case.

[4]A copy of Dr. Tornatore's *curriculum vitae* can be found at P. Ex. 19.

[5]A copy Dr. Chaudhry's *curriculum vitae* can be found at R. Ex. S.

October 5, 2004, respondent filed a motion to publish the undersigned's September 24, 2004 decision. The undersigned subsequently granted the motion and the decision was reissued for publication on October 12, 2004.

On October 19, 2004,[6] petitioner filed a Motion for Reconsideration of the entitlement decision and Exhibit 20, a letter to the editor, entitled "Immunization and Risk of Relapse of Guillain-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy."[7] Petitioner argued that this article, which was not a part of the record at the time the undersigned rendered his opinion, "is not only relevant but critical to the issue of his [Ryan's] entitlement to compensation." Petitioner's Motion for Reconsideration ("P. Motion"), filed Oct. 19, 2004, at 1. Specifically, petitioner referred to the statement by the authors of Exhibit 20 that "'Pollard and Selby reported a man whose first ***three relapses of CIDP*** were each triggered by tetanus toxoid. His subsequent illness pursued a spontaneously relapsing and remitting course responsive to plasma exchange (Pollard, personal communication).'" P. Motion at 4 (quoting P. Ex. 20 at 1230). Thus, petitioner contends that "Dr. Pollard believed TT caused his patient to suffer three separate relapses of CIDP, not GBS. This, Ryan submits, unequivocally establishes that TT *can* cause CIDP." P. Motion at 4 (citing Capizzano v. Secretary of HHS, No. 00-759V, 2003 WL 22425000, at * 4 (Fed. Cl. Spec. Mstr. Aug. 5, 2003) ("In essence, rechallenge cases are such strong proof of causality that it is unnecessary to determine the mechanism of cause -- it is understood to be occurring.")).

Petitioner believes that, in light of the new evidence discussed above, TT can cause CIDP. P. Motion at 4. Accordingly, petitioner posits that the only issue remaining for the Chief Special Master's reconsideration is whether TT in fact caused Ryan's CIDP. Id. Based on the record, petitioner submits that the TT was the cause of Ryan's CIDP. Id. Thus, petitioner avers that "as a matter of 'fundamental fairness,'" the Chief Special Master should reconsider petitioner's case based on the newly submitted evidence. Id. at 6.

At a status conference held with the parties on October 18, 2004, the undersigned explained his belief that, given the nature of the Vaccine Program, it is incumbent upon all, petitioner, respondent and the court, to ensure as complete a record as possible upon which to render a decision. Thus, on October 22, 2004, the undersigned granted petitioner's motion for reconsideration in order to evaluate the additional evidence submitted by petitioner and ordered the Clerk of the Court to withdraw the September 24, 2004 decision, which was reissued for publication on October 12, 2004, in the above-captioned case. The parties were directed to coordinate in determining a schedule for: 1) the filing of their written submissions regarding this evidence in November; and 2) a telephonic hearing with their experts in December. See Order,

[6]Petitioner's counsel faxed a copy of this submission to respondent's counsel and the Office of Special Masters on October 14, 2004.

[7]P. Ex. 20 (Richard A.D. Hughes et al., Immunization and Risk of relapse of Guillian-Barré Syndrome or Chronic Inflammatory Demyelinating Polyradiculoneuropathy, MUSCLE & NERVE 1230-1231 (Sept. 1996)).

filed Oct. 22, 2004.

On November 1, 2004, petitioner filed "Petitioner's Memorandum in Support of Motion for Reconsideration." On November 30, 2004, respondent filed his "Response to Petitioner's 'Motion for Reconsideration'." The crux of respondent's argument was that the "newly submitted evidence adds nothing to petitioner's claim that tetanus toxoid can cause CIDP, or that it did so in this case." Response to Petitioner's "Motion for Reconsideration" ("R. Response to P. Motion"), filed Nov. 30, 2004, at 6. Thus, respondent requested that the undersigned reissue his September 24, 2004 decision for publication. Id.

On December 17, 2004, petitioner filed Exhibit 21, a medical article, entitled "Chronic Inflammatory Demyelinating Polyneuropathy Presenting with Features of GBS."[8]

On December 21, 2004, a supplemental telephonic hearing was conducted. To aid the undersigned in his evaluation of the new evidence, petitioner and respondent once again presented Carlo Tornatore, M.D., and Vinay Chaudhry, M.D., as their respective expert witnesses.

The record is now closed and the case is ripe for decision. After reviewing the entire record, and for the reasons set forth below, the court finds petitioner has failed to carry the burden of proof required under the Act, and thus is not entitled to compensation. A full discussion follows.

II. FACTUAL BACKGROUND

The following is a condensed version of the facts as they appear in the medical records. A more thorough exposition of the pertinent disputed facts, as well as an extensive examination of testimonial evidence, appears in Part III (Discussion Section).

On March 22, 1999, Ryan visited his pediatrician, Dr. Judith Hochstadt. P. Ex. 1 at 2-3. Notes from this visit indicate that Ryan was healthy, with no known health problems. Id. at 3. At this visit, Ryan received a tetanus booster. Id. at 7.

According to the affidavit of Ryan's father, during the second week post-vaccination, Ryan began to "become more tired and fatigued...to the point where he would even stumble on the [tennis] court and lose his balance...soon his symptoms worsened. Ryan began to lose his balance more often, endured increasing aches and pains throughout his body, felt tingling in his feet and hands with some loss of feeling, and was continuously more tired and weak when he came home." P. Ex. 9 (Affidavit of Douglas Kelley).

On April 24, 1999, Ryan presented at his pediatrician's office with complaints of lower back stiffness and numbness and tingling of both hands and feet, as well as dizzy spells at night,

[8]M. Mori et al., Chronic Inflammatory Demyelinating Polyneuropathy Presenting with Features of GBS, 58 NEUROLOGY 979-982 (2002).

which had begun 2-3 weeks earlier. P. Ex. 1 at 9. Ryan also reported increased fatigue, weight loss, a sore throat for 1-2 days, and a 100 degree fever the previous night. Id.

On May 4, 1999, Ryan was admitted to Bridgeport Hospital for possible Lyme disease and subacute cerebritis. P. Ex. 2 at 1. Subsequent tests for Lyme disease were negative. Id. at 2. Ryan was discharged on May 6, 1999, with instructions to follow up with Dr. Nallainathan, a pediatric neurologist, in one week. Id. at 44. His discharge diagnosis included encephalitis. Id. at 2.

On May 17, 1999, Ryan visited Dr. Nallainathan, who ordered a nerve conduction study to rule out GBS. P. Ex. 3 at 2. Based on the results of this study, Dr. Nallainathan concluded that Ryan's clinical picture was an atypical form of GBS presenting as ataxia, but without opthalmoplegia. Id.

On June 4, 1999, Dr. Nallainathan prescribed a course of Prednisone for Ryan. P. Ex. 3 at 18-19. By June 14, 1999, Ryan was walking much better and his headaches had disappeared. Id. Over the next two weeks, Dr. Nallainathan gradually tapered Ryan off prednisone. Id. Once off prednisone, Ryan suffered a recurrence of neurological symptoms and the corticosteroid was restarted. Id. at 16.

On August 9, 1999, Dr. Nallainathan wrote to Dr. Hochstadt that his impressions of Ryan's clinical picture had changed. P. Ex. 3 at 20. Dr. Nallainathan no longer believed that Ryan had GBS. Id. After further evaluation, Dr. Nallainathan now felt that Ryan more likely suffered from CIPD. Id. The doctor also noted that Ryan's neurologic problems persisted, despite treatment with prednisone. Id. at 20.

In November 1999, Ryan was seen by Dr. David Cornblath, a neurologist at Johns Hopkins University. P. Ex. 7 at 7-12. Dr. Cornblath noted, "after receiving a TT injection, he [Ryan] developed an acquired demyelinating polyneuropathy." Id. Nerve conduction studies performed the day of Ryan's evaluation showed "considerable worsening of his neuropathy," and features of a "moderately-severe asymmetric sensory-motor demyelinating polyneuropathy consistent with an acquired disorder, such as CIDP among others." Id. at 11. Dr. Cornblath concluded that "[t]he differential diagnoses for his [Ryan's] syndrome considers GBS, CIDP, CIDP with a paraprotein, or CIDP following tetanus administration (if this exists)." Id. at 9.

Over the next two years, Ryan was treated by Dr. Kenneth Seigel with intravenous immunoglobin ("IVIG") and underwent plasmaphoresis in an attempt to control his symptoms. P. Ex. 5. These treatments were largely unsuccessful, and Ryan was seen again by Dr. Cornblath in March 2002. P. Ex. 7 at 1-6. At that time, Dr. Cornblath noted, "Mr. Kelley has a severe acquired demyelinating neuropathy which has been resistant to treatment with prednisone, IVIG, plasma exchange and CellCept. He is, if anything, slightly worse than when he was seen 2 years ago." Id. at 2-3. Nerve conduction studies confirmed that petitioner's condition was slightly worse than on examination two years earlier. Id. at 4-5.

Notes from Dr. Siegel in May and September 2002 list Ryan's diagnosis as CIDP. P. Ex. 5 at 1-4; P. Ex. 15 at 3. The most current records submitted by petitioner from Ryan's evaluation at Johns Hopkins on November 11, 2003, reveal that Ryan had been maintained on azathioprine and was doing extremely well. P. Ex. 16 at 1. It was also noted that "Ryan is improving. His reflexes have returned, and he has begun to improve his strength in both biceps, wrist extension, and left ankle dorsiflexion." Id. at 2.

III. DISCUSSION

Causation in Vaccine Act cases can be established in one of two ways: either through the statutorily prescribed presumption of causation or by proving causation-in-fact. Petitioners must prove one or the other in order to recover under the Act. According to §13(a)(1)(A), claimants must prove their case by a preponderance of the evidence.[9]

For presumptive causation claims, the Vaccine Injury Table lists certain injuries and conditions which, if found to occur within a prescribed time period, create a rebuttable presumption that the vaccine caused the injury or condition. 42 U.S.C. §300aa-14(a). Neither GBS nor CIDP are an injury listed on the Vaccine Injury Table and thus do not benefit from the Act's presumed causation. Id. Thus, petitioner must prove that the vaccine in-fact caused Ryan's injury, a so-called "off-Table" case.

To demonstrate entitlement to compensation in an off-Table case, petitioners must affirmatively demonstrate by a preponderance of the evidence that the vaccination in question more likely than not caused the injury alleged. See, e.g., Bunting v. Secretary of HHS, 931 F.2d 867, 872 (Fed. Cir. 1991); Hines v. Secretary of HHS, 940 F.2d 1518, 1525 (Fed. Cir. 1991); Grant v. Secretary of HHS, 956 F.2d 1144, 1146, 1148 (Fed. Cir. 1992). See also §§11(c)(1)(C)(ii)(I) and (II). To meet this preponderance of the evidence standard, "[petitioners must] show a medical theory causally connecting the vaccination and the injury." Grant, 956 F.2d at 1148 (citations omitted); Shyface v. Secretary of HHS, 165 F.3d 1344, 1353 (Fed. Cir. 1999). A persuasive medical theory is shown by "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury." Hines, 940 F.2d at 1525; Grant, 956 F.2d at 1148; Jay v. Secretary of HHS, 998 F.2d 979, 984 (Fed. Cir. 1993); Hodges v. Secretary of HHS, 9 F.3d 958, 961 (Fed. Cir. 1993); Knudsen v. Secretary of HHS, 35 F.3d 543, 548 (Fed. Cir. 1994). Furthermore, the logical sequence of cause and effect must be supported by "[a] reputable medical or scientific explanation" which is "evidence in the form of scientific studies or expert

[9] A preponderance of the evidence standard requires a trier of fact to "believe that the existence of a fact is more probable than its nonexistence before the [special master] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence." 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).

medical testimony." Grant, 956 F.2d at 1148; Jay, 998 F.2d at 984; Hodges, 9 F.3d at 960.[10] See also H.R. Rep. No. 99-908, Pt. 1, at 15 (1986), reprinted in 1986 U.S.C.C.A.N. 6344.

---

[10]The general acceptance of a 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. Daubert v. Merrell Dow Pharmaceuticals, Inc., 509 U.S. 579, 594 (1993). Although the Federal Rules of Evidence do not apply in Program proceedings, the United States Court of Federal Claims has held that "Daubert is useful in providing a framework for evaluating the reliability of scientific evidence." Terran v. Secretary of HHS, 41 Fed. Cl. 330, 336 (1998), aff'd, 195 F.3d 1302, 1316 (Fed. Cir. 1999), cert. denied, Terran v. Shalala, 531 U.S. 812 (2000). In Daubert, the Supreme Court noted that scientific knowledge "connotes more than subjective belief or unsupported speculation." Daubert, 509 U.S. at 590. Rather, some application of the scientific method must have been employed to validate the expert's opinion. Id. In other words, the "testimony must be supported by appropriate validation – i.e., 'good grounds,' based on what is known." Id. Factors relevant to that determination may include, but are not limited to:

> Whether the theory or technique employed by the expert is generally accepted in the scientific community; whether it's been subjected to peer review and publication; whether it can be and has been tested; and whether the known potential rate of error is acceptable.

Daubert v. Merrell Dow Pharmaceuticals, Inc., 43 F.3d 1311, 1316 (9th Cir. 1995) (Kozinski, J.), on remand, 509 U.S. 579 (1993); see also Daubert, 509 U.S. at 592-94.

However, the court also cautioned about rejecting novel scientific theories that have not yet been subjected to peer review and/or publication. The court pointed out that the publication "does *not* necessarily correlate with reliability," because "in some instances well-grounded but innovative theories will not have been published." Daubert, 509 U.S. at 594. However, the Supreme Court's only guidance to lower courts in determining the reliability of a novel proposition is that

> . . . submission to the scrutiny of the scientific community is a component of "good science," in part because it increases the likelihood that substantive flaws in methodology will be detected. The fact of publication (or lack thereof) in a peer reviewed journal thus will be a relevant, though not dispositive, consideration in assessing the scientific validity of a particular technique or methodology on which an opinion is premised.

Id. at 593-94; see Gall v. Secretary of HHS, No. 91-1642V, 1999 WL 1179611, at *8 (Fed. Cl. Spec. Mstr. Oct. 31, 1999).

While petitioners need not show that the vaccine was the sole or even predominant cause of the injury, petitioners bear the burden of establishing "that the vaccine was not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Shyface, 165 F.3d at 1352-53. Petitioners do not meet their affirmative obligation to show actual causation by simply demonstrating an injury which bears similarity to a Table injury or to the Table time periods. Grant, 956 F.2d at 1148. See also H.R. Rep. No. 99-908, Pt. 1, at 15 (1986), reprinted in 1986 U.S.C.C.A.N. 6344. Nor do petitioners satisfy this burden by merely showing a proximate temporal association between the vaccination and the injury. Grant, 956 F.2d at 1148 (quoting Hasler v. United States, 718 F.2d 202, 205 (6th Cir. 1983), cert. denied, 469 U.S. 817 (1984) (stating "inoculation is not the cause of every event that occurs within the ten day period [following it]. . . . Without more, this proximate temporal relationship will not support a finding of causation")); Hodges, 9 F.3d at 960. Finally, petitioners do not demonstrate actual causation by solely eliminating other potential causes of the injury. Grant, 956 F.2d at 1149-50; Hodges, 9 F.3d at 960.

The Court of Federal Claims has also weighed in on the evidentiary standards for causation-in-fact in Vaccine Act cases. For example, with respect to the issue of timing, although showing a proximal temporal relationship to the administration of the vaccine is not enough to satisfy a petitioner's burden, a temporal relationship can be a critical part of a diagnosis. Hocraffer v. Secretary of HHS, No. 99-533V, 2005 WL 352552 at *13 (Fed. Cl. 2005). In a case where "the temporal relationship between the exposure to the questioned event and the onset of symptoms is critical to a diagnosis, the temporal relationship is highly probative." Hocraffer, 2005 WL 352552 at * 13. When a petitioner demonstrates a strong temporal relationship between the injury and the administration of a vaccine, a petitioner must also provide "a reliable medical or scientific theory explaining a causal link, but under a less stringent standard than would be required if the temporal relationship was less probative of a causal link." Id. (citing Golub v. Secretary of HHS, No. 99-5161, 2000 WL 1471643, at *3) (Fed. Cir. Oct. 3, 2000) (unpublished opinion).

Additionally, the Court of Federal Claims has also found that when a petitioner presents scientific or medical evidence that demonstrates a direct causal relationship between a vaccine and an injury, "then proof of causation-in-fact via *direct causation* is the preferred path." Pafford v. Secretary of HHS, No. 01-0165V, 2005 WL 4575936 at *8 (Fed. Cl. 2005). Evidence of direct causation can be found from "an epidemiologic study demonstrating a relative risk greater than two . . . or dispositive clinical or pathological markers evidencing a direct causal relationship." Id. (citing Stevens v. Secretary of HHS, No. 2001 WL 387418 at *12). When a petitioner is unable to present epidemiologic evidence or vaccine footprints, a petitioner must prove causation in fact through the use of circumstantial evidence. Pafford, 2005 WL 4575936 at *9. Such circumstantial evidence may include: epidemiology (evidencing a relative risk greater than two), animal studies, case reports/case series studies, anecdotal reports, manufacturing disclosures, Physician Desk Reference citations, journal articles, institutional findings (such as those reported by the Institute of Medicine), novel medical theories, treating physician testimony, and non-dispositive, but inferential clinical and laboratory studies. Id.

The court in Pafford went on to explain that the speculative nature of proving causation-in-fact in Vaccine Act cases with circumstantial evidence, however, requires that a petitioner do much more of the 'heavy lifting' than in an on-Table case or even in an off-Table case where there is direct evidence. Id. (citing Lampe v. Secretary of HHS, 219 F. 3d 1357, 1360 (Fed. Cir. 2000)). Once biologic plausibility is established, a petitioner must demonstrate a nexus between the proposed mechanism and the actual injury, that the vaccine more likely than not caused the injury. Pafford, 2005 WL 387418 at *9.[11] If a petitioner's proposed biologic mechanism is beyond the realm of plausibility, than other circumstantial evidence, no matter how probative, cannot overcome the petitioner's failure to establish biologic plausibility. Id. at *11. A special master, however, "does not act contrary to the law and commit reversible error if he determines that a plausible biologic mechanism and a literal temporal relationship, alone, do not establish a causal link." Id.

Nonetheless, proof that a vaccine can cause a particular injury is not per se proof that it caused the injury in petitioner's case. Id. To be sure, a petitioner should present more proof than just biologic plausibility combined with a strong temporal relationship to buttress his argument that the vaccine in-fact caused his injury. Id. The elimination of other causes as well as the establishment of a scientifically appropriate temporal relationship weigh significantly in a special master's evaluation of the evidence. Id.

This case presents primarily three issues. First, whether CIDP and GBS are distinct and separate diseases. Second, whether Ryan suffers from CIDP or GBS. And third, whether the TT vaccination can cause CIDP and, if so, did it do so in this case. After a review of the record, the court finds that: (1) GBS and CIDP are two separate diseases; (2) Ryan suffers from CIDP; and (3) petitioners failed to offer sufficient proof that the TT vaccination can and did cause Ryan's CIDP. A complete discussion of these issues follows.

A. The Parties' Arguments

Petitioner's Position

Petitioner argues that Ryan has satisfied the requirements of § 300aa-13(a)(1) in demonstrating by a preponderance of the evidence that the TT vaccination likely caused his acquired inflammatory demyelinating polyneuropathy. P. Brief at 13. Petitioner contends Ryan has demonstrated that "there is not a preponderance of evidence that his injury was due to 'factors unrelated' to the TT." Id. (citing § 13(a)(1)(B)). Petitioner maintains that Ryan's medical records alone establish his right to entitlement. Id. at 14. Petitioner further asserts that this evidence is bolstered by the expert opinion of Dr. Tornatore and petitioner's belief that respondent's expert provides further support for Ryan's claim. Id. at 15, 25.

[11] But see, Capizzano v. Secretary of HHS, No. 00-759V, 2004 WL 1399178, at *8 n.21 (Fed. Cl. Spec. Mstr. June 8, 2004) (citing Knudsen v. Secretary of HHS, 35 F.3d 543, 549 (Fed. Cir. 1994) (to require identification and proof of specific biologic mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program)).

Respondent's Position

Based on Ryan's medical records and Dr. Chaudhry's testimony, respondent argues that Ryan suffers from CIDP, not GBS. R. Brief at 10. Respondent contends that CIDP and GBS are distinct clinical entities, which petitioner has not shown to have the same cause. Id. at 11. Respondent asserts that petitioner has failed to provide any compelling evidence to establish that TT can cause CIDP, specifically no medical literature or epidemiological evidence. Id. at 15. Assuming *arguendo* that the undersigned concludes that the TT vaccine can cause CIDP, respondent maintains that petitioner has not proven by a preponderance of the evidence that the TT vaccine actually caused Ryan's CIDP. Id. at 18.

B. Analysis

Dr. Tornatore's fundamental argument is that any distinctions between GBS and CIDP are artificial and unnecessary because GBS and CIDP are merely acute and chronic variations of the same disease process. As he views GBS and CIDP as essentially the same disease, Dr. Tornatore maintains that, because the TT vaccination has been causally linked to GBS, the TT vaccination may also cause CIDP.[12] However, based on the medical literature and Dr. Chaudhry's more convincing testimony, the court cannot accept Dr. Tornatore's position. Accordingly, before addressing the issue of whether the tetanus toxoid vaccine can cause CIDP, the court will discuss its reasoning for concluding that, despite Dr. Tornatore's contention to the contrary, GBS and CIDP are in fact two separate diseases.

Before addressing that issue, it must be noted that the court found Dr. Tornatore to be a marginal witness. While he is clearly well qualified, his testimony strayed from accepted medical principals into speculative, argumentative, and unsupported statements. The undersigned suspects that Dr. Tornatore misunderstood his role. As stated by the American Medical Association ("AMA"): "The medical witness must not become an advocate or a partisan in the legal proceeding." AMA COUNCIL ON ETHICAL AND JUDICIAL AFFAIRS, Code of Medical Ethics (2002-2003 ed.), at 9.07 "Medical Testimony." Dr. Tornatore, however, appeared to make every effort,

[12]Special Masters have found a tetanus vaccine in fact caused GBS. See White v. Secretary of HHS, No. 98-426V, 2002 WL 1488764 (Fed. Cl. Spec. Mstr. May 10, 2002); Watson v. Secretary of HHS, No. 96-639, 2001 WL 1682537 (Fed. Cl. Spec. Mstr. Dec. 18, 2001); Housand v. Secretary of HHS, No. 94-441V, 1996 WL 282882 (Fed. Cl. Spec. Mstr. May 13, 1996); Guy v. Secretary of HHS, No. 92-779V, 1995 WL 103348 (Fed. Cl. Spec. Mstr. Feb. 21, 1995); Alberding v. Secretary of HHS, No. 90-3177V, 1994 WL 110736 (Fed. Cl. Spec. Mstr. March 18, 1994); Robinson v. Secretary of HHS, No. 91-01V, 1991 WL 268650 (Fed. Cl. Spec. Mstr. Nov. 27, 1991). **However**, they have not found causation in-fact for CIDP. See Trojanowicz v. Secretary of HHS, No. 95-215, 1998 WL 774338 (Fed. Cl. Spec. Mstr. July 1, 1998), aff'd, 43 Fed. Cl. 469 (Fed. Cl. 1999); O'Leary v. Secretary of HHS, No. 90-1729V, 1997 WL 254217 (Fed. Cl. Spec. Mstr. Apr. 4, 1997); Coultas v. Secretary of HHS, No. 93-0081V, 1995 WL 605559 (Fed. Cl. Spec. Mstr. Sept. 29, 1995).

no matter how thinly supported, to advocate petitioner's position. It was unhelpful testimony for the court and ultimately unhelpful to petitioner.[13]

In contradistinction, Dr. Chaudhry's testimony was marked by experience, cogent explanations, and literature and textbook support. His testimony was highly persuasive and its contrast to Dr. Tornatore's testimony highlighted even further the dubious quality of Dr. Tornatore's assertions.

Are GBS and CIDP two distinct diseases?

Both experts agreed that CIDP and GBS are immune inflammatory peripheral nerve diseases. Tr at 9, 121. However, beyond that their agreement ended. Arguing that CIDP, GBS and all other autoimmune disorders, share a "commonality," petitioner's expert, Dr. Tornatore,[14] stated, "It's important not to be a splitter in these things, but to be a lumper and to realize that these are inflammatory conditions, in that our treatment modalities frequently are identical for all of them." Id. at 10. Respondent's expert, Vinay Chaudhry, M.D.,[15] disagreed. Although

---

[13]In Thompson v. Secretary of HHS, No. 99-436V, 2003 WL 21439672 (Fed. Cl. Spec. Mstr. May 23, 2003), Special Master Millman similarly noted Dr. Tornatore's position as an advocate rather than expert in both the case at hand and two earlier cases:

> Dr. Tornatore referred in his testimony to "our case." He apparently takes a highly personal view of his testimony so that he has become a team player. In order to make "his" case, Dr. Tornatore ignores the medical records and creates facts which support his assertions which, in themselves, are not based on his clinical practice or knowledge. He has done this before. See Morris v. Secretary of HHS, NO. 99-412V, 2002 WL 31965739 (Fed. Cl. Spec. Mstr. Dec. 18, 2002), appeal docketed (Fed. Cl. Jan. 18, 2003), and Bruesewitz v. Secretary of HHS, No. 95-0266V, 2002 WL 31965744 (Fed. Cl. Spec. Mstr. Dec. 20, 2002) (DPT does not cause afebrile seizures).

Thompson, 2003 WL 21439672 at * 6.

[14]Dr. Tornatore is a board-certified neurologist and professor of medicine at Georgetown University Medical Center. P. Ex. 19. Dr. Tornatore is the Director of the MS and Associated Autoimmune Disorders Clinic at Georgetown Hospital. Tr. at 6. Dr. Tornatore estimated that he sees about 10 to 15 patients over a couple of months with CIDP. Id. at 16. Dr. Tornatore has published approximately 45 articles; however, none of these publications are on either CIDP or GBS. Id. at 10, 14.

[15]Dr. Chaudhry is a professor of neurology at the Johns Hopkins University School of Medicine. Tr. at 100. Dr. Chaudhry has nine publications in peer-reviewed journals, three book chapters, and four review articles on CIDP and GBS. Id. at 102. He has made at least 15 to 20 (continued...)

concurring with Dr. Tornatore that CIDP and GBS are both autoimmune diseases, Dr. Chaudhry maintained that "it's not just a question of lumper and splitter, as was related to before. They [CIDP and GBS] are two distinct diseases. Id. at 124.

Dr. Tornatore opined that the TT vaccination caused Ryan's inflammatory neuropathy. Tr. at 60. Dr. Tornatore testified that Ryan was initially given several diagnoses; first, GBS and then more of a "CIDP-like picture." Id. at 19-20. Dr. Tornatore explained that an acute course of inflammatory neuropathy, such as GBS, can become chronic. Id. at 22-24. He further opined that distinctions between relapsing, progressive and chronic inflammatory demyelinating polyneuropathies are artificial. Id. 25-26.

In support of his assertion that any distinction between the chronic and relapsing form of GBS is artificial, Dr. Tornatore cited a passage from Respondent's Exhibit Q,[16] in which the authors state:

> Acute Inflammatory polyneuritis, CIDP, and the experimental autoimmune diseases EAN and chronic EAN, produced by sensitizing animals to peripheral nerve myelin or the P-2 basic protein of peripheral nerve myelin, bear a close resemblance to each other clinically and pathologically, suggesting that these are related diseases with a common pathogenesis.

Id. at 1509; Tr. at 27-29; P. Brief at 23.

However, in the same paragraph the authors also note that "[t]he evidence of cellular or humoral sensitivity to a myelin antigen in the human diseases is, however, inconclusive." R. Ex. Q at 1509. The court probed Dr. Tornatore about whether his assertion that CIDP and relapsing, recurring AIDP are the same was contradicted by an earlier page in the same article, which states, "AIDP may recur, but such recurring cases are not generally included in CIDP." Tr. at 31; R. Ex. Q at 1500. Dr. Tornatore maintained that it was not. Tr. at 31; R. Ex. Q at 1500. Dr. Tornatore could not cite to any literature submitted by petitioner to further support his position. Tr. at 32.

Moreover, Dr. Chaudhry countered Dr. Tornatore's assertions that Ryan suffered from relapsing GBS. Tr. at 116. Dr. Chaudhry testified that, although the entity is uncommon, he has

---

[15](...continued)
presentations on GBS and CIDP in national meetings and conducted several courses at both the national and international level regarding the treatment and diagnosis of different immune-mediated neuropathies. Id. at 103. Dr. Chaudhry performs electrodiagnostic testing on about 25 patients per week with peripheral nerve disease. Id. at 104. According to Dr. Chaudhry, during this testing the main question asked is whether the disease is CIDP or a demyelinating disease. Id.

[16]P.J. Dyck et al., Chronic Inflammatory Demyelinating Polyradicalreuropathy, in PERIPHERAL NEUROPATHY 1498, 1509 (W.B. Sanders Co., 1993).

seen patients with relapsing GBS. Id. He explained that such a diagnosis involves rapidly-progressive weakness lasting less than four weeks per episode. Id. at 116-117. Dr. Chaudhry believed that Ryan did not experience such rapid weakness; instead Ryan suffered from a "progressive evolving disease." Id. at 117. Dr. Chaudhry stated that the only time at which there was a possibility for a diagnosis of relapsing GBS was due to the tapering of Ryan's medication, which caused a temporary return of symptoms. Id.

Dr. Tornatore's fundamental argument was that CIDP and GBS share the same pathogenesis, and the only difference between the two is that GBS is acute while CIDP is chronic. Tr. at 41. According to Dr. Tornatore, the real distinction between the two is the "tempo," or strength of the immune response. Id. at 37. Dr. Tornatore stated that with GBS, patients would experience "greater symptoms within a very short period of time," while with CIDP, patients would have a "more prolonged course" due to a weaker immune response. Id. Dr. Tornatore observed that there also exists a "sub-acute" form of inflammatory neuropathy, which lies somewhere between GBS and CIDP on the spectrum. Id. at 49. According to Dr. Tornatore, the sub-acute variety shares the same immunology and the same "cellular infiltrate with demyelination" as GBS and CIDP. Id. at 49, 50.

In support of the hypothesis that GBS and CIDP share the same pathogenesis, Dr. Tornatore cited a passage from a 1993 article by J.D. Pollard. Tr. 66-67; see P. Ex. 18.[17] In this article, based on an animal study, Dr. Pollard concluded that "[t]his spectrum of clinical manifestations of EAN [experimental allergic neuritis] lends support to the view that GBS and its chronic variant, chronic inflammatory demyelinating polyradiculoneuropathy (CIDP), are part of a single disease process." P. Ex. 18 at 147. However, this statement alone is not strong enough for the court to conclude that GBS and CIDP are the same disease process. As discussed at hearing, and acknowledged by Dr. Tornatore, animal studies do not translate exactly into humans. Tr. at 67.

Contrary to Dr. Tornatore's testimony, Dr. Chaudhry asserted that GBS and CIDP are "absolutely distinct, different diseases." Tr. 108. First, two-thirds of patients diagnosed with GBS have an inciting event, with approximately fifty to sixty percent of those patients presenting with a campylobacter infection. Id. at 109. On the other hand, Dr. Chaudhry estimated that less than one percent of those patients diagnosed with CIDP note an inciting event. Id. Second, Dr. Chaudhry explained that ninety percent of GBS patients peak within four weeks, and at Johns Hopkins, a patient progressing beyond this point would not be given a diagnosis of GBS. Id. With CIDP, however, the disease continues to progress for long periods of time. Id. at 115-114. Further, the clinical features are different. Id. at 110. Most people suffering from GBS experience symptoms of ascending paralysis, involving facial and respiratory muscles, which are not typically seen in CIDP. Id. at 110-111. Also, the course of progression of the two diseases differs. Id. at 112. According to Dr. Chaudhry, typically GBS is monophasic and CIDP is

[17] J.D. Pollard, Immunopathology of Guillain-Barré Syndrome, in GUILLAIN BARRÉ SYNDROME 146, 147 (Thieme Med. Pub., 1993).

chronic or relapsing/remitting. Id. at 113. This means that GBS is usually a one-time illness, while CIDP is an illness in which one continues to worsen over time. Id. Medication tends to improve CIDP, and relapses may occur when the medicine is tapered or when the optimal dose is not administered. Id. In sum, Dr. Chaudhry concluded that there are five reasons that lead him to believe that GBS and CIDP differ. These are: "the inciting event, the time that it peaks, the factor that the breathing muscles and the aurinomic [sic] and the facials are not affected, the phasic illness, monophasic, and the response to treatment."[18] Id.

The court notes that Dr. Tornatore's argument attempting to analogize CIDP to GBS is almost identical to the argument made by petitioner's expert, Dr. Bean, in Trojanowicz v. Secretary of HHS, No. 95-215, 1998 WL 774338 (Fed. Cl. Spec. Mstr. July 1, 1998), aff'd, 43 Fed. Cl. 469 (Fed. Cl. 1999), a case heard and decided by the undersigned. In fact, in Trojanowicz, Dr. Bean presented the same article that was offered by Dr. Tornatore in this case to support petitioner's analogy argument. See Trojanowicz, 1998 WL 774338 at * 4. Specifically, Dr. Bean testified that the Dyck and Thomas paper "supports the close association between the acute and chronic cases and points out the immunogenic triggers that are supposed in both..." Id. However, the court noted that Dr. Bean had failed to submit a prior page which contradicted his argument. This contradictory passage stated:

> CIDP, like AIDP, is an inflammatory demyelinating polyradiculoneuropathy with cytoalbuminologic dissociation. As the mechanisms underlying AIDP and CIDP are unknown, it is possible that both syndromes are variants of the same disorder, as their shared pathologic features might suggest. ***On the other hand, cogent reasons for separating CIDP and AIDP, whether this separation ultimately proves to have fundamental validity or not, can be advanced***.

Id. (citing R. Ex. F at 1500 (emphasis added)). The court related that, when questioned about this quote, "Dr. Bean's response was a very unacceptable and highly unpersuasive 'there's a lot that's unknown, but there is some evidence that there are similar causes for both conditions at times, possibly.'" Id. (quoting Tr. at 43).

In striking resemblance, Dr. Tornatore offered the same Dyck article, found in this case as Respondent's Exhibit Q, to support his theory that CIDP and GBS share the same inciting events. Tr. at 69-73; see R. Ex. Q at 1501. In attempting to demonstrate this, Dr. Tornatore cited from a section of the article which offered examples of antecedent events in patients with CIDP. Tr. at 70-71; R. Ex. Q at 1501.

> THE WITNESS: ...And then if we go to the very last paragraph, or the very last sentence. It says, "The controls as cited are not totally satisfactory.

[18]While not wanting to "go into the pathogenesis," Dr. Chaudhry noted that "there is some evidence now accumulating, and it's all coming up that in fact the pathogenesis of the two diseases is different." Tr. at 113.

And whether the incidence of antecedent infection in CIDP cases exceeds what would be expected by chance alone is not known." However, they go on to make point here that initially they thought there was antecedent events.

THE COURT: Sure. But I think the conclusion is kind of important, don't you? It's not known.

THE WITNESS: ***Well, we don't know. But it doesn't mean that there isn't.*** But at some point, people –

THE COURT: Well, when you're being asked for positive support, ***not knowing is not positive support.***

THE WITNESS: ***I don't know if I agree with that.***

Tr. at 71 (emphasis added).

As determined in Trojanowicz, the court finds in this case that no medical article submitted or cited analogizes CIDP to GBS. Further, as the undersigned noted about Dr. Bean in Trojanowicz, Dr. Tornatore similarly has been "either highly selective in pulling support from the submitted articles or ignored clearly contradictory portions of the same articles." Id. at * 4.

The court has canvassed the medical literature submitted in this case and has determined that petitioner failed to provide sufficient support for his argument that GBS and CIDP are essentially the same entity. In fact, the literature demonstrates that GBS and CIDP are distinct and separate diseases. For example, Petitioner's Exhibit 13-D states:

> The classification of inflammatory polyneuropathies into acute and chronic is based partly on clinical course. Both conditions present as predominantly motor neuropathies with elevated CSF protein levels and have similar electrophysiological features of conduction block and slowing of motor conduction velocities. The pathologic signs of demyelination and inflammation are common to acute and chronic varieties. ***However, there are striking variations in the clinical course and other differences that include prognosis, precipitating events, pathological features, and response to treatment.***

P. Ex. 13-D at 214 (J.D. Pollard, A Critical Review of Therapies in Acute and Chronic Inflammatory Demyelinating Polyneuropathies, 10 MUSCLES & NERVE 214 (1987)) (emphasis added).

The distinction between the two diseases is supported further by other medical articles in the record. See, e.g., R. Ex. C at 54 (listing clinical features that distinguish CIDP from GBS); R. Ex. O at 626 (stating that "CIDP may be a chronic variety of GBS, although there certainly are clear clinical and immunological differences"). This distinction is also evidenced by excerpts from a medical textbook, in which the two disorders are discussed in two different chapters, each of which specifically indicates the clinical and

laboratory features, diagnostic criteria, and pathogenesis separately. See, e.g., R. Ex. K, L (Chapter 10, entitled "Chronic Inflammatory Demyelinating Polyradiculoneuropathy," and Chapter 9, entitled "Guillain-Barré Sydnrome," respectively, found in DIAGNOSIS AND TREATMENT OF PERIPHERAL NERVE DISORDERS).

The court finds that Dr. Tornatore failed to offer sufficient, objective support demonstrating that CIDP and GBS have the same pathogenesis. Accordingly, petitioner's argument that the tetanus vaccine can cause CIDP merely because the tetanus vaccine can cause GBS must fail. As the undersigned concluded previously in Trojanowicz v. Secretary of HHS, No. 95-215, 1998 WL 774338 (Fed. Cl. Spec. Mstr. July 1, 1998):

> Despite Dr. Bean's good faith efforts to support this case, it is logically and legally impermissible to extrapolate from similarities in pathogenesis to a conclusion of shared causative agents in light of the lack of support from the medical literature, or some type of objective support from the relevant medical community, and in the face of medical literature indicating strong differences in antecedent events.

Id. at * 5. Based on a review of the medical literature and the experts' testimony, the court finds that CIDP and GBS are two distinct and separate diseases.

Did Ryan Suffer from CIDP or GBS?

As the court found that CIDP and GBS are separate diseases, it must now determine which disease Ryan suffered. Consistent with Dr. Tornatore's hearing testimony, the medical records demonstrate that Ryan's diagnosis during the early stages of his illness was uncertain. P. Ex. 3 at 1-2; Tr. at 19-20. Initially, it was believed that Ryan suffered from GBS; however, later Ryan was diagnosed with CIDP. P. Ex. 1 at 20. Dr. Tornatore maintained that classifying Ryan as suffering from CIDP versus GBS was based on "arbitrary definitions." Tr. 31. However – although unwilling to deviate from his opinion that CIDP and GBS are essentially the same entity – in response to questioning about specifically what type of inflammatory neuropathy Ryan suffered, Dr. Tornatore conceded, "if we had to use these diagnostic criteria, he would fall into the, probably into the CIDP category, just based on the fact that he wasn't at his nadir by three to four weeks." Tr. at 61.

Dr. Chaudhry gave clear and compelling testimony, presenting the clinical criteria used for distinguishing between CIDP and GBS. Dr. Chaudhry, a leading expert on GBS and CIDP, provided five reasons for clinically distinguishing the two "distinct, different diseases." Tr. at 108, 110-113; see discussion, supra, page 10. Dr. Chaudhry testified that the condition with which Ryan was diagnosed is CIDP. Tr. at 105. Dr. Chaudhry reached this conclusion by evaluating the clinical features of Ryan's illness against the clinical course and diagnostic criteria for CIDP. Tr. at 106. Specifically in Ryan, Dr. Chaudhry observed that the weakness in all four of his limbs, absence of reflexes, high spinal fluid protein, and an EMG showing demyelination were all "absolutely typical features" of CIDP. Id. In addition, Dr. Chaudhry found Ryan's responsiveness

to steroids, another typical feature of CIDP, to be indicative of a diagnosis of CIDP.[19] Id. at 107. Dr. Chaudhry concluded that Ryan presented with all of the features of CIDP, "including the onset, the progression, the chronicity, the response to medications, the [responsiveness to] steroid in the beginning, the spinal fluid protein, the EMG." Id. at 108. Dr. Chaudhry opined that it is not uncommon, and in fact the practice of his institution, for patients to receive several different diagnoses before reaching a final diagnosis of CIDP. Tr. at 114. Dr. Chaudhry explained that this is due to the nature of CIDP, which in differing from GBS, progresses for long periods of time. Id. at 114-115.

Dr. Tornatore refers to the "arbitrary criteria" used in distinguishing CIDP and GBS. However, as discussed above, he could not provide solid evidence that CIDP and GBS are a single disease process. Dr. Chaudhry, on the other hand, provided more convincing testimony, based on his extensive clinical experience, demonstrating his reasoning for concluding that Ryan suffered from CIDP. Hence, based on Dr. Chaudhry's testimony coupled with medical records and medical literature, the court concludes that Ryan suffered from CIDP.

Can the Tetanus Toxoid Vaccination Cause CIDP?

Finding that Ryan suffered from CIDP, the court must now decide whether the tetanus toxoid vaccination can cause CIDP and, if so, whether it caused Ryan's CIDP. Based on the temporal relationship between the vaccine and Ryan's illness, the biologic plausibility demonstrated in the Pollard and Selby article, and the antecedent event of the tetanus toxoid vaccination, Dr. Tornatore concluded that the TT vaccination caused Ryan's illness. Tr. at 96-97. As discussed previously, Dr. Tornatore testified that GBS and CIDP share the same underlying pathogenesis and the same causation mechanism. Id. at 66. Thus, petitioner takes the position that because the tetanus toxoid vaccine is known to cause GBS,[20] it can also cause CIDP, which is merely a chronic variant to the same disorder. Id. at 59-60; P. Brief at 24 (citing P. Ex. 13 at Tabs A,C, E, and F). However, the court found above that Dr. Tornatore failed in his attempt to analogize the two diseases. As such, petitioner cannot link the tetanus vaccination to CIDP based solely on the vaccination's possible causal relationship with GBS.

In support of the assertion that the TT vaccination can cause CIDP, Dr. Tornatore cited the

[19]Dr. Chaudhry noted that in fact, treatment with steroids in GBS patients causes them to get worse. Tr. at 108.

[20]Regarding whether tetanus toxoid containing vaccines are capable of causing GBS, the Institute of Medicine has concluded that the evidence "favors a causal relation." Kathleen R. Stratton et al., Institute of Medicine, Diphtheria and Tetanus Toxoids, in ADVERSE EVENTS ASSOCIATED WITH CHILDHOOD VACCINES: EVIDENCE BEARING ON CAUSALITY 67, 86-90 (1994) ("IOM Report"). See also n. 9, supra, for a list of cases in which special masters have found that tetanus in fact caused GBS.

Fenichel article. Id. at 55; see P. Ex. 17.[21] In this article, Dr. Gerald Fenichel discusses the well-known case report by Selby and Pollard.[22] Specifically, Dr. Fenichel's article states that the patient discussed in the Selby and Pollard report,

> developed three episodes of Guillain Barré syndrome (GBS) after three doses of tetanus toxoid. The episodes were separated by 9 years, and the intervals between immunization and onset of symptoms were 3 weeks, 2 weeks, and 9 days. The patient subsequently experienced additional relapses without prior immunization and was diagnosed as having chronic inflammatory demyelinating polyneuropathy (CIDP). ***It is not possible to know whether tetanus toxoid caused or triggered the CIDP in the susceptible individual.***

P. Ex. 17 at 1548 (emphasis added); see also Tr. at 55-56. However, despite Dr. Fenichel's statement that the patient in the Selby and Pollard case report suffered CIDP, the Selby and Pollard case report itself provides only that the patient suffered from an "acute idiopathy polyneuropathy (Landry-Guillain-Barré-Strohl Syndrome)"or GBS, and does not indicate that he suffered from CIDP.[23] See P. Ex. 13, Tab E. Further, although Dr. Tornatore failed to read the last sentence of the paragraph he cited (provided above with emphasis) during his testimony, the complete excerpt demonstrates that the Fenichel article is inconclusive about the relationship between TT and CIDP. Dr. Tornatore stated that other than the Selby and Pollard article, he was unaware of any articles that draw a relationship between CIDP and the TT vaccine. Id. at 98.

Dr. Chaudhry testified that he was not aware of any epidemiological studies, medical literature or support from the medical community indicating that tetanus toxoid can cause CIDP. Id. at 131. He also stated that he does not believe that CIDP and GBS share the same causative agents. Id. Dr. Chaudhry concluded that Ryan suffered from CIDP, which was not caused by the tetanus shot he received on March 22, 1999. Id.

Dr. Tornatore provided insufficient evidence to support his argument that the TT vaccine can cause CIDP. Dr. Tornatore testified that the tetanus toxoid can cause CIDP; however, he conceded that there are no epidemiologic studies to support this assertion. Tr. at 76. While the IOM did conclude that there is sufficient evidence favoring the acceptance of a causal relationship between the tetanus vaccination and GBS, it did not find a causal relationship between tetanus and

---

[21]G.M. Fenichel, Assessment: Neurologic Risk of Immunization, 52 NEUROLOGY 1546, 1548 (1999).

[22]See P. Ex. 13, Tab E (J.D. Pollard & G. Selby, Relapsing Neuropathy Due to Tetanus Toxoid, 37 J. NERUOL. SCI. 113 (1978)).

[23]The parties attempted to contact Dr. Pollard regarding the possibility that the patient discussed in their case report suffered from CIDP. However, during a telephonic status conference held on April 29, 2004, the parties reported that they had been unsuccessful in their attempt.

CIDP. See P. Ex. G at 1604.[24] Moreover, as discussed above, the little medical literature presented by petitioner was lacking in any evidence establishing such a causal relationship.

Further, the court notes that, despite petitioner's contention that Ryan's treating physicians believed that the tetanus toxoid vaccination caused his acquired inflammatory demyelinating polyneuropathy, see P. Brief at 15, the medical records do not reflect this assertion. While Dr. Nallainathan did note the possibility of Guillain-Barré secondary to tetanus toxoid in a record of May 17, 1999, P. Ex. 3 at 1-3, after he determined that Ryan suffered from CIDP instead, Dr. Nallainathan no longer noted any connection between Ryan's CIDP and the TT vaccination. See, e.g., P. Ex. 3 at 20. Further, although Dr. Cornblath noted the temporal relationship between Ryan's CIDP and the vaccination, and provided as one of several possible differential diagnoses, "CIDP following the tetanus administration (if this exists)," P. Ex. 7 at 9, the records do not indicate that Dr. Cornblath concluded that the TT vaccination caused Ryan's CIDP.

Based on the lack of support from the medical community and absence of medical literature, the court finds that petitioner's assertion that the TT vaccine can cause CIDP must also fail.[25]

As the court found above that Ryan suffered from CIDP and that there is insufficient evidence to support a finding that the tetanus vaccine can cause CIDP, it follows that the tetanus vaccination could not have caused Ryan's CIDP in this case.

Supplemental Hearing

At the December 21, 2004, supplemental hearing, Dr. Tornatore presented a summary of Petitioner's Exhibits 20 and 21.[26] Transcript II ("Tr. II") at 5-11. Dr. Tornatore explained that Petitioner's Exhibit 20, a letter to the editor of the journal "Muscle and Nerve," is significant in that it presents a personal communication from Dr. Pollard, which helps to clarify the Pollard and Selby article discussed at the March 30, 2004 hearing. Id. at 6. Specifically, the personal

---

[24]K.R. Stratton et al., Adverse Events Associated with Childhood Vaccines Other Than Pertussis and Rubella – Summary of a Report from the Institute of Medicine, 271 JAMA 1602, 1605 (May 25, 1994).

[25]The undersigned rejected a similar argument in Trojanowicz:

> No other articles supported a known studied relationship between CIDP and the tetanus vaccination. In fact, Dr. Bean [petitioner's expert] acknowledged that he was unaware of any case reports of CIDP following the tetanus vaccine. Tr. at 61. Thus, the court finds that there is insufficient evidence in this record to support a finding that the tetanus vaccine can cause CIDP.

Id. at * 5.

[26]The undersigned notes that Dr. Tornatore's testimony at the supplemental hearing was very helpful and did not suffer from the infirmities expressed, supra, at 10-11.

communication reveals that the patient studied in the Pollard and Selby article subsequently was felt to have CIDP. Id. at 8.

With respect to Petitioner's Exhibit 21, Dr. Tornatore explained that the article suggests that the inflammatory neuropathies are a spectrum of disorders, with clinical features that are very hard to distinguish. Tr. II at 10. Thus, the two articles strengthen petitioner's arguments that AIDP and CIDP are hard to distinguish and that a CIDP-like presentation following a tetanus vaccination is possible due to the two disorders' similarities in clinical criteria. Id. at 11.

When questioned about whether letters to the editor are generally peer reviewed, Dr. Tornatore replied vaguely, "[t]hey are usually reviewed by someone....but it may not be peer-reviewed by two separate people." Tr. II at 12. He also acknowledged that it is fair to say that a letter to the editor does not receive as much scrutiny as a regular article might. Id. Further, Dr. Tornatore was questioned about the reference to the IOM's personal communication with Dr. Pollard in "Adverse Events Associated with Childhood Vaccines," found at Respondent's Exhibit T. Id. at 17-19. Dr. Tornatore conceded that it appears the IOM had personal communications with Dr. Pollard when it published the chapter entitled "Guillain-Barré Syndrome." Id. at 19. He also admitted that "the Institute of Medicine clearly says that Guillain-Barre and tetanus, there is an association. They *do not* say that it's CIDP." Id. at 20 (emphasis added). Dr. Tornatore also acknowledged that the IOM has never found a causal relationship between CIDP and tetanus toxoid. Id.

Dr. Chaudhry observed that Petitioner's Exhibit 20 is unclear as to whether the Pollard "personal communication" is referring to CIDP existing within the time frame of the first three relapses. Tr. II at 27-28. Instead, Dr. Chaudhry believes that the authors meant that the patient had three episodes of GBS and then, at some later date after the 1978 Pollard and Selby report, the patient developed CIDP. Id. at 28-29. Moreover, Dr. Chaudhry pointed out that the Pollard and Selby case involved a rechallenge event. Id. at 30. Thus, Dr. Chaudhry explained, to fit petitioner's interpretation of the Pollard and Selby study, even if it is not necessary to prove that Ryan's disease is GBS rather than CIDP, one must show that Ryan received repeated immunizations. Id. In Ryan's case, however, there is no evidence of such. Id.

Dr. Chaudhry testified that he still believes Ryan has CIDP. Tr. II at 31. Acknowledging that there are rare instances where patients do not fall "neatly into place," Dr. Chaudhry did not find Ryan's case to be confusing, stating that it "was absolutely a clear-cut case of CIDP [and] diagnosed and treated as such." Id. Dr. Chaudhry stated that he found neither Exhibit 20 nor 21 changed his position from the earlier hearing that CIDP and GBS are distinct clinical entitles or that tetanus toxoid cannot cause CIDP. Id. at 31-32.

After reviewing the newly submitted evidence and evaluating the expert testimony, the undersigned concludes that this new evidence does not support a finding that tetanus toxoid can cause CIDP, nor does it support a finding that the TT vaccine caused Ryan's CIDP in this case. Petitioner relies on the personal communication between Dr. Pollard and the authors of Exhibit 20 to argue that the patient in the Pollard and Selby study suffered CIDP and thus, tetanus can cause CIDP. However, the undersigned concurs with Dr. Chaudhry that reference to CIDP is not clear

regarding when the illness began. Further, the citation to the "personal communication" with Dr. Pollard is too speculative to rely upon for proof of causation. As noted, the IOM also had personal communication with Dr. Pollard. Subsequent to that communication, the IOM, utilizing Pollard's article, found an association between GBS and tetanus; no mention of CIDP was made. It is reasonable to conclude that Dr. Pollard made no mention of CIDP in his communication with the IOM.

Finally, while the articles may raise questions of blurring of the lines between GBS and CIDP, to which Dr. Chaudhry agreed, the articles do not support Dr. Tornatore's argument that because GBS and CIDP share the same pathology they should also share causation. The sole support for the IOM's conclusion that tetanus can in-fact cause GBS was based on the 1978 Pollard and Selby study involving rechallenge. No such support exists for CIDP. Petitioner argues that the lines blur between GBS and CIDP and thus a rechallenge case of GBS can be extended to apply to CIDP. This may be proven correct someday, but as it stands now there is insufficient evidence that GBS and CIDP are cut from the same cloth so that proof of causation related to GBS can be applied to CIDP.

IV. CONCLUSION

Based on the foregoing, the court finds, after considering the entire record in this case, that petitioner is not entitled to compensation under the Vaccine Act. The court found above that this case fails because the medical records and the experts' testimony support petitioner's diagnosis of CIDP. The court also found that petitioner failed to provide sufficient evidence that the tetanus toxoid vaccination can cause CIDP. Further, petitioner failed to demonstrate that GBS and CIDP are so similar that, merely because the tetanus toxoid vaccine can cause GBS, it can also cause CIDP. Thus, petitioner failed to demonstrate that Ryan's CIDP was caused-in-fact by the TT vaccination. For the reasons discussed above, petitioner fails to qualify for an award under the Program. In the absence of a motion for review filed pursuant to RCFC, Appendix B, the Clerk is directed to enter judgment accordingly.

IT IS SO ORDERED.

Gary J. Golkiewicz
Chief Special Master