In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 11-631V

(to be published)

* *		
ROY GREENE,	*	
	*	
Petitioner,	*	Filed: August 2, 2019
	*	
v.	*	
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Tetanus-Diphtheria ("Td") Vaccine; Evidentiary Support for Onset Timeframe; Expert Opinions; Vaccine Trial Risk Intervals
	*	
	*	
Respondent.	*	
	*	
* *		

Richard Gage, Law Offices of Richard Gage, Cheyenne, WY, for Petitioner.

Brittany Ditto, U.S. Dep't of Justice, Washington, DC, for Respondent.

DECISION DENYING ENTITLEMENT[1]

On September 29, 2011, Roy Greene filed a petition for compensation in the National Vaccine Injury Compensation Program (the "Vaccine Program"),[2] alleging that he developed brachial neuritis as a result of his receipt of the tetanus-diphtheria ("Td") vaccine on July 22, 2009. Pet. (ECF No. 1). Mr. Greene originally asserted both a Table injury claim and a "non-Table" causation-in-fact claim *(id.* at 2), but I dismissed the Table claim after a March 2015 fact hearing, at which time I determined that Petitioner's symptoms arose forty-one days after the vaccination,

[1] This Decision has been designated "to be published," and will therefore be posted on the United States Court of Federal Claims website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, § 205, 116 Stat. 2899, 2913 (Dec. 17, 2002) (current version at 44 U.S.C. § 3501 (2014)). As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision in its present form will be available to the public. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended, 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act.

and thus outside of the twenty-eight-day limit for a brachial neuritis Table claim. 42 C.F.R. § 100.3(a)(I)(B)).

This case has had a tortuous procedural history, but at long last the parties participated in an entitlement hearing on May 9, 2019, at which time both sides offered expert testimony—primarily addressing whether Mr. Greene's brachial neuritis began in a medically acceptable timeframe as measured from the date of vaccination. After listening to that expert testimony, and considering the expert reports and literature offered, I find that Petitioner has not met his burden of establishing by a preponderance that a six-week timeframe for onset of brachial neuritis after receipt of the tetanus vaccine is medically reasonable—or that the vaccine "more likely than not" *did* injure him given the undisputed facts.

Factual History

The facts relevant to the present decision are set forth in my earlier onset fact ruling. *See Greene v. Sec'y of Health & Human Servs.*, No. 11-631V, 2015 WL 9056034, at *1 - 4 (Fed. Cl. Spec. Mstr. July 31, 2015) ("Fact Ruling"). They are incorporated by reference herein. The Fact Ruling was issued after a 2015 hearing at which several witnesses testified, including Petitioner. For present purposes, the most important of the Fact Ruling's findings are as follows:

> (a) Petitioner received the Td vaccine on July 22, 2009, in his right arm after experiencing a significant construction-related injury to his hand at his workplace;
>
> (b) Petitioner saw no healthcare providers in connection with his injury until September 7, 2009 (Labor Day of that year), when he went to a hospital emergency room in Houston, Texas, complaining of sharp pain in his right upper arm that he stated had begun only a few days before—not any time in the month of July or August;
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> (c) after hearing witness testimony and comparing it to the medical records filed in the case, I determined that onset of Petitioner's subsequently-diagnosed brachial neuritis[3] had occurred no earlier than September 1, 2009 (or 41 days post-vaccination); and
>
> (d) based on this fact determination, I dismissed the Table claim, since Petitioner's onset had not been established preponderantly to have occurred within 28 days of administration of the tetanus vaccine.

See generally Fact Ruling at *1–4, *17.

[3] As recognized by the parties and their experts, the term "brachial neuritis" is medically synonymous with the terms "Parsonage Turner Syndrome" or neuralgic amyotrophy. *See, e.g.*, Tr. at 10, 69. I shall use the former as an overall descriptor of Mr. Greene's injury herein, although certain items of literature filed in this case use the other terms in discussing the condition's nature and etiology.

Brief Summary of Relevant Procedural History

Between the date of the Fact Ruling and the fall of 2016, the parties could not settle the non-Table claim. In that period, Petitioner submitted two expert reports from an orthopedist, Thomas W. Wright, M.D. *See* Report dated Dec. 18, 2015, filed as Ex. 22 (ECF No. 62); Report dated Apr. 25, 2016, filed as Ex. 29 (ECF No. 66). But Respondent took issue with the adequacy of the opinions expressed therein—arguing in particular that more was needed on the third *Althen* prong because of the conclusory nature of Dr. Wright's opinion, which relied heavily on the fact that a 41-day onset was only about two weeks longer than what the Table contemplates, rendering the extra time a *de minimis* difference.

In light of Respondent's objections, I proposed that Petitioner obtain an additional expert report addressing the *Althen* prong three issue. *See* Status Conference Order, dated Sept. 29, 2016 (ECF No. 72). Mr. Greene thereafter filed an expert report from Dr. Marcel Kinsbourne on January 6, 2017. Respondent, however, deemed this report similarly inadequate and conclusory. In response (and mindful that the case was now nearly six years old) I proposed that Respondent either file his own expert report or move for a ruling on the record as it stood. *See* Status Conference Order, dated Jan. 26, 2017 (ECF No. 86).

Respondent took the second option, filing a motion to dismiss in March 2017. *See* Motion to Dismiss, dated Mar. 31, 2017 (ECF No. 90) ("Mot."). Respondent argued that the record itself (which at that time included *only* the two Wright expert reports plus the supplemental Kinsbourne report, as well as my fact determination on onset) established "legally insufficient proof" for a favorable entitlement decision and should therefore be dismissed. *Id.* at 1. In particular, Respondent challenged Dr. Wright's attempt to "piggyback" on the Table timeframes for appropriate onset, despite clear Program law establishing that non-Table claims could not do so. *Id.* at 5–6; (citing *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1147–48 (Fed. Cir. 1992) ("[s]imple similarity to conditions or time periods listed in the Table is not sufficient evidence of causation")). Respondent also maintained that Dr. Kinsbourne's report set forth a scientifically unreliable opinion, and was just as conclusory as Dr. Wright's reports in assuming that a 41-day onset period was within what is "generally recognized" as medically reasonable for other autoimmune illnesses, without providing reliable scientific or medical substantiation for that proposition. *Id.* at 8–9.

After the Motion was fully briefed, I granted it, but Petitioner subsequently filed a motion for reconsideration on June 16, 2017, along with two new, supplemental expert reports (one from Dr. Kinsbourne and an additional report from Dr. Vera Byers[4]) as well as several items of previously-unfiled medical literature. ECF Nos. 94–97. I subsequently withdrew my initial dismissal decision in order to evaluate the merits of the reconsideration request, but then denied

[4] The Byers report (which was accompanied by five items of literature) was mistakenly filed twice. *See* Ex. 59. Its contents were not referenced during the May 2019 hearing, and Dr. Byers did not testify there.

entitlement a second time. *Greene v. Sec'y of Health & Human Servs.*, No. 11-631V, 2017 WL 5382856 (Fed. Cl. Spec. Mstr. Sept. 26, 2017) ("Second Dismissal Decision"). The Second Dismissal Decision arose from my determination that Petitioner's experts were improperly relying on the Table timeframes to defend the medical sufficiency of the timeframe for onset of his brachial neuritis. Second Dismissal Decision at *6–7.

In dismissing Mr. Greene's claim, I erroneously conflated the standards applied to evaluating a reconsideration request with the legal standards applicable to entitlement claims generally. Petitioner thus sought review of the Second Dismissal Decision, and the Court of Federal Claims granted his motion on February 27, 2018, remanding this matter back to me for a new disposition of Respondent's original motion, based on all evidence Petitioner had submitted and applying the proper legal standards. *Greene v. Sec'y of Health & Human Servs.*, No. 11-631V, 2018 WL 1514440 (Fed. Cl. Feb. 27, 2018). I thereafter determined that, because Petitioner had now offered sufficient evidence to meet his preponderant burden of proof *if unrebutted*, I could not grant Respondent's request to dismiss the case. Remand Ruling, dated May 7, 2018 (ECF No. 116) ("Remand Ruling"). However, I also found that fairness required that I permit Respondent (based on a prior request) the chance to submit his own expert report on the timeframe question, and that I would hold a hearing thereafter. *Id.* at 11, 16.[5]

Respondent subsequently filed an expert report from Dr. Eric Lancaster on June 14, 2018 (ECF No. 121-1), with Petitioner filing a report from Dr. Laurence Steinman on November 13, 2018 (ECF No. 128-1). This prompted a supplemental expert report filing from Dr. Lancaster on April 9, 2019 (ECF No. 133-1), and then a third expert report from Dr. Kinsbourne on April 29, 2019 (ECF No. 134-1). The hearing was held as scheduled on May 9, 2019, with no post-hearing briefing. The matter is now fully ripe for resolution.

Summary of Expert Testimony

1. *Dr. Marcel Kinsbourne*

Dr. Kinsbourne filed three written reports and testified at hearing. *See* Report, dated Jan. 6, 2017, filed as Ex. 38 (ECF No. 82-1) ("Kinsbourne Rep."); Report, dated June 13, 2017, filed as Ex. 45 (ECF No. 94-1); Report, dated Apr. 26, 2019, filed as Ex. 73 (ECF No. 134-1); Tr. at 5–38. Dr. Kinsbourne opined that onset of Mr. Greene's brachial neuritis occurred in a medically acceptable timeframe, based upon his characterization of brachial neuritis as a vaccine-caused, neuropathic autoimmune injury comparable to Guillain-Barré syndrome ("GBS"). Tr. at 8.

[5] Petitioner contested my determination to permit Respondent the opportunity to file his own expert report, rather than simply decide the case based on the existing record, but the Court of Federal Claims ruled that these objections had no legal merit. *See* Order, dated May 30, 2018 (ECF No. 119).

As his curriculum vitae ("CV") indicates, Dr. Kinsbourne is a pediatric neurologist. CV, filed as Ex. 39 (ECF No. 82-2) ("Kinsbourne CV"). He received his medical degree in England, and he has been licensed to practice medicine in North Carolina since 1967. *Id.* at 1. From 1967 to 1974, Dr. Kinsbourne served as an associate professor in pediatrics and neurology and a senior research associate at Duke University Medical Center before holding a series of academic positions, including professorships in pediatrics, neurology, and psychology. *Id.* at 2. His clinical experience includes serving as a senior staff physician in Ontario from 1974–80, and as a clinical associate in neurology at Massachusetts General Hospital from 1981–91. *Id.* at 2–3.

Dr. Kinsbourne has published several articles examining neurological diseases (though none directly addressing brachial neuritis), and he is on the editorial board of several journals, such as *Brain and Cognition* and *Archives of Clinical Neuropsychology*. Kinsbourne CV at 4, 7–40. His focus has been on teaching cognitive and behavioral neuroscience for twenty years, retiring from his teaching position at the New School a few years ago. Tr. at 28–29. Although a neurologist, and although he claims to see patients occasionally, Dr. Kinsbourne has not had a regular clinical practice for over twenty-five years, and has no specialized expertise in studying or treating peripheral neuropathies like brachial neuritis (although he asserts that he has encountered it in his patients). *Id.* at 6, 28, 36. Accordingly, the opinion Dr. Kinsbourne offered in this case was rooted not in his personal experience (whether from clinical or research exposure) with brachial neuritis, but arose from his own knowledge of neurology generally plus research performed specifically for the purpose of offering an expert opinion for Petitioner.

Dr. Kinsbourne began with a general discussion of what brachial neuritis is, noting that it usually presents abruptly in the shoulder and upper arm, with pain that can last for days or weeks, and which can be accompanied by limb weakness and muscle atrophy. Tr. at 10. Because it is heterogeneous in nature, its etiology can include both mechanical and immunologic causes, although in either situation its symptoms follow a similar course. *Id.* at 11. When immunologic in origin, brachial neuritis occurs because of an attack by autoantibodies, generated in response to some external signal (i.e., infection), against either the outer myelin sheath of peripheral nerves in the arms or the nerve axons (although Dr. Kinsbourne acknowledged that attack on the axon was the main feature of brachial neuritis). *Id.*

In so maintaining, Dr. Kinsbourne analogized brachial neuritis to a different autoimmune-in-origin peripheral neuropathy, GBS. Tr. at 12–15. He deemed GBS the most common autoimmune peripheral neuropathy, stating that it "comes in two variants," with one primarily featuring demyelination of the nerve sheath, while the other, acute motor axonal neuropathy (AMAN)[6] involves attack on the nerve axon itself. *Id.* at 12, 14. He noted in particular that, regardless of the variant and situs of nerve attack, medical literature supports the conclusion that

[6] AMAN is a GBS subtype commonly seen in China, generally caused by the campylobacter virus. *Dorland's Illustrated Medical Dictionary* 1268 (32nd ed. 2012) [hereinafter "*Dorland's*"]. It features motor axonal degeneration with little inflammation or demyelination. P. Dyck & P.K. Thomas, 2 *Peripheral Neuropathy* 2200 (4th ed. 2005) [hereinafter "Dyck & Thomas"].

the same antiganglioside autoantibodies are involved in the pathologic process leading to GBS (thus allowing the inference that these autoantibodies might also be part of the process leading to brachial neuritis). *See* R. Yu, et al., *Ganglioside Molecular Mimicry and its Pathological Roles in GBS and Related Diseases*, 74 Infection & Immunity 6517 (2006), filed as Ex. 54 (ECF No. 95-1) (AMAN and acute motor and sensory axonal neuropathy both associated with antibodies against the ganglioside component of nerve membrane). GBS variants otherwise present with the same symptoms and course, proceeding in a monophasic manner and peaking within thirty days at most from onset (although the timeframe from instigation to onset can be longer). *Id.* at 13.[7]

To support his contention that GBS was sufficiently comparable to brachial neuritis to apply the former's timeframe for onset, Dr. Kinsbourne referenced several pieces of medical or scientific literature. *E.g.*, R. Verma, et al., *Neuralgic Amyotrophy Associated with Dengue Fever: Case Studies of Three Patients*, 57 J. Postgraduate Med. 329 (2011), filed as Ex. 52 (ECF No. 94-8) ("Verma"). Verma considered three patients who presented with brachial neuritis associated with dengue infection (as opposed to vaccination). Verma at 329; Tr. at 51. All three developed brachial neuritis in days to a week after experiencing an acute fever and/or rash brought on by the instigating infection, suggesting to Verma's authors that immune-mediated mechanisms explained each case. Verma at 331. Dr. Kinsbourne felt Verma supported the possibility that brachial neuritis could have a similar mechanism as GBS, such as molecular mimicry. Tr. at 19–20.

Other literature was offered to establish the existence of certain autoantibodies common to both GBS and brachial neuritis – thus strengthening the idea that they have a common pathogenesis. *See, e.g.*, N. Van Alfen, *The Clinical Spectrum of Neuralgic Amyotrophy in 246 Cases*, 129 Brain 438, 448 (2006), filed as Ex. 51 (ECF No. 94-7) ("Van Alfen I"). Van Alfen I observed that a certain percentage of individuals with brachial neuritis tested positive for antiganglioside antibodies comparable to those associated with GBS. Tr. at 20–21; Van Alfen I at 444 (nine of thirty-four patients tested, or 26 percent). Van Alfen I itself, however, acknowledges (consistent with what is understood about brachial neuritis generally in the medical community) that "motor symptoms are said to predominate" in the condition, with sensory symptoms (which would reflect impact on sensory nerves) secondary. Van Alfen I at 447–48.

To further support the contention that GBS and brachial neuritis are analogous autoimmune conditions (making it reasonable to use the same timeframes for onset in both), Dr. Kinsbourne also offered three Japanese case studies. Tr. at 22–23, 24–27, 33–34 (discussing K. Naito, et al., *Intravenous Immunoglobulin (IVIg) Therapy with Methylprednisolone Pulse Therapy for Motor Impairment of Neuralgic Amyotrophy: Clinical Observation in 10 Cases*, 51 Internal Med. 1493

[7] Dr. Kinsbourne also endeavored to identify record evidence suggesting that Mr. Greene had experienced demyelination rather than simply an axonal attack. *See, e.g.*, Tr. at 9–10 (citing Ex. 4 at 15 (notes from October 5, 2009 testing indicated presence of "a focal demyelinating process")). However, Dr. Lancaster forcefully disputed that this particular evidence established the presence of demyelination, allowing at best that the interpreting physician's comment "was an extremely indirect inference based upon very limited data," and opining instead, based upon his own reading of the same test results, that they did not establish demyelination. *Id.* at 96–97.

(2012), filed as Ex. 74 (ECF No. 134-2) ("Naito"); R. Morishima, et al., *Chronic Brachial Plexus Neuritis that Developed into Typical Neuralgic Amyotrophy and Positively Responded to Immunotherapy*, 57 Internal Med. 1021, filed as Ex. 75 (ECF No. 134-3) ("Morishima"); K. Moriguchi, et al., *Four Cases of Anti-Ganglioside Antibody-Positive Neuralgic Amyotrophy with Good Response to Intravenous Immunoglobulin Infusion Therapy*, 238 J. Neuroimmunology 107 (2011), filed as Ex. 76 (ECF No. 135-1) ("Moriguchi")).

Naito and Morishima both examined the efficacy of immunotherapy treatments (common to the treatment of GBS) such as intravenous immunoglobulin ("IVIG")[8] for brachial neuritis. In Naito, for example, nine of ten brachial neuritis patients who received a form of IVIG treatment showed improvement in motor impairment. Naito at 1499; Tr. at 22–23. Dr. Kinsbourne felt Naito therefore supported his overall contention about GBS and brachial neuritis's similarities—although Naito's authors were careful to limit their conclusions to the concept that only those experiencing a severe form of brachial neuritis characterized by a prolonged autoimmune response might benefit from immunosuppressive treatments like IVIG, adding that the overall efficacy of such treatments for brachial neuritis as a whole remained unresolved—similar to the contention (central to Petitioner's theory herein) that antiganglioside antibodies are commonly a component of brachial neuritis's pathogenesis. Naito at 1499.

Morishima is a single-patient case study involving a fifty-five-year-old man who developed brachial neuritis after a marine sports accident that caused direct injury to his arm and shoulder. Morishima at 1021. After an initial acute response, the man's pain and related symptoms subsided for three months, but reappeared thereafter, proving resistant to treatment even a year later. *Id.* at 1021–22; Tr. at 24, 37. Lab tests revealed the man possessed the kind of antiganglioside antibodies discussed above (suggesting to treaters that he was experiencing an ongoing autoimmune process), leading them to employ IVIG effectively. Morishima at 1023. Morishima's authors thus proposed that this kind of treatment might also be of use in cases of chronic brachial neuritis. *Id.* at 1024–25. Dr. Kinsbourne acknowledged that the lengthy, approximately twelve-week onset for the patient's brachial neuritis was an outlier, but maintained nonetheless that Morishima's authors appeared to have accepted it as reasonable. Tr. at 33–34.

Moriguchi considered four brachial neuritis patients, three of whom were believed to have developed it from an antecedent infection (while the fourth had a history of symptom complaints in connection with arm surgery). Moriguchi at 107; Tr. at 26. All of the studied individuals tested positive for increased levels of antiganglioside antibodies, and two of the three patients whose condition was associated with infection showed improvement after IVIG treatment—more support, Dr. Kinsbourne maintained, for the similarities between GBS and brachial neuritis as common autoimmune conditions with a similar pathogenesis. Moriguchi at 107–08; Tr. at 26–27.

[8] IVIG is a blood product used to treat patients with antibody deficiencies. *Caruso v. Sec'y of Health & Human Servs.*, No. 15-200V, 2017 WL 5381154, at *4 n.11 (Fed. Cl. Spec. Mstr. Oct. 18, 2017) (citing *Clinical Uses of Intravenous Immunoglobulin*, NCBI (2005), https://www ncbi.nlm.nih.gov/pmc/articles/PMC1809480). Because it increases the effectiveness of a patient's immune response, IVIG is commonly employed to treat autoimmune conditions. *Id.*

Significantly, however, the individuals believed to have developed brachial neuritis after infection (and hence those most analogous to a person, like Mr. Greene, whose brachial neuritis is posited to have been vaccine-caused) experienced onset between *one day and two weeks* after infection—not *six* weeks, as alleged is reasonable herein. Moriguchi at 107–08.

Dr. Kinsbourne subsequently addressed in more direct form the timeframe issue central to Petitioner's claim. Because of GBS's "prototypical" nature as an autoimmune peripheral neuropathy, Dr. Kinsbourne reasoned that the timeframe "risk intervals" that the scientific and medical community uses for evaluating when GBS onset might properly be associated with a vaccination could also be applied to a similar neuropathy like brachial neuritis. Tr. at 15. He thus considered a number of items of literature he felt supported a longer timeframe—some, but not all, of which involved brachial neuritis. *E.g.*, H. Tseng, et al., *Safety of a Tetanus-Diphtheria-Acellular Pertussis Vaccine When Used Off-Label in an Elderly Population*, 53 Clinical Infectious Diseases 315 (2012), filed as Ex. 49 (ECF No. 94-5) ("Tseng").

Tseng, for example, considered 119,573 adults over the age of sixty-five who received the tetanus-diphtheria-aceullar pertussis ("TDaP") vaccine, following them over a four-year period to evaluate the risk of adverse events (including GBS and brachial neuritis) after vaccination, in comparison to the whole-cell form of vaccine that was previously in wide use. Tseng at 315; Tr. at 30. Tseng's authors noted that there was reliable evidence (derived from Institute of Medicine ("IOM") publications) supporting a possible causal relationship between TDaP and brachial neuritis, thus justifying consideration of it as a possible adverse event. Tseng at 316 (citing K.R. Stratton, et al., *Adverse Events Associated with Childhood Vaccines Other Than Pertussis and Rubella*, 271 J. Am. Med. Ass'n 1602 (1994), filed as Ex. A (ECF No. 91-1) ("Stratton")). They found that brachial neuritis was no more common after Tdap vaccination than after Td. *Id.* at 316, 319; Tr. at 15–16, 30–31. In so doing, however, Tseng's authors utilized a one to forty-two day risk interval to look for brachial neuritis or GBS, although they did not explain why this interval was used. *Id.* at 4.[9]

Van Alfen I included in its studied subject group individuals whose brachial neuritis was considered hereditary or idiopathic, observing that a little over half (53.2 percent) reported an antecedent event, with less than 5 percent of that subgroup reporting having been vaccinated before symptoms arose. Van Alfen I at 440, 443. Although the time between vaccine administration and onset was not reported, other onset times (after infection, exercise, or surgery) were measured as under twenty-four hours, one to seven days, one to two weeks, and over two weeks, with most

[9] In prior filings in this action, Respondent has observed the incongruity of Petitioner's reliance on the IOM determination of a causal association between tetanus-containing vaccines and brachial neuritis (*see, e.g.*, Kinsbourne Rep. at 3) and the fact that the IOM—which Tseng also relied upon as a basis for including it as a possible adverse event—goes only to *three or four weeks* in proposing what a reasonable timeframe under such circumstances might be. Mot. at 5–6, 8.

cases seeing onset within a week (although 10 percent of post-infection cases occurred more than two weeks later). *Id.* at 443.

Other articles offered on the risk interval period said nothing about brachial neuritis specifically. W. Yih, et al., *An Assessment of the Safety of Adolescent and Adult Tetanus-Diphtheria-Acellular Pertussis (Tdap) Vaccine, Using Active Surveillance for Adverse Events in the Vaccine Safety Datalink*, 27 Vaccine 4257 (2009), filed as Ex. 53 (ECF No. 94-9) "(Yih"); Tr. at 30. Yih considered five categories of potential adverse events after TDaP vaccination: encephalopathy-encephalitis-meningitis; paralytic syndromes, seizure, cranial nerve disorders, and GBS. Yih at 4258. Nearly 700,000 TDaP recipients ten to sixty-four years old were followed for 145 weeks, relying on a forty-two-day risk interval for all potential adverse outcomes save seizure. *Id.* at 4259, 4261. In doing so, Yih's authors explained that, "not knowing the true window of risk and not wanting to miss late events," they relied on the same six-week period known to be applicable to GBS, in a desire to be intentionally over-inclusive. *Id.* at 4259. Dr. Kinsbourne deemed Yih significant despite its failure to include brachial neuritis as an adverse event because it demonstrated the range of possible adverse outcomes involving possible autoimmune-mediated conditions. Tr. at 17.

Another article discussed by Dr. Kinsbourne at hearing explored the larger question of what kind of risk intervals should be used as a general matter in vaccine safety studies. *See generally* A. Rowhani-Rahbar, et al., *Biologically Plausible and Evidence–Based Risk Intervals in Immunization Safety Research*, 31 Vaccine 271 (2012), filed as Ex. 48 (ECF No. 94-4) ("Rowhani-Rahbar"); Tr. at 17–18. Rowhani-Rahbar discussed the process for identifying and defining biologically plausible and evidence-based risk intervals (the period of time after vaccination) for adverse events following vaccination. Rowhani-Rahbar at 272. Risk intervals allow researchers to study the incidence of a potential adverse effect by following risk and control intervals, and should be determined by considering features of the adverse event, presumed or known pathologic mechanism, and the vaccine itself. *Id.* at 272–73. The researchers cautioned against a too-long interval, noting that the risk of adverse events is likely concentrated in a short period of time, and that applying a long interval could dilute results pointing to the actual period of highest risk (although a too-short period could also underestimate risk). *Id.* at 275. For these reasons, the researchers emphasized the need to identify the most biologically-plausible timeframes possible. *Id.*

Rowhani-Rahbar specifically proposed risk intervals for only two adverse events following vaccine administration—febrile seizures and acute disseminated encephalomyelitis ("ADEM"). Rowhani-Rahbar at 273. For ADEM (which, like brachial neuritis, is neurologic in nature, but is a central nervous system disease rather than peripheral), Rowhani-Rahbar concluded that the most trustworthy time period from vaccination to onset "best substantiated by available biological and epidemiologic data" was five to twenty-eight days. Rowhani-Rahbar at 274. A secondary, longer

interval of two to forty-two days was also deemed "biologically plausible,"[10] and therefore worthy of consideration in order to fully assess a potential safety problem, but was more uncertain, since "there might be reason to suspect that most of the excess risk, if any, is concentrated in a much shorter period of time." *Id.* at 275; Tr. at 18–19.

Based on the above, Dr. Kinsbourne opined that onset of Petitioner's brachial neuritis forty-one days after vaccination was medically reasonable. He noted that he would actually support a similar six-week period for virtually any autoimmune disease with "no hesitation," adding that although he was aware of reliable scientific support for even longer onsets in the case of other autoimmune conditions like GBS, he was not comfortable proposing a timeframe beyond forty-two days. Tr. at 31–33. He admitted, however, that even if such a six-week period was reliable, the probability of developing brachial neuritis diminished as the tail end of the temporal "curve" was reached. *Id.* at 38. He also acknowledged that none of the evidence offered for the timeframe question directly involved cases measuring onset from vaccination, as opposed to a different cause (although he emphasized that some evidence, like Van Alfen I, did support the more general proposition that vaccines *could* cause brachial neuritis). *Id.* at 37; Van Alfen I at 443.

2. *Dr. Laurence Steinman*

Dr. Steinman prepared one brief written report for this case and testified at hearing. Tr. at 39–71, 135–43; Report, dated Nov. 12, 2018, filed as Ex. 68 (ECF No. 128-1). He offered an opinion that largely overlapped with what Dr. Kinsbourne presented.

Dr. Steinman obtained his medical degree from Harvard Medical School, where he completed a fellowship in chemical neurobiology. Tr. at 39; *see also* CV at 1, filed as Ex. 77 (ECF No. 136-1) ("Steinman CV"). After medical school, Dr. Steinman went on to complete both a pediatrics and neurology residency at Stanford University. Tr. at 39; Steinman CV at 1. He then joined the faculty at Stanford in 1980, where he presently serves as the George A. Zimmerman Professor of Neurological Sciences, Neurology, Genetics and Pediatrics. Tr. at 39; Steinman CV at 1. Dr. Steinman claimed to have encountered brachial neuritis at least one hundred times in his career (although he did not specify when he most recently encountered it—or whether his encounters came via his role as professor overseeing the work of medical residents, as opposed to his own treatment of patients). Tr. at 136. Dr. Steinman has also published extensively in peer-reviewed journals on topics including neuroimmunology and GBS. Steinman CV at 5–45. He has demonstrated expertise in both a wide variety of central nervous system diseases (multiple sclerosis in particular) and immunologic issues, and claimed great familiarity in treating brachial neuritis, although he does not appear to have focused on it over other central nervous system

[10] The only support for this timeframe offered in Rowhani-Rahbar is the statement that "[b]ased on prior reports of the onset of ADEM following immunization, the occurrence of such events appears to decrease substantially beyond 6 weeks." Rowhani-Rahbar at 274. This statement is footnoted in turn by nine items of literature—all involving ADEM, however, and some of which appear to discuss passive surveillance reports of post-vaccine cases rather than to weigh actual biologic risk.

diseases (such as multiple sclerosis).

Dr. Steinman characterized brachial neuritis as a kind of neuropathic autoimmune disease. Although his testimony on this point was mostly indistinguishable from Dr. Kinsbourne's, he did attempt to refine some of Dr. Kinsbourne's earlier assertions. Thus, Dr. Steinman emphasized that brachial neuritis is in his opinion likely an inflammatory autoimmune condition, similar to GBS in pathophysiology, although more focal (i.e., restricted to an arm and shoulder as opposed to having an impact bilaterally/symmetrically). Tr. at 43–44. He allowed that it has different triggers than GBS, although Dr. Steinman emphasized its immunologic character, based on his experience as well as existing literature. Tr. at 135–36 (referencing Van Alfen I in support of his contention that the most common cause for brachial neuritis is not trauma but an immunologic trigger, such as vaccination or infection), 138 (noting that based upon his experience "at grand rounds or some teaching conference," the etiology of brachial neuritis can be identified half of the time).

Consistent with Dr. Kinsbourne's testimony, Dr. Steinman asserted that brachial neuritis involves attacks on myelin and nerve axon equally. Tr. at 56–57. He admitted, however, that brachial neuritis is not *exclusively* immunologic in origin, but can also be the result of some direct trauma (like the case study discussed in Morishima), although the subsequent pathogenesis in all cases would be "autoimmune or at least inflammatory," concepts he contradictorily claimed were the same but also distinguishable. *Id.* at 70–71.

Also consistent with Dr. Kinsbourne's prior testimony was Dr. Steinman's overarching presumption that GBS, and what is known about it in terms of vaccine causation, provides an analog to brachial neuritis for purposes of determining timeframe in this case. Thus, he deemed significant the fact that brachial neuritis has been shown (in case reports like Naito and Moriguchi) to respond to immune-modulating treatments like IVIG,[11] and that the specific antiganglioside antibodies allegedly associated with GBS were observed to be present for the patients considered by these case studies, thereby confirming its autoimmune character and/or similarity to GBS—along with the fact that (reflecting the role the immune system writ large plays in its pathogenesis) brachial neuritis does not always occur in the same arm that receives trauma. Tr. at 57–64. He also maintained, like Dr. Kinsbourne, that "host factor" variations in how an individual reacts to an event triggering an autoimmune response would impact that individual's course. *Id.* at 44–45.

Based upon the foregoing, Dr. Steinman echoed Dr. Kinsbourne's conclusion that timeframes viewed as medically reasonable for onset of GBS were equally applicable to brachial neuritis. In so maintaining, he relied on the fact that both illnesses are directed at the peripheral nerves and (in his view) likely involve similar mechanistic paths. Tr. at 45. He also invoked a seminal item of medical literature (not directly relevant to brachial neuritis) from the swine flu

[11] Dr. Steinman went so far as to assert that IVIG is used at his own hospital at Stanford to treat brachial neuritis, although he did not substantiate this assertion with any independent evidence. Tr. at 138.

epidemic in the 1970s. L. Schonberger, et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program*, 110 Amer. J. Epidemiology 105 (1979), filed as Ex. 69 (ECF No. 128-2) ("Schonberger"). Schonberger, Dr. Steinman observed, supported up to a *ten-week* timeframe for onset of GBS, but suggested risk was highest in the shorter, six to seven-week interval (although Schonberger itself expressly states that GBS risk "was concentrated primarily within the 5-week period after vaccination"). Schonberger at 105; Tr. at 47, 49. Because Schonberger was "based on an enormous data set of surveillance," he felt it was a particularly reliable epidemiologic study that had applicability herein. *Id*. at 47, 55.[12]

As a result, Dr. Steinman (like Dr. Kinsbourne) endorsed a six-week timeframe for onset of brachial neuritis after vaccination as medically reasonable. Tr. at 53. He claimed that, in his own clinical experience, he had seen a variety of onsets ranging throughout the proposed six-week period, but that onset toward the end of that period "would not worry me." *Id.* at 141. In explaining this conclusion, Dr. Steinman repeated many of the same points made by Dr. Kinsbourne, commenting on the same items of literature reviewed during Dr. Kinsbourne's testimony. He was thus asked about Verma (Tr. at 50–51), Yih (*id.* at 52–53), Rowhani-Rahbar (*id.* at 54–56), Naito (*id.* at 58–59), Morishima (*id.* at 60–61), and Moriguchi (*id.* at 62–63). He acknowledged, however, when asked generally about the utility of risk intervals, that they were often primarily the product of what a particular study's authors wanted to achieve in a particular study, rather than a reflection of biological plausibility for a given potentially adverse event. *See, e.g.*, Tr. at 52–53, 55 ("where you set the dial is in the hands of the person or teams turning the dial").

On cross examination, Dr. Steinman admitted some limitations to the bases for his conclusions regarding timeframe for onset herein. He allowed that case studies like Naito equivocated as to the efficacy of immunotherapies for brachial neuritis, although he maintained nonetheless that such evidence pointed in the direction of the utility of such treatments, even if more formal scientific corroboration was still absent. Tr. at 67–68. He agreed that such treatments were not uniformly called for either, especially since certain individuals suffering from brachial neuritis might see improvement absent IVIG treatment (although he stressed the medical importance of doing whatever possible to aid patients). *Id.* at 68. And he conceded that one of the articles referenced in his own expert report only supported a three-week timeframe for post-vaccination onset of brachial neuritis. *Id*. at 65–66 (discussing P. Tsairis, et al., *Natural History of Brachial Plexus Neuropathy*, 27 Arch. Neurol. 109, 111–12 (1972), filed as Ex. 58 (ECF No. 95-5) ("Tsairis")).

[12] Dr. Steinman also noted that the tetanus vaccine package insert indicates that individuals with GBS are advised against getting a second vaccine within six weeks of the disease's appearance as further bulwarking the reliability of the six-week timeframe. Tr. at 49–50. Petitioner did not, however, file this document—and even if he had, the fact that it applies to GBS (coupled with existing sound Vaccine Program caselaw suggesting that package inserts deserve little evidentiary weight) greatly reduces the value of this argument (especially given the existence of several more reliable items of evidence that were filed in this action and which support the timeframe argument (e.g., Rowhani-Rahbar)).

3. *Dr. Eric Lancaster*

Dr. Eric Lancaster provided two expert reports in this action on Respondent's behalf, and also testified at hearing. *See* Report, dated May 25, 2018, filed as Ex. B (ECF No. 121-1) ("Lancaster Rep."); Report, dated Mar. 28, 2019, filed as Ex. D (ECF No. 133-1) ("Supp. Lancaster Rep."). Dr. Lancaster opined that the tetanus vaccine did not likely cause Petitioner's brachial neuritis, and that the timeframe in which Petitioner's injury began was not a medically acceptable period for post-vaccination causation. In so proposing, he disputed the assumption of Petitioner's experts that what is known about GBS onset and timeframe can be applied equally to brachial neuritis.

Dr. Lancaster is a clinical physician at the Center for Autoimmune Neurology at the University of Pennsylvania, as well as an assistant professor of neurology at the University of Pennsylvania. Ex. C at 1 (ECF No. 121-11) ("Lancaster CV"). He completed a neurology residency at the University of Pennsylvania from 2004–07, and is board certified in neurology, with subspecialties in neuromuscular medicine and electrodiagnostic medicine. *Id.*; Tr. at 73–74. His research focuses on antibody-mediated neurological disorders, and he sees patients with complex autoantibody disorders on a regular basis. Lancaster CV at 1; Tr. at 75. He has considerable expertise performing the tests used to evaluate peripheral neuropathies (e.g., EMGs, nerve conduction studies)[13], and devotes half his time to a clinical practice, although he estimates only to have treated ten to twenty patients with brachial neuritis over his career. Tr. at 74–75; Lancaster Rep. at 1.

Dr. Lancaster's description of brachial neuritis was mostly consistent with that provided by Petitioner's experts, although it diverged in a few significant respects. He characterized the condition as "inflammation in a particular area of the nervous system called the brachial plexus," located in the shoulder near the neck. Tr. at 76–77. It has a monophasic course, with a sudden onset followed by a slow recovery over weeks or months, although patients often recovery fully. *Id.* at 77. He was less confident than Petitioner's experts that brachial neuritis is primarily autoimmune in nature, however, noting that although "it certainly involves inflammation," not enough is known about its pathophysiology and the mechanisms driving it (for example, whether it is solely antibody-mediated or the result of a T-cell response) to firmly state that it is primarily an autoimmune condition. *Id.* at 107. But several items of literature filed by Respondent seem to accept that autoimmunity plays a role in brachial neuritis's pathogenesis. *See, e.g.*, N. Van Alfen, *Clinical and Pathophysiological Concepts of Neuralgic Amyotrophy*, 7 Nat. Rev. Neurol. 315, 320

[13] An EMG, or electromyography test, is a diagnostic procedure used to assess the health of muscles and the nerve cells that control them (motor neurons). *Dorland's* at 602. A nerve conduction study, or "nerve conduction velocity test," measures the speed of conduction of an electrical impulse through a nerve, to evaluate the presence of nerve damage or destruction. *Nerve Conduction Studies*, Health Library, Johns Hopkins Medicine, https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/nerve-conduction-velocity-ncv (last visited July 23, 2019).

(2011), filed as Ex. B-1 (ECF No. 121-2) ("Van Alfen II") (acknowledging that brachial neuritis "is thought to be autoimmune in origin").

Dr. Lancaster nevertheless stressed (in contrast to Drs. Kinsbourne and Steinman) that brachial neuritis is primarily an "axonal" injury to the nerves, readily ascertainable via an EMG study, rather than one featuring multifocal demyelination—and in fact it is this axonal characteristic that causes recovery after onset to take time. Tr. at 77, 96–97; Lancaster Rep. at 6; J. Feinberg, et al., *Parsonage-Turner Syndrome*, 6 Hosp. for Spec. Surg. 199, 202 (2010), filed as Ex. B-2 (ECF No. 121-3) ("Feinberg"); *see also* Van Alfen II at 319 ("[a]s in other [peripheral nervous system] disorders, the amount of axonal damage provides a fair prediction of the possibility for nerve recovery," adding that subsequent reinnervation can take months or years). He indicated no awareness, based on existing literature or medical community views, that brachial neuritis should be considered primarily a demyelinating injury, noting that testing (such as nerve conduction studies) used to diagnose brachial neuritis does not usually reveal "severe conduction slowing or reversible conduction blocks"—either of which would reflect the existence of demyelination. Tr. at 79.

Dr. Lancaster similarly took issue with the efforts of Petitioners' experts to borrow GBS onset timeframes for this case, stressing the differences in the two conditions: the focal and localized nature of the inflammation present in brachial neuritis, as opposed to the multifocal, bilateral character of GBS, and the demyelination featured most often in GBS, as opposed to the "axonal process" in brachial neuritis. Tr. at 87–88, 89 ("these are very different disorders and it isn't that hard to tell them apart"), 94–95 (referencing Van Alfen II). He also disputed the efficacy of immunosuppressive treatments for brachial neuritis, arguing that although such treatments have been medically established to be effective for GBS, there is a lack of persuasive and reliable scientific data establishing their utility for brachial neuritis, and that he had not in his experience encountered the use of such treatments for this condition (or the testing of brachial neuritis patients for the presence of autoantibodies). *Id.* at 88–89, 124–26. And he noted that given what is known about the two diseases and their differing courses (with brachial neuritis having a higher likelihood of a fast onset after triggering event than GBS), it made sense to him that the latency period relevant to one would be inapplicable to the other. *Id.* at 89–91. In fact, he felt it far more medically likely that brachial neuritis would have a shorter, post-trigger onset than GBS. *Id.* at 91–92.

Another distinction between GBS and brachial neuritis that Dr. Lancaster deemed significant was the fact that the latter can be initiated by "different triggering events" not associated with the former. Tr. at 77. Brachial neuritis can be caused by injury from exertion or other arm trauma, infection, or vaccination—although half of the time in his experience no causal factor could be identified. *Id.* at 77, 104 (discussing vaccination as possible cause). Regardless, the presentation and progression of most cases of brachial neuritis was the same. *Id.* at 78. He also emphasized that direct injury to the nerve itself was not a requirement for triggering of brachial

neuritis. *Id.* at 78, 83–84. On the contrary, "relatively minor events" could produce brachial neuritis, from overexertion after exercise to maintaining the same body position on a lengthy airplane flight. *Id.* at 99–100, 111–12; Lancaster Rep. at 7. He particularly stressed that unlike GBS, brachial neuritis *could* have direct arm trauma as its triggering event. Tr. at 90 ("[t]his has not, to my knowledge, ever been accepted as the cause of [GBS]").

Turning to the present record, Dr. Lancaster agreed that Petitioner has brachial neuritis. He did not, however, see evidence that would suggest Mr. Greene's immune system was undergoing an autoimmune process in the forty-one-day period between receipt of the tetanus vaccine and onset of his symptoms in September 2009. Tr. at 80. Indeed, he disputed that such a long timeframe was reasonable for onset of brachial neuritis after *any* inciting event.

Relying on Van Alfen I as well as Tsairis, along with his personal clinical experience, Dr. Lancaster observed that half of studied cases in those items of literature began within a week of the putative trigger,[14] with 90 percent occurring in two weeks. Tr. at 81, 85–87; Tsairis at 111–12; Van Alfen I at 443 (nearly 70 percent of all studied cases reporting an antecedent event involved symptom onset within a week or less). He therefore deemed an onset beyond three to four weeks of the triggering incident as "increasingly implausible." Tr. at 81. He also disputed the validity of employing a single timeframe for all neuroimmune disorders attributable to vaccination, as Petitioner urges. *Id.* at 82. He did, however, acknowledge that certain items of literature, like Van Alfen I, seemed to allow for the possibility that brachial neuritis could still occur at a timeframe beyond two weeks (even though his reading of the data from that item of literature suggested that the syndrome was "very heavily concentrated" on the immediate two to three weeks after a trigger). *Id.* at 117–19; Van Alfen I at 443 (approximately 10 percent of forty-nine studied cases involved onset of post-infection brachial neuritis occurring more than two weeks after the infection, with the remaining 90 percent occurring two weeks or less after infection).

Dr. Lancaster noted several findings in the medical record that he deemed significant to his opinion. The electrodiagnostic test results for Mr. Greene suggested to him the presence of "severe axonal injury," with far less convincing evidence of demyelination, and nothing that would suggest the presence of GBS either. Tr. at 96–97; Lancaster Rep. at 10–11. Such results were therefore consistent with brachial neuritis. Tr. at 97. He also observed no treater support for the tetanus vaccine being causal. *Id.* at 98. And overall, based on his view of the totality of the record, he thought it most likely that in Mr. Greene's specific case physical exertion close in time to onset in

[14] Dr. Lancaster acknowledged that it might be difficult in cases of infection-induced brachial neuritis to pin down the date of the inciting event, but noted that incidents involving arm trauma (whether from exertion, accident, or surgery) were a different matter—and in such cases latency was often very short. Tr. at 81.

early September was the most likely trigger[15] —otherwise the cause was idiopathic (although he did not discount the initial hand injury that Petitioner suffered, which caused him to receive the tetanus vaccine in the first place, as also possibly causative). *Id.* at 98–104; Ex. 12 at 19 (September 7, 2009 ER record with handwritten note referencing Petitioner's exertion in the days prior to onset as a possible "recent injury" explanation for pain).[16]

Dr. Lancaster went on to review the literature and evidence offered in the case to support, or refute, a forty-one-day timeframe for onset of post-vaccination brachial neuritis. While articles like Tsairis did allow that a vaccine might be causative, they lent support for a timeframe for onset of no more than four weeks. Tr. at 82–84; Tsairis at 111. Rowhani-Rahbar also did not consider brachial neuritis in proposing the two risk intervals it discusses, and in fact its authors were in Dr. Lancaster's view "extremely careful" to highlight that any selected interval needed "to be customized to the specific disease in question." Tr. at 85. Schonberger similarly only involved GBS, and in Dr. Lancaster's reading underscored the fact that brachial neuritis and GBS have distinguishable pathophysiologies. *Id.* at 92–93.

Dr. Lancaster also directly questioned the significance of the three Japanese case studies (Naito, Morishima, and Moriguchi) filed by Petitioner and commented upon by his experts. He challenged their findings about the efficacy of immunosuppressive treatments for brachial neuritis (which, Petitioner argues, corroborates the similarity of the condition to more purely autoimmune neuropathies like GBS), allowing that even if those articles suggested IVIG might be effective in treating brachial neuritis, "the jury is still out" on the mechanism by which brachial neuritis occurs (and in particular whether it is mediated by autoantibodies at all, let alone the same ones implicated in GBS). Tr. at 94, 121–22. He also did not deem significant the fact that some evidence of autoantibodies also relevant to GBS was identified in tests performed in these case reports, noting that he could not exclude the possibility that they were merely false positive findings, reflecting the fact that many people in the population might also possess them (but not establishing that their presence meant the patient's brachial neuritis had been *mediated* by the autoantibody). *Id.* at 108–09.

[15] *See, e.g.*, Lancaster Rep. at 9. Because this Decision does *not* turn on identifying an alternative cause, but instead is the product of my determination that the proposed forty-one-day onset has not been established with sufficient preponderant evidence, I do not analyze Respondent's success in establishing such factual contentions.

[16] Mr. Greene was called at hearing to testify to rebut this point, and he maintained that he had not in fact overexerted or otherwise harmed his arm or shoulder in the days immediately prior to his ER visit in September 2009—and that he did *not* so inform treaters at the time, despite what the contemporaneous medical document states. Tr. at 127–29, 130–31. Respondent, however, offered a later-in-time medical record, filled out by Petitioner, in October 2009 (a month after the ER visit) in which he himself again identified "hard work" around Labor Day of that year triggering a "neuro explosion." *Id.* at 132; *see also* Ex. 2 at 4; Ex. 12 at 19. Petitioner could not credibly explain these medical record references and their contradiction of his recollection, other than by asserting that the degree of pain he was in at the time (coupled with his lack of knowledge about what brachial neuritis was) somehow impacted his judgment or what he told treaters. Tr. at 133–34.

On cross examination, Dr. Lancaster granted the medical plausibility of a causation theory relying on vaccination as initiating brachial neuritis, even if the absence of evidence of the correct autoantibodies relating to the process resulting in the condition. Tr. at 110. He also acknowledged (in response to questions that seemed aimed at establishing that Dr. Lancaster's standard for medical acceptability was far too high for purposes of a Vaccine Program case) that he personally would not accept a forty-one-day timeframe for onset of brachial neuritis without some kind of corroborative scientific or medical study specific to brachial neuritis and vaccination. *Id.* at 107.

Applicable Legal Standards

A. *Claimant's Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Human Servs*., 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1320 (Fed. Cir. 2006).[17] As already noted, Petitioner's Table claim was dismissed after issuance of the Fact Ruling.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs*., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Human Servs*., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions;

[17] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd*, 104 F. App'x 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim (which is the kind of claim asserted in this matter), a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Secretary of Health & Human Services*, 418 F.3d 1274 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, the petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id*. at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Human Servs.*, 121 Fed. Cl. 230, 245 (2015) ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)). But this does not negate or reduce a petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant*, 956 F.2d at 1148. In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the

vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 136 (2011), *aff'd*, 463 F. App'x 932 (Fed. Cir. 2012); *Veryzer v. Sec'y of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff'd without op.*, 475 F. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review denied* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to

the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc*., 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). "The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community." *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

The fact that a claimant offers an expert opinion does not render the opinion that expert espouses scientifically reliable or persuasive. Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339).

C. *Review of Medical and Scientific Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all of the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also*

Paterek v. Sec'y of Health & Human Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

As I have previously noted, the first, "can cause" *Althen* prong has been met in this case, given the ample prior decisions associating vaccines containing a tetanus component with brachial neuritis, as well as the showing made by Petitioner's experts. Remand Ruling at 11 n.7; *Devonshire v. Sec'y of Health & Human Servs.*, No. 99-031V, 2006 WL 2970418, at *15 (Fed. Cl. Spec. Mstr. Sept. 28, 2006) (stating that it is well known that brachial neuritis can occur following a tetanus vaccination), *aff'd*, 76 Fed. Cl. 452 (2007); *DeGrandchamp v. Sec'y of Health & Human Servs.*, No. 01-413V, 2003 WL 21439670, at *7 (Fed. Cl. Spec. Mstr. May 15, 2003) (relying on IOM publications to find that in theory, the tetanus toxoid in Td vaccine can cause brachial neuritis). Literature offered in this case also supports this determination. *See, e.g.*, Van Alfen II at 320. This case instead turns on the remaining *Althen* prongs, which I address in order of their significance to my Decision.

I. Petitioner Has Not Established that his Brachial Neuritis Began in a Medically-Acceptable Post-Vaccination Timeframe (*Althen* Prong Three)

There is no dispute in this case that Mr. Greene experienced brachial neuritis, but the medical reasonableness of the timeframe in which his symptoms began, measured from date of vaccination forty-one days earlier, is very much contested. I have already determined that Petitioner cannot meet his preponderant burden of proof merely by relying on the approximately thirteen-day differential between the end date for a viable Table claim and his own onset. *Grant*, 956 F.2d at 1147–48.[18] Petitioner was thus tasked with establishing the medical reasonableness of the timeframe herein through resort to expert testimony and whatever other evidence bears on that question.

Petitioner has offered some literature addressing the amount of time medical science expects brachial neuritis will occur after an instigating trigger—but it largely does *not* support a

[18] The reasonableness of not allowing a petitioner to rely on the Table claim timeframe for a non-Table claim is underscored by considering how this is fair to both sides. In this case, for example, Dr. Lancaster has opined that the defined Table period for a tetanus/brachial neuritis claim is based on the most persuasive medical and scientific evidence available—thus suggesting to him that anything beyond it would by definition *not* be medically reasonable. Tr. at 91 (mentioning that the twenty-eight-day range reflects the "consensus of experts"). If petitioners are permitted to rely on how *close* onset in a given case is to the defined Table period in support of a non-Table claim, then Respondent should be similarly entitled to wield the Table period offensively, as proof that science does *not* preponderantly support a longer timeframe. Program law instead requires petitioners (and Respondents in seeking to rebut a Vaccine Act non-Table claim) to rely on *evidence* to bulwark the reasonableness of the proposed period—independent of the Table timeframe (although evidence used to establish that timeframe may still be relevant).

six-week onset for the illness, instead suggesting more persuasively that the maximum time from trigger to onset would be no more than two or three weeks. *See, e.g.*, Van Alfen I[19], Tsairis. Indeed, and as Respondent previously argued, one of the same pieces of evidence that supports Petitioner's *Althen* prong one showing—the IOM review of tetanus-caused brachial neuritis—supports at most a four-week onset risk period. Stratton at 55 (noting that latency period for brachial neuritis after vaccination "ranges from a few days to 3 or *at most 4 weeks*") (emphasis added). And Dr. Lancaster convincingly explained why a shorter onset timeframe made more sense for brachial neuritis—and conversely, why a timeframe exceeding four weeks was far less medically acceptable.

Accordingly, what direct proof exists on the topic does not preponderate in Petitioner's favor. Of course, petitioners may establish their Vaccine Act claim with circumstantial evidence, so the absence of sufficient direct proof does not end the analysis. Mr. Greene sought to meet his preponderant burden through (a) invocation of risk intervals applied to other autoimmune diseases, and (b) a comparison of the timeframes that reliable scientific evidence establishes for onset of GBS. Neither argument was ultimately persuasive, however (although both were supported by some reliable scientific/medical evidence).

A. *Risk Intervals for Other Autoimmune Diseases Are Not Preponderant Evidence Alone of A Reasonable Timeframe*

Petitioner relied on some literature involving risk intervals generally, best explained in Rowhani-Rahbar. This evidence unquestionably has probative value. *See generally* Rowhani-Rahbar. The fact that longer risk intervals are often utilized in measuring vaccine safety is circumstantial evidence supporting the conclusion that it would be medically reasonable to expect *some* analogous vaccine-caused neurologic injuries to occur within a similar timeframe. It is for this reason that the longer risk interval that Petitioner's experts propose to apply in this case has also been found persuasive by other special masters in cases alleging autoimmune injuries comparable to those evaluated in Rowhani-Rahbar. *See, e.g.*, *Day v. Sec'y of Health & Human Servs.*, No. 12-630V, 2015 WL 8028393, at *22 (Fed. Cl. Nov. 13, 2015) (applying Rowhani-Rahbar secondary risk interval for ADEM to case alleging that petitioner's multiple sclerosis ("MS") was vaccine-caused).

There are, however, significant countervailing points that weigh against endorsement of a forty-one-day timeframe herein. First and foremost, not *all* neurologic injuries with an autoimmune component are the same, even if they have some common features. Ample Program authority has noted that, while petitioners may reasonably analogize an injury to other autoimmune conditions, they cannot prevail *solely* by doing so. *See, e.g.*, *R.V. v. Sec'y of Health & Human Servs.*, No. 08-504V, 2016 WL 3882519, at *41 (Fed. Cl. Spec. Mstr. Feb. 19, 2016) (not crediting

[19] Although Van Alfen I does allow that 10 percent of studied, infectious-originating cases of brachial neuritis might have had an onset of greater than two weeks, this finding is too vague to give it significant weight – it does not allow for the conclusion that an onset of longer than four weeks is acceptable, and it does not involve vaccination directly. Van Alfen I at 443.

petitioner's reliance on unsupported analogy to other illnesses in proving causation theory), *mot. for review denied*, 127 Fed. Cl. 136 (2016); *R.K. v. Sec'y of Health & Human Servs.*, No. 03-0632V, 2015 WL 10936124, at *105 (Fed. Cl. Spec. Mstr. Sept. 28, 2015) (same), *mot. for review denied*, 125 Fed. Cl. 57 (2016), *aff'd*, 671 F. App'x 792 (Fed. Cir. 2016). And as Dr. Lancaster noted, brachial neuritis is in fact *not* congruent with a central nervous system disease like ADEM—the latter being the basis for the forty-one-day period relied upon in Rowhani-Rahbar (and found to be analogous to the MS injury considered in *Day*). It is therefore unpersuasive for Petitioner to argue that timeframes involving distinguishable neurologic diseases should be applied as a consistent yardstick to all cases involving nerve-related autoimmune injuries.

A secondary problem posed by Petitioner's argument is its overreliance on a longer risk interval as a proxy for preponderant evidence establishing a medically reasonable onset timeframe. Rowhani-Rahbar addresses the application of risk intervals for epidemiologic studies evaluating overall vaccine safety. Although they are employed based on some consideration of when an adverse post-vaccination event might be expected to occur, they are also intentionally "broad nets" intended to catch as many putative adverse events as possible. Dr. Steinman himself noted that an adopted interval can mean whatever the study's author wants it to (although he nevertheless added that a study would for the most part seek to define an interval based on when a plausible reaction or illness might occur). Tr. at 52–53, 55.

Thus, a *secondary* interval explicitly understood to be less specific, and hence medically accurate, but included nonetheless in a study to avoid missing possible related adverse events is not particularly robust proof as to the scientific/medical consensus as to when a particular injury would *most likely* occur post-vaccination. The fact that a risk interval is selected for a particular study because its authors deem it to have utility therein does not mean that the same period can be deemed preponderantly established in the context of a Vaccine Act claim. Thus, while I acknowledge that Petitioner's invocation of the risk interval concept generally had some evidentiary value, it was not by itself enough to preponderantly establish that vaccine-caused brachial neuritis could reasonably occur within six weeks of vaccination—especially since the evidence specific to brachial neuritis says otherwise.

B. *GBS and Brachial Neuritis Are Not Congruent for Purposes of Determining Reasonableness of Onset*

To bulwark adoption of a six-week timeframe, Petitioner's experts consistently proposed that brachial neuritis is analogous to GBS—an autoimmune, peripheral nervous system-affecting neurologic condition that substantial Program caselaw (based in turn on reliable science) has determined can reasonably begin in as long as six to eight weeks after vaccination. *See, e.g.*, *Barone v. Sec'y of Health & Human Servs.*, No. 11-707V, 2014 WL 6834557, at *13 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (eight weeks is the longest reasonable timeframe for a non-Table

flu/GBS injury). But this argument was ultimately unpersuasive. Despite some of their common features, GBS is simply not sufficiently comparable to brachial neuritis to apply the same onset timeframe to both.

Literature filed in this case relating to brachial neuritis, and the reports and testimony discussing it, clearly establishes that it is a neurologic injury primarily to the nerve axon—a finding confirmed by nerve electrophysiologic studies and also consistent with the motor loss associated with brachial neuritis. Feinberg at 202; *see also* Lancaster Rep. at 10–11.[20] It also can be caused by direct trauma that would never result in GBS, further suggesting it is highly distinguishable. And while the same literature also suggests a person with brachial neuritis may have some sensory symptoms that could reflect secondary nerve demyelination, that is not the fundamental character of the condition—unlike GBS. *See Auch v. Sec'y of Health & Human Servs.*, No. 12-673V, 2017 WL 1034396, at *9 (Fed. Cl. Spec. Mstr. Jan. 13, 2017) (Dr. Steinman opining that GBS is characterized by autoimmune attack on nerve myelin sheath, resulting in demyelination).

To establish the contrary, Petitioner's experts simply asserted that this was not the case—that brachial neuritis is as characterized by demyelination as GBS. *See* Tr. at 11 (Dr. Kinsbourne), 56–57 (Dr. Steinman). Alternatively, they attempted to blur the lines between the two conditions by noting the existence of AMAN, a nerve axon-oriented GBS variant. *See, e.g.*, *id.* at 21–22. While this GBS variant clearly exists, Petitioner's experts did not elaborate on the fact that AMAN is also (a) uncommon in comparison to other GBS variants, (b) largely confined to a pediatric population, and (c) mainly occurs outside of the U.S., in eastern Asian countries. *Dorland's Illustrated Medical Dictionary* 1268 (32nd ed. 2012); P. Dyck & P.K. Thomas, 2 *Peripheral Neuropathy* 2200–01 (4th ed. 2005). It is also readily distinguishable from brachial neuritis, as Dr. Lancaster observed. Supp. Lancaster Rep. at 1. The existence of an axonal-impacting GBS variant, therefore, provides limited assistance to Petitioner's argument that GBS and brachial neuritis are comparable for purposes of determining a medically acceptable onset timeframe.

Petitioner also invoked case studies indicating that certain autoantibodies associated with GBS (antiganglioside antibodies) have been found in testing of brachial neuritis patients. The significance of the presence of these autoantibodies in individuals with brachial neuritis is far from determined, however, as more persuasive scientific literature indicates. *See, e.g.*, Van Alfen II at 320 ("some studies have reported antiganglioside [peripheral nervous system] antibodies in patients, but this finding could well be *a consequence of axonal damage rather than its cause*") (emphasis added). As Dr. Lancaster noted, none of these autoantibodies have been scientifically demonstrated to *drive* an autoimmune process resulting in brachial neuritis. And even if it is assumed that the pathophysiology of brachial neuritis could involve similar autoantibodies, such

[20] Well-recognized authorities confirm the same. *See, e.g.*, Dyck & Thomas at 2303 ("most of the currently available evidence from electrophysiologic studies suggest multifocal or patchy abnormalities *consistent with axonal damage (loss)* within the brachial plexus and isolated peripheral nerves of the upper extremities") (emphasis added).

points do *not* establish, based on existing persuasive science and medical literature about brachial neuritis specifically, that the timeframes relevant for a *different* autoimmune disease can simply be applied wholesale here—that the time in which it would take for brachial neuritis (which Dr. Lancaster successfully established would usually be an acute process) to manifest would be consistent with GBS.

The same goes for the Japanese case studies offered to establish that brachial neuritis has been successfully treated with the kind of immune-modulating treatments long understood to be effective for GBS. *See* Naito; Morishima; Moriguchi. It is routinely recognized in the Program that case reports do not merit significant weight as a class of evidence (*see, e.g.*, *Crutchfield v. Sec'y of Health & Human Servs.*, No. 09-39V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014), *aff'd*, 125 Fed. Cl. 251 (2014)), but here the actual reports filed are on their face even less probative than usual. Indeed, only Morishima involved a lengthy onset (admitted even by Dr. Kinsbourne to be an outlier under Petitioner's own theory), with the other case studies observing post-infectious onset (the most analogous circumstance to vaccine-induced brachial neuritis) in a timeframe consistent with *Respondent's* position of less than four weeks. Accordingly, even if I give *some* weight to the suggestion of these case reports that IVIG treatments have been efficacious for certain cases of brachial neuritis, these articles are greatly outweighed by other evidence establishing the many accepted differences between GBS and brachial neuritis.

C. *Respondent Successfully Rebutted Petitioner's Timeframe Evidence, as well as his Expert Reports and Testimony*

Reliable evidence offered by Respondent and discussed by Dr. Lancaster persuasively suggests brachial neuritis is more acute in nature, and will likely begin within two or three weeks of whatever triggers it—not six weeks, as Petitioner argues here. This is consistent with what is known about the orientation of the injury (to the axon) and its acute nature, and does not rely on analogy to distinguishable autoimmune conditions. Dr. Lancaster's opinion stemmed from a practice-derived understanding of how brachial neuritis presents and what causes it—not just from research performed for the purpose of offering an opinion herein. He has knowledge of the condition and regularly performs the EMG and nerve conduction study tests employed to evaluate its existence and course.

Dr. Lancaster's opinion is also consistent with my determinations in other cases involving similar injuries. *See, e.g.*, *Garner v. Sec'y of Health & Human Servs*., No. 15-063V, 2017 WL 1713184 (Fed. Cl. Spec. Mstr. Mar. 24, 2017), *mot. for review denied*, 2017 WL 3483352 (Fed. Cl. July 31, 2017). In *Garner*, I considered a claim that the Hepatitis A and B vaccines had caused brachial neuritis. The earliest onset possible in *Garner* was forty-five days after vaccination, based on the first record documentation of any complaints by the petitioner about arm or shoulder pain. *Id.* at *1. Respondent, however, persuasively argued (also via Dr. Lancaster's testimony) that the

condition was far more acute in nature (and in terms of the causative mechanism as well), making twenty-eight days the outer limit for latency. *Id.* at *8. I found this point to be dispositive, even though the claimant's *Althen* prong one showing was (as here) sufficient, and dismissed the case on the record. *Id.* at *16. That decision was upheld on review. 2017 WL 3483352. Nothing about this case distinguishes it from *Garner*'s analysis or outcome.

Petitioner's expert testimony, by contrast, was simply less effective and persuasive.[21] Both experts he relied upon are credentialed and competent to testify generally on neurologic injuries and the potential immunologic triggers for them—and they do so often in the Vaccine Program. Also, and as discussed extensively above, they offered some reliable items of evidence to support many of their contentions. And the opinion they jointly voiced -- that similarities between GBS and brachial neuritis were enough to allow application of the timeframe for onset of the former to the latter -- was reasonable, and coherently presented as well.

But this does not mean that Drs. Kinsbourne and Steinman offered *persuasive* expert opinions that compel a favorable determination for Petitioner on the third *Althen* prong. Not only were their contentions effectively rebutted, but these two experts do not possess demonstrated, specific experience studying or treating brachial neuritis and its causes, even if they may have intermittently encountered it in their professional lives.[22] As a result, their assertions vouching for the reasonableness of timing in this case lacked the evidentiary heft of an expert who could credibly explain how his own experience studying or treating the disease in question informed his opinion.

Overall, Petitioner did not offer sufficient evidence to meet his preponderant burden on this third *Althen* prong. As the Federal Circuit has noted, establishing the timeframe prong is dependent upon "the medical understanding of the *disorder's* etiology . . . ." *Bazan*, 539 F.3d at

[21] Although Drs. Wright's and Byers's expert reports remain in evidence, Petitioner made no reference to their contentions at hearing, and I do not find that they appreciably assisted Petitioner's case with respect to the third *Althen* prong.

[22] Dr. Kinsbourne was particularly deficient in this regard. By his own admission, he has essentially treated almost *no* patients for more than twenty-five years (although he routinely seeks to offer opinions in Vaccine Program cases on an individual's diagnosis or symptoms course). Tr. at 28; *Holmes v. Sec'y of Health & Human Servs.*, No. 08-185V, 2011 WL 2600612, at *2 n.9 (Fed. Cl. Spec. Mstr. April 26, 2011) (noting that Dr. Kinsbourne has not had a clinical neurology practice for thirty-seven years), *aff'd*, 115 Fed. Cl. 469 (2014). His neurology background cannot make up for this lack of personal understanding of the condition in question.

Dr. Steinman, by contrast, appears to see patients more regularly, even if a clinical practice is not his main focus. But he does not possess specific demonstrated expertise with respect to brachial neuritis. Indeed, although he regularly testifies for Vaccine Act petitioners, by my count he has only offered an expert opinion in cases involving brachial neuritis twice before. *See, e.g.*, *Winterfeld v. Sec'y of Health & Human Servs.*, No. 15-933V, 2018 WL 2225178 (Fed. Cl. Spec. Mstr. Mar. 9, 2018) (awarding attorney's fees and costs in settled case alleging brachial neuritis after receipt of influenza vaccine). And tellingly, in that case the petitioner claimed an onset of approximately *two weeks* after vaccination—not six. *See* Petition at 2, *Winterfeld*, 2018 WL 2225178 (ECF No. 1).

1352 (emphasis added). The most compelling and reliable evidence offered herein pertinent to brachial neuritis (the relevant "disorder") establishes that it proceeds fairly acutely after a trigger, and is highly unlikely to occur more than three or four weeks after instigation. The fact that other autoimmune-mediated and somewhat comparable neurologic diseases might reasonably be understood to have a longer potential onset timeframe does not compel the conclusion that the same is true for brachial neuritis.

II. Petitioner Has Not Established that his Brachial Neuritis Was Caused By the Tetanus Vaccine He Received in July 2009

Even if I had found that a forty-one-day timeframe for onset of brachial neuritis was medically acceptable, the record in this case fails to support the conclusion that the tetanus vaccine is the most likely explanation for Mr. Greene's injury.

The record provides no objective evidence whatsoever—direct, circumstantial, or otherwise—that Petitioner was experiencing an autoimmune-derived injury attributable to vaccination. Mr. Greene had no symptoms at all before he presented to the ER in early September 2009, and then only reported he had been feeling pain for a few days before—consistent with the acutely-presenting nature of brachial neuritis.[23] There is nothing from the pre- or post-vaccination record suggesting that an autoimmune reaction was brewing in a subclinical form. And none of Mr. Greene's treaters implicated the tetanus vaccine as causative of his injuries—nor did they ever propose IVIG treatment to remedy it (something that further undercuts the concept that brachial neuritis is properly deemed congruent with GBS—or that medical science is increasingly viewing immunosuppressive treatments as effective for brachial neuritis).

I have similarly found in other cases involving a long, silent post-vaccination period before onset of injury that the "did cause" prong was not met. *See, e.g.*, *Bender v. Sec'y of Health & Human Servs.*, No. 11-693V, 2018 WL 3679637, at *34 (Fed. Cl. Spec. Mstr. July 2, 2018) (finding that petitioner's transverse myelitis, which began forty-two days after receipt of the meningococcal and Hepatitis A vaccines, could not have been caused by the vaccines when there was no evidence of subclinical process occurring over that six-week period), *mot. for review denied*, 141 Fed. Cl. 262 (2019). No evidence was offered here establishing that brachial neuritis would otherwise be characterized by a subclinical period that could explain the medical record silence. And Petitioner did not fully rebut Dr. Lancaster's point that certain medical records seem to contain statements to

[23] Although at the 2015 fact hearing Petitioner attempted to establish that he had been experiencing some initial symptoms in mid-August 2009, I found that these allegations were not corroborated by the medical record, and that he did not otherwise establish why the presumption of accuracy that attaches to such records under the law should not control here as well. *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) (crediting special master's decision to rely on contemporaneous medical records over later-in-time statements from the petitioner to the contrary).

providers by Mr. Greene that at the time of onset, he associated his sudden pain with very recent hard work at his construction job (something Dr. Lancaster deemed as an intervening, possibly explanatory occurrence).[24]

Respondent also raised fair questions about whether the true cause of Petitioner's brachial neuritis was the injury to his hand in July 2009 that prompted receipt of the tetanus vaccination in the first place. Based upon the existing record (and although I do not find the burden ever shifted to Respondent), I cannot find that an alternative cause has been established by a preponderance, any more than that the vaccine has been established to be causal. This does, however, raise a reasonable point further undercutting Petitioner's claim. For, if the tetanus vaccine could explain Petitioner's injury forty-one days later, on a medical record devoid of reference to any symptoms or signs in the intervening period, why would the injury that occurred immediately prior to its administration *not* also potentially be causal? Petitioner did not establish that brachial neuritis due to vaccine is distinguishable in course or presenting symptoms from that caused by trauma, or why the record otherwise better supports vaccination as the cause.[25]

CONCLUSION

This claim should have been resolved far sooner. Although some delay was attributable to the parties' good faith efforts to settle, Petitioner's unwillingness to abandon the legally-untenable position that the Table timeframe applicable to brachial neuritis could be leveraged in a non-Table context also interfered with its timely resolution. Petitioner did eventually marshal reliable evidence to support his claim, thus justifying a hearing. But after considering the expert witnesses and evidence upon which they relied, it is my reasoned conclusion that Petitioner has failed—despite abundant opportunity—to preponderantly substantiate his contention that a forty-one-day onset period for his brachial neuritis was medically acceptable, or that the tetanus vaccine more likely than not "did cause" his subsequent injury.

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[24] I do not find in this case, however, that Petitioner's brachial neuritis was "more likely than not" caused by such trauma close-in-time to the ER visit, since the record does not support that conclusion preponderantly. But this *does* constitute evidence undermining Petitioner's argument that only the vaccine could be causal, and was ineffectively rebutted.

[25] Of course, the causal interplay of hand injury versus vaccine does not solely cut in Respondent's favor. If this were a case where Petitioner's onset had occurred in a more medically-reasonable timeframe, application of the *Shyface* "substantial factor" could compel a finding *for* Petitioner under the present circumstances despite the possible role of the hand injury in producing brachial neuritis. *Heinzelman v. Sec'y of Health & Human Servs.*, No. 07–01V, 2008 WL 5479123, at *4 (Fed. Cl. Spec. Mstr. Dec. 11, 2008) (vaccine may still be substantial factor in causing injury sufficient to justify entitlement award, even where "two forces act in concert"). But this only illuminates the significance of my finding that the timeframe of onset was too remote from *either* occurrence to even weigh such competing causative factors.

I accordingly **DISMISS** Petitioner's causation claim. In the absence of a timely-filed motion for review (see Appendix B to the Rules of Court), the Clerk shall enter judgment in accord with this decision.[26]

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Special Master

[26] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.