In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 18-1601V
Filed: January 7, 2026

STACY L. DEFENZA *as administrator of* ESTATE OF LINDA L. CHERVENOK, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner

Jeffrey S. Pop, Jeffrey S. Pop & Associates, Beverly Hills, CA, for petitioner.
Nina Ren, U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

On October 16, 2018, Linda Chervenok filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2018),[2] alleging that she developed Guillain-Barré Syndrome ("GBS") as a result of an influenza ("flu") vaccination she received on October 21, 2015. After her passing, her estate representative, Stacy L. Defenza, was substituted as petitioner on November 20, 2025. For the reasons set forth below, I conclude that petitioner is *not* entitled to an award of compensation.

[1] Because this document contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the document will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] Within this decision, all citation to § 300aa will be the relevant sections of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations, including showing that an individual received a vaccination covered by the statute; received it in the United States; suffered a serious, long-standing injury; and has received no previous award or settlement on account of the injury. Finally – and the key question in most cases under the Program – the petitioner must also establish a *causal link* between the vaccination and the injury. In some cases, the petitioner may simply demonstrate the occurrence of what has been called a "Table Injury." That is, it may be shown that the vaccine recipient suffered an injury of the type enumerated in the "Vaccine Injury Table," corresponding to the vaccination in question, within an applicable time period following the vaccination also specified in the Table. If so, the Table Injury is presumed to have been caused by the vaccination, and the petitioner is automatically entitled to compensation, unless it is affirmatively shown that the injury was caused by some factor other than the vaccination. § 300aa-13(a)(1); § 300 aa-11(c)(1)(C)(i); § 300aa-14(a).

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines ex rel. Sevier v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991).

In this case, petitioner has alleged that the flu vaccine caused Ms. Chervenok to suffer GBS. GBS is listed on the Vaccine Injury Table relative to flu vaccine when onset of symptoms occurs "not less than 3 days and not more than 42 days" following vaccination. 42 C.F.R. § 100.3(a)(XIV)(D). However, petitioner does not allege Ms. Chervenok suffered a Table claim because her onset of symptoms began about 24-hours after her vaccination, outside of the timeframe provided by the Vaccine Table. (ECF Nos. 1, 99, 102.) Therefore, petitioner must demonstrate causation-in-fact.

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1278-79; *Hines*, 940 F.2d at 1525. Under that standard, petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. She need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition at issue and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999).

Thus, petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Ultimately, petitioner must satisfy what has come to be known as the *Althen* test, which requires: (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 proximate temporal relationship between vaccination and injury. *Id*.

A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1). Medical records are generally viewed as particularly trustworthy evidence because 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. § 300aa-13(b)(1). A petitioner may rely upon circumstantial evidence. *See Althen*, 418 F.3d at 1280. Moreover, the *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. While scientific certainty is not required, that expert's opinion must be based on "sound and reliable" medical or scientific explanation. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1359 (Fed. Cir. 2019).

Cases in the Vaccine Program are assigned to special masters who are responsible for "conducting all proceedings, including taking such evidence as may be appropriate, making the requisite findings of fact and conclusions of law, preparing a decision, and determining the amount of compensation, if any, to be awarded." Vaccine Rule 3(b)(1). Special masters must ensure each party has had a "full and fair opportunity" to develop the record but are empowered to determine the format for taking evidence based on the circumstances of each case, including having the discretion to decide cases without an evidentiary hearing. Vaccine Rule 3(b)(2); Vaccine Rule 8(a); Vaccine Rule 8(d). Special masters are not bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence in keeping with fundamental fairness to both parties. Vaccine Rule 8(b)(1). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." § 300aa-13(b)(1). The special master is required to consider the entirety of the evidentiary record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler v. Sec'y of Health & Human Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023) (citing *Hines*, 940 F.2d at 1528).

II. Procedural History

This case was originally assigned to another special master. (ECF No. 5.) Petitioner filed medical records, affidavits, and an opinion letter by her treating neurosurgeon, marked as Exhibits 1-15. (ECF Nos. 9, 13.) The case was reassigned to the undersigned in August of 2019. (ECF Nos. 25-26.) Respondent filed his Rule 4 Report in November of 2019, primarily arguing that the case should be dismissed based on the 24-hour post-vaccination period of onset. (ECF No. 29.) Respondent contended that the treating neurosurgeon's opinion as stated in her letter was limited to recognizing that the GBS was temporally proximate to the vaccination. (*Id*. at 18 (discussing Ex. 12).)

In September of 2020, petitioner filed an expert report and supporting medical literature by neuroimmunologist Lawrence Steinman, M.D.[3] (ECF Nos. 34-36; Exs. 16-40.) Dr. Steinman opined that the flu vaccine can cause GBS via an immunologic concept known as molecular mimicry and that such a process can occur within 24 hours of vaccination in the context of a recall antibody response following repeat vaccination. (Ex. 16, pp. 21-25.) In February of 2021, respondent filed a responsive expert report and supporting medical literature by immunologist J. Lindsay Whitton, M.D., Ph.D.[4] (ECF No. 43; Exs. A-B.) In pertinent part, Dr. Whitton challenged Dr. Steinman's opinion that molecular mimicry can lead to disease within 24 hours, even in the context of a recall response. (Ex. A, pp. 18-23.)

Thereafter, a Rule 5 Order was filed on February 19, 2021. (ECF No. 44.) The undersigned stressed that the timing of onset was likely to be the dispositive issue, noting skepticism that petitioner would be able to meet her burden of proof under *Althen* prong three. (*Id*.) Petitioner then filed a supplemental report by Dr. Steinman in June of 2021 (ECF Nos. 46-47; Exs. 42-53), and respondent responded with a supplemental report by Dr. Whitton in November of 2021 (ECF No. 51; Ex. C). Petitioner filed a second opinion letter by her treating neurosurgeon, along with a third report by Dr. Steinman. (ECF No. 53; Exs. 54-62.) Respondent then filed a further report by Dr. Whitton. (ECF No. 55; Ex. D.)

An entitlement hearing was subsequently set for March of 2024 (ECF No. 58), and petitioner filed updated medical records (ECF Nos. 60-61; Exs. 63-77). Additional

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[3] Dr. Lawrence Steinman obtained his medical degree from Harvard University. (Ex. 56, p. 1.) He completed two residencies at Stanford University, one in pediatrics and one in pediatric and adult neurology, as well as a fellowship at the Wiezmann Institute of Science and National Institutes of Health. (*Id.*) Dr. Steinman is board certified in psychiatry and neurology. (*Id.* at 2.) He currently works as a professor at Stanford University in the Departments of Neurology and Neurological Sciences, Pediatrics, and Genetics. (*Id.*) He has published 606 peer reviewed articles. (*Id.* at 5-52.)

[4] Dr. J. Lindsay Whitton received his medical degree and Ph.D. from the University of Glasgow in Scotland. (Ex. B, p. 1.) He worked as a professor in the Department of Immunology and Microbial Science at Scripps Research Institute until retiring in March of 2023. (*Id.*; Tr. 114.) However, he maintains a position as a professor emeritus at Scripps Research Institute. (Tr. 114.) Dr. Whitton has published 192 peer reviewed articles. (Ex. B, pp. 2-15.)

exhibits were filed during the pre-hearing proceedings, prompting a contested motion for leave to file exhibits out of time (ECF Nos. 81-84). Petitioner was permitted to introduce the disputed articles (Exs. 115-18) into the record, but was limited to presenting the articles in connection with a supplemental report and precluded from discussing the articles during the hearing. (ECF No. 86.) The hearing was held on March 4 and 5, 2024. (*See* Transcript of Proceeding ("Tr."), at ECF Nos. 92-93.) Drs. Steinman and Whitton testified. Dr. Steinman was proffered without objection as an expert in neurology, immunology, and neuroimmunology, and Dr. Whitton was proffered without objection as an expert in immunology.[5] (Tr. 28, 120.) During the hearing, both parties attempted to introduce still further new evidence into the records. (Exs. 120-21; Exs. F-H.) The parties were again permitted to present this new evidence in connection with supplemental reports. (ECF No. 90.)

Following the hearing, petitioner filed a supplemental report by Dr. Steinman (ECF No. 95; Ex. 122), respondent filed a responsive supplemental report by Dr. Whitton (ECF No. 97; Ex. I), and Dr. Steinman provided a final report in reply (ECF No. 98; Ex. 123). The parties then filed post-hearing briefs. (ECF No. 99-100, 102.) In their briefs, petitioner argued that she has satisfied all three prongs of the *Althen* test, while respondent argued primarily that petitioner failed to satisfy the third *Althen* prong. (ECF Nos. 99, 100.) However, respondent also argued that petitioner's failure to meet her burden of proof under *Althen* prong three by extension is also fatal to her showing under *Althen* prongs one and two. (ECF No. 100, pp. 27-30.)

In light of the above, I have concluded that the parties have had a full and fair opportunity to develop the record of this case and that the case is ripe for resolution of entitlement.

III. Summary of the Record Evidence

The facts of this case are largely undisputed and relatively few are germane to the analysis that follows. Accordingly, a very brief factual summary is appropriate. Ms. Chervenok received the flu vaccination at issue on October 21, 2015, and developed numbness, tingling, and flaccid paralysis in her extremities, as well as shortness of breath, approximately 24 hours later. (Ex. 2; Ex. 3, pp. 80, 98, 112-15, 130, 140; Ex. 41, p. 18; Ex. 71.) Although the treating physicians initially suspected her symptoms had a spinal etiology (*e.g.*, Ex. 3, p. 429), her ultimate diagnosis of GBS is not disputed. (ECF No. 62.) No other cause of Ms. Chervenok's GBS has been asserted. (*E.g.*, Tr. 204-05 (respondent's expert characterizing the GBS as idiopathic).)

Pertinent to the expert opinions, two details of Ms. Chervenok's prior medical history need to be noted. First, Dr. Steinman's opinion assumes that she had

[5] In their post-hearing briefs, both parties include arguments relative to Dr. Steinman's credibility, with petitioner stressing his direct involvement with the concepts (and papers) at issue and respondent arguing that he has no particular expertise with GBS and often acts as an advocate. (ECF No. 99, p. 7; ECF No. 100, n.1.) I have considered these arguments, but conclude that these factors do not change the analysis that follows or the outcome of the case.

experienced prior exposure to the specific component of the flu vaccine he opines was causal. (Tr. 64-65.) In that regard, she received the flu vaccine in prior years, including years during which H1N1 was included in the vaccine. (Ex. 41, pp. 31, 38.) Second, Ms. Chervenok had a history of lower back pain for which she underwent a radiofrequency ablation ("RFA") procedure approximately two weeks prior to the vaccination at issue. (Ex. 11, pp. 1-2, Tr. 7.) The experts agree that RFA can result in an elevation of cytokines that persists for up to a month, though they disagree as to the significance of that point. (Ex. 122, pp. 1-2; Ex. I, pp. 2-3.)

Treating neurosurgeon, Dr. Allison Rathman, prepared a letter supporting this claim.[6] (Ex. 12.) Dr. Rathman opined that "[i]t is with a high degree of medical certainty that I can say that [Ms. Chervenok's] sudden onset of quadriplegia and respiratory failure were due to GBS, which was, more likely than not, caused by the flu vaccination of October 21, 2015." (*Id*. at 3.) Dr. Rathman's letter, though it discussed the details of Ms. Chervenok's clinical presentation, did not explicitly indicate why Dr. Rathman attributed her GBS to her flu vaccination. (Ex. 12.) However, Dr. Rathman also prepared a second letter. (Ex. 54.) In the second letter, Dr. Rathman advised that she had been prompted to indicate whether any medical literature supported the post-vaccination period of onset at issue in this case.[7] Dr. Rathman cited two epidemiologic studies by Salmon et al. and Park et al., which were also discussed in greater detail by the experts. (*Id*.) Otherwise, she "defer[ed] to a neurologist or immunologist on how biologically this can occur." (*Id*.) Dr. Rathman indicated that her opinion is based on the temporal association between Ms. Chervenok's GBS and her prior flu vaccine, as well as on the fact that "no other cause for her GBS was identified following a robust work up." (*Id*.)

Dr. Steinman further explains that GBS is an autoimmune peripheral nerve injury in which the myelin tissue of the nerve is attacked. (Ex. 16, pp. 8-9; Tr. 31-32.) It is mediated both by T lymphocytes and by antibodies. (Ex. 16, pp. 10-12; Tr. 32.) Dr. Steinman opines that the flu vaccine is an established cause of GBS. (Ex. 16, p. 12; Ex. 42, p. 11; Tr. 33.) In particular, Dr. Steinman opines that antibodies produced in response to the A/California/7/2009, H1N1, component of the seasonal influenza vaccine, are capable of cross-reacting with gangliosides contained within the myelin and

[6] Ms. Chervenok underwent a cervical laminectomy performed by Dr. Rathman, which failed to relieve her symptoms, before the treating physicians ultimately concluded that her condition was GBS. (Ex. 3, pp. 833-34, 847; Ex. 8, p. 31-32; Ex. 12, p. 3.)

[7] Dr. Rathman's assessment of the actual onset of GBS is a bit unclear. In her second letter, she refers to onset as having been "within 2 days of vaccination." (Ex. 54, p. 1.) In her first letter, however, she indicated that Ms. Chervenok first presented to her for care on October 23, 2015, which was two days post-vaccination, but noted that "she began to experience sensory changes and motor weakness" on October 22, 2015, which was only one day post-vaccination. (Ex. 12, p. 1.) Although the second letter referred to onset of GBS occurring on the second day post-vaccination, Dr. Rathman never (in either letter) sought to parse the October 22 symptoms as being unrelated to GBS. In any event, petitioner's post-hearing briefing affirmatively agreed that the onset of GBS was on October 22, 2015, the day after the vaccination. (ECF No. 99, p. 6.) Based on my own review of the record as a whole, I also conclude that it is more likely than not that the onset of GBS occurred approximately 24 hours post-vaccination.

axons in the peripheral nerves, resulting in GBS via the process of molecular mimicry. (Ex. 16, pp. 11-20.) In this case, though Dr. Whitton challenged whether molecular mimicry explains why there is an elevated risk of GBS following flu vaccination, the question of whether the flu vaccine can cause GBS is not meaningfully at issue as a general matter.

Instead, as previously noted, the primary issue in this case is whether an autoimmune disease process driven by molecular mimicry, as relied upon by Dr. Steinman, can occur within one day. Dr. Steinman opines that it can, whereas respondent's expert, Dr Whitton, disagrees, opining that this is implausible. (Tr. 35-36. 121-22.) The reasons for the experts' disagreement on this point, and the evidence underlying their competing views, is discussed extensively throughout the analysis under *Althen* prong three.

IV. Analysis

As explained above, under the three-part *Althen* test, petitioner must demonstrate (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 proximate temporal relationship between vaccination and injury. *Althen*, 418 F.3d at 1278. In this case, however, *Althen* prongs one and two need little attention while *Althen* prong three is dispositive.

a. *Althen* prong three is dispositive (analysis of *Althen* prongs one and two)

Regarding *Althen* prong one, Dr. Steinman presented a theory of causation whereby the flu vaccine can cause GBS via molecular mimicry. (Ex. 16, p. 9; Robert S. Fujinami et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 CLINICAL MICROBIOLOGY REVS. 80 (2006) (Ex. 23, p. 1-2).) Specifically, he opined that the H1N1 component of the flu vaccine can elicit anti-ganglioside antibodies, which cross-react against gangliosides contained within the myelin and axons in the peripheral nerves, resulting in GBS. (Ex. 16, pp. 11-20.) On respondent's behalf, although he did not agree that molecular mimicry has been established as the cause of post-vaccination GBS, Dr. Whitton agreed that there is an increased risk of GBS following vaccination against the flu. (Tr. 172-75.) He also testified that, apart from the timing at issue in this case, he did not have any significant disagreement with Dr. Steinman's discussion of molecular mimicry. (*Id.* at 156.)

Regarding *Althen* prong two, petitioner relies primarily on the opinion of treating neurosurgeon, Dr. Rathman, who opined that Ms. Chervenok's GBS was caused by her preceding flu vaccine.[8] Dr. Rathman's opinion is limited insofar as her opinion was

[8] In her post-hearing brief, petitioner also notes statements by several other treating physicians, including Ms. Chervenok's urologist, primary care provider, rheumatologist, and occupational therapist, that she argues had attributed the GBS to the prior flu vaccination. (ECF No. 99, pp. 37-38.) Some of the cited statements are ambiguous with respect to whether they include causal opinions or merely state the fact

based on close temporal proximity and the elimination of alternative causes. (Ex. 54.) These considerations standing alone do not carry petitioner's burden of proof. *Althen*, 418 F.3d at 1278 (explaining that "[a]lthough probative, neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation"). Nonetheless, the Federal Circuit has held that, where a petitioner has demonstrated *Althen* prongs one and three, treating physician opinions are "quite probative" and in that context relying in part on temporal proximity is not disqualifying. *Capizzano v. Sec'y of Health & Human Servs*., 440 F.3d 1317, 1326 (Fed. Cir. 2006).

In light of the above, and considering the record as a whole, I conclude that petitioner has satisfied *Althen* prong one. However, petitioner's showing under *Althen* prong two is dependent upon her success under *Althen* prong three. If petitioner had succeeded under *Althen* prong three, I would have credited Dr. Rathman's opinion and petitioner would have met her burden under *Althen* prong two. However, because petitioner has not demonstrated, under *Althen* prong three, that the timing of onset in this case is appropriate for a causal inference, she likewise cannot meet her burden under *Althen* prong two, especially given the degree to which Dr. Rathman's assessment was predicated on an appropriate temporal relationship between the vaccination and the injury. *Accord Tripp v. Sec'y of Health & Human Servs*., 178 Fed. Cl. 688, 699 (2025) (rejecting as circular the argument that petitioner's treaters' diagnosis should dictate the issue of general causation). Accordingly, *Althen* prong three is dispositive in this case.

b. Petitioner has not met her burden of proof under *Althen* prong three

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* at 1281. 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-in-fact." *de 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 coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id*.; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd per curiam*, 503 F. App'x 952 (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 rev. denied sub nom. C.K. v. Sec'y of Health & Human Servs.*, 113 Fed. Cl. 757 (2013), *aff'd sub nom. Koehn v. Sec'y of Health & Human Servs.*, 773 F.3d 1239 (Fed. Cir. 2014).

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that the GBS arose post-vaccination. But in any event, none of these notations, as highlighted by petitioner, identify any reasoning beyond what was articulated by Dr. Rathman in her two letters.

In this case, there is no dispute that the onset of Ms. Chervenok's GBS occurred about 24 hours post-vaccination. (ECF No. 99, p. 6; ECF No. 100, p. 8.) Instead, the core dispute in this case is whether that period of onset is appropriate for a causal inference based on Dr. Steinman's theory of causation, which is based on molecular mimicry. (ECF No. 99, pp. 6-29; ECF No. 100, pp. 9-22; ECF No. 102, pp. 9-17.) Discussion of this issue can be broken down into two distinct topics: First, whether Dr. Steinman has provided an immunologic explanation as to how an injury due to molecular mimicry can occur within 24 hours; and second, whether available epidemiology bolsters Dr. Steinman's view or otherwise persuasively establishes an increased risk of post-vaccination GBS inclusive of the first day post-vaccination.

i. Dr. Steinman's immunologic explanation

Although the pathophysiology of GBS is not completely understood (Ex. 16, p. 8), Dr. Steinman has been clear in asserting that his causal opinion implicating the flu vaccine is based on the development of injury-causing autoantibodies as part of the adaptive immune response (*Id*. at 12 (explaining that "one of the components of Petitioner's vaccine, the H1N1 component, could elicit antibodies that are associated with GBS"). On respondent's behalf, however, Dr. Whitton opines that, while there is no bright line, an adaptive immune response usually takes at least six or so days to develop. (Tr. 175-76.) While some shorter latencies, such as three days, may be arguable, he opines that a 24-hour latency for the development of a meaningful antibody response capable of resulting in an autoimmune disease is clearly *implausible*. (*Id*. at 160, 175-76.)

Dr. Steinman disagrees with Dr. Whitton's assessment of the appropriate timing, opining that it is medically reasonable to conclude that the disease process at issue can occur within one day of vaccination. (Tr. 35-36.) Although he noted as a general matter that a primary adaptive immune response usually takes between 10 to 30 days to develop (*Id*. at 69-70), Dr. Steinman offers several reasons for concluding that, under the right circumstances, the adaptive immune response can produce disease causing levels of autoantibodies within 24 hours. Specifically, Dr. Steinman opines that the following factors explain how the adaptive immune response can produce disease-causing levels of autoantibody within 24 hours (*Id*. at 101):

(1) When an individual encounters a repeat antigen, such as following repeated vaccinations, they experience a recall (B cell) response, which produces antibodies faster than a primary immune response (Tr. 66-73);

(2) In addition, such an individual also has circulating plasmablasts, which are also separately and concurrently stimulated to produce further antibodies (Tr. 80-82, 85-86);

(3) Further still, circulating IL-6, both as a result of the vaccination at issue, as well as in this case from a prior RFA procedure, has an additional

stimulating effect on antibody production (Tr. 89-94, 226-27; Ex. 122, pp. 1-2); and

(4) Ultimately, because the individual necessarily has preexisting antibodies in circulation from prior antigen exposure(s) that are already potentially cross-reactive, the immune response at issue need only produce enough additional antibodies to reach the tipping point for autoimmunity (Tr. 76-77, 94-95).

Dr. Steinman opines that, because these processes are happening concurrently, and because the only thing that matters is the aggregate increase in antibody levels, it is possible for an immune response to break self-tolerance within 24 hours. (Tr. 101.) For the reasons discussed below, Dr. Steinman is not persuasive in contending that any of the points that have been raised, either alone or in combination, demonstrate that the immune response underlying his theory can develop as quickly as he opines.

1. Recall response

In his initial report, Dr. Steinman relied primarily on the concept of a recall response. (Ex. 16, pp. 21-23.) (It was only later that he further developed his opinion to involve other concurrent immune responses.) Specifically, because Ms. Chervenok had been previously vaccinated with the H1N1 component of the flu vaccine, she already had a population of preformed antibodies, as well as memory B and T cells, which can respond rapidly to restimulation with H1N1. (*Id*.) In particular, Dr. Steinman cited a graph from an immunology textbook by Siegrist et al., which showed that while a primary immune response (as measured by IgG antibody titers) begins more slowly and peaks after 10 days, a secondary, or recall, response rises much more rapidly and peaks much more quickly. (*Id*. at 23 (citing Claire-Anne Siegrist, *Vaccine Immunology*, *in* PLOTKIN'S VACCINES 16 (2018) (Ex. 38, fig. 2.3)).)

During the hearing, however, Dr. Steinman acknowledged that, while it is clear that the secondary immune response "shoots up" earlier, "we don't know how long it takes to start" and, though he opines it can occur within one day, he cannot reach "certainty" on that point. (Tr. 70-73.) Notably, the Seigrist authors are no more specific than to characterize the "rapid" antibody increase reflected in the graph cited by Dr. Steinman as being less than seven days. (Siegrist, *supra*, at Ex. 38, fig. 2.3.) Nonetheless, Dr. Steinman also cites tuberculin tests as an example of a recall response that occurs within 24 hours, given that the test result (a small skin reaction) can be observed within that time. (Tr. 96-99 (citing Tiroumourougane V. Serane & Bhuvaneswari Kothendaraman, *Tuberculin Test Can Be Read After 24 Hours in Adolescent Children*, 60 J. TROPICAL PEDIATRICS 157 (2014) (Ex. 110)).)

Dr. Whitton explains, however, that the recall response is memory B cell dependent and B cells do not produce antibodies directly. (Tr. 132 (citing Kim L. Good & Stuart G. Tangye, *Decreased Expression of Krüppel-like Factors in Memory B Cells Induces the Rapid Response Typical of Secondary Antibody Responses*, 104 PNAS

13420 (2007) (Ex. A22)); Ex. I, pp. 8-10 (citing Robert Markewitz et al., *Kinetics of the Antibody Response to Boosting with Three Different Vaccines Against SARS-CoV-2*, FRONTIERS IMMUNOLOGY, Jan. 2022, at 1 (Ex. H)).) When a memory B cell is activated by an antigen, it starts dividing into immature cells known as plasmablasts (also discussed further below), which can then in turn lead to antibody production. (Tr. 135-36, 139-40.) According to Dr. Whitton, a study by Good and Tangye, while admittedly only an ex vivo experiment, evidences that this process does not even begin until at least 24 hours post-stimulation and then each generation of division takes about six hours. (*Id*. at 143, 202-03.) Even acknowledging that divisions increase exponentially with each generation, Dr. Whitton opines that it takes at least three days for this division to occur at meaningful levels. (*Id*. at 135, 140, 143.)

Ultimately, Dr. Whitton explains that the time it takes for a memory B cell to divide to create antibody-producing plasmablasts is a fundamental limiting factor in how long it takes to initiate a meaningful recall response. (Tr. 141.) Dr. Whitton notes that tuberculin tests cited by Dr. Steinman, by contrast, measure an expected immune response and operate via T cell response, specifically a delayed hypersensitivity response. (*Id*. at 153-55; Serane & Kothendaraman, *supra*, at Ex. 110, p. 3.) They are therefore not informative of how long it would take to produce an injurious, cross-reactive, antibody response as is at issue in this case. (Tr. 153-54.)

Petitioner challenges the relevance of the two papers cited by Dr. Whitton (Good & Tangye and Markowitz et al.). (ECF No. 99, pp. 31-32; ECF No. 102, pp. 11-12; Tr. 77-79.) Adopting the phrasing of the IOM,[9] she also argues that the recall response eliminates the so-called "lag phase" of the immune response and proceeds directly to the "logarithmic phase," *i.e.*, the exponential increase in divisions with each generation noted by Dr. Whitton. (ECF No. 99, pp. 18-19.) Importantly, however, consistent with Dr. Whitton's explanation, the IOM likewise indicates that the logarithmic phase is a 3-5 day process. (ADVERSE EFFECTS OF VACCINES: EVIDENCE AND CAUSALITY (Kathleen Stratton et al. eds., 2012) [hereinafter 2012 IOM Report] (Ex. A, Tab. 12, p. 2).) Moreover, the IOM suggests that the recall response shortens the lag phase from about 7-10 days to between 1-3 days (*Id*.), which remains consistent with Dr. Whitton's testimony that it takes at least 24 hours to initiate B cell division. Thus, petitioner's suggestion that the lag phase is eliminated altogether is not supported. And, as noted above, Siegrist, as cited by Dr. Steinman, is not to the contrary in that the author characterizes a "rapid" secondary response merely as being less than 7 days. (Siegrist, *supra*, at Ex. 38, fig. 2.3.)

[9] The Institute of Medicine (known as the National Academy of Medicine since 2015) is the medical arm of the National Academy of Sciences. The National Academy of Sciences ("NAS") was created by Congress in 1863 to be an advisor to the federal government on scientific and technical matters (*see* An Act to Incorporate the National Academy of Sciences, ch. 111, 12 Stat. 806 (1863)), and the Institute of Medicine ("IOM") is an offshoot of the NAS established in 1970 to provide advice concerning medical issues. When Congress enacted the Vaccine Act in 1986, it directed that the IOM conduct studies concerning potential causal relationships between vaccines and illnesses. *See* § 300aa-1 note.

Accordingly, although Dr. Steinman has demonstrated that a secondary or recall response does develop more quickly than a primary immune response, the concept of a recall response does not explain how molecular mimicry can occur within 24 hours.

2. Concurrent immune processes proposed by Dr. Steinman

After being challenged with regard to the notion that a recall response in itself could explain a 24-hour post-vaccination onset, Dr. Steinman later introduced two other means of antibody stimulation that he opined would act concurrently as the recall response developed. However, Dr. Steiman is not persuasive in seeking to demonstrate that these immune responses would act to further accelerate the recall response beyond its otherwise understood limitations.

a. Plasmablasts

Dr. Steinman opines that in addition to the B cell-driven recall response, "plasmablasts," which he contends can be long-lived, are already circulating in the bloodstream and therefore will also act in the same manner as memory B cells, interacting with antigens to undergo somatic hypermutation and producing high affinity antibodies. (Tr. 81-86.) According to Dr. Steinman, plasmablasts are "antibody factories" that are dividing and producing antibodies at all times (*Id*. at 81-82, 86), and so, "[m]olecules that turn up the production of antibodies are elicited, somewhere between three and seven hours" of vaccination and therefore "[y]ou don't need more cell divisions." (*Id*. at 75-76; *see also* Ex. 123, p. 5.) Thus, Dr. Steinman effectively opines that the existence of circulating plasmablasts obviates the above-discussed lag phase of the recall response and foreshortens the logarithmic phase. Dr. Whitton, however, charges that Dr. Steinman is conflating plasmablasts, which are short-lived and do not produce significant antibodies, with long-lived plasma cells, which reside in the bone marrow and do continuously produce antibodies without antigen stimulation. (Tr. 132-36.) According to Dr. Whitton, it is the long-lived plasma cells of the bone marrow, and not any circulating plasmablasts, that constitute the "antibody factories." (Ex. I, p. 6.) This is, perhaps, the most fundamental disagreement between the experts in this case.

As Dr. Whitton explains it, after a B cell (either memory or naive) makes contact with an antigen, it differentiates into a plasmablast, which is an immature plasma cell that itself has a lifespan of only 3-5 days and produces few antibodies. However, that plasmablast will then start dividing, resulting in either short-lived plasma cells, which last for about 3-4 weeks, or end stage plasma cells. (Tr. 132-34.) End stage plasma cells, or long-lived plasma cells, do not proliferate, but instead migrate to the bone marrow where they go on to produce antibodies. (*Id*.) Dr. Whitton points to a 2022 immunology textbook by Murphy et al. to suggest that the distinction between plasmablasts and long-lived plasma cells represents basic immunologic understanding. (Ex. I, pp. 6-7 (quoting MURPHY ET AL., JANEWAY'S IMMUNOBIOLOGY (10th ed. 2022) (Ex. F)).) That textbook explains that

> Plasmablasts are cells that have begun to secrete antibody, yet are still dividing and express many of the characteristics of activated B cells that allow their interaction with T cells. After a few more days, the plasmablasts in the primary focus stop dividing and may eventually die. Subsequently, plasmablasts or B cells arising from the germinal centers can migrate to the bone marrow and become long-lived plasma cells, where they continue antibody production.

(MURPHY ET AL., *supra*, at Ex. F, p. 1.)

Dr. Steinman counters, however, that this description by Murphy et al. is "outdated" insofar as it does not account for the findings of the papers he has cited. (Ex. 122, p. 4.) Dr. Steinman's own research group demonstrated, in the context of research into multiple sclerosis, that plasmablasts were the source of clonal antibodies to rubella, varicella, and measles, despite exposure to those antigens having occurred years prior. (Ex. 123, p. 2 (discussing Tobias V. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis Bind EBV EBNA1 and GlialCAM*, 603 NATURE 321 (2022) (Ex. 58)).) A further paper showed that plasmablasts persisted and produced antibodies that prevented tumor growth in cancer patients. (Ex. 122, p. 2 (discussing Jeff DeFalco et al., *Non-Progressing Cancer Patients Have Persistent B Cell Responses Expressing Shared Antibody Paratropes That Target Public Tumor Antigens*, 187 CLINICAL IMMUNOLOGY 37 (2018) (Ex. 120)); Tr. 211-18.) And, finally, a commentary by Sato et al. likewise indicates that plasmablasts are perpetually produced in the context of neuromyelitis optica. (Tr. 211-13, 218-19 (discussing Douglas Kazutoshi Sato et al., *Cerebrospinal Fluid Aquaporin-4 Antibody Levels in Neuromyelitis Optica Attacks*, 76 ANNALS NEUROLOGY 305 (2014) (Ex. 121)).)

Yet, Dr. Whitton has pointed out that none of the papers cited by Dr. Steinman actually evidence the notion that the plasmablasts are themselves long-lived as he had seemed to suggest. (Ex. I.) Thus, for example, Dr. Steinman appeared to be careful in his hearing testimony to assert only that plasmablasts persist as a class of cells, rather than as individual cells. (Tr. 212-13.) He stated that "[w]hether the existing antibody levels are due to, let's say, plasmablast A or its progeny, who are identical, remains unknown." (*Id.* at 212.) And, indeed, in his penultimate report, Dr. Steinman characterized antibody-producing plasmablasts as "either long-lived *or constantly replenished*." (Ex. 122, p. 7 (emphasis added).) In fact, Dr. Steinman specifically testified that his Lanz research group did not investigate the longevity of plasmablasts. (Tr. 83-84 (discussing Lanz et al., *supra*, at Ex. 58).)

Following on from that point, Dr. Whitton asserts that the papers cited by Dr. Steinman (Lanz et al., DeFalco et al., and Sato et al.) all involved subjects experiencing chronic disease wherein the immune system is continually activated by the antigen exposure. (Ex. I, pp. 3-5.) This is distinguishable from vaccination where exposure to the antigen is only transient. (*Id.*) In his final report, Dr. Steinman responded to Dr. Whitton's critique by stressing how the studies he has cited support plasmablasts as a source of a long-lived *antibody* response, but upon my review, continued to equate a transient immune response from vaccination with a chronic immune response without directly addressing the question of plasmablasts being constantly replenished versus long-lived. (Ex. 123.)

Thus, Dr. Whitton has suggested, and Dr. Steinman has not refuted, that the long-lived antibody response Dr. Steiman cites, even if generated by plasmablasts, is made possible by the ongoing stimulation of the B cell immune response. Dr. Whitton suggests that, absent that added factor, there is no evidence supporting the notion that plasmablasts, which are otherwise understood to be short lived, continue to circulate perpetually. (*E.g.*, Ex. I, pp. 3-7.) As observed by Murphy et al., the general understanding is that "the plasmablasts in the primary focus stop dividing" after a few days. (MURPHY ET AL., *supra*, at Ex. F, p. 1.) Therefore, the record evidence, including the specific studies cited by Dr. Steinman, do not readily evidence that a person like Ms. Chervenok, prior to the vaccination at issue, having prior exposures but not ongoing immune dysfunction, would have perpetually circulating antibody-producing plasmablasts. Otherwise, the paper by Rodda and Pepper as cited by Dr. Steinman simply demonstrates (especially Figure 1) that it is memory cells in circulation that give rise to antibody-secreting plasmablasts following flu vaccination, *i.e.*, the recall response. (Tr. 81-82 (discussing Lauren B. Rodda & Marion Pepper, *Naive and Memory B Cells Respond to Flu Vaccine*, 586 NATURE 34 (2020) (Ex. 48)).)

But in any event, none of the papers cited by Dr. Steinman address timing. Even if fully crediting Dr. Steinman's opinion regarding the existence of "long-lived plasmablasts," nothing on this record apart from Dr. Steinman's *ipse dixit* applies this concept to the timing question at issue in this case. Instead, the three papers cited by Dr. Steinman (Lanz et al., Sato et al., and DeFalco et al.) appear to demonstrate, in the context of ongoing autoimmune disease or cancer, merely that a B cell immune response can be persistent and that, in its persistence, it will continue to produce plasmablasts. Moreover, if Dr. Steinman were correct that any prior antigen exposure would result in perpetually circulating plasmablasts in healthy subjects, then the activity of these plasmablasts would necessarily be a part of the overall secondary immune response we observe in people experiencing repeat antigen exposure. Therefore, without evidence more specifically addressing how these proposed plasmablasts affect the timing of the overall immune response, Dr. Steinman's reference to plasmablasts is not sufficient to supplant the otherwise generally accepted understanding regarding the time needed to mount a meaningful antibody response, as in the preceding section regarding the recall response. Indeed, Dr. Steinman notes that plasmablasts are a part of the B cell family (Tr. 87) and the only paper he cited specifically examining post-flu vaccine plasmablasts is consistent with Dr. Whitton's explanation of memory B cell division following antigen contact (Rodda & Pepper, *supra*, at Ex. 48, fig. 1).

Accordingly, I am not persuaded by Dr. Steinman's assertion that perpetually circulating plasmablasts are implicated in this case. Dr. Steinman has not shown that plasmablasts are themselves long-lived or that they will persist in circulation in the absence of ongoing immune stimulation. Moreover, even if they were, Dr. Steinman is not persuasive in opining that this would meaningfully alter the medically appropriate timeframe for a causal inference based on a recall antibody response leading to molecular mimicry.

b. IL-6

Dr. Steinman further opines that the cytokine IL-6 has been shown to promote antibody production. (Tr. 89-94; Ex. 122, pp. 1-2 (citing Oliver Dienz et al., *The Induction of Antibody Production by IL-6 Is Indirectly Mediated by IL-21 Produced by CD4+ T Cells*, 206 J. EXPERIMENTAL MED. 69 (2009) (Ex. 59)).) Specifically: "Dienz immunized mice with inactive influenza virus in combination with IL-6 and analyzed virus-specific antibody production. Dienz found that the presence of IL-6 during immunization substantially enhanced the levels of influenza-specific IgG1. Dienz concluded that IL-6 enhances the in vivo antibody response." (*Id.* at 2 (citing Dienz et al., *supra*, at Ex 59, p. 7).) Dr. Steinman indicates that IL-6 promotes antibody production by increasing IL-21 production which, in turn, promotes the B cell helper capabilities of CD4+ T cells. (*Id*. (citing Dienz et al., *supra*, at Ex 59, p. 1).) Dr. Steinman also opines that this process could help to stimulate the above-discussed plasmablasts. (*Id.* at 3.)

In Ms. Chervenok's case, Dr. Steinman notes two significant sources of IL-6. (Ex. 122, pp. 1-2.) First, she underwent a RFA procedure approximately two weeks prior to vaccination. Dr. Steinman cites several papers for the proposition that this procedure elevates cytokines, including IL-6, for about four weeks. (*Id*. (citing Ryan Slovak et al., *Immuno-Thermal Ablations – Boosting the Anticancer Immune Response*, J. IMMUNOTHERAPY CANCER, Oct. 2017, at 1 (2017) (Ex. 115); H. Takaki et al., *Thermal Ablation and Immunomodulation: From Preclinical Experiments to Clinical*, 98 DIAGNOSTIC & INTERVENTIONAL IMAGING 651 (2017) (Ex. 116); Kanishka Rangamuwa et al., *Thermal Ablation in Non-Small Cell Lung Cancer: A Review of Treatment Modalities and the Evidence for Combination with Immune Checkpoint Inhibitors*, 10 TRAVEL LUNG CANCER RES. 2842 (2021) (Ex. 117)).) Second, the flu vaccination would itself have been a source of elevated IL-6. Citing Talaat et al., Dr. Steinman notes that a recall response to the flu vaccine can elevate cytokines within 7 hours of vaccination. (*Id*. at 1 (citing Kawsar R. Talaat et al., *Rapid Changes in Serum Cytokines and Chemokines in Response to Inactivated Influenza Vaccination*, 12 INFLUENZA OTHER RESPIRATORY VIRUSES 202 (2018) (Ex. 55, pp. 6-7, fig. 3)).) Thus, he concludes that the fact that petitioner had elevated levels of IL-6 helps to explain how she could experience a heightened and faster recall response leading to GBS within one day. (*Id*. at 2.)

Dr. Whitton does not dispute that the flu vaccine and prior RFA procedure would have produced IL-6. (Tr. 140-41; Ex. I, pp. 2-3.) However, he disagrees with Dr. Steinman's interpretation of the Dienz paper. Whereas Dr. Steinman presented the Dienz study as demonstrating that IL-6 affects circulating plasma cells, Dr. Whitton explains that the study examines IL-6 as a driver of B cell differentiation into plasma cells. (Tr. 146.) In particular, he stressed that the study showed that IL-6 enhanced levels of influenza-specific IgG when it was administered in combination with the flu antigen. (Ex. I, pp. 2-3.) Thus, the paper shows that IL-6 may enhance downstream antibody levels after the memory B cell is activated by antigen contact. (*Id*. at 3; Tr. 146-49.) It does not show that IL-6 would act to increase preexisting antibody levels in the absence of that antigen contact. (*Id*.) Moreover, the paper did not examine antibody levels until day 7 and after. (Tr. 146-49; Ex. I, pp. 2-3.) Thus, the Dienz paper does not provide evidence supporting the notion that IL-6 would further accelerate the recall response or otherwise meaningfully increase antibody levels within 24 hours. Similarly, while the Talaat paper does show that vaccination can elevate cytokines within 24 hours, this represents only the first step in the process identified by Dr. Steinman. That IL-6 would in turn need to promote IL-21 that would in turn promote CD4+ T cells to "help" B cells. And, in any event, the Talaat study did not include any measure of antibodies prior to the 14th day post-vaccination. (Tr. 150-52; Ex. D, p. 5.)

Thus, while Dr. Steinman has shown that elevated IL-6 would potentially contribute to an overall more robust antibody response to vaccination, he has not shown either that IL-6 would accelerate the antibody response generally or that it would do so within one day.

3. Preexisting antibodies and the "tipping point"

Finally, Dr. Steinman stressed that, due to her prior vaccinations, Ms. Chervenok would have had preexisting antibodies circulating in her system with high affinity and strong binding for flu antigens. (Tr. 73-74.) He opined that the antibodies generated by the immune responses discussed in the preceding sections would be additive to existing and ongoing antibody production. (*Id.* at 75.) Moreover, Dr. Steinman suggested that, because the baseline level of preexisting antibodies may already be high, it would be more likely that the body could produce sufficient additional antibodies to reach the tipping point to autoimmunity within one day. (Ex. 123, p. 5.) Dr. Steinman characterized this as "the heart of my theory." (Tr. 76.) Dr. Steinman cited two papers for this point. (Tr. 94-96 (discussing Ludwig Kappos et al., *Induction of a Non-Encephalitogenic Type 2 T Helper-Cell Autoimmune Response in Multiple Sclerosis After Administration of an Altered Peptide Ligand in a Placebo-Controlled, Randomized Phase II Trial*, 6 NATURE MED. 1176 (2000) (Ex. 60), and Bibiana Bielekova et al., *Encephalitogenic Potential of the Myelin Basic Protein Peptide (Amino Acids 83-99) in Multiple Sclerosis: Results of a Phase II Clinical Trial with an Altered Peptide Ligand*, 6 NATURE MED. 1167 (2000) (Ex. 61)).) According to Dr. Steinman, these studies show that, in some instances, it can take multiple injections of the same molecular mimic to result in an adverse event. (Ex. 55, p. 5.)

Dr. Whitton agreed that, as a result of her prior vaccinations, petitioner likely had high affinity antibodies for flu antigens in her bloodstream. (Tr. 137.) He also agreed that it is "perfectly reasonable" for Dr. Steinman to argue that "vaccination could drive those levels of antibody up over a threshold or a tipping point, which caused the development of [GBS]." (*Id.* at 138-39.) However, as with the recall response, Dr. Whitton did not agree that this would accelerate the immune response to the degree suggested by Dr. Steinman.

First, Dr. Whitton stressed that, by definition, any level of pre-existing antibody production petitioner was experiencing would *not* have been pathologic because she was not experiencing disease. (Tr. 137-38.) Whereas Dr. Steinman opined that petitioner "had pre-existing antibodies to influenza at the time of her immunization on 10/21/15 that triggered a strong recall response" (Ex. 122, p. 6), Dr. Whitton explains that it is the memory B cell response, not any preexisting antibodies, that determines the strength of the recall response (Ex. I, p. 12). Antibodies would simply act to neutralize antigen. (Tr. 129.) Second, he observed that the two studies cited by Dr. Steinman (Kappos et al. and Bielekova et al.) involved the triggering of a T cell response. (*Id.* at 149-50.) While Dr. Whitton does not dispute that a T cell response can occur quickly, this is not informative of the time it would take for an antibody response to develop and to reach a tipping point. (*Id.*) Third, even as Dr. Steinman proposes concurrent immune responses, this hypothesis from Dr. Steinman still relies on the same mechanisms of antibody development that are otherwise at issue. (*Id.* at 138-39.) Accordingly, without more, the tipping point concept does not in itself explain how a disease-causing antibody response could occur within 24 hours. (*Id.* at 139-40.) And, finally, Dr. Whitton cited animal studies by Patrick & Lindstrom, Berman & Patrick, and Baggi et al., showing that even pushing the animal models as hard as possible, they could not get symptoms to manifest prior to 24 hours even after repeat exposures. (Ex. A, pp. 24-25 (citing Jim Patrick & Jon Lindstrom, *Autoimmune Response to Acetaminophen Receptor*, 180 SCIENCE 871 (1973) (Ex. A23); Phillip W. Berman & Jim Patrick, *Experimental Myasthenia Gravis: A Murine System*, 151 J. EXPERIMENTAL MED. 204 (1980) (Ex. A24); Fulvio Baggi et al., *Breakdown of Tolerance to a Self-Peptide of Acetylcholine Receptor α-Subunit Induces Experimental Myasthenia Gravis in Rats*, 172 J. IMMUNOLOGY 2697 (2004) (Ex. A25)); Tr. 140, 152-54.)

While the tipping point concept raised by Dr. Steinman is not disputed as a general matter, petitioner has not substantiated that this concept would explain how onset of an autoimmune condition could occur within 24 hours of a trigger. In particular, although Dr. Steinman opined that both preexisting plasmablasts and IL-6 stimulated plasmablasts would generate antibodies additional to the recall response, he has not succeeded in demonstrating that either of these processes would operate any more quickly than the recall response itself. Nor has he provided any evidence beyond his own speculation to suggest that these processes acting concurrently would accelerate the development of GBS. None of the evidence or concepts presented by Dr. Steinman overcome the fundamental understanding that even a recall response takes time to develop meaningful levels of antibodies. Moreover, even if Dr. Steinman were persuasive in asserting that the body could produce sufficient levels of autoantibodies to breach self-tolerance within 24 hours, this would still leave the question of whether that breach of self-tolerance would then go on to produce injury and symptom manifestation within that same 24-hour period. As noted above, Dr. Whitton cited animal model studies that demonstrated that, no matter how far the researchers pushed the model, they could not produce autoimmune injury within one day. (Ex. A, pp. 24-25 (citing Patrick & Lindstrom, *supra*, at Ex. A23; Berman & Patrick, *supra*, at Ex. A24; Baggi et al., *supra*, at Ex. A25); Tr. 140, 152-54)

Thus, considering all of the above, Dr. Steinman has not come forward with a preponderantly supported explanation of how petitioner's flu vaccine could have produced an autoantibody response leading to a loss of immune self-tolerance and subsequent development of GBS within 24 hours of vaccination.

ii. Epidemiology and case reports

Petitioner has also presented several case reports, along with four epidemiologic studies, which she contends provide support for the proposition that the flu vaccine can cause GBS within one day. (ECF No. 99, pp. 8-15, 33-34.) Dr. Steinman asserts that these studies indicate that there is a period of elevated risk for GBS that begins on the first day post-vaccination. (Ex. 42, p. 11; Tr. 36-64, 102-06 (discussing Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979) (Ex. 40), Yong-Shik Park et al., *Clinical Features of Post-Vaccination Guillain-Barré Syndrome (GBS) in Korea*, 32 J. KOREAN MED. SCI. 1154 (2017) (Ex. 52), Daniel A. Salmon et al., *Association Between Guillain-Barré Syndrome and Influenza A (H1N1) 2009 Monovalent Inactivated Vaccines in the USA: A Meta-Analysis*, 381 LANCER 1461 (2013) (Ex. 53), and Laura L. Polakowski et al., *Chart-Confirmed Guillain-Barré Syndrome After 2009 H1N1 Influenza Vaccination Among the Medicare Population, 2009-2010*, 178 AM. J. EPIDEMIOLOGY 962 (2013) (Ex. 92)).) Although he acknowledges these papers provide some associational evidence linking the flu vaccine to GBS generally, Dr. Whitton stresses that epidemiology can only demonstrate correlation, not causation, and that these studies are inadequate to conclude that the day-one cases observed in the studies are causally related to the vaccination. (Tr. 122-28, 176-79.)

Of course, the Federal Circuit has previously stressed that a petitioner is not obligated to prove a case with epidemiology. *Capizzano*, 440 F.3d at 1325. In that regard, petitioner argues that failing to credit this literature that recognizes a 1-42 day risk period for GBS would impermissibly elevate her burden of proof. (ECF No. 99, p. 29; ECF No. 102, p. 9.) However, contrary to what petitioner seems to suggest, the fact that petitioners are not obligated to present epidemiology does not mean that the special master cannot assess whether the epidemiology that is filed *meaningfully* supports the petitioner's contentions. *Andreu v, Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009) ("Although *Althen* and *Capizzano* make clear that a claimant need not produce medical literature or epidemiological evidence to establish causation under the Vaccine Act, where such evidence is submitted, the special master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury."); *D'Toile v. Sec'y of Health & Human Servs.*, 726 F. App'x 809, 811 (Fed. Cir. 2018) ("Nothing in *Althen* and *Capizzano* requires the Special Master to ignore probative epidemiologic evidence that undermines petitioner's theory."). I have considered that the studies cited by petitioner are not entirely lacking teachings potentially supportive of petitioner's view. *Accord Doles v. Sec'y of Health & Human Servs*., No. 2023-2404, 2025 WL 1177875 (Fed Cir. 2025). Yet, while the articles at issue do include overall risk periods extending from the date of vaccination for weeks onward as petitioner stresses, they do not ultimately add significantly to petitioner's claim with respect to her burden of demonstrating specifically that "given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation" at one day post-vaccination. *de Bazan*, 539 F.3d at 1352.

1. Schonberger et al.[10]

Schonberger et al. is a seminal study that is generally accepted as providing support for the proposition that the flu vaccine can cause GBS. (Schonberger et al., *supra*, at Ex. 40.) Following a 1976 campaign to vaccinate for a specific pandemic strain of influenza (A/New Jersey or Swine flu), a surveillance study uncovered 1,098 cases of GBS occurring during the period of the vaccination campaign, with 532 having recently received a dose of the A/New Jersey influenza vaccine prior to onset. (Schonberger et al., *supra*, at Ex. 40, p. 1.) Comparing those incidences against the non-vaccinated population, Schonberger et al. concluded that there was an excess risk of GBS of 9.5 cases per million vaccinees or just under 1 case per 100,000 vaccinations (*Id*. at 7), with the period of increased risk being "concentrated primarily within the 5-week period after vaccination" (*Id*. at 1).

As support for the proposition that this study supports a one-day onset for post-vaccination GBS, Dr. Steinman compared several figures within the study. First, Figures 6 and 7 of the study show the attributable risk of GBS following vaccination by week. (Tr. 39-41.) According to these figures, the study population included 1.03 cases of GBS per million vaccinees arising within the first week post-vaccination. Compared to the expected incidences of 0.22 cases per million persons, the 95% confidence interval demonstrated an increased risk of GBS during that first week. (Schonberger et al., *supra*, at Ex. 40, p. 9; Tr. 41.) Turning to Figure 5, data is broken down into two-day increments. (Schonberger et al., *supra*, at Ex. 40, p. 8.) That figure shows that approximately 11 cases of GBS occurred on days 0-1, with day 0 being the day of vaccination.[11] (*Id*.) Because the number of cases occurring on days 0 and 1 are higher than some of the other days included in the overall five-week period of elevated risk, and because the authors did not explicitly exclude day 1 from their conclusion, Dr. Steinman opines that this study supports a causal inference based on a one-day latency. (Tr. 44-45.) I do not find Dr. Steinman's reasoning persuasive for two reasons.

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[10] Lawrence B. Schonberger et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 AM. J. EPIDEMIOLOGY 105 (1979) (EX. 40).

[11] Dr. Steinman interprets Figure 5 as showing 11 cases of GBS occurring on days 0-1. (Tr. 41-42.) However, the y-axis showing the number of cases is marked at 4-case intervals, leaving it difficult to ascertain exactly how many cases fall in each time period. (Schonberger et al., *supra*, at Ex. 40, fig 5.) For 0-1 days post-vaccination, the corresponding bar rises to just below the 12-case mark. (*Id*.) This is the only instance in the paper where the data is broken down in this manner. While some uncertainty remains, Dr. Steinman's reliance on 11 cases of GBS arising on days 0-1 appears reasonable.

First, it is not clear that Schonberger et al. demonstrates any elevated risk of GBS on days 0-1 post-vaccination. As Dr. Whitton noted, the study authors did not draw this conclusion. (Tr. 122.) Although the paper does indicate that incidence of GBS were elevated relative to the first *full week* post-vaccination, nothing in the study details the relative risk by day or specifically concludes that the data depicted in Figure 5 (11 cases of GBS on days 0-1) represented an increased risk in itself. According to Dr. Steinman, Figures 5 and 6 can be juxtaposed to reach such a conclusion. However, these figures are not directly comparable. The examined populations are not the same. Figure 5 depicts all the cases of GBS observed throughout the available national data over 15 months, whereas Figure 6 depicts data from an approximately three-month period in 1976 from a select number of states having maximal case ascertainment. (Schonberger et al., *supra*, at Ex. 40, pp. 7-8.) Additionally, Figure 6 has calculated an incidence rate per million vaccinees, whereas Figure 5 represents an absolute number of cases of GBS. (*Id*.) Moreover, although Figure 6 shows that week 1 as a whole demonstrated elevated risk, Figure 5 shows that there were fewer cases on day 0-1 than any other two-day period during that first week. (*Id.*) Accordingly, without any actual calculation available, it should not be merely assumed that the day 0-1 cases explain the finding of elevated risk for the whole of week one. Ultimately, if there is sufficient data in the Schonberger paper to actually calculate whether the number of cases of GBS occurring zero-to-one day post-vaccination represents an elevated risk, it has not been demonstrated on this record.

Second, as Dr. Whitton suggested, epidemiologic data is limited to demonstrating correlation, rather than causation. (Tr. 123-24, 178-79.) Accordingly, the fact that some cases of GBS occurred on the first day post-vaccination does not invariably lead to the conclusion that a causal relationship exists. To the extent the study authors were focused on the "strikingly nonrandom distribution of the intervals between vaccination and the onset of GBS" (Schonberger et al., *supra*, at Ex. 40, p. 16), Dr. Whitton noted that the overall distribution revealed by the study, with some increased incidence in the first week but the highest incidences being in the second and third weeks post-vaccination, is generally consistent with his opinion that the relevant antibody response does not begin until day 3 (Tr. 181-82). Thus, for example, a follow up study by Langmuir et al., also introduced by Dr. Steinman for other reasons, reexamined the same data as Schonberger et al., and refined the analysis. (Alexander D. Langmuir et al., *An Epidemiologic and Clinical Evaluation of Guillain-Barré Syndrome Reported in Association with the Administration of Swine Influenza Vaccine*, 119 AM. J. EPIDEMIOLOGY 841 (1984) (Ex. 51).) In order to account for incubation periods, the Langmuir authors applied a lognormal curve.[12] (*Id*. at 19, 35-36.) The authors

[12] A probability distribution is "a mathematical function that assigns to each measurable event in a sample space the probability that the event will occur." *Probability distribution*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=71334 (last visited Dec. 31, 2025). A lognormal distribution is a probability distribution "of a random variable x such that y = ln x has a normal distribution." *Log-normal distribution*, DORLAND'S MEDICAL DICTIONARY ONLINE, https://www.dorlandsonline.com/dorland/definition?id=71331 (last visited Dec. 31, 2025). Lognormal distributions are often used to study medical incubation times for diseases. *Id.* Langmuir et al. determined that a lognormal distribution was necessary, in part, "to measure the influence of biologic randomness and chance on the observed incubation periods of the attributable cases," *i.e.*, cases that are

explained that "[t]he lognormal distribution explains over 99 per cent of the observed variability in the incubation periods of the attributable cases. This degree of regularity is statistically persuasive evidence of an orderly disease mechanism . . . ." (*Id*. at 36.) When the lognormal curve was fitted to the data (for extensive cases[13]), "[t]he resulting theoretical curve starts near 0 during the first 3.5-day period after vaccination" and only then "rises sharply to a peak in the last half of the second 7-day interval." (*Id*. at 19.)

While Dr. Steinman is correct that Schonberger et al. did not explicitly exclude the day 1 cases from their conclusion, they did specifically state that "[i]t is also noteworthy that the latent period for both experimental allergic neuritis and allergic encephalomyelitis is similar to the latent period between A/New Jersey influenza vaccination and GBS in the present series." (Schonberger et al., *supra*, at Ex. 40, p. 18.) This is an explicit indication by the study authors that, despite expressing the period of elevated risk simply as five weeks post-vaccination, they did believe that a relevant latency period was implicated even though they did not examine it in detail. Although the specific experimental studies cited by Schonberger et al. are not included in the record of this case, this passage is consistent with Dr. Whitton's testimony and strongly suggests the authors would agree that casual attribution relative to the earliest post-vaccination cases of GBS would necessarily be circumscribed by the otherwise expected latency period, which, for the reasons discussed above, does not favor Dr. Steinman's view. Accordingly, I am not persuaded that the Schonberger authors broadly stated conclusion meaningfully supports the notion that the day 0-1 cases of GBS observed in the study were causally related to the preceding vaccination.

2. Salmon et al.[14] and Polakowski et al.[15]

In 2009, another mass vaccination campaign against a pandemic flu strain, specifically the 2009 monovalent H1N1 influenza vaccine, led to follow up studies examining whether this vaccine produced an elevated risk of GBS similar to what was observed following the Swine flu vaccination campaign of the 1970's. Petitioner has cited two such studies: Salmon et al. (Ex. 53) and Polakoswki et al. (Ex. 92).

Salmon et al. conducted a meta-analysis of data from six adverse event monitoring systems. The resulting analysis captured about 23 million vaccinated

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presumably attributed to the vaccine. (Langmuir et al., *supra*, at Ex. 51, p. 35.) Thus, "[i]n calculating the theoretical curve, the assumption was made that the observed incidence rate represented a composite of two factors, one the average background endemic incidence plus the additional effect that could be attributed to the swine influenza vaccine." (*Id.* at 19.)

[13] "Extensive" cases included patients with "extensive motor involvement," including trunk and/or cranial muscles with respiratory impairment, as well as patients with "extensive" paresis or paralysis but without recorded respiratory impairment. (Langmuir et al., *supra*, at Ex. 51, pp. 7, 13.)

[14] Daniel A. Salmon et al., *Association Between Guillain-Barré Syndrome and Influenza A (H1N1) 2009 Monovalent Inactivated Vaccines in the USA: A Meta-Analysis*, 381 LANCER 1461 (2013) (EX. 53).

[15] Laura L. Polakowski et al., *Chart-Confirmed Guillain-Barré Syndrome After 2009 H1N1 Influenza Vaccination Among the Medicare Population, 2009-2010*, 178 AM. J. EPIDEMIOLOGY 962 (2013) (EX. 92).

subjects. (Salmon et al., *supra*, at Ex. 53, p. 1.) The authors detected what they characterized as a "modest" or "small" increased risk of GBS, representing about 1.6 excess cases of GBS per million vaccinated people. (*Id*.) The exposure window analyzed in the study was 1-42 days. (*Id*. at 4.) The paper does not include any explicit discussion of how that period was selected, but the introduction appears to indicate that it was likely derived from the above-discussed findings relative to the Swine flu vaccine. (*Id*. at 1.) Dr. Steinman points out that this study, like Schonberger et al., observed multiple cases occurring on the first and second days (specifically, two cases on day 1) (Tr. 56-58 (citing Salmon et al., *supra*, at Ex. 53, p. 5, fig. 2)) and that the study results confirm that the first week results were statistically significant (*Id.* at 60-62 (citing Salmon et al., *supra*, at Ex. 53, p. 5, tbl. 2)).

Polakowski et al. examined chart-confirmed cases of GBS following the 2009 H1N1 influenza vaccination campaign. (Polakowski et al., *supra*, at Ex. 92, p. 1.) Like Salmon et al., they examined cases arising during a six-week period following vaccination, noting this to be the period of elevated risk identified by earlier studies examining the Swine flu vaccine. (*Id*.) From a population of 3,436,452 vaccinees, they identified 95 potential incident cases of GBS. (*Id*. at 3.) The study found an elevated risk of GBS for the six-week post-vaccination period, but also noted a higher risk occurring between days 8-21. (*Id*. at 7-8.) Dr. Steinman points out that this study does document a single case of GBS occurring on day 1 and, like Schonberger et al., the authors do not exclude the earliest data from their overall risk period, which is noted to run from days 1-42. (Tr. 41-53.)

Again, however, as discussed above relative to the Schonberger study, the day-one cases identified by the Salmon and Polakowski studies are not very informative both because epidemiology is limited to demonstrating correlation and because the study authors did not include any calculations that would establish that day 1, in particular, included any excess risk of GBS. At best, Salmon et al., similar to Schonberger et al., assessed the first 6 days as a whole as demonstrating increased risk. In that regard, Dr. Whitton further stresses that it is significant that, although some level of increased risk was expressed for the broader period of 1-42 days, the Polakowski study confirmed a higher risk was detected for days 8-21. (Tr. 128.) He opines that the fact that the authors specifically narrowed the discussion of increased risk in this manner suggests that the core finding for days 8-21 should carry greater weight than the flanking periods. (*Id*. at 125.) Moreover, Dr. Whitton noted that this study has some significant limitations. First, the authors noted that they were unable to fully account for preceding infections. Second, the study only resulted in statistically significant findings when the broadest diagnostic criteria were used. (*Id*. at 127-28.)

It should also be noted that the increased risk observed by the Salmon and Polakowski studies was noted to be lower than what had been observed following the Swine flu vaccination campaign. (Salmon et al., *supra*, at Ex. 53, p. 5 (noting 1.6 excess cases of GBS per million vaccinees, whereas Schonberger et al, *supra*, at Ex. 40, p. 7, noted 9.5 per million); Polakowski et al., *supra*, at Ex. 92, p. 8 (noting increased risk was "approximately 5 times lower than that observed in the same period in 1976").)

Thus, it is notable that the number of day-one cases observed in these studies is much lower than the 11 cases Dr. Steinman cited relative to Schonberger et al. In Salmon et al., only two day-one cases were observed. (Salmon et al., *supra*, at Ex. 53, p. 5, fig. 2.) And in Polakoswki et al., only one case was observed on day 1. (Polakowski et al., *supra*, at Ex. 92, p. 6, fig. 2.) In that regard, Salmon et al. noted that there is no single accepted background rate for GBS and that use of a different background rate would alter the attributable risk calculation. (Salmon et al., *supra*, at Ex. 53, p. 7.) To that point, Dr. Whitton cited a study by Black et al. for the proposition that, at a population level, about 5 cases of GBS are expected each day. (Tr. 124-25; Ex. A, pp. 25-26 (citing Steven Black et al., *Importance of Background Rates of Disease in Assessment of Vaccine Safety During Mass Immunisation with Pandemic H1N1 Influenza Vaccines*, 374 LANCET 2115 (2009) (Ex. A26)).) This strongly suggests that one or two cases of GBS occurring on any given day could be the result of chance, greatly tempering the potential significance of these findings. Ultimately, these are not robust findings with respect to day 1.

3. Park et al.[16]

Park et al. is an examination of the demographic characteristics of GBS patients who submitted a claim for compensation to a vaccine injury compensation program in South Korea. (Park et al., *supra*, at Ex. 52.) Specifically, the authors examined 48 cases of GBS that were compensated within that program. (*Id.* at 1.) Among these cases, 97.9% developed symptoms within 3 weeks and more than half developed symptoms within 2 days. (*Id*. at 2.) Dr. Steinman suggested that this paper, despite significant limitations, provides some evidence supporting petitioner's claim in that the proportion of cases occurring between 0-2 days is "a striking preponderance." (Tr. 103.) Dr. Whitton observes, however, that it is difficult to draw any conclusions from this study due to its context. Specifically, Dr. Whitton noted that the paper represents a highly selective group in that it observes people seeking compensation for perceived vaccine injury and, moreover, we do not have information on what criteria the compensating authority uses to adjudge eligibility. (*Id.* at 125-27.) I agree. It is particularly noteworthy that the very high number of cases occurring within two days of vaccination is inconsistent with the overall distribution curve observed in the three studies discussed above, strongly suggesting that the parameters of the compensating scheme may have contributed to the distribution. *Accord Block v. Sec'y of Health & Human Servs*., No. 19-969V, 2021 WL 2182730, at *8 (Fed. Cir. Spec. Mstr. Apr. 26, 2021) (noting the Park paper is "not a particularly strong piece of evidence"); *Velasquez v. Sec'y of Health & Human Servs*., No. 19-1703V, 2024 WL 829599, at *17 (Fed. Cir. Spec. Mstr. Jan. 31, 2024) (indicating this paper reveals "only that another country's vaccine compensation program paid damages in cases of short onset" (emphasis omitted)). *But see Harris v. Sec'y of Health & Human Servs*., No. 18-944V, 2023 WL 2583393, at *33 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (crediting the Park paper for the more limited proposition that onset of GBS appeared to occur more quickly among minor claimants as compared to adult claimants).

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[16] Yong-Shik Park et al., *Clinical Features of Post-Vaccination Guillain-Barré Syndrome (GBS) in Korea*, 32 J. KOREAN MED. SCI. 1154 (2017) (Ex. 52).

4. Case reports

I have also considered the case reports cited by petitioner. (ECF No. 99, pp. 33-34 (citing A. Kakar & P. K. Sethi, *Guillain Barre Syndrome Associated with Hepatitis B Vaccination*, 64 INDIAN J. PEDIATRICS 710 (1997) (Ex. 97); L Bennetto & N Scolding, *Inflammatory/Post-Infectious Encephalomyelitis*, 75 J. NEUROLOGY NEUROSURGERY PSYCHIATRY i22 (2004) (Ex. 112)); *see also* Charles M. Poser, *Neurologic Syndromes That Arise Unpredictably*, CONSULTANT, Jan. 1987 (Ex. 93); Charles M. Poser, *Neurological Complications of Swine Influenza Vaccination*, 66 ACTA NEUROLOGICA SCANDINAVICA 413 (1982) (Ex. 94).) However, although case reports are not entirely devoid of evidentiary value, they are not strong evidence with respect to causation. *Paluck v. Sec'y of Health & Human Servs.*, 104 Fed. Cl. 457, 475 (2012) (noting that "case reports 'do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value,'" but "the fact that case reports can by their nature only present indicia of causation does not deprive them of all evidentiary value" (quoting *Campbell v. Sec'y of Health & Human Servs.*, 97 Fed. Cl. 650, 668 (2011))); *Crutchfield v. Sec'y of Health & Human Servs.*, No. 09-0039V, 2014 WL 1665227, at *19 (Fed. Cl. Spec. Mstr. Apr. 7, 2014) (noting that a "single case report of Disease X occurring after Factor Y . . . do not offer strong evidence that the temporal relationship is a causal one – the temporal relationship could be pure random chance" (emphasis omitted)), *mot. for rev. denied*, 125 Fed. Cl. 251 (2014). Here, even setting aside the inherent limitations of case reports, the reports cited by petitioner are not on point. They mostly do not address GBS and the single case report that did demonstrate GBS occurring within 24 hours of vaccination (Kakar & Sethi), involved a different vaccine administered to a 3-year-old child. (Kakar & Sethi, *supra*, at Ex. 97.)

iii. <u>Prior program experience</u>

There have been several prior cases that have addressed whether onset of GBS or other demyelinating conditions within one day of vaccination can support a causal inference. These cases have come to mixed results. In their briefing, each party highlights those prior cases that support that party's view and seek to distinguish the others. (ECF No. 99, p. 19; ECF No. 100, pp. 25-26.) Petitioner cites: *E.M. v. Sec'y of Health & Human Servs*., No. 14-753V, 2021 WL 3477837 (Fed. Cl. Spec. Mstr. July 9, 2021) (finding entitlement to compensation where onset of small fiber neuropathy occurred four to six hours post-vaccination); *Lehrman v. Sec'y of Health & Human Servs.*, No. 13-901, 2018 WL 1788477 (Fed. Cl. Spec. Mstr. Mar. 19, 2018) (finding that a flu vaccine acted synergistically with an upper respiratory infection to cause GBS within 24 hours of vaccination); *Quackenbush-Baker v. Sec'y of Health & Human Servs*., No. 14-1000V, 2018 WL 1704523 (Fed. Cl. Spec. Mstr. Mar. 14, 2018) (accepting that the flu vaccine can cause a significant aggravation of multiple sclerosis within 40 hours of vaccination); *Harris v. Sec'y of Health & Human Servs*., No. 18-944V, 2023 WL 2583393 (Fed. Cl. Spec. Mstr. Feb. 21, 2023) (finding that a repeat Tdap vaccine can cause GBS within one day of vaccination). Respondent cites: *Velasquez v. Sec'y of Health & Human Servs*, No. 19-1703V, 2024 WL 829599 (Fed. Cl. Spec.

Mstr. Jan. 31, 2024) (finding *Althen* prong three was not met where onset of GBS occurred less than three days post-vaccination); *Block v. Sec'y of Health & Human Servs*., No. 19-969V, 2021 WL 5709764 (Fed. Cl. Spec. Mstr. Oct. 29, 2021) (finding *Althen* prong three not met where onset of GBS occurred approximately 24 hours post-vaccination); *Rowan v. Sec'y of Health & Human Servs*., No. 17-760V, 2020 WL 2954954 (Fed. Cl. Spec. Mstr. Apr. 28, 2020) (finding *Althen* prong three not met where onset of GBS occurred 30-36 hours post vaccination); *Dennington v. Sec'y of Health & Human Servs*., No. 18-1303V, 2023 WL 2965239 (Fed. Cl. Spec. Mstr. Mar. 23, 2023) (denying compensation where petitioner suffered GBS 48 hours after receiving a Tdap vaccine).

I have considered all of the cases cited by the parties. On the whole, the cases cited by respondent are closer to being on point. Ultimately, however, none of these prior decisions or rulings are binding. *Boatmon v. Sec'y of Health & Human Servs*., 941 F.3d 1351, 1358 (Fed. Cir. 2019); *Hanlon v. Sec'y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998), *aff'd*, 191 F.3d 1344 (Fed. Cir. 1999). Of particular note is the parties' debate regarding the significance of the undersigned's own prior ruling in *Harris, supra*, in which a minor petitioner was found entitled to compensation for GBS that arose about 24 hours following a Tdap vaccination. (ECF No. 99, p. 19; ECF No. 76, pp. 46-47; ECF No. 100, pp. 25-26.) As respondent suggested, the *Harris* case is distinguishable from this case for a number of reasons: it involved a different vaccine administered to a minor child and was based on expert opinion proffered by the petitioner that is not comparable to this case. But in any event, respondent's experts in *Harris* were unpersuasive because they limited their opinions merely to stressing that a rapid onset would be atypical. 2023 WL 2583393, at *34 (explaining that "Dr. Platt does explicitly state in his first report that in his opinion a latency of one to two days is 'implausible.' However, the basis for that assertion is not fully explained. Dr. Platt never explicitly describes any medically reasonable timeframe to infer causation beyond the one-to-four week period he repeatedly cites merely as 'typical.'" (internal citation omitted).) In this case, by contrast, Dr. Whitton's opinion as to why a 24-hour onset is implausible under Dr. Steinman's theory is thoroughly explained and persuasive. Notably, and in contrast to *Harris*, the undersigned has issued two other decisions denying compensation where petitioners asserted that molecular mimicry can explain a neurologic condition arising within 24 hours of vaccination. *Greenslade v. Sec'y of Health & Human Servs*., No. 14-1140V, 2024 WL 3527665 (Fed. Cl. Spec. Mstr. June 28, 2024) (denying compensation for a significant aggravation of transverse myelitis occurring 24 hours after flu vaccination); *McGill v. Sec'y of Health & Human Servs*., No. 15-1485V, 2023 WL 3813524 (Fed. Cl. Spec. Mstr. May 11, 2023) (denying compensation for small fiber neuropathy arising 8-9 hours post-vaccination).

V. Conclusion

Regardless of the cause of Ms. Chervenok's condition, I am mindful of the pain and suffering she has endured, and I extend my condolences to petitioner for Ms. Chervenok's subsequent passing. Moreover, I appreciate that, at first blush, the onset of GBS occurring the day after a flu vaccine offers a striking coincidence. However, for all the reasons discussed above, the onset of Ms. Chervenok's GBS occurred *too soon* after vaccination to be causally related. As discussed above, after weighing the competing expert opinions, respondent is persuasive in contending that it is biologically implausible for GBS to develop in such a short period of time. Thus, a causal inference implicating Ms. Chervenok's vaccine as the cause of her condition is not supported.

Therefore, pursuant to § 300aa-12(d)(3)(A) and Vaccine Rule 10, this decision concludes that petitioner is not entitled to an award of compensation. Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).

IT IS SO ORDERED.

s/Daniel T. Horner
Daniel T. Horner
Special Master