In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: July 19, 2022

RONALD STURDEVANT,	*	PUBLISHED
	*	
Petitioner,	*	No. 17-172V
	*	
v.	*	Special Master Nora Beth Dorsey
	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Entitlement; Motion to Strike Testimony; Influenza ("Flu") Vaccine; Bell's Palsy.
	*	
Respondent.	*	
	*	

William Cochran, Black McLaren Jones Ryland & Griffee, PC, Memphis TN, for petitioner.
Zoe Wade, U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

I. INTRODUCTION

On February 6, 2017, Ronald Sturdevant ("petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2012).[2] Petitioner alleges that he suffered Bell's palsy as the result of an influenza ("flu") vaccination administered on November 3, 2015. Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating that "this case is not appropriate for

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling 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, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2012). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 2 (ECF No. 16).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards, the undersigned finds that petitioner has provided preponderant evidence that his flu vaccine caused his Bell's palsy, satisfying petitioner's burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, petitioner is entitled to compensation.

II. ISSUES TO BE DECIDED

Diagnosis is not at issue. Joint Status Rept., filed June 29, 2021, at 1 (ECF No. 68). The parties stipulated that petitioner received a flu vaccine on November 3, 2015, and that onset of his right-sided Bell's palsy was November 4, 2015. Id.

The central issue is whether petitioner has provided preponderant evidence of causation for all three Althen prongs. Petitioner asserted that he has met his burden under the Althen prongs. Petitioner's Post-Hearing Brief ("Pet. Post-Hearing Br."), filed Oct. 25, 2021, at 6-15 (ECF No. 79); Pet. Post-Hearing Reply Br. ("Pet. Post-Hearing Reply"), filed Feb. 7, 2022, at 1-6 (ECF No. 85). Respondent disagreed and argued that petitioner failed to submit preponderant evidence (1) "of a reliable medical theory causally connecting the vaccination and the injury," (2) "of a logical sequence of cause and effect connecting the vaccination and the injury," and (3) "showing a medically reasonable timeframe from which to infer causation." Resp. Posthearing Br. on Entitlement ("Resp. Posthearing Br."), filed Jan. 24, 2022, at 1-21 (ECF No. 84). Respondent also contended that petitioner's Bell's palsy was more likely than not caused by a factor unrelated to his vaccination—a herpes viral infection. Id. at 21-23.

The second issue to be resolved relates to respondent's expert, Dr. Vinay Chaudhry's hearing testimony. During the entitlement hearing, and in a subsequent Motion to Strike, petitioner moved to strike Dr. Chaudhry's hearing testimony regarding alternative causes of petitioner's Bell's palsy. Pet. Motion to Strike ("Pet. Mot."), filed Oct. 25, 2021 (ECF No. 80).

III. BACKGROUND

A. Medical Terminology

Bell's palsy is defined as "unilateral facial paralysis of sudden onset, due to [a] lesion of the facial nerve[,] [] resulting in characteristic distortion of the face." Bell Palsy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=95779 (last visited June 2, 2022). "[P]atients with Bell's palsy may experience dryness of the eye or mouth, taste disturbance or loss, hyperacusis,[3] and sagging of the eyelid or corner of the mouth." Pet.

[3] Hyperacusis is "exceptionally acute hearing, the hearing threshold being unusually low. It may or may not be accompanied by pain." Hyperacusis, Dorland's Online Med. Dictionary, https://www.dorlandsonline.com/dorland/definition?id=23650 (last visited June 2, 2022).

Ex. 8 at 2.[4] "Bell's palsy is a diagnosis of exclusion requiring the careful elimination of other causes of facial paresis or paralysis." Id.

Although Bell's palsy is a well-known and common disease, its etiology remains unclear. Pet. Ex. 21 at 1;[5] Pet. Ex. 36 at 1.[6] There are several known risk factors for Bell's palsy, including obesity, hypertension, diabetes, and more. Pet. Ex. 8 at 2-3; Pet. Ex. 15 at 1;[7] Resp. Ex. E at 2;[8] Resp. Ex. Q at 5.[9] "[G]enetic, vascular, infective[,] and immunological causes have all been postulated." Pet. Ex. 12 at 1.[10] It is believed that "herpes simplex virus [("HSV")] activation is the likely cause of Bell's palsy in most cases." Resp. Ex. E at 2; see also Resp. Ex. Q at 7; Pet. Ex. 15 at 8. "Facial paresis or paralysis is thought to result from facial nerve inflammation and edema. As the facial nerve travels in a narrow canal within the temporal bone, swelling may lead to nerve compression and result in temporary or permanent nerve damage." Pet. Ex. 8 at 2; see also Resp. Ex. Q at 6-7 (noting "[e]dema of the facial nerve within the narrow fallopian canal has been observed," and "[t]he cause of the edema may be ischemia in predisposed patients").

Reich detailed the anatomy of the facial canal,[11] in pertinent part, as follows:

> The facial nerve travels with the vestibulocochlear nerve in the internal auditory meatus before entering the facial canal (fallopian canal), a narrow bony canal within the temporal bone. It is because of its course through this narrow

[4] Reginald F. Baugh et al., Clinical Practice Guideline: Bell's Palsy Executive Summary, 149 Otolaryngology & Neck Surgery 656 (2013).

[5] Weigong Zhou et al., A Potential Signal of Bell's Palsy After Parenteral Inactivated Influenza Vaccines: Reports to the Vaccine Adverse Event Reporting System (VAERS) United States, 1991-2001, 13 Pharmacoepidemiology & Drug Safety 505 (2004).

[6] Cheng-Hsiu Chou et al., Bell's Palsy Associated with Influenza Vaccination: Two Case Reports, 25 Vaccine 2839 (2007).

[7] Donald H. Gilden, Bell's Palsy, 351 New Eng. J. Med. 1323 (2004).

[8] Michael Ronthal, Bell's Palsy: Pathogenesis, Clinical Features, and Diagnosis in Adults, UpToDate, https://www.uptodate.com/contents/bells-palsy-pathogenesis-clinical-features-and-diagnosis-in-adults (last updated Feb. 10, 2016).

[9] Stephen G. Reich, Bell's Palsy, 23 Continuum 447 (2017).

[10] S. Rowlands et al., The Epidemiology and Treatment of Bell's Palsy in the UK, 9 Eur. J. Neurology 63 (2002).

[11] For an illustration of the lymph nodes and glands of the head and neck, see Pet. Ex. 56 at 4, 7 (Hannah M. Chason & Brian W. Downs, Anatomy, Head and Neck, Parotid Gland, StatPearls, https://www.ncbi.nlm.nih.gov/books/NBK534225/ (last updated June 17, 2021)).

> canal, with little room for expansion, that inflammation of the nerve (due to any cause) is thought to cause compression resulting in paralysis . . . .
>
> The chorda tympani is the final branch of cranial nerve VII [facial nerve] before it exits the skull at the stylomastoid foramen. . . . From the stylomastoid foramen, the facial nerve courses through the parotid gland before dividing into branches that innervate all of the muscles of facial expression as well as the buccinator.

Resp. Ex. Q at 4-5.

B. Procedural History

Petitioner filed his petition on February 6, 2017, and filed medical records on February 9, 2017. Petition; Pet. Exhibits ("Exs.") 1-5. On August 24, 2017, petitioner filed an expert report from Dr. M. Eric Gershwin. Pet. Ex. 6. On September 7, 2017, petitioner filed additional medical records and respondent filed his Rule 4(c) Report, in which he recommended against compensation. Pet. Ex. 26; Resp. Rept. at 2.

On February 16, 2018, respondent filed an expert report from Dr. Chaudhry. Resp. Ex. A. Petitioner filed a supplemental expert report from Dr. Gershwin on April 4, 2018. Pet. Ex. 27. On June 1, 2018, respondent filed a supplemental expert report from Dr. Chaudhry, and petitioner filed a responsive expert report from Dr. Gershwin on July 26, 2018. Resp. Ex. R; Pet. Ex. 30. On October 29, 2018, respondent filed an expert report from Dr. Neil Romberg. Resp. Ex. S. In April 2019, petitioner filed a responsive expert report from Dr. Gershwin and medical records. Pet. Exs. 37-40. On July 21, 2019, respondent filed a supplemental report from Dr. Romberg. Resp. Ex. LL. An entitlement hearing was set for July 2021. Prehearing Order dated June 10, 2019 (ECF No. 39).

This case was reassigned to the undersigned on January 21, 2020. Notice of Reassignment dated Jan. 21, 2020 (ECF No. 46). The undersigned held a Rule 5 Conference on February 5, 2020. Order dated Feb. 5, 2020 (ECF No. 47). Based on the undersigned's preliminary review of the case, she made a preliminary finding that petitioner had provided preponderant evidence of causation. Id. at 2. Thereafter, petitioner filed photographs and updated medical records, and the parties filed supplemental medical literature. Pet. Exs. 41-55; Resp. Exs. MM-OO. In September 2020, respondent filed a status report stating that he "does not intend to pursue a 'factors unrelated' [to vaccination] theory" at the entitlement hearing. Resp. Status Rept., filed Sept. 25, 2020 (ECF No. 54).

An entitlement hearing was held on July 13 and July 14, 2021. Order dated July 14, 2021 (ECF No. 72). Dr. Gershwin, Dr. Chaudhry, and Dr. Romberg testified. Transcript ("Tr.") 3, 187. After the hearing, both parties filed additional evidence. Pet. Exs. 56-57; Resp. Ex. PP.

On October 25, 2021, petitioner filed a post-hearing brief along with a motion to strike Dr. Chaudhry's hearing testimony. Pet. Post-Hearing Br.; Pet. Mot. Respondent filed his response to petitioner's motion to strike on November 4, 2021 and his post-hearing brief on

January 24, 2022. Resp. Response to Pet. Mot. ("Resp. Response"), filed Nov. 4, 2021 (ECF No. 81); Resp. Posthearing Br. On February 7, 2022, petitioner filed his reply post-hearing brief and reply to respondent's response to petitioner's motion to strike. Pet. Post-Hearing Reply; Pet. Reply to Resp. Response ("Pet. Reply"), filed Feb. 7, 2022 (ECF No. 86).

This matter is now ripe for adjudication.

C. Factual History

1. Medical History

Prior to the vaccination at issue, petitioner had a prior medical history significant for obesity, diabetes mellitus (type 2), hypertension, osteoarthritis, allergic rhinitis, and sleep apnea. Pet. Ex. 3 at 2, 5, 11; Pet. Ex. 5 at 1. There is no indication that petitioner had ever had Bell's palsy prior to vaccination. There is also no suggestion that petitioner had ever had a herpes virus infection prior to the vaccination at issue.

On November 3, 2015, at fifty-one years old, petitioner received a flu vaccine in his left arm. Pet. Ex. 2 at 2; Pet. Ex. 40 at 1-2.

Two days later, on November 5, 2015, petitioner presented to his primary care physician, Dr. Christopher R. Depner for right-sided facial numbness that "started yesterday afternoon." Pet. Ex. 3 at 18. Petitioner also reported he received a flu vaccine on Tuesday.[12] Id. Bell's palsy was added to petitioner's problem list. Id. Dr. Depner's physical examination revealed normal neck with no masses, normal thyroid, "[n]o visible regional lymphadenopathy," and paresis of petitioner's right seventh cranial nerve.[13] Id. at 19-20. Assessment was Bell's palsy. Id. at 20. Dr. Depner commented, "[p]atient with fairly classic Bell's palsy with paresis in the distribution of the right [seventh] nerve and also involving the forehead to some degree. He's unable to close his left[14] eyelid fully."[15] Id. Dr. Depner planned to patch petitioner's eye, start

[12] Tuesday was November 3, 2015.

[13] The seventh cranial nerve is another name for the facial nerve. Pet. Ex. 15 at 5 fig.4; Nervus Facialis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92293 (last visited June 2, 2022).

[14] It appears Dr. Depner inadvertently referred to petitioner's left eyelid, instead of his right.

[15] Dr. Depner added petitioner was "able to close his right eye adequately, so [it was] no longer drying out and bothering him. He still ha[d] rather dense right facial nerve paresis." Pet. Ex. 3 at 20. It is not clear whether this is from a follow up visit, and if so, which visit.

petitioner on prednisone[16] and Famvir,[17] and have him follow up in one week. Id. Dr. Depner did not document the presence of any rash or vesicles, or otherwise suggest or diagnose petitioner with a herpes virus.

Petitioner followed up with Dr. Depner on November 10, 2015. Pet. Ex. 3 at 21. Petitioner continued to have paresis of his right seventh cranial nerve. Id. at 23.

At a follow up examination on November 23, 2015, petitioner reported he was doing well but had some eye tearing and fuzzy distance vision. Pet. Ex. 3 at 24. Dr. Depner's physical examination revealed paresis of right seventh nerve, paralyzed right facial muscles, unable to raise right eyebrow, and weakness closing right eyelid. Id. at 25-26. Assessment remained Bell's palsy. Id. at 26. Dr. Depner found petitioner had not "made much improvement with his facial paralysis," and ordered him to attend physical therapy. Id. Again, Dr. Depner did not note any rash or vesicles or diagnose a herpes virus.

Petitioner had his initial physical therapy evaluation on December 9, 2015 with Shanna Winters. Pet. Ex. 4 at 44. Petitioner presented for right-sided Bell's palsy. Id. He "report[ed] that he had a flu shot on 11/4,[18] and Bell's palsy symptoms began on 11/5. Symptoms came on quickly and have improved since. Went to Dr. Depner and began steroids. [Petitioner] report[ed] that pain lasted for a month but has resolved." Id. He reported "trouble with hand eye coordination, blurry vision, drinking from a cup or straw," and "increased light sensitivity secondary to not being able to close his eye." Id. Ms. Winters' physical examination revealed petitioner had a drooped eyebrow. Id. at 45. Petitioner was unable to show his teeth, close his eyes, blink, squint, or inflate cheeks. Id. He was able to drink from a straw with difficulty and was unable to drink from a cup. Id.

On December 14, 2015, petitioner returned to Dr. Depner for follow up. Pet. Ex. 3 at 27. Physical examination was unchanged. Id. at 28-29. Assessment remained Bell's palsy. Id. at 31. Dr. Depner wrote "[petitioner] still ha[d] a rather dense paralysis in the right face . . . . He [was] able to close his eye to within about 90% of full closure." Id. Dr. Depner ordered bloodwork, including a Lyme disease titer. Id. Petitioner's blood work showed low lymphocytes, but was otherwise normal. Id. at 29-30. He tested negative for Lyme disease. Id. at 30.

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[16] Prednisone is "a synthetic glucocorticoid derived from cortisone, administered orally as an antiinflammatory and immunosuppressant in a wide variety of disorders." Prednisone, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=40742 (last visited June 2, 2022).

[17] Famvir, or famciclovir, is "used in the treatment of herpes zoster and . . . of mucocutaneous herpes simplex in immunocompromised patients." Famciclovir, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=18166 (last visited June 2, 2022).

[18] Petitioner's correct date of vaccination is November 3, 2015. Pet. Ex. 2 at 2.

Petitioner returned to Dr. Depner next on May 25, 2016. Pet. Ex. 3 at 32. Petitioner's Bell's palsy had not resolved completely. Id. Dr. Depner noted petitioner "still ha[d] right facial weakness, but it ha[d] improved. He [was] able to blink and he [was] able to drink liquids and drink with a straw." Id. at 34. Petitioner reported he was six months out of physical therapy. Id. Dr. Depner found petitioner was "near the point of maximum medical improvement." Id. Assessment remained Bell's palsy. Id.

On July 4, 2016, petitioner presented to the Jones Memorial Hospital Emergency Department for a laceration on the left side of his face from an air tool. Pet. Ex. 4 at 5. Petitioner's past medical history included Bell's palsy. Id. at 12. On examination, no mention of facial muscle weakness was noted. Id. at 13.

In 2017, petitioner presented to Dr. Depner on numerous occasions for unrelated issues. Pet. Ex. 26 at 1-20. In each of the physical examinations from January to April 2017, petitioner's cranial nerves were grossly intact. Id. at 2, 6, 12, 15. On July 11, 2017, Dr. Depner's physical examination revealed right facial paralysis. Id. at 19. Dr. Depner noted "[petitioner's] Bell's palsy ha[d] improved quite slowly, but [was] still quite marked. He [was] able to blink. He [could] close his right eye, but not tightly. His cornea [was] not drying out." Id. Dr. Depner told petitioner he likely will not improve further. Id. Dr. Depner added petitioner "developed this about a week after he received the flu vaccine. It is conceivable that the flu vaccine precipitated this. [Petitioner] has a [lawsuit] and []is hoping to get recompense[d] through the vaccine compensation program." Id.

Petitioner visited Dr. Depner various times from 2018 to 2020 for annual examinations or unrelated issues. Pet. Ex. 49 at 3-49. No complaints or comments regarding his Bell's palsy were documented at these visits. See id. At no time did Dr. Depner's diagnose petitioner with a herpes rash or with herpes, or document that herpes was the etiology of petitioner's Bell's palsy.

2. Petitioner's Affidavit

Petitioner averred that prior to his flu vaccination on November 3, 2015, he had never suffered from Bell's palsy. Pet. Ex. 1 at ¶¶ 3-4. The day after vaccination, "[he] began to have pain, weakness[,] and paralysis in the right side of [his] face. Id. at ¶ 5. He "was unable to close his right eye," his "eyesight became blurry," and he "was unable to drink from a cup." Id.

He sought treatment. Pet. Ex. 1 at ¶ 6. He also underwent physical therapy and was given at-home exercises. Id. As of the date of his affidavit, January 30, 2017, he "continue[d] to have right-sided numbness and loss of sensation, particularly around the right side of [his] mouth." Id. at ¶ 7. Additionally, "[his] right eye water[ed] more . . . , [he] continue[d] to blink abnormally, and [was] unable to raise [his] right eyebrow." Id.

D. Expert Reports

1. Petitioner's Expert, Dr. M. Eric Gershwin

a. Background and Qualifications

Dr. M. Eric Gershwin is board certified in internal medicine, rheumatology, and allergy and clinical immunology. Pet. Ex. 7 at 2. He received his M.D. from Stanford University in 1971. Id. at 1. Thereafter, he completed an internship and residency at Tufts-New England Medical Center in Boston, Massachusetts and worked as a clinical associate in immunology at National Institutes of Health in Bethesda, Maryland. Id. at 2. Dr. Gershwin has been a professor at University of California School of Medicine in Davis, California since 1975 and the Chief of the Division of Rheumatology/Allergy and Clinical Immunology at University of California School of Medicine in Davis, California since 1982. Id. at 1-2. He has served as an editor and reviewer on various editorial boards. Id. at 5-7. Dr. Gershwin has also authored or co-authored over 1,000 publications during his career. Id. at 8-125.

b. Opinion

i. Althen Prong One

Dr. Gershwin opined the flu vaccine can cause Bell's palsy through an innate immune response. Pet. Ex. 6 at 2-3; Pet. Ex. 27 at 1; Tr. 14. He explained that the immune system is divided into innate immunity, or "first responder cells," and adaptive immunity, or "delayed responder cells." Tr. 16. Innate immunity "begins almost immediately following vaccination, and adaptive immunity takes days to weeks." Id. Further, "every adaptive response requires innate immune recognition." Pet. Ex. 30 at 1; see Pet. Ex. 31 at 5 (explaining how "the innate and adaptive immune responses [are] integrated . . . as a single immune system, with the innate response preceding, and being necessary for, the adaptive immune response").[19]

For a vaccine to be successful, cytokines are elicited and produce inflammation through signaling and activating cells. Tr. 18-19. Lymphocytes, or mononuclear cells, can be found in lymph nodes. Tr. 17-18. Once activated, they travel within regional lymph nodes. Tr. 14. Macrophages are another component of innate immunity that are active within lymph nodes and migrate within tissues. Tr. 19; see also Pet. Ex. 37 at 1. Dr. Gershwin explained that following vaccination, the vaccine is "processed [] by regional cells, [] macrophages, [] dendritic cells, [and] other mononuclear cells that [are] rapidly transported to regional lymph nodes." Tr. 20. Cytokines are also released and travel through the blood and through the lymphatic system to distant organs and other lymph nodes. Id. Cytokine production is systemic, going all over the body. Tr. 26.

[19] Ruslan Medzhitov & Charles A. Janeway, Innate Immunity: Impact on the Adaptive Immune Response, 9 Current Op. Immunology 4 (1997).

Dr. Gershwin cited Hervé et al.[20] and Chatziandreou et al.[21] for support of this process. Pet. Ex. 27 at 1 (citing Pet. Ex. 28); Tr. 22-28 (citing Pet. Exs. 28, 55). Hervé et al. explained that vaccination "leads to the stimulation of local cells, followed by the recruitment of blood immune cells to the local site and the production of different soluble factors including vasodilators and cytokines, which may trigger the development of signs and symptoms of local inflammation." Pet. Ex. 55 at 3 fig.1. Those factors, like cytokines, travel into the bloodstream and "may contribute to the development of general symptoms (fever, myalgia, headache etc) in the vaccinee." Id. "Resident immune cells, mast cells, monocytes[,] and macrophages are activated within minutes of injection and release soluble factors" such as proinflammatory cytokines. Id. at 4 fig.2. "These newly recruited immune cells, mainly composed of blood-born neutrophils, monocytes[,] and T lymphocytes, also contribute to pain sensation by releasing soluble factors, such as cytokines, . . . that can directly interact with local sensory receptors." Id. Once cytokines are produced, they "act both locally . . . and may act systemically at distant organs." Id. Additionally, Hervé et al. noted cytokines in mouse muscle were detected as early as three hours after injection with an adjuvant-containing-vaccine. Id. at 2. And although the vaccine at issue here did not contain an adjuvant, Dr. Gershwin explained that "an adjuvanted vaccine would [] slow things down," and thus, "[a] soluble vaccination would be at least as fast if not faster." Tr. 27.

Dr. Gershwin cited Chatziandreou et al. to demonstrate how within hours of vaccination, there is "immediate activation of macrophages and other cells" in the "regional lymph nodes of the shoulder." Tr. 53. Chatziandreou et al. "examine[d] the role of lymph node macrophages (LNMs) in the induction of the cytokine storm triggered by inactivated [flu] virus vaccine." Pet. Ex. 28 at 2. The authors "demonstrate[d] that it is the lymph node macrophages that rapidly initiate an inflammatory response and they detected a rapid and significant secretion of the inflammatory mediators, IL1-α and IFNβ, within 90 minutes." Pet. Ex. 27 at 1.

Dr. Gershwin opined the mechanisms involved in Bell's palsy include a "highly focal inflammatory response." Pet. Ex. 6 at 2. "[T]here is [] an inflammatory response and . . . Bell's [p]alsy most often occurs in the absence of significant systemic features." Id. With Bell's palsy, there is "inflammation of the facial nerve with subsequent compression and permanent damage to the nerve." Id.; see also Pet. Ex. 37 at 1. "[T]he nerve is surrounded by small, round inflammatory cells from the internal acoustic meatus to the stylomastoid foramen. . . . There is an increased space between the neurons, which is consistent with edema." Pet. Ex. 37 at 1.

To summarize his theory, Dr. Gershwin testified that "[f]ollowing vaccination, there would have been the expected innate immune response which would [] occur[] within hours. It would involve activation and trafficking of mononuclear cells," that "would traffic throughout the body." Tr. 42. The trafficking cells would move to the lymph nodes in the facial area,

[20] Caroline Hervé et al., The How's and What's of Vaccine Reactogenicity, 9 NPJ Vaccines 1 (2019).

[21] Nikolaos Chatziandreou et al., Macrophage Death Following Influenza Vaccination Initiates the Inflammatory Response that Promotes Dendritic Cell Function in the Draining Lymph Nodes, 18 Cell Reps. 2427 (2017).

particularly in the parotid gland[22] and adjacent regions, where the facial nerve is located. Tr. 42, 249. "Those trafficking cells within the tissue surrounding the nerve[] . . . would produce further obstruction in someone that likely already had a degree of obstruction producing ischemia and . . . paralysis of the facial nerve." Tr. 42-43.

During the hearing, Dr. Gershwin referenced Ronthal, an exhibit cited by respondent, to further support his opinion that an inflammatory response is the causal mechanism implicated with Bell's palsy. Tr. 30-32. Ronthal explained "the facial nerve has a thickened, edematous perineurium with a diffuse infiltrate of small, round, inflammatory cells between nerve bundles and around intraneural blood vessels." Resp. Ex. E at 2. According to Dr. Gershwin, "those inflammatory cells came from local regional lymph nodes." Tr. 32. "[T]he lymphatic system is not a stationary system. It is mobile, much like the blood." Pet. Ex. 30 at 1. Thus, vaccination can cause lymph node swelling in remote locations. Tr. 24-25 (citing Pet. Ex. 51 at 8 (listing the side effect of "swollen, painful, or tender lymph glands in the neck, armpit, or groin" for the diphtheria, tetanus, and acellular pertussis booster vaccination));[23] see also Pet. Ex. 30 at 1.

Dr. Gershwin acknowledged that viral infections have been suspected to be involved in the development of Bell's palsy. Pet. Ex. 6 at 2; see, e.g., Pet. Ex. 8 at 2 ("While a viral etiology is suspected, the exact mechanism of Bell's palsy is currently unknown."); Pet. Ex. 12 at 1 ("The aetiology of Bell's palsy remains unclear although genetic, vascular, infective[,] and immunological causes have all been postulated."); Pet. Ex. 15 at 8 ("HSV type 1 (HSV-1) is probably the cause of most cases of Bell's palsy."). But see Pet. Ex. 12 at 4 (examining 2,473 cases of Bell's palsy from 1992 to 1996 in the United Kingdom and finding "no suggestion that [Bell's palsy] is triggered [] by episodes of herpes simplex infection").[24] He opined, however, that the mechanism thought to be at play here is similar to what is thought to occur in cases of Bell's palsy that are caused by a viral infection such as herpes. Pet. Ex. 6 at 2; Tr. 14.

[22] Dr. Gershwin explained the parotid glands are salivary glands located "below the neck, . . . and can extend up the lymphatics, almost below the ears. And lymphatics around them will drain throughout the facial area." Tr. 46-47. "[T]he parotid gland has two sets of lymph nodes. They drain both into the superficial but also the deep cervical lymph node chain, and they're intimately involved in the drainage from the ear, from the eyelid, and from other parts of the face as well." Tr. 249.

[23] Diphtheria, Tetanus, and Acellular Pertussis Booster Vaccine (Intramuscular), Drugs.com, https://www.drugs.com/cons/diphtheria-tetanus-and-acellular-pertussis-booster-vaccine-intramuscular.html (last updated Dec. 19, 2020).

[24] The authors found only 19 of the 2,473 cases had herpes labialis (cold sores) in the 90 days prior to onset of Bell's palsy, and 17 cases in the 90 days following onset. Pet. Ex. 12 at 3. Three of the 2,473 cases recorded herpes labialis both before and after onset of Bell's palsy. Id. They concluded there was "no evidence of any tendency for herpes simplex infections to precede Bell's palsy." Id. at 1.

Dr. Gershwin also cited articles that discussed vaccinations, including the flu vaccine, as an etiology of Bell's palsy. Pet. Ex. 6 at 2; see, e.g., Pet. Ex. 18 at 2-3;[25] Pet. Ex. 19 at 1;[26] Pet. Ex. 20 at 1;[27] Pet. Ex. 21 at 1.

Four of the studies cited by Dr. Gershwin acknowledged finding a signal or increased risk of Bell's palsy after administration of the flu vaccination. First, the authors in Zhou et al. reviewed and analyzed reports in the Vaccine Adverse Event Reporting System ("VAERS")[28] between 1991 and 2001 to determine whether there was an association between Bell's palsy and the flu vaccine.[29] Pet. Ex. 21 at 1-2. The authors identified 197 possible cases of Bell's palsy after receipt of a flu vaccine. Id. Of the 197 reports, Bell's palsy diagnosis was verified in 154, and among those, 145 cases received the flu vaccine alone. Id. The authors concluded there "may be a signal of possible association between [flu] vaccines and an increased risk of Bell's palsy." Id. at 5. They noted the etiology and pathogenesis of Bell's palsy is not clear, but that

[25] Barbara Roth et al., "All That Palsies Is Not Bell's"–The Need to Define Bell's Palsy As an Adverse Event Following Immunization, 26 Vaccine 1 (2007).

[26] Ali Rowhani-Rahbar et al., Immunization and Bell's Palsy in Children: A Case-Centered Analysis, 175 Am. J. Epidemiology 878 (2012). This article examined the association between Bell's palsy and vaccines in children, not adults. Pet. Ex. 19 at 1-2. Of the 822 children in the study, 233 received at least one vaccine in the 12 months prior to onset. Id. at 4. The authors found no association between vaccination (flu, hepatitis B, or any vaccine) and Bell's palsy during their risk intervals of 1-14 days, 1-28 days, and 29-56 days. Id.

[27] Margot Mutsch et al., Use of the Inactivated Intranasal Influenza Vaccine and the Risk of Bell's Palsy in Switzerland, 350 New Eng. J. Med. 896 (2004). This article reported an increased risk of Bell's palsy associated with the inactivated intranasal flu vaccine administered in Switzerland from October 2000 to April 2001 after 46 sentinel cases were reported. Pet. Ex. 20 at 1-2. Of the 412 patients, the authors identified 91 patients who developed Bell's palsy following the intranasal flu vaccine and found "[t]he risk was highest during the second month after intranasal vaccination." Id. at 5-6. The authors concluded "the intranasal [flu] vaccine used in Switzerland during the 2000–2001 [flu] season greatly increased the risk of Bell's palsy among vaccinees," and they described the association as "strong, temporal, and specific." Id. Forty of the 412 patients received a parenteral flu vaccine. Id. at 6 tbl.3. They found "61.5 percent of parenterally vaccinated case patients had an onset interval of more than 91 days." Id. at 5-6. They concluded there was "no significant risk of Bell's palsy . . . associated with the parenteral [flu] vaccines." Id.

[28] Zhou et al. also acknowledged the issues and limitations with VAERS, stating, in relevant part, "[d]ata from VAERS should be interpreted with caution because they represent adverse events that occurred after vaccination, not all of which may have been caused by vaccination. Temporal association alone does not mean that the vaccine caused the illness or symptoms." Pet. Ex. 21 at 5. Dr. Gershwin agreed that there are "multiple limitations to VAERS" and that VAERS reports "have to be placed in the context of their limitations." Tr. 67, 75-77.

[29] This flu vaccine was similar, but not identical to petitioner's flu vaccine. Tr. 36.

there is "concern that latent [HSV-1] infections of the geniculate ganglia of facial nerves may be one of the causes of Bell's palsy" and that "[i]mmune response mechanisms have also been considered." Id.

Second, Dr. Gershwin cited a recent 2020 study authored by Kamath et al.[30] who "analyzed [VAERS] data to determine whether the facial paralysis reporting rate is higher in those who received the [flu] vaccination compared with those who received other vaccines." Pet. Ex. 47 at 1. The authors evaluated VAERS reports from January 2015 to October 2019, and they identified 250 reports of facial paralysis in patients who received flu vaccines and 346 reports of facial paralysis for all other vaccines. Id. at 3. "[Their] study show[ed] that the likelihood of reporting facial paralysis following [flu] vaccination [was] higher compared with other vaccines." Id. at 4. The authors found an onset median of 3 (range of 1-10) days, but noted "the number of patients for whom the time of onset data were recorded was limited." Id. at 5. Most of the cases of facial paralysis occurred within the first 2 weeks following vaccination with the seasonal trivalent or quadrivalent intramuscular flu vaccine. Id. at 6. They noted "[t]he appearance of Bell's palsy after the vaccination supports the immunological hypothesis." Id. at 4.

Dr. Gershwin next cited Bardage et al.,[31] a population-based study in Sweden with H1N1 vaccine (Pandemrix) from October 2009 and March 2010 who found an increased risk of Bell's palsy. Pet. Ex. 35 at 2, 4. The authors found "a significantly increased risk for Bell's palsy" in "those vaccinated in the early phase of the vaccination campaign (≤ 45 days), when high risk groups predominated." Id. at 4. "In contrast, among people vaccinated after the first 45 days of the campaign, representing more closely the general population, [they] found no statistically significant associations between vaccination and autoimmune or neurological diseases." Id. The authors concluded that they "[could not] explain the small increase in risk for Bell's palsy seen in this study." Id. at 5.

Lastly, Dr. Gershwin cited Huang et al.,[32] who used a "capture-recapture method to (1) assess the reporting completeness of Taiwan's passive safety surveillance system for selected adverse events after 2009 H1N1 vaccines; and (2) evaluate the risks of these events for the biologically plausible postvaccination risk intervals." Pet. Ex. 54 at 2. The authors identified

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[30] Ashwin Kamath et al., Facial Paralysis Following Influenza Vaccination: A Disproportionality Analysis Using the Vaccine Adverse Event Reporting System Database, 20 Clinical Drug Investigation 883 (2020). Like the authors in Zhou et al., the authors acknowledged the limitations in their findings, including the "inherent limitations of the VAERS database analysis and the fact that disproportionality measures only indicate the presence of a signal." Pet. Ex. 47 at 4-6.

[31] Carola Bardage et al., Neurological and Autoimmune Disorders After Vaccination Against Pandemic Influenza A (H1N1) with a Monovalent Adjuvanted Vaccine: Population Based Cohort Study in Stockholm, Sweden, 343 BMJ 1 (2011).

[32] Wan-Ting Huang et al., The Reporting Completeness of a Passive Safety Surveillance System for Pandemic (H1N1) 2009 Vaccines: A Capture-Recapture Analysis, 30 Vaccine 2168 (2012).

1,475 cases of Bell's palsy, with 298 patients developing Bell's palsy 0-42 days after flu vaccination. Id. at 3 tbl.2. The authors also determined the estimated number of Bell's palsy cases to occur between 0-42 days after flu vaccination to be 525, while the expected number of cases was 354. Id. at 3, 3 tbls.3-4. Huang et al. concluded "[t]here was an increased risk for Bell's palsy in the interval 0-42 days after vaccination." Id. at 3.

Dr. Gershwin acknowledged, however, that some studies have found no association between the flu vaccine and Bell's palsy. Pet. Ex. 6 at 2; see, e.g., Pet. Ex. 19 at 4 ("In this study, we did not find an association between immunization with [flu vaccine], [hepatitis b] vaccine, or any vaccine and Bell's palsy during risk intervals of 1–14 days, 1–28 days, and 29–56 days following immunization among children aged 18 years or younger."); Pet. Ex. 22 at 1 (identifying no elevated risk of adverse events after flu vaccination);[33] Pet. Ex. 23 at 3 (finding "no evidence of an increased risk of Bell's palsy in the three months following parenteral inactivated [flu] vaccine").[34]

In addition to studies, Dr. Gershwin also cited Chou et al., an article that discussed two case reports of Bell's palsy following flu vaccination. Pet. Ex. 36 at 1. The first was of a 30-year-old male who developed symptoms 10 days after administration of a flu vaccine. Id. He had received a flu vaccine in the past and had no history of any adverse drug reactions, had no personal or family history of neurological disorders, and had no history of recent infections. Id. The authors found "no other explanation for the Bell's palsy except for the [flu] vaccine. Furthermore, because the peripheral facial palsy presented within 1 month between the vaccination and the onset of neurological symptoms, a causal relationship was suspected . . . when there was no evidence of infection." Id. at 2. The second case report was of an 80-year-old man who developed symptoms three days after flu vaccination. Id. He had a history of type II diabetes and hypertension. Id. He had also received a flu vaccine previously and gave no history of an adverse drug reaction, had no personal or family history of neurological disorders, and had no history of recent infections. Id. Because of his history of diabetes and hypertension, the authors were unable to "definitively implicate the [flu] vaccine as etiologic for Bell's palsy." Id.

ii. Althen Prong Two

As described above, Dr. Gershwin opined that petitioner's flu vaccine caused his Bell's palsy through a local innate inflammatory response. Pet. Ex. 6 at 1-3; Pet. Ex. 27 at 1-2; Tr. 40. More specifically, Dr. Gershwin opined "the lymph nodes and the innate immune system that are found surrounding the facial nerve and, particularly, in the region from the internal acoustic meatus to the stylomastoid foramen became acutely inflamed, leading directly to compression." Pet. Ex. 37 at 2. During the entitlement hearing, he stated that he was "referring to the facial lymph nodes, particularly in the parotid gland." Tr. 46. He clarified that petitioner did not only

[33] Sharon K. Greene, Near Real-Time Surveillance for Influenza Vaccine Safety: Proof-of-Concept in the Vaccine Safety Datalink Project, 171 Am. J. Epidemiology 177 (2010).

[34] Julia Stowe et al., Bell's Palsy and Parenteral Inactivated Influenza Vaccine, 2 Hum. Vaccines 110 (2006).

have swelling of his parotid glands, but he suspected that petitioner's other lymph nodes around his body would have been swollen if they had been examined. Tr. 47.

He explained petitioner "mount[ed] an innate response which [] [began] almost immediately" following vaccination. Pet. Ex. 6 at 2. After vaccination, "the inflammation produced by innate immune cells within the lymph nodes [of petitioner's] facial area would have transported lymphocytes into that local tissue environment, . . . produc[ing] [petitioner's] clinical symptoms of Bell's palsy." Tr. 14; see also Pet. Ex. 27 at 1. This "localized inflammatory reaction within the facial nerve," according to Dr. Gershwin, "is a tissue-specific innate response that is unique to individuals that are genetically susceptible to Bell's [p]alsy." Pet. Ex. 6 at 2-3; see also Pet. Ex. 27 at 1. "[D]ue to his genetic susceptibility, he reacted differently than would a normal host." Pet. Ex. 37 at 1. He suspected that petitioner "likely had more lymph node tissue on his right side where his Bell's palsy was located." Id. at 2. At the hearing, he opined that genetics are not important "unless there are genetic components to how one might respond to a vaccine and/or genetic components [like] obesity[] [and] hypertension." Tr. 30.

Dr. Gershwin clarified that he does not believe petitioner had an excessive innate response, and there was no evidence that petitioner had a local reaction at the vaccine injection site. Pet. Ex. 37 at 1-2; Tr. 54-55. At the hearing, Dr. Gershwin explained "that the absence of a local swelling at the site of injection may not correlate with the development of lymphocytic inflammation or swelling elsewhere." Tr. 24 (citing Pet. Ex. 55 at 4). He found that given petitioner's body stature, the absence of records noting swollen parotid glands or swollen lymph nodes on physical examination "would not be unusual." Tr. 26. He suspected that "more likely than not, [petitioner's] lymph nodes were probably already enlarged [] from his obesity, superimposed on his sleep apnea and his chronic nasal obstruction and allergic rhinitis." Tr. 14. On cross-examination, Dr. Gershwin testified that petitioner's clinical diagnosis of Bell's palsy is sufficient evidence that "more likely than not, overwhelmingly more likely than not, . . . an inflammatory response" occurred. Tr. 256.

Although Dr. Chaudhry, respondent's expert, argued petitioner's Bell's palsy should have been symmetric, meaning that it should have occurred on the same side that petitioner received the vaccine at issue,[35] Dr. Gershwin testified that "Bell's palsy is characteristically [asymmetric]." Tr. 252. Dr. Gershwin agreed with Dr. Romberg, respondent's expert, that "[w]e are symmetric at birth," and opined that we "become asymmetric as we get older." Id. Given petitioner's predisposing factors, Dr. Gershwin opined petitioner was "likely to have more asymmetry on one side than the other." Id.

Dr. Gershwin opined that petitioner "has many predisposing reasons to be more susceptible to closure of the relatively small space that involves the facial nerve." Tr. 13-14, 29. However, these reasons or factors, such as petitioner's obesity, would only predispose petitioner and not directly lead to his development of Bell's palsy. Pet. Ex. 6 at 2. During the hearing, Dr. Gershwin explained that "if [petitioner] didn't have those predisposing factors . . . , I don't think he would have got[ten] Bell's palsy." Tr. 42.

[35] Petitioner received the flu vaccine in his left arm and developed Bell's palsy on the right side of his face.

Additionally, Dr. Gershwin found Dr. Depner's note, stating petitioner "developed [Bell's palsy] about a week after he received the flu vaccine" and that "[i]t is conceivable that the flu vaccine precipitated this," supports his opinion that the flu vaccine caused petitioner's Bell's palsy. Pet. Ex. 26 at 19; Tr. 43. He testified that he would not recommend the flu vaccine to petitioner if he were his patient. Tr. 248.

He also found that the fact Dr. Depner prescribed antivirals does not mean petitioner had a viral infection. Tr. 251. "[I]n [his] opinion, in Bell's palsy, [] steroids most dramatically have an effect. In the absence of evidence of a herpetic infection, [he] would still argue that the reason steroids were given is that they remain the mainstay for the treatment of Bell's palsy, as they have for decades." Tr. 255-56.

Lastly, he found "no other antecedent events that can explain the development of Bell's [p]alsy in [petitioner]." Pet. Ex. 30 at 2; see also Tr. 44. "There was no infection identified here." Tr. 30.

iii. Althen Prong Three

Dr. Gershwin opined that petitioner's numbness on the right side of his face began on November 4, 2015, one day after his flu vaccination. Pet. Ex. 6 at 1; Tr. 40-41. He found this onset to be "consistent with an innate response and tissue specificity, [] similar to that of any virus-induced Bell's [p]alsy." Pet. Ex. 6 at 3; see also Tr. 44.

Dr. Gershwin explained that "[i]nnate immune responses occur rapidly." Pet. Ex. 6 at 3; see also Pet. Ex. 37 at 2; Tr. 23 (citing Pet. Ex. 28). He cited Hervé et al., who noted that cytokines were detected as early as three hours post-adjuvant-containing-vaccination. Pet. Ex. 55 at 2. Although petitioner's vaccine did not contain an adjuvant, he opined that "an adjuvanted vaccine would [] slow things down," and "[a] soluble vaccination would be at least as fast if not faster." Tr. 27.

He opined "the majority of cases of Bell's palsy following the [flu] vaccine have an onset within the first month of vaccination." Pet. Ex. 30 at 2. Zhou et al. found "[a]pproximately 40% of the Bell's palsy reports had an onset interval of 1–3 days," and around 77% had an onset between 1 and 30 days after flu vaccination.[36] Pet. Ex. 21 at 4. Kamath et al. similarly found a median onset of 3 (1-10) days. Pet. Ex. 47 at 5. Huang et al. identified 298 patients who developed Bell's palsy 0-42 days after flu vaccination. Pet. Ex. 54 at 3 tbl.2. And Chou et al. discussed two case reports of Bell's palsy after flu vaccination and reported onset intervals of three days and 10 days. Pet. Ex. 36 at 1-2.

[36] The authors stated that "reporting bias may explain the short onset intervals observed in [their] study. Therefore, the short onset interval should be interpreted with caution. It may not represent the true onset time due to the differential reporting bias in a passive surveillance system." Pet. Ex. 21 at 5.

2. Respondent's Expert, Dr. Vinay Chaudhry

a. Background and Qualifications

Dr. Vinay Chaudhry is board certified in neurology, neuromuscular diseases, electrodiagnostic medicine, and clinical neurophysiology. Resp. Ex. A at 1. He received his M.B. and B.S. in India and then completed an internship and various residencies and fellowships from 1980 to 1989. Resp. Ex. B at 1-2. He was a Professor of Neurology at Johns Hopkins University School of Medicine and the Co-Director of the Neurology EMG Laboratory at Johns Hopkins Hospital. Id. at 1, 3. Currently, Dr. Chaudhry is the chief of the neuromuscular division and vice chair of faculty affairs at UNC-Chapel Hill. Tr. 84. Dr. Chaudhry specializes in the field of neuromuscular diseases. Resp. Ex. A at 1. He has an active clinical practice where he sees over 2,000 patients per year. Id. He has authored or co-authored over 200 publications. Resp. Ex. B at 3-17.

b. Opinion

i. Althen Prong One

Dr. Chaudhry opined that although "the cause of Bell's palsy is not clear," there is a lack of evidence to support a causal relationship between the flu vaccine and Bell's palsy. Resp. Ex. A at 4-6.

Dr. Chaudhry criticized Dr. Gershwin's proposed theory that the flu vaccine invoked an innate immune response specifically against the seventh cranial nerve. Resp. Ex. A at 7. Dr. Chaudhry agreed that "immunization is known to cause an early innate immune response that facilitates development of an antigen-specific adaptive immune response," but found "it difficult to understand" how an innate immune response would exclusively target the seventh cranial nerve. Id. at 7-8; see also Tr. 110-14, 121. He explained that "[t]he innate immune response is a nonspecific response" that "is not specialized for specific antigens." Resp. Ex. R at 1; see also Tr. 111-12, 121. "Because of this broad effect, it is only capable to a certain degree of stopping germs from entering and spreading in the body." Resp. Ex. R at 1.

While Dr. Chaudhry agreed that a flu vaccine can initiate an innate response in the lymph nodes, he argued this would occur locally at the injection site. Tr. 159. Dr. Chaudhry explained that a vaccine administered in the arm would affect axillary lymph nodes, which are not near the facial nerve. Resp. Ex. R at 1; see also Tr. 159-60. He stated there are no lymph nodes near the facial nerve.[37] Tr. 111, 120. He added that "no lymph node swelling has ever been documented or noted to cause facial palsy" in any available literature, nor has it been postulated as a potential etiology for Bell's palsy. Resp. Ex. R at 1; see also Tr. 160, 179. He agreed that the seventh

[37] But see Pet. Ex. 56 at 4, 7 (illustrating the lymph nodes and glands of the head and neck).

cranial nerve goes through and divides in the parotid gland, but stressed that the parotid gland is not a lymph node, but a salivary gland.[38] Tr. 160-61, 179-80.

Moreover, Dr. Chaudhry cited studies that acknowledged that the flu vaccine could trigger Bell's palsy. See, e.g., Resp. Ex. M at 6 ("[Bell's palsy] "could have multiple triggers, of which—considering the hypothetical autoimmune aetiology—[flu] and [flu] vaccination could be one.").[39] But he also cited several studies to support the lack of a causative relationship between the flu vaccine and Bell's palsy. Resp. Ex. A at 5-6 (citing, e.g., Pet. Exs. 19, 22, 23; Resp. Exs. M, F, P).

For example, Dr. Chaudhry cited to Wijnans et al., a self-controlled case series from the United Kingdom aimed "to determine whether there was an increased risk of Bell's palsy following vaccination with any [flu] vaccine containing A/California/7/2009 (H1N1)-like viral strains,"[40] and "whether risks were different following pandemic [flu] A(H1N1)pdm09 vaccines and seasonal [flu] vaccines containing the [flu] A(H1N1)pdm09 strain." Resp. Ex. M at 1. The study population was comprised of all Bell's palsy cases identified in THIN, a primary health care database in the United Kingdom, from June 1, 2009 to June 30, 2013. Id. at 1-2. The authors identified 6,381 cases of Bell's palsy in 6,288 people, 6,198 of whom developed Bell's palsy only once during the study period. Id. at 4. Their data revealed a relative incidence rate of Bell's palsy between 1 and 42 days post-flu vaccination to be 0.88. Id. at 5. When adjusted for seasonality, episodes of acute respiratory infection, and pregnancies, the relative incidence rate decreased to 0.85. Id. The authors "found no evidence of an increased incidence of Bell's palsy consultations following seasonal [flu] vaccination overall, nor for monovalent pandemic [flu] vaccine in 2009." Id. at 7. Additionally, they did not find "evidence of an increased risk of Bell's palsy following vaccination with any [flu] vaccine containing A/California/7/2009 (H1N1)-like viral strains, either pandemic or seasonal vaccines." Id. at 8. However, the authors did note "[i]nflammation is thought to play an important role in the aetiology of Bell's palsy." Id. at 2.

Similarly, Stowe et al. conducted a large population-based study of 2,128 individuals who developed Bell's palsy from 1992 to 2005 and found "no evidence of an increased risk [of Bell's palsy] in the three months following parenteral inactivated [flu] vaccine." Pet. Ex. 23 at 1-2. The highest incidence rate Stowe et al. found was during the risk period of 1 to 30 days. Id.

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[38] The parotid gland, however, does contain lymph nodes. It "is the only salivary gland with two nodal layers, which drain into the superficial and deep cervical lymph system." Pet. Ex. 56 at 2. Additionally, "[l]ymph nodes occur in the skin overlying the parotid gland (pre-auricular nodes) and in the substance of the gland. There are usually ten lymph nodes present in the gland . . . ." Gray's Anatomy: The Anatomical Basis of Clinical Practice 505 (Susan Standring et al. eds., 41st ed. 2016).

[39] Leonoor Wijnans et al., Bell's Palsy and Influenza(H1N1)pdm09 Containing Vaccines: A Self-Controlled Case Series, 12 PLoS ONE e0175539 (2017).

[40] Petitioner's flu vaccine contained this strain. Pet. Ex. 57 at 17 (package insert).

at 2. Stowe et al. and Wijnans et al. found a significant increase in Bell's palsy cases on day 0 (date of vaccination).[41] Id. at 1-3, 2 tbl.1; Resp. Ex. M at 5, 7 tbl.3.

Dr. Chaudhry also cited to Greene et al., a near real-time prospective surveillance study for prespecified adverse events, including Bell's palsy, among enrollees in the Vaccine Safety Datalink Project who received seasonal trivalent inactivated flu vaccines during the 2005/2006 to 2007/2008 flu seasons. Pet. Ex. 22 at 1, 3. Dr. Chaudhry noted the Vaccine Safety Datalink Project collects medical information from numerous medical systems and has a population of nine million people, almost six million of whom received a flu vaccine, as reported by Greene et al. Tr. 143-44. The authors found "no evidence of elevated risk following [flu vaccination] for any of [the] predefined adverse event categories." Pet. Ex. 22 at 9.

The Institute of Medicine ("IOM"),[42] now the National Academy of Medicine, concluded "[t]he evidence favors rejection of a causal relationship between inactivated [flu] vaccine and Bell's palsy." Resp. Ex. F at 10. However, in coming to this conclusion, they only reviewed Stowe et al. and Greene et al. Id. at 8-9.

Dr. Chaudhry cited Lee et al.,[43] another study that examined data from the Vaccine Safety Datalink Project, like Greene et al., during the 2009/2010 flu season, to support his opinion that there is no evidence the seasonal flu vaccine can cause Bell's palsy. Resp. Ex. A at 6 (citing Resp. Ex. P at 1). At the hearing, Dr. Chaudhry stated that the data in Lee et al. may overlap with the data in Greene et al. Tr. 144. The authors in Lee et al. found no statistical signals for Bell's palsy and the live attenuated monovalent flu vaccine, trivalent inactivated flu vaccine, and the live attenuated flu vaccine. Resp. Ex. P at 4.

However, for the monovalent inactivated flu vaccine, they observed "a signal for Bell's palsy for adults aged ≥ 25 years on March 31, 2010, with 141 observed cases compared to 88 expected, for a relative risk of 1.60." Id. "By May 1, 2010, there were 157 cases of Bell's palsy identified . . . in the risk interval and 94 in the comparison interval among [monovalent flu] vaccinees . . . for a relative risk of 1.67." Id. When the authors "evaluat[ed] for temporal clustering and conduct[ed] a case-centered logistic regression analysis that controlled for seasonality, further evidence of a causal association between [the monovalent inactivated flu vaccine] and Bell's palsy was not demonstrated." Id.

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[41] Stowe et al. explained the "increase in risk on the day of vaccination is unlikely to represent a causal association on grounds of biological plausibility and can be explained by opportunistic recording of cases at time of vaccination." Pet. Ex. 23 at 3. Wijnans et al. agreed with Stowe et al. that the finding of an increased risk of Bell's palsy on the date of vaccination was due to "a likely opportunistic recording of cases." Resp. Ex. M at 6.

[42] Inst. of Med., Influenza Vaccine, in Adverse Effects of Vaccines: Evidence and Causality 293 (Kathleen Stratton et al. eds., 2012).

[43] Grace M. Lee et al., H1N1 and Seasonal Influenza Vaccine Safety in the Vaccine Safety Datalink Project, 41 Am. J. Preventative Med. 121 (2011).

Lastly, Dr. Chaudhry criticized Dr. Gershwin's medical literature. For example, Dr. Chaudhry cited Mutsch et al., who examined the intranasal flu vaccine as well as the parenteral flu vaccine. Resp. Ex. A at 7 (citing Pet. Ex. 20); see also Tr. 122-23. Dr. Chaudhry noted no risk was found between parenteral flu vaccines, which is the vaccine at issue here, and Bell's palsy. Resp. Ex. A at 7; Tr. 122-23. "Indeed, the control[] group (without any vaccine) had a higher number (41 cases) of Bell's palsy compared to the parenteral [flu] vaccine group (10 cases)." Resp. Ex. A at 7. Thus, he agreed with the Mutsch et al. authors who found the parenteral flu vaccine was not associated with a higher risk of Bell's palsy. Id. Additionally, with regard to the intranasal flu vaccine, he noted "there is a possibility that you are . . . actually inducing inflammation next to the temporal bone or the area" due to the location of administration. Tr. 123. He also examined Zhou et al. and Kamath et al., and he opined that no causal association can be made using VAERS data. Tr. 125-26, 135-36 (citing Pet. Exs. 21, 47).

Dr. Chaudhry also opined that the case reports do not provide support for Dr. Gershwin's theory. Tr. 133-34 (citing Pet. Ex. 36). The onset in the first case report was ten days, which Dr. Chaudhry found would not support an innate immune response mechanism. Tr. 133. The patient in the second case report was elderly and suffered from both diabetes and hypertension, and Dr. Chaudhry agreed with the authors that ischemia could not be ruled out. Tr. 134-35. Dr. Chaudhry also noted the authors did not propose an innate immune reaction mechanism. Tr. 135.

Alternatively, Dr. Chaudhry noted two mechanisms that are thought to cause Bell's palsy: "(1) herpes simplex-mediated viral inflammatory/immune mechanism and (2) ischemia associated with diabetes and arteriosclerosis." Resp. Ex. A at 4. First, to support his viral hypothesis,[44] he noted herpes virus "is probably the cause of most cases of Bell's palsy." Id. (quoting Pet. Ex. 15 at 8); see also Resp. Ex. E at 2 ("[HSV] activation has become widely accepted as the likely cause of Bell's palsy in most cases, though the evidence is not entirely conclusive."). He opined that herpes is known to be latent in the geniculate ganglion. Tr. 112. He explained this mechanism has specificity to it, unlike the nonspecific innate immune response posited by Dr. Gershwin. Id.

Ronthal, an UptoDate article referenced by Dr. Chaudhry, described the mechanism applicable to the herpes virus as follows:

> [T]he facial nerve has a thickened, edematous perineurium with a diffuse infiltrate of small, round, inflammatory cells between nerve bundles and around intraneural blood vessels. Myelin sheaths undergo degeneration. These changes are seen throughout the bony course of the facial nerve, although nerve damage is maximal in the labyrinthine part of the facial canal where edema causes compression and the tenuous blood supply adds to the damage.

[44] Dr. Chaudhry also noted that varicella zoster virus reactivation "is well known to cause Ramsay Hunt syndrome (facial nerve palsy with rash)," and it "may be the cause" of Bell's palsy "[i]n up to 19 % of patients." Resp. Ex. A at 4 (citing Resp. Ex. H at 6 (C. J. Sweeney & D. H. Gilden, Ramsay Hunt Syndrome, 71 J. Neurology Neurosurgery Psychiatry 149 (2001))); see also Tr. 99. However, Dr. Chaudhry did not cite to the actual study referenced.

Resp. Ex. E at 2-3.

At the entitlement hearing, citing to Gilden and Reich, Dr. Chaudhry explained the mechanism further as it relates to the anatomy of the facial structure. Tr. 103 (citing Pet. Ex. 15 at 5 fig.4; Resp. Ex. Q at 4-5, 4 fig.5-3). He explained the pons is "the nerve that comes out of the brain stem area" and "loops around the sixth nerve nucleus, and . . . comes out to . . . the internal auditory meatus before entering the facial canal." Id. The geniculate ganglion, known as "the sensory ganglia of the facial nerve," is where herpes resides until it is reactivated for a number of different reasons. Tr. 103-04. "[T]hat virus then travels along the course of the nerve and causes damage and inflammation to the nerve in the facial canal, which is a tight area of the nerves." Tr. 104. Reich explained the facial canal is "a narrow bony canal within the temporal bone" and the "little room for expansion" leads to "inflammation of the nerve" and "compression resulting in paralysis." Resp. Ex. Q at 5; see also Tr. 152 (agreeing that the herpes mechanism involves inflammation of the seventh cranial nerve). Then, the facial nerve exits into the stylomastoid foramen, which is located under the ear. Tr. 104 (citing Resp. Ex. Q at 4 fig.5-3). Upon exiting, the facial nerve can go in multiple directions. Tr. 107-09. Dr. Chaudhry noted he is unaware of any lymph nodes in the facial canal. Tr. 104.

He then cited studies[45] that found HSV-1 genomes in Bell's palsy patients and explained that it is thought that reactivation of HSV in the geniculate ganglion causes Bell's palsy. Resp. Ex. A at 4. In Burgess et al.,[46] for example, the authors, using polymerase chain reaction ("PCR") with DNA from the temporal bone at autopsy, found HSV-1 genomic DNA in the geniculate ganglion of a patient with Bell's palsy, and concluded that "[t]his association suggest[ed] that . . . HSV-1 may have caused Bell's palsy" in that patient. Resp. Ex. K at 1, 3-4. They hypothesized that

[45] One study Dr. Chaudhry cited was Takahashi et al., who produced "a mouse model of facial nerve paralysis induced by the reactivation of latently infected HSV-1" to investigate the mechanism of Bell's palsy. Resp. Ex. L at 1 (Hirotaka Takahashi et al., Mouse Model of Bell's Palsy Induced by Reactivation of Herpes Simplex Virus Type 1, 60 J. Neuropathology & Experimental Neurology 621 (2001)). The authors inoculated a strain of HSV-1 into the mice. Id. Mice that developed a transient facial nerve paralysis after primary infection were then subjected to either (1) auricular skin scratch at the site of the previous inoculation, (2) an intraperitoneal injection of anti-CD3 monoclonal antibody, or (3) a combination of both. Id. at 1-2. Their "[h]istopathological findings showed neuronal degeneration in the geniculate ganglion and demyelination of the facial motor nerve in paralyzed mice. These findings suggest[ed] that a combination of stimuli, local skin irritation, and general immunosuppression is essential for successfully inducing facial nerve paralysis in mice with latent HSV-1 infection." Id. at 1.

[46] Robert C. Burgess et al., Polymerase Chain Reaction Amplification of Herpes Simplex Viral DNA From the Geniculate Ganglion of a Patient with Bell's Palsy, 103 Annals Otology Rhinology Laryngology 775 (1994).

> Since spread of HSV-1 is via sensory or autonomic nerve fibers, infection of the geniculate ganglion may have occurred by viral spread along the chorda tympani. Since HSV-1 is a common infection in the oral cavity, the oral cavity could have been the primary site from which the chorda tympani became infected. Triggering events leading to viral reactivation and subsequent inflammation and edema of the ganglion may have resulted in nerve entrapment and the ensuing facial paralysis this patient experienced.

Id. at 4. Lazarini et al.[47] used PCR to test the occurrence of HSV-1 in the saliva of Bell's palsy patients. Resp. Ex. I at 1. They found 11 of 38 patients (29%) with Bell's palsy tested positive for the presence of HSV-1 in their saliva. Id. at 1, 3. "This result was statistically significant if compared to the control group, in which [they] did not find any positive case." Id. at 1. Like the authors in Burgess et al., the Lazarini et al. authors concluded that viral reactivation may be the etiology of Bell's palsy. Id. at 1, 3-4.

Similarly, Murakami et al.[48] used PCR on facial nerve endoneurial fluid specimens and specimens of posterior auricular muscle innervated by the facial nerve and found 11 of 14 patients (79%) with Bell's palsy had HSV-1 genomes. Resp. Ex. J at 1, 3. They "conclude[d] HSV-1 infection in the facial nerve is directly related to the pathogenesis of Bell['s] palsy." Id. at 4. The authors acknowledged that "identification of viral DNA may not always be definitive evidence that a particular agent causes a disease process, because PCR can amplify viral DNA regardless of whether the virus is in the infective, lytic, or latent state." Id. at 3. However, "HSV-1 . . . would probably not be detected in the endoneurial fluid or auricular muscle unless [it was] reactivated," which they argued was supported by the fact that HSV-1 was not detected in any control. Id. at 4. The authors explained that once latent HSV-1 in the geniculate ganglion is reactivated, "it destroys ganglion cells and spreads into the endoneurial fluid. The virus also infects Schwann cells, leading to demyelinization and inflammation of the facial nerve." Id.

With regard to the second hypothesis (ischemia associated with diabetes and arteriosclerosis), Dr. Chaudhry did not describe this mechanism in detail in his expert reports. See Resp. Ex. A at 5; Resp. Ex. R at 1-3. But he cited medical literature that briefly discussed this hypothesis. Gilden, for example, wrote "[s]ome cases of Bell's palsy have been attributed to ischemia from diabetes and arteriosclerosis, which helps to explain the increased incidence of Bell's palsy in elderly patients; the disorder is analogous to ischemic mononeuropathy of other cranial nerves in patients with diabetes." Pet. Ex. 15 at 8. Similarly, Reich explained "[e]dema of the facial nerve within the narrow fallopian canal has been observed during decompressive surgery for Bell's palsy," and the cause of the edema has been suspected to be due to "ischemia in predisposed patients, such as the elderly or those with diabetes mellitus or hypertension, akin to other known ischemic cranial neuropathies." Resp. Ex. Q at 6-7. Ronthal noted "[a]

[47] Paulo Roberto Lazarini et al., Herpes Simplex Virus in the Saliva of Peripheral Bell's Palsy Patients, 72 Brazilian J. Otorhinolaryngology 7 (2006).

[48] Shingo Murakami et al., Bell Palsy and Herpes Simplex Virus: Identification of Viral DNA in Endoneurial Fluid and Muscle, 124 Annals Internal Med. 27 (1996).

retrospective study[49] found that 190 (74 percent) of 257 patients with Bell's palsy first noticed facial weakness in the morning, suggesting that actual development of facial palsy occurred during sleep; the authors speculated that nocturnal onset suggested an ischemic mechanism." Resp. Ex. E at 3 (internal citations omitted).

At the entitlement hearing, Dr. Chaudhry explained ischemia as a cause of Bell's palsy typically occurs in elderly patients in the setting of hypertension or diabetes when there is a narrowing of blood vessels resulting in reduced blood flow. Tr. 90-91. Although the mechanism is not fully understood, he noted ischemia would occur quickly. Tr. 92-93, 96. Dr. Chaudhry testified that it is "possible" for ischemia to cause a nerve to swell "but [it is] not necessary." Tr. 93. He added that such swelling, when it occurs, would not be considered inflammation. Tr. 94-95.

ii. Althen Prong Two

Dr. Chaudhry opined that petitioner's flu vaccine did not play a causative role in the development of his Bell's palsy. Resp. Ex. A at 6, 8; Resp. Ex. R at 2; Tr. 150. If Dr. Chaudhry were to pick one cause of petitioner's Bell's palsy, it would be due to the herpes virus before ischemia and hypertension. Tr. 119. Based on the medical literature and his personal experience treating patients, he opined "that viral cause is more likely than not."[50] Tr. 119-20.

Dr. Chaudhry agreed petitioner developed Bell's palsy and had no other neurological deficits. Resp. Ex. A at 3; Resp. Ex. R at 2; Tr. 89. He agreed that petitioner had never been diagnosed or treated with Bell's palsy prior to the vaccine at issue, nor did he have any clinical evidence of Bell's palsy prior to the vaccine at issue. Tr. 161. However, he found petitioner's clinical presentation, treatment, and improvement typical for Bell's palsy. Resp. Ex. A at 4; Tr. 163-64. Dr. Chaudhry noted petitioner was treated with antiviral therapy which is recommended along with corticosteroids "based on the hypothesis that viral infection (herpes simple[x]) or shingles (varicella zoster) causes Bell's palsy." Resp. Ex. A at 5.

Given the anatomy of the facial structure, Dr. Chaudhry inferred that the damage to the nerve occurred within petitioner's facial nerve canal. Tr. 181. Because petitioner's vaccine was administered intramuscularly in his arm, Dr. Chaudhry argued the lymph nodes affected by an innate immune response would be the axillary lymph nodes, not near petitioner's facial nerve, and not only on one side of petitioner's face. Resp. Ex. R at 1. However, Dr. Chaudhry testified that there was no evidence of lymph node swelling in petitioner. Tr. 120. Additionally, Dr. Chaudhry testified that parotid gland inflammation is painful and to not feel it would be unusual. Tr. 111, 182. He opined "[it is] unlikely that [petitioner's] injury occurred in the parotid gland region[] [] because all the components of facial nerve were affected." Tr. 181.

[49] This study was not filed.

[50] This line of testimony is at issue in petitioner's motion to strike. See Pet. Mot.; Pet. Reply.

Dr. Chaudhry disagreed with petitioner's treating physician, Dr. Depner's statement that "[i]t is conceivable that the flu vaccine precipitated [petitioner's Bell's palsy]." Tr. 167 (quoting Pet. Ex. 26 at 19).

Further, Dr. Chaudhry testified that an ischemic cause of petitioner's Bell's palsy could not be ruled out. Tr. 98, 169-72. Petitioner had a history of hypertension for at least two years prior to the onset of his Bell's palsy. Tr. 98. He argued petitioner's two risk factors for developing Bell's palsy—hypertension and obesity—would have made him more prone to developing Bell's palsy. Resp. Ex. A at 4; Tr. 168-69. On cross-examination, Dr. Chaudhry opined it is likely that petitioner's hypertension played a part in causing his Bell's palsy. Tr. 171-72.

Dr. Chaudhry also testified that a viral cause, specifically a herpes virus, could not be ruled out as the cause of petitioner's Bell's palsy. Tr. 118-19, 170-72. He cited to the fact that petitioner improved on antivirals and his clinical course was consistent with what would happen with a viral Bell's palsy. Tr. 119. He conceded, however, that it is possible that petitioner could have improved on his own. Id.

iii. Althen Prong Three

Dr. Chaudhry agreed petitioner developed Bell's palsy on November 4, 2015. Resp. Ex. A at 3; Tr. 161. He found "the onset of the symptoms within twenty four hours of the vaccine administration would be too quick to produce [] Bell's palsy." Resp. Ex. A at 6.

He explained that there are three phases to describe the latency between exposure to an antigen and development of an immune response: a lag phase, logarithmic phase, and plateau phase. Resp. Ex. A at 6. "The lag phase . . . is classically thought to be between 4 to 7 days for the primary response and between 1 to 3 days for subsequent exposure to the antigen. The logarithmic phase . . . is 7-10 days for the primary response and 3 to 5 days for the secondary response." Id. (citing Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57, 58 (Kathleen Stratton et al. eds., 2012)).[51] Thus, he argued petitioner's 24-hour onset "is too short an interval for an immune reaction to an antigen." Id.

In his supplemental report, Dr. Chaudhry noted a specific adaptive immune response sets in four to seven days after vaccination. Resp. Ex. R at 1. However, he did agree that an innate immune response "does not need a long start-up phase," and is typically "very quick, within hours." Id.; Tr. 159. He also agreed that "there [was] an overlap between the innate response and the adaptive response kicking in," but was not able to opine on the duration of an innate immune response. Tr. 159.

[51] Respondent did not provide this chapter; however, this text is well known to the undersigned. The section Dr. Chaudhry cites to discusses the "latency between antigen exposure and peak adaptive immune response." See Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57, 57-58 (Kathleen Stratton et al. eds., 2012).

Dr. Chaudhry cited medical literature noting onset periods. Stowe et al. found the highest incidence rate of Bell's palsy in their study to be during the risk period of 1 to 30 days. Pet. Ex. 23 at 2. Additionally, Stowe et al. and Wijnans et al. found a significant increase in Bell's palsy cases on day 0 (date of vaccination).[52] Id. at 1-3, 2 tbl.1; Resp. Ex. M at 5, 7 tbl.3.

3. Respondent's Expert, Dr. Neil D. Romberg

a. Background and Qualifications

Dr. Neil Romberg is board certified in pediatrics and allergy and immunology. Resp. Ex. T at 1-2. He is an assistant professor of pediatrics at the University of Pennsylvania and an attending immunology physician at the Children's Hospital of Philadelphia. Resp. Ex. S at 1; Resp. Ex. T at 1. After receiving his M.D. from Pennsylvania State College of Medicine in 2004, he completed a residency in pediatrics at New York University School of Medicine in 2008 and a fellowship in allergy and clinical immunology at Yale University in 2011. Resp. Ex. T at 1. Dr. Romberg focuses his career on "car[ing] for patients with inherited immunological disorders and [] investigat[ing] the molecular mechanisms that underlie their diseases. [He] head[s] a research laboratory that investigates several topics in human immunology . . . including failures of immunologic tolerance, autoantibody production and excessive activation of the innate immune system." Resp. Ex. S at 1.

b. Opinion

i. Althen Prong One

Dr. Romberg opined that there is no evidence to support Dr. Gershwin's theory that an innate immune response initiated by a flu vaccine in the deltoid can lead to unilateral facial nerve paralysis. Resp. Ex. S at 3.

Dr. Romberg, like Dr. Gershwin, explained that the immune system is categorized into the innate and the adaptive system. Resp. Ex. S at 3; Tr. 205. He explained that the innate immune system responds rapidly. Resp. Ex. S at 3. "Components of the innate system called pattern recognition receptors (PRR) detect danger signals including common parts of microbes or specific types of non-organic matter. Several parts of [flu] virus are detected by PRRs" and "[u]pon detection by PRRs, the innate immune system can quickly, within minutes to hours, produce a local immune response including production of cytokines." Id.; see also Tr. 235-36. He opined that lymph nodes would not be involved at the time of an initial innate immune response at the site of vaccination, but would be involved in a vaccine response when an "antigen presenting cell like a dendritic cell [] move[s] from a local tissue into the lymph node," which would take hours to a day. Tr. 196-97, 234. He clarified that such a response would not typically occur in all lymph nodes, and would be expected in only the draining lymph nodes. Tr. 238.

[52] See supra note 41.

Dr. Romberg added that "[m]ost vaccines are [] designed to activate the innate immune system" and "it is clear the innate system activation was the intended . . . outcome" of the vaccine at issue here. Resp. Ex. S at 3; see also Tr. 234. "Most innate immune responses are small and cause symptoms near the site [of vaccination]." Resp. Ex. S at 3. "A small amount of inflammation is desirable as it recruits lymphocytes from the adaptive immune to regional lymph nodes resulting in the eventual production of high titer neutralizing antibodies." Id.

In addition, Dr. Romberg agreed with Dr. Gershwin that innate immune responses can cause systemic responses. They "can be excessive causing both an injection site reaction and even systemic symptoms," which "are the consequence of inflammatory cytokines entering the circulation to have effects on organs distant from the injection site." Resp. Ex. S at 3; see also Tr. 236. When systemic inflammation occurs, "the local reaction is so robust that the mediators are [] no longer kept locally and are pouring out into the blood supply." Tr. 212. "[T]he body starts reacting to those mediators[] . . . at areas and organs distal or distant to the local site." Id. Signs of systemic inflammation include fever, myalgia, headache, hypotension, and more. Tr. 213-15.

At the entitlement hearing, Dr. Romberg discussed Chatziandreou et al., an article relied upon by Dr. Gershwin. Tr. 198-202, 205-07 (citing Pet. Ex. 28). The authors examined the role of macrophages in lymph nodes of mice. Tr. 198. He explained that subscapular sinus macrophages act as "flypaper," or "a filter that keeps [] bacteria in the lymph node so that they can't cause infection but they can be interrogated by the immune system." Tr. 199-200. This process would also apply to vaccine components. Tr. 201. Dr. Romberg further explained that Chatziandreou et al. speculated that the purpose of these macrophages was to contain an infection to a local area of the body, and to integrate dendric cells into the process, which "are specialized to communicate directly with the adaptive immune system." Tr. 200.

Given the way our immune system operates, Dr. Romberg explained that "any vaccine component or inflammatory mediator produced at the injection site or in a draining lymph node could only travel to the facial nerve after first passing through a network of lymphatic vessels, then into venous circulation, then systemic arterial circulation to be disseminated to all vascularized tissues including facial nerves." Resp. Ex. LL at 2; see also Tr. 218-19. He cited Trevaskis et al.[53] to demonstrate the path a vaccine administered in the left deltoid would take to reach the right cranial nerve. See Tr. 219-20 (citing Resp. Ex. MM at 2 fig.1).

He noted "gravity drains deltoid lymphatics down to axillary lymph nodes and not up into the skull." Resp. Ex. S at 3. In Chatziandreou et al., as Dr. Romberg explained, mice were injected with the vaccine in their foot pads, which would drain into the popliteal lymph nodes located behind the knees. Tr. 206. This would be equivalent to receiving a vaccine in the deltoid muscle, with the vaccine draining into the axillary lymph nodes found in your armpit. Id. In response to Dr. Gershwin's citation to a website indicating "swollen, painful, or tender lymph glands in the neck, armpit, or groin" as a side effect of vaccination, Dr. Romberg explained that if a vaccine is administered into the thigh, then the vaccine would drain into the inguinal lymph

[53] Natalie L. Trevaskis et al., From Sewer to Saviour—Targeting the Lymphatic System to Promote Drug Exposure and Activity, 14 Nature Revs.781 (2015).

nodes found in the groin. Tr. 207-08 (quoting Pet. Ex. 51 at 8). "[I]f [a] vaccine was given especially high in the deltoid, it might end up swelling lymph nodes . . . around the collarbone." Tr. 209.

Thus, Dr. Romberg opined that there is "no rational anatomical nor immunological explanation for how innate inflammation initiated by a vaccine injected into the deltoid could contribute to unilateral facial nerve paralysis." Resp. Ex. S at 3 (emphasis omitted). He explained that "[l]ocal spread of inflammation through soft tissues would require visible induration." Id.; see also Tr. 211. He further opined that spread of inflammation through blood vessels would be "nonsensical as the arterial blood supply to and venous blood return from the deltoid is not shared with either cranial nerve." Resp. Ex. S at 3. Relying on Trevaskis et al., he found it "very unlikely" that an innate inflammatory reaction would occur in only one facial nerve. Tr. 220-21 (citing Resp. Ex. MM at 2 fig.1).

Dr. Romberg agreed "that while some cases of Bell's palsy are likely caused by an inflammatory mechanism, there are as many cases which are not." Resp. Ex. S at 5. He opined that infections have been implicated as a cause of Bell's palsy. Tr. 230-31. He noted two mechanisms that are thought to be at play in those cases: (1) direct pressure to the facial nerve and (2) reactivation of HSV, leading to inflammation that causes the nerve to swell. Tr. 231.

He also criticized Dr. Gershwin's medical literature.[54] Resp. Ex. S at 4-5. First, with regard to Dr. Gershwin's reliance on Zhou et al., Dr. Romberg addressed the inherent limitations in using VAERS data and found "a conclusion drawn from a dataset with such massive underreporting is nearly meaningless." Id. at 4. He also cited an editorial that discussed issues with the findings in Zhou et al. Id. (citing Resp. Ex. FF at 1-2).[55]

Dr. Romberg cited to Stowe et al., who found "no evidence of an increased risk of Bell's palsy in the three months following parenteral inactivated [flu] vaccine." Resp. Ex. S at 4 (quoting Pet. Ex. 23 at 3); see also Resp. Ex. LL at 1. He opined Stowe et al. was "a methodologically superior study" than Zhou et al. Resp. Ex. LL at 1. Because Stowe et al. "is the only large population-based study on the topic of interest, [he] conclude[d] the potential association between Bell's palsy and parenteral [flu] vaccine identified by Zhou et al[.] is unlikely a real biological phenomenon but instead reflects methodological flaws intrinsic to the VAERS database." Resp. Ex. S at 4. (emphasis omitted).

[54] Dr. Romberg also criticized Dr. Gershwin's reliance on Mutsch et al. and found it "undermines, not supports, Dr. Gershwin's theory." Resp. Ex. S at 4-5 (emphasis omitted). At the hearing, Dr. Romberg agreed that the intranasal flu vaccine has been linked to an increased risk of Bell's palsy and "[found] that data persuasive." Tr. 232-33.

[55] Samuel Shapiro, Clinical Judgement, Common Sense and Adverse Reaction Reporting, 13 Pharmacoepidemiology & Drug Safety 511 (2004).

ii. Althen Prong Two

Dr. Romberg agreed that petitioner's symptoms were consistent with a diagnosis of Bell's palsy. Tr. 192. He also agreed that "inflammation [] plays a role in some Bell's palsy cases," but opined that there is no evidence to support the conclusion that petitioner's flu vaccine "triggered an exaggerated innate inflammatory response injuring [petitioner's] facial nerve." Resp. Ex. S at 5; see also Tr. 228-29. He based his opinion on three reasons: (1) there was no evidence of inflammation at petitioner's site of vaccination, "and even if there was, there [was] no plausible anatomic explanation how it could extend discretely to his facial nerve;" (2) many cases of Bell's palsy are non-inflammatory in nature; and (3) "all well conducted epidemiologic studies agree that injected inactivated seasonal [flu] vaccine is not linked to increased incidence of Bell's [p]alsy." Resp. Ex. S at 5.

Dr. Romberg opined "it is clear that innate system activation was the intended and likely outcome of vaccinating [petitioner] with [the] seasonal [flu] vaccine." Resp. Ex. S at 3. Dr. Romberg agreed with Dr. Gershwin that "[t]here is no evidence that the adaptive immune system contributed to [petitioner's] development of Bell's [p]alsy." Id. at 2. "[G]iven [petitioner's] onset of symptoms [] approximately 24 hours after receiving his flu vaccine, if the flu vaccine was responsible [] in some way for causing his Bell's palsy, that would be way too soon for an adaptive immune response to have occurred." Tr. 193.

Dr. Romberg stated that "[he] [could not] imagine how [petitioner's] seasonal [flu] vaccine could induce inflammation at a location so remote from the injection site." Resp. Ex. S at 3. After a review of petitioner's medical records from November 5 to December 3, 2015, he found "no evidence that [petitioner] experienced a visible local reaction at the vaccination site," and no evidence of any systemic inflammatory symptoms in petitioner. Id. (emphasis omitted); see also Resp. Ex. LL at 2. Dr. Romberg found petitioner "was never noted nor suspected to be febrile which is consistent with non-elevated serum IL-1beta concentrations," and "[h]e was never noted nor suspected to be hypotensive suggesting he did not have elevated systemic TNF-alpha concentrations." Resp. Ex. S at 3 (emphasis omitted). In fact, petitioner was hypertensive (134/90) when he was evaluated by Dr. Depner on November 5, 2015. Id.

Because "gravity drains deltoid lymphatics down to axillary lymph nodes and not up into the skull," Dr. Romberg would have expected the lymph nodes in petitioner's axillary chain on his left side to be swollen if there was an excessive reaction. Resp. Ex. S at 3; see also Tr. 221. Thus, even if petitioner's flu vaccine induced systemic inflammation in petitioner, Dr. Romberg would have expected a "more widely disseminated disease including symmetric not unilateral facial paralysis." Resp. Ex. S at 3. He concluded "Dr. Gershwin's theory of inflammatory facial injury is unlikely and his theory that injected [flu] vaccine triggered unilateral facial nerve inflammation in the absence of systemic symptoms is implausible." Resp. Ex. LL at 2.

However, Dr. Romberg conceded that petitioner's flu vaccine could trigger a response at a site distant from vaccination. At the hearing, he explained that the flu vaccine petitioner received could trigger a focal response at a distant location "[i]f a small quantity of flu protein . . . entered [petitioner's] bloodstream [] at a level too low to be detected and cause systemic inflammatory reaction." Tr. 226. Then, "through some sort of unlikely event[,] only lodge in his

right facial nerve." Id. However, he opined "if it was not large enough to provoke an inflammatory reaction as it was traveling through the blood, it would also not be in great enough quantity to create an inflammatory reaction at a local site like a nerve." Tr. 226-27.

When questioned about Dr. Depner's note stating petitioner "developed [Bell's palsy] about a week after he received the flu vaccine" and that "[i]t is conceivable that the flu vaccine precipitated this," Dr. Romberg opined Dr. Depner's opinion "is not implausible." Tr. 236-37; see Pet. Ex. 26 at 19. He also opined that when Dr. Depner prescribed antiviral medication, he was "playing the odds" because "herpes viruses [are] the most likely contributing factor" of Bell's palsy. Tr. 241-42. Additionally, steroids would have been prescribed as an anti-inflammatory. Tr. 242. Overall, he found Dr. Depner's medication regimen for petitioner "suggest[ed] . . . that Dr. Depner, from an etiological perspective, did highly suspect a herpes virus as a contributor or cause of [petitioner's] Bell's palsy." Tr. 245.

Dr. Gershwin testified that petitioner was genetically predisposed or susceptible to Bell's palsy; however, Dr. Romberg found no evidentiary basis for this aspect of Dr. Gershwin's opinion. Resp. Ex. S at 5; Resp. Ex. LL at 1. He noted, after reviewing the medical records, petitioner had no family history of Bell's palsy or any other inflammatory disease. Resp. Ex. S at 5. Although petitioner has a family history of type II diabetes, and diabetes is a risk factor for Bell's palsy, Dr. Romberg opined that the mechanism for diabetes-induced Bell's palsy is vascular and not inflammatory. Id.

Dr. Gershwin also opined that petitioner had more lymph node tissue on his right side where his Bell's palsy was located, which Dr. Romberg did not find to be a plausible explanation given Dr. Gershwin's proposed mechanism. Tr. 227-28. Dr. Romberg viewed the "lymphatic tissue in the neck, face[,] and skull as being symmetric." Tr. 227. He agreed that "human anatomy changes over time, but [he was] not aware if lymph node symmetry changes from birth to being an adult." Tr. 237. He also "[did not] know why [cells] would [] exit lymph nodes into local tissues to attack a nerve." Tr. 228. "If they're going to leave a lymph node, they're going to leave [] and enter systemic circulation through the thoracic duct, so they're not going to directly leave the lymph node into the tissue." Tr. 230. However, he also stated if a vaccine were to travel to the facial nerve, it would be disseminated to the facial nerve once there was systemic circulation. Resp. Ex. LL at 2; see Tr. 218-19.

Lastly, Dr. Romberg noted there is no evidence showing "[petitioner's] right facial nerve was actually damaged by inflammation." Resp. Ex. LL at 1. He stated "[a]s we do not have a facial nerve specimen from [petitioner], nor any specific laboratory or imaging data suggesting an excessive inflammatory response, [he] [found] no reasonable basis to speculate inflammation was a contributor or cause of his unilateral facial paralysis." Resp. Ex. S at 5 (emphasis omitted). Dr. Romberg concluded that even if "[petitioner's] facial nerve was damaged by innate immune cells, Dr. Gershwin's theories that these cells were activated by the [flu] vaccine or that [petitioner] was genetically susceptible to this specific injury are scientifically implausible." Resp. Ex. LL at 2.

iii. Althen Prong Three

Dr. Romberg agreed that petitioner's onset of symptoms was approximately 24 hours after administration of the flu vaccine. Tr. 193.

He opined that once the flu vaccine was detected by pattern recognition receptors, "the innate immune systems can quickly, within minutes to hours, produce a local immune response including production of cytokines." Resp. Ex. S at 3; see also Tr. 233. Because petitioner received an unadjuvanted flu vaccine, Dr. Romberg stated he would expect the immune system response to be slower than an adjuvanted flu vaccine. Tr. 233. However, this response would still begin within hours, and it would last days. Tr. 233-34.

IV. MOTION TO STRIKE EXPERT TESTIMONY

A. Parties' Contentions

During the entitlement hearing, and in a subsequent Motion to Strike, petitioner moved to strike Dr. Chaudhry's hearing testimony regarding alternative causes of petitioner's Bell's palsy. Pet. Mot.

The basis of the petitioner's motion was that in September 2020, respondent filed a status report stating that he "[did] not intend to pursue a 'factors unrelated' [to vaccination] theory" at the hearing. Resp. Status Rept., filed Sept. 25, 2020 (ECF No. 54); Tr. 172. In response, respondent argued that the testimony at issue could be considered for the purpose of determining "whether [p]etitioner ha[d] satisfied [his] burden . . . that the vaccine [was] more likely than not the cause of the injury." Tr. 172. Further, respondent asserted that Dr. Chaudhry's testimony was consistent with his previous testimony. Id.

The specific testimony petitioner seeks to strike is in Dr. Chaudhry's direct testimony and cross-examination testimony. See Pet. Mot. at 4-5. On direct, the specific colloquy is as follows:

> Q. But at this point, you couldn't reliably rule out either cause. Would that be fair?
>
> A. Correct. But based on the literature review and based on my personal experience treating patients over the last 30-some years, I would think that viral cause is more likely than not. But I can't rule out hypertension either.

Id. at 3 (quoting Tr. 119-20). There was no contemporaneous objection made at the hearing to the above testimony by Dr. Chaudhry of an alternative cause (viral cause).

As it pertains to Dr. Chaudhry's cross-examination testimony, petitioner seeks to strike the following exchange:

Q. Okay. But you are not saying that either one of these conditions [obesity and hypertension] was the cause of [petitioner's] Bell's palsy, are you?

A. Well, I just, I think, discussed it earlier, that ischemia is one of the proposed hypotheses, and one of the reasons ischemia is thought to be a proposed hypothesis is because people who have hypertension are more prone to it. So not directly hypertension, but whatever is causing his hypertension, which is arteriosclerosis of the blood vessels is also the causative agent for the ischemia to progress. So indirectly, hypertension can cause and be related to arteriosclerosis, and so I think I --

Let me read my report, if I may, because when I mention the ischemic hypothesis, the -- and this is taken directly from the New England Journal article where they said that ischemia is thought to be another etiology because it's analogous to ischemic mononeuropathy occurring in patients with diabetes and hypertension. This is a quote from the New England Journal.

So the ischemic hypothesis is partially based on the fact that people who have diabetes and people who have hypertension have vascular disease which can lead to Bell's palsy. So I don't think hypertension caused it directly, but it is one of the theories that ischemic mononeuropathy happens more in people who have hypertension because of the blood vessels' anatomy.

Q. So just to be clear, you are not giving an opinion in this case to a reasonable degree of medical probability as to the cause of [petitioner's] Bell's palsy. Is that right?

A. Well, yes and no. I've given two possible theories. I didn't say these were the cause. I said, this could be the viral hypothesis. It could be the ischemic hypothesis. Either one or both could be playing a part. I did not say that that's the cause of the Bell's palsy. He had Bell's palsy. So that's -- I cannot dissociate myself from my opinion when I've already written it, that there are two potential causes of Bell's palsy, and [petitioner] could have one, both, and neither, too.

I'm not -- but I think, knowing what we know about Bell's palsy, those are the two etiologies. Certainly he was given antiviral treatment by the same doctor who feels that it was precipitated. He was given hypertensive treatment by the same doctor, so I cannot rule out them being the etiology.

* * *

Q. Do you have an opinion as to what caused [petitioner's] Bell's palsy to a reasonable degree of medical probability.

A. Well, I think I said it in my report, and I don't know why you didn't see it. But on the -- when I said antiviral treatment, I said, indeed [petitioner] was treated

> with antiviral therapy. So I'm saying that this could be the cause. When I say that he had hypertension, that hypertension can be associated with ischemic mononeuropathy, the next sentence is, "[i]ndeed, [petitioner] had hypertension." So to the reasonable degree of certain cause is what we know about Bell's palsy. He had Bell's palsy. He was treated for antiviral treatment. He had hypertension, which is known risk factor. Would they be playing a part? Absolutely.
>
> Q. I'm sorry. I don't think you ever answered my questions with a yes or no.
>
> A. It's not a yes or no answer. I can only tell you what I just stated. It's not a yes or no. He has Bell's palsy. Bell's palsy has two hypotheses. Both of those hypotheses could be true. Is it 100 percent? No. Is it likely? Yes.

Pet. Mot. at 4-5 (quoting Tr. 169-72). At the hearing, petitioner moved to strike the above testimony by Dr. Chaudhry. Tr. 172.

The parties have now briefed the issue. Petitioner moved to strike Dr. Chaudhry's testimony "to the extent it is [] interpreted as evidence of a factor unrelated" for three reasons: (1) "it would be unfairly prejudicial to petitioner to allow the testimony in evidence;" (2) "respondent unduly delayed disclosing such evidence," which "unfair[ly] prejudice[d] [] petitioner;" and (3) "Dr. Chaudhry's testimony was unresponsive." Pet. Mot. at 6; Pet. Reply at 1-3. In the alternative, petitioner argued Dr. Chaudhry's testimony "should not be considered relevant to the issue of a factor unrelated for the reasons set forth above" and "[g]iven the confusing, ambiguous[,] and contradictory nature of his testimony." Pet. Mot. at 7; Pet. Reply at 1-3.

Respondent asserted the Court should deny petitioner's motion to strike "because (1) petitioner is estopped under the 'invited error' doctrine from claiming Dr. Chaudhry's testimony should be stricken because petitioner adduced the testimony he now complains of, (2) petitioner's request to strike Dr. Chaudhry's direct testimony is untimely, and (3) . . . striking said testimony is not an appropriate remedy." Resp. Response at 1, 6-8, 11-12. Respondent added that (1) "[r]espondent did not unduly delay in disclosing evidence, in that respondent did not intend to present the evidence at issue;" (2) "Dr. Chaudhry's testimony responded to the question posed by petitioner's counsel;" and (3) Dr. Chaudhry's testimony was not "confusing, ambiguous[,] and contradictory," nor was he "required to use the words 'reasonable degree of medical probability.'" Id. at 8-11.

B. Legal Standard

A special master must "afford[] each party a full and fair opportunity to present its case." Vaccine Rule 3(b)(2). Vaccine Rule 8(b)(1) provides that "[i]n receiving evidence, the special master will not be bound by common law or statutory rules of evidence but must consider all relevant and reliable evidence governed by principles of fundamental fairness to both parties." This rule echoes the statutory requirement that a "special master . . . shall consider . . . all [] relevant medical and scientific evidence contained in the record." § 13(b)(1). Together, Vaccine Rule 8 and § 13 "direct[] the special master to consider all relevant and reliable evidence,

unencumbered by traditional rules of admissibility, while being guided by principles of fairness." Hazelhurst v. Sec'y of Health & Hum. Servs., 604 F.3d 1343, 1349 (Fed. Cir. 2010). The Vaccine Act further mandates "flexible and informal standards of admissibility of evidence." § 12(d)(2)(B).

Although the Vaccine Rules do not specifically include a mechanism for a motion to strike testimony, Vaccine Rule 1 provides that for any matter not specifically addressed by the Vaccine Rules, the special master may regulate applicable practice consistent with the rules and the purpose of the Vaccine Act. Vaccine Rule 1(b). Vaccine Rule 1 also provides that the Rules of the Court of Federal Claims ("RCFC") may apply to the extent they are consistent with the Vaccine Rules. Vaccine Rule 1(c).

The Court of Federal Claims has found that at a hearing, when an expert attempts to present an opinion not disclosed before the hearing, the opposing party may seek to strike that testimony.[56] Childers v. United States, 116 Fed. Cl. 486, 596-99 (2013) (granting motion to strike testimony). Under RCFC 26(a)(2)(B)(i), an expert report must contain "a complete statement of all opinions the witness will express and the basis and reasons for them." The Court of Federal Claims explained "expert reports must be 'detailed and complete'" and "[a] complete report must include the substance of the testimony which an expert is expected to give on direct examination together with the reasons therefor." Id. at 597 (quoting Salgado v. Gen. Motors Corp., 150 F.3d 735, 741 n.6 (7th Cir. 1998)); see Fed. R. Civ. P. 26 Advisory Committee's note. Additionally, "[t]he report must be complete such that opposing counsel is not forced to depose an expert in order to avoid ambush at trial." Id. (quoting Salgado, 150 F.3d at 741 n.6).

According to RCFC 37(c)(1), "[i]f a party fails to provide information or identify a witness . . . , the party is not allowed to use that information or witness to supply evidence on a motion, at a hearing, or at a trial, unless the failure was substantially justified or is harmless." Courts applying the Federal Rules of Civil Procedure counterparts[57] have found exclusion mandatory "unless the offending party can show that its violation of Fed. R. Civ. P. 26(a) was either justified or harmless." Childers, 116 Fed. Cl. at 598 (quoting Scott Timber, Inc. v. United States, 93 Fed. Cl. 221, 226 (2010)).

In Vaccine Program cases, however, "exclusion from the record is an exceptional remedy, and should only be applied by the Court where the material sought to be excluded is so unreliable, it patently forfeits every trace of being helpful to the Court's consideration of the facts of the case." Veryzer v. Sec'y of Health & Hum. Servs., No. 06-522V, 2010 WL 2507791, at *21 (Fed. Cl. Spec. Mstr. June 15, 2010). "Advance notice of evidence and theories is particularly important in a highly scientific or technical context such as the Vaccine Program, where an adequate response to new information may require additional scientific research and

[56] The Federal Circuit has noted a "special master can order the experts to confine their testimony to the issues addressed in their reports." Simanksi v. Sec'y of Health & Hum. Servs., 671 F.3d 1368, 1382 (Fed. Cir. 2012).

[57] RCFC 37(c)(1) is identical in relevant part to Rule 37(c)(1) of Federal Rules of Civil Procedure.

evaluation." Sumner v. Sec'y of Health v. Hum. Servs., No. 99-946V, 2015 WL 5173644, at *16 (Fed. Cl. Spec. Mstr. Aug. 13, 2015). Yet, "[t]he Vaccine Rules favor broad inclusion, and 'the probative value of the evidence or the credibility of the witnesses . . . are matters within the purview of the fact finder.'" R.K. v. Sec'y of Health v. Hum. Servs., No. 03-0632V, 2015 WL 10911950, at *36 (Fed. Cl. Spec. Mstr. May 23, 2016) (quoting Munn v. Sec'y of Health v. Hum. Servs., 970 F.2d 863, 871 (Fed. Cir. 1992)), mot. for rev. denied, 125 Fed. Cl. 57 (2016), aff'd mem., 671 F. App'x 792 (Fed. Cir. 2016).

C. Analysis

The undersigned finds it is not necessary to strike Dr. Chaudhry's testimony. After considering all of the evidence in the record, including the testimony petitioner seeks to strike, as well as all medical records, expert reports, medical literature, and testimony, the undersigned finds petitioner entitled to compensation, as described in detail below. Thus, petitioner is not prejudiced by this testimony. Further, any issues with the testimony in question has been addressed by the undersigned in the assignment of the weight she afforded the testimony rather than exclude it in its entirety. Pursuant to the Vaccine Rules and statute, which favor broad inclusion and principles of fundamental fairness to both parties, the undersigned **DENIES** petitioner's motion to strike.

V. CAUSATION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). The petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, petitioner must prove 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 & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The

received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the respondent, the special master may consider the evidence presented by the respondent in determining whether the petitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner's evidence on a requisite element of the petitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Causation

To receive compensation through the Program, petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Because petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received caused his injury. To do so, petitioner must establish, by preponderant evidence: "(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.

The causation theory must relate to the injury alleged. The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting

that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in petitioner's favor).

VI. CAUSATION ANALYSIS

A. Althen Prong One

Under Althen Prong One, petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1375 (Fed. Cir. 2009); Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359 (Fed. Cir. 2019); see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds petitioner has set forth a sound and reliable medical theory to explain how the flu vaccine can cause Bell's palsy for the following reasons.

First, that the innate immune response is initiated after vaccination is well-described in the medical literature and acknowledged by all experts. Petitioner's expert, Dr. Gershwin, and respondent's immunology expert, Dr. Romberg, agreed that cytokines are produced within minutes to hours after vaccination and produce inflammation, and they provided supporting medical literature. Hervé et al., for example, explained that "[r]esident immune cells, mast cells, monocytes[,] and macrophages are activated within minutes of injection and release soluble factors" such as proinflammatory cytokines. Pet. Ex. 55 at 4 fig.2.

Additionally, the immunology experts agreed that this innate immune response can occur in lymph nodes, but disagreed as to which lymph nodes would be involved. Respondent's experts argued a vaccine administered in the deltoid would produce an innate immune response in the axillary or draining lymph nodes, while petitioner's expert argued the innate immune response would result in the activation and trafficking of mononuclear cells that would travel to lymph nodes throughout the body.

For support, Dr. Gershwin cited Hervé et al., who explained that once cytokines are produced, they "act both locally . . . and may act systemically at distant organs." Pet. Ex. 55 at 4 fig.2. Although Dr. Romberg opined only the draining or axillary lymph nodes would be involved when a vaccine is administered in the deltoid, he did not disagree that a local response

initiated by a vaccine could travel and produce a response in the cranial nerves. He explained that "any vaccine component or inflammatory mediator produced at the injection site or in a draining lymph node" would need to "pass[] through a network of lymphatic vessels, then into venous circulation, [and] then systemic arterial circulation to be disseminated to all vascularized tissues including facial nerves." Resp. Ex. LL at 2; see also Tr. 218-19. Additionally, Dr. Romberg noted "some cases of Bell's palsy are likely caused by an inflammatory mechanism." Resp. Ex. S at 5.

Second, the experts all agree that herpes virus has been suspected to cause Bell's palsy. Dr. Gershwin opined that his proposed mechanism is similar to the mechanism thought to occur in Bell's palsy cases caused by viral infections such as herpes, in that both lead to inflammation and compression of the seventh cranial nerve. In describing the mechanism thought to be at play in Bell's palsy cases caused by reactivation of herpes, Dr. Chaudhry testified that the herpes mechanism involves inflammation of the seventh cranial nerve and cited Reich who confirmed that "inflammation of the nerve" and "compression resulting in paralysis" occurs. Resp. Ex. Q at 5. Likewise, Dr. Romberg explained that with reactivation of herpes, inflammation occurs and causes the nerve to swell. Thus, the mechanism proposed by Dr. Gershwin is recognized and accepted as it relates to a viral infection.

Third, the relevant anatomy of the facial nerve as it passes through the bony fallopian canal has been implicated as playing a causal role in the medical literature. Reich noted "[e]dema of the facial nerve within the narrow fallopian canal has been observed" in Bell's palsy. Resp. Ex. Q at 6-7. He explained the facial canal is "a narrow bony canal within the temporal bone" and the "little room for expansion" leads to "inflammation of the nerve" and "compression resulting in paralysis." Id. at 5.

Petitioner's theory in this case is similar to that in Beraki v. Secretary of Health & Human Services, No. 17-243V, 2021 WL 4891119 (Fed. Cl. Spec. Mstr. Sept. 20, 2021). In Beraki, petitioner's expert opined that the innate immune system's Toll-Like Receptor system "release[s] proinflammatory cytokines following vaccination, and describe[d] the process as one that 'mimics the response to natural infection.'" Beraki, 2021 WL 4891119 at *15. Petitioner's expert noted "the relevant anatomy and the vulnerability of the facial nerve . . . has been implicated as playing a causal role in the medical literature," and cited literature discussing the inflammatory process thought to take place. Id. In that case, the undersigned found petitioner's theory sound and reliable. Id. at *15-16.

Lastly, studies have discussed the flu vaccine as a cause of Bell's palsy. Dr. Gershwin cited four articles that acknowledged a signal or increased risk of Bell's palsy after administration of the flu vaccination. Zhou et al. concluded there "may be a signal of possible association between [flu] vaccines and an increased risk of Bell's palsy." Pet. Ex. 21 at 5. Kamath et al. found "the likelihood of reporting facial paralysis following [flu] vaccination [was] higher compared with other vaccines." Pet. Ex. 47 at 4. Bardage et al. found "a significantly increased risk for Bell's palsy" in "those vaccinated in the early phase of the vaccination campaign (≤ 45 days), when high risk groups predominated." Pet. Ex. 35 at 4. And Huang et al.concluded "[t]here was an increased risk for Bell's palsy in the interval 0-42 days after vaccination." Pet. Ex. 54 at 3. While the authors of these studies did not reach any conclusions

as to the pathogenesis of Bell's palsy, some hypothesized an immune inflammatory response mechanism to be at play. See Pet. Ex. 21 at 5; Pet. Ex. 47 at 4. Additionally, studies cited by respondent's experts noted that inflammation is thought to play a part in the development of Bell's palsy. See, e.g., Resp. Ex. M at 2.

The lack of supportive epidemiological evidence is not dispositive. It is difficult to use epidemiology to determine whether a vaccine is implicated in causation. Moreover, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." Andreu, 569 F.3d at 1378 (quoting Capizzano, 440 F.3d at 1325-26); see also Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in petitioner's favor).

Petitioner's causal theory combines a sound and reliable mechanism of innate inflammatory response (like that which may occur with infection) with the known anatomical vulnerability of the facial nerve to inflammation in the fallopian canal. For these reasons, the undersigned finds that petitioner has provided preponderant evidence of a sound and reliable causal theory, satisfying Althen Prong One.

B. Althen Prong Two

Under Althen Prong Two, petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical 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 trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). The petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In regard to Althen Prong Two, the undersigned finds petitioner provided preponderant evidence of a logical sequence of cause and effect showing that his vaccination was the cause of his Bell's palsy.

First, the undersigned agrees with the experts and finds petitioner's medical records show that his clinical course is consistent with Bell's palsy. On November 3, 2015, petitioner received a flu vaccine in his left arm. Two days later, on November 5, 2015, petitioner presented to Dr. Depner for right-sided facial numbness that began the prior afternoon. Dr. Depner's assessment was Bell's palsy. He prescribed an anti-inflammatory as well as an anti-viral.

Next, the undersigned finds petitioner's clinical course is consistent with the proposed causal mechanism. To summarize, Dr. Gershwin explained that an innate immune response began "almost immediately" after vaccination. Pet. Ex. 6 at 2. "[T]he inflammation produced by innate immune cells within the lymph nodes [of petitioner's] facial area . . . transported lymphocytes into that local tissue environment, . . . produc[ing] [petitioner's] clinical symptoms of Bell's palsy." Tr. 14; see also Pet. Ex. 27 at 1. More specifically, "the lymph nodes and the innate immune system that are found surrounding the facial nerve and, particularly, in the region from the internal acoustic meatus to the stylomastoid foramen became acutely inflamed, leading directly to compression." Pet. Ex. 37 at 2.

Dr. Depner's physical examination on November 5, 2015 did not document any signs and symptoms of inflammation at the site of vaccination, and the experts agreed. Dr. Gershwin explained "that the absence of a local swelling at the site of injection may not correlate with the development of lymphocytic inflammation or swelling elsewhere." Tr. 24.

Although Dr. Depner's physical examination on November 5, 2015 did not note any signs of swollen lymph nodes, Dr. Gershwin opined that given petitioner's body stature, it is not unusual for there to be an absence of records noting swollen parotid glands or swollen lymph nodes. He suspected that petitioner "likely had more lymph node tissue on his right side where his Bell's palsy was located." Pet. Ex. 37 at 2. He opined that "more likely than not, [petitioner's] lymph nodes were probably already enlarged [] from his obesity, superimposed on his sleep apnea and his chronic nasal obstruction and allergic rhinitis." Tr. 14. These factors, Dr. Gershwin opined, did not directly cause petitioner's Bell's palsy, but predisposed petitioner to the condition.

Further, the undersigned is not persuaded by respondent's argument of an alternative cause. Dr. Chaudhry, respondent's expert, opined that petitioner's Bell's palsy was more likely than not caused by a herpes virus. Dr. Chaudhry supported this finding with the fact that petitioner improved on antivirals and his clinical course was consistent with what would happen with a viral Bell's palsy. Dr. Romberg also found Dr. Depner's medication regimen for petitioner "suggest[ed] . . . that Dr. Depner, from an etiological perspective, did highly suspect a herpes virus as a contributor or cause of [petitioner's] Bell's palsy." Tr. 245.

However, Dr. Chaudhry conceded that it is possible that petitioner could have improved on his own. Additionally, Dr. Depner prescribed both an anti-inflammatory and an anti-viral. There is no evidence to support Dr. Chaudhry's contention that petitioner improved on the anti-viral medication, given he was also taking an anti-inflammatory medication at the same time. Respondent also provided no evidence that petitioner ever had the herpes virus, a necessary requirement for the latent herpes infection to be reactivated, or that he had the virus at the time of his Bell's palsy. Nor did any treating physician opine that petitioner's Bell's palsy was caused

by a herpes virus. In fact, Dr. Depner, petitioner's treating physician, stated "[i]t is conceivable that the flu vaccine precipitated [petitioner's Bell's palsy]." Pet. Ex. 26 at 19.

Dr. Romberg argued there is no proof "[petitioner's] right facial nerve was actually damaged by inflammation" because there was no "facial nerve specimen from [petitioner], nor any specific laboratory or imaging data suggesting an excessive inflammatory response." Resp. Ex. LL at 1-2. However, no such testing was done. Further, requiring such proof would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program"). Petitioner's burden of proof is by a preponderance of the evidence, and "petitioner need not make a specific type of evidentiary showing." Capizzano, 440 F.3d at 1325.

Therefore, the undersigned finds there is no evidence to support respondent's position that a herpes viral infection was more likely than not the cause of petitioner's Bell's palsy. The undersigned further finds the evidence does not support a finding of any alternative cause other than vaccination in this case.

Thus, the undersigned finds that petitioner provided preponderant evidence of a logical sequence of cause and effect, satisfying Althen Prong Two.

C. Althen Prong Three

Althen Prong Three requires petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disease's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1239, 1243 (Fed. Cir. 2014); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 542 (2011), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

The parties stipulated, and the experts agree, that petitioner received a flu vaccine on November 3, 2015, and one day later, on November 4, 2015, he developed Bell's palsy. The experts also agree that the innate immune system is activated within minutes to hours following vaccination and cited medical literature supporting cytokine release during this time. This timeframe is appropriate given the petitioner's causal theory of inflammation leading to injury of the facial nerve. Therefore, petitioner has provided preponderant evidence satisfying Althen Prong Three.

VII. CONCLUSION

For the reasons discussed above, the undersigned finds that petitioner has established by preponderant evidence that his flu vaccine caused his Bell's Palsy. Therefore, petitioner is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master