In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 18-1735V
Filed: February 9, 2024

W.G., Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Special Master Horner

William E. Cochran, Jr., Black McLaren, et al., PC, Memphis TN, for petitioner.
Bridget Corridon, U.S. Department of Justice, Washington, DC, for respondent.

Decision[1]

On November 8, 2018, petitioner, W.G., filed a petition under the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa-10, *et seq.* (2018),[2] alleging that the influenza ("flu") vaccine and hepatitis B ("hep B") vaccines he received on September 19, 2017 caused him to suffer "Bell's palsy, hearing loss and neuropathy." (ECF No. 1.) For the reasons set forth below, I conclude that petitioner is *not* entitled to compensation.

I. Applicable Statutory Scheme

Under the National Vaccine Injury Compensation Program, compensation awards are made to individuals who have suffered injuries after receiving vaccines. In general, to gain an award, a petitioner must make a number of factual demonstrations,

[1] When this decision was originally filed the undersigned advised his intent to post it on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). In accordance with Vaccine Rule 18(b), petitioner filed a timely motion to redact certain information. This decision is being reissued with petitioner's name reduced to initials. Except for those changes and this footnote, no other substantive changes have been made. This decision will be posted on the court's website with no further opportunity to move for redaction.

[2] All references to "§ 300aa" below refer to the relevant section of the Vaccine Act at 42 U.S.C. § 300aa-10-34.

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

In many cases, however, the vaccine recipient may have suffered an injury *not* of the type covered in the Vaccine Injury Table. In such instances, an alternative means exists to demonstrate entitlement to a Program award. That is, the petitioner may gain an award by showing that the recipient's injury was "caused-in-fact" by the vaccination in question. § 300aa-13(a)(1)(B); § 300aa-11(c)(1)(C)(ii). In such a situation, of course, the presumptions available under the Vaccine Injury Table are inoperative. The burden is on the petitioner to introduce evidence demonstrating that the vaccination actually caused the injury in question. *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *Hines v. Sec'y of Health & Human Servs.*, 940 F.2d 1518, 1525 (Fed. Cir. 1991). In this case, petitioner has alleged that the flu vaccine and the hep B vaccine caused him to suffer Bell's palsy, hearing loss, and neuropathy. (ECF No. 1.) Because these conditions are not listed on the Vaccine Injury Table relative to the flu vaccine or the hep B vaccine, petitioner must establish that his injury was "caused-in-fact" by his vaccination(s).

The showing of "causation-in-fact" must satisfy the "preponderance of the evidence" standard, the same standard ordinarily used in tort litigation. § 300aa-13(a)(1)(A); *see also Althen*, 418 F.3d at 1279; *Hines*, 940 F.2d at 1525. Under that standard, the petitioner must show that it is "more probable than not" that the vaccination was the cause of the injury. *Althen*, 418 F.3d at 1279. The petitioner need not show that the vaccination was the sole cause but must demonstrate that the vaccination was at least a "substantial factor" in causing the condition, and was a "but for" cause. *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999). Thus, the petitioner must supply "proof of a logical sequence of cause and effect showing that the vaccination was the reason for the injury[,]" with the logical sequence being supported by "reputable medical or scientific explanation, *i.e.*, evidence in the form of scientific studies or expert medical testimony." *Althen*, 418 F.3d at 1278; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner may not receive a Vaccine Program award based solely on his or her assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. § 300aa-13(a)(1).

In what has become the predominant framing of this burden of proof, the *Althen* court described the "causation-in-fact" standard, as follows:

> Concisely stated, Althen's burden is to show by preponderant evidence that the vaccination brought about her injury by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury. If Althen satisfies this burden, she is "entitled to recover unless the [government] shows, also by a preponderance of the evidence, that the injury was in fact caused by factors unrelated to the vaccine."

Althen, 418 F.3d at 1278 (citations omitted). The *Althen* court noted that a petitioner need not necessarily supply evidence from medical literature supporting petitioner's causation contention, so long as the petitioner supplies the medical opinion of an expert. *Id*. at 1279-80. The court indicated that, in finding causation, a Program fact-finder may rely upon "circumstantial evidence," which the court found to be consistent with the "system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants." *Id*. at 1280.

As with a Table Injury, respondent may ultimately prove petitioner's injury to be due to a factor unrelated to vaccination even if petitioner meets his initial burden of proof. § 300aa-13(a)(1)(B). Respondent bears the burden of demonstrating the presence of any alternative cause by preponderant evidence only if petitioner satisfies his *prima facie* burden. § 300aa-13(a)(1)(B); *Walther v. Sec'y of Health & Human Servs*., 485 F.3d 1146, 1150 (Fed. Cir. 2007). However, respondent may also present evidence relating to an alternative cause to demonstrate the inadequacy of petitioner's evidence supporting his case in chief. Nonetheless, petitioner does not bear the burden of eliminating alternative causes where the other evidence on causation is sufficient to establish a *prima facie* case under *Althen*. *de Bazan v. Sec'y of Health & Human Servs*., 539 F.3d 1347, 1352-53 (Fed. Cir. 2008); *Walther*, 485 F.3d at 1150.

II. Procedural History

Initially, this case was assigned to Special Master Christian Moran. (ECF No. 4.) Petitioner filed medical records, an affidavit, and a statement of completion, in December of 2018. (ECF Nos. 9-10; Exs. 1-3.) Petitioner later filed an additional affidavit on in January of 2019. (ECF No. 13; Ex. 4.) The case was reassigned to the undersigned on August 27, 2019, while still awaiting respondent's medical review. (ECF Nos. 22-24.) Thereafter, petitioner filed additional evidence, including an immunization authorization form and additional medical records. (ECF Nos. 28, 32; Exs. 5-6.) Petitioner filed an expert report and supporting medical literature on April 20, 2020. (ECF No. 33; Exs. 7-62.)

Respondent filed his Rule 4(c) Report, as well as his own expert report and supporting medical literature, on September 29, 2020. (ECF Nos. 38-40; Exs. A through A, Tab 4.) Respondent contended that this case is not appropriate for compensation both because "the report provided by [petitioner's expert] fails to meet petitioner's burden to demonstrate vaccine causation" and "petitioner's diabetes alone, is more likely than not the cause of the Bell's palsy." (ECF No. 38, pp. 2, 13, 14.) Petitioner filed a responsive expert report and supporting medical literature on March 11, 2021. (ECF No. 45; Exs. 63-68.) Respondent filed a supplemental expert report on July 29, 2021. (ECF No. 49; Ex. C.) After receiving preliminary guidance pursuant to Vaccine Rule 5 (*see* ECF No. 50), petitioner filed a further expert report and supporting medical literature in January of 2022. (ECF No. 52; Exs. 69-70.)

A one-day fact hearing was held remotely on August 25, 2022. (*See* ECF No. 57; Transcript of Proceedings ("Tr"), filed Sept. 19. 2022, at ECF No. 60.) Thereafter, both parties agreed that the record should be closed. (ECF No. 62.) Petitioner filed a Motion for Ruling on the Record on December 23, 2022. (ECF No. 64.) Respondent responded on January 30, 2023, and additionally requested that petitioner file Walmart pharmacy records. (ECF No. 65, p. 4, n.6.) Petitioner filed a reply and a record from Walmart certifying that there were no additional records to be filed. (ECF Nos. 66-67; Ex. 71.)

Petitioner argues that he was correctly diagnosed as suffering Bell's palsy and that he has demonstrated each of the three *Althen* prongs by preponderant evidence, thereby proving his Bell's palsy to have been caused by his flu and/or hep B vaccines. (ECF No. 64, pp. 6-10.) Petitioner asserts that respondent has failed to present reliable evidence explaining petitioner's condition and that no other antecedent events are evidenced to explain petitioner's Bell's palsy. (*Id*. at 11.) Respondent disputes that petitioner has satisfied any of the three *Althen* prongs. (ECF No. 65, pp. 19-33.) Respondent contends that the burden of proof did not shift to respondent in this case; however, respondent stresses in the context of *Althen* prong two that petitioner's uncontrolled diabetes is the likely cause of petitioner's Bell's palsy. (*Id*. at 30-32.) In reply, petitioner contends that respondent's expert did not actually challenge petitioner's expert on any of the details of his opinion relative to the requirements of *Althen*, but instead only offered petitioner's diabetes as an alternative cause of his condition. (ECF No. 66.)

This case is now ripe for resolution. I have concluded that the parties have had a full and fair opportunity to develop the record and that it is appropriate to resolve this case without an entitlement hearing. S*ee Kreizenbeck v. Sec'y of Health & Human Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020) (citing *Simanski v. Sec'y of Health & Human Servs.*, 671 F.3d 1368, 1385 (Fed. Cir. 2012); *Jay v. Sec'y of Health & Human Servs.*, 998 F.2d 979, 983 (Fed. Cir. 1993)); *see also* Vaccine Rule 8(d); Vaccine Rule 3(b)(2).

III. Factual History

a. As reflected in the medical records

i. Pre-vaccination

Petitioner was diagnosed with type 2 diabetes prior to 2014. (Tr. 8-9, 35.) The medical records reflect that, throughout 2014, petitioner's diabetes was uncontrolled and, as a result, he suffered several injuries, including osteomyelitis in his right toe, diabetic peripheral vascular disease, peripheral neuropathy, hypertension, benign hyperkeratotic lesions, and diabetic retinopathy. (Ex. 2, pp. 6-153, 735-60, 813-19.)

After petitioner's appointment on August 19, 2014, there is a significant gap in treatment until August 22, 2017, when he saw optometrist Dr. Haleh Shafa. (Ex. 2, pp. 136-53, 157-84.) During these three years, petitioner was taking "Insulin 70/30 25 units in the mornings and 17 units in the evenings." (*Id.* at 162.) At this appointment, petitioner's glucose was 134 mg/dL (reference range 70-140 mg/dL) and his hemoglobin A1C was high at 11.0% (reference range 4.8-5.6%). (*Id.* at 169-76.) Additionally, petitioner's cholesterol was high at 239 mg/dL (reference range 0-199 mg/dL). (*Id.* at 172, 766.) During this appointment, petitioner received a pneumococcal vaccination, as well as the first dose of a three-part hep B vaccine series. (*Id.* at 161, 182.) Additionally, at this appointment, petitioner underwent retinal imaging screening for diabetic retinopathy, which found minimal diabetic retinopathy in petitioner's right eye. (*Id.* at 168-69.)

Petitioner's next appointment was on September 14, 2017, where he was seen by podiatrist Dr. Lisa B. Michaels. (Ex. 2, pp. 188-99.) During this appointment, petitioner's chief complaint was "an open lesion" on his left foot, and it was noted that petitioner had a history of "peripheral neuropathy." (*Id.* at 189.) Dr. Michaels recorded a history of "uncontrolled" type 2 diabetes and diagnosed petitioner with a "diabetic neuropathic ulceration." (*Id.* at 189, 191.) During this appointment, Dr. Michaels performed a selective wound debridement and removed the devitalized tissue from petitioner's foot. (*Id.* at 191.) This tissue was tested, and the results revealed a staphylococcus infection. (*Id.* at 194-95, 770.) Additionally, petitioner underwent an x-ray of this foot, which found "no fracture dislocation or osseous destruction." (*Id.* at 193, 819.) At this appointment, petitioner's glucose was high at 213 mg/dL (reference range 70-140 mg/dL). (*Id.* at 194, 773.) Additionally, petitioner's urine contained high levels of microalbumin at 47.3 mg/dL (reference range 0.0-2.0 mg/dL) and albumin at 201.5 mg/gm (reference range 0.0-30.0 mg/gm). (*Id.* at 771.)

On September 15, 2017, petitioner returned to Dr. Shafa for a diabetic eye exam. (Ex. 2, pp. 203-14.) During this appointment, petitioner was diagnosed with bilateral moderate nonproliferative retinopathy, as well as amblyopia and low visual impairment of the left eye. (*Id.* at 211.)

ii. Vaccination and subsequent course

On September 19, 2017, petitioner saw Dr. Delphanie D. Head for a routine follow up. (Ex. 2, pp. 218-38.) During this appointment, petitioner received the flu and hep B vaccines that are at issue in this case. (*Id.* at 224-26, 236; Ex. 5, p. 6.) Petitioner underwent a right upper quadrant ultrasound during this appointment, which found no abnormalities. (Ex. 2, p. 225.) Petitioner's glucose was high at 216 mg/dL (reference range 70-140 mg/dL), his microalbumin was high at 45.3 mg/dL (reference range 0.0-2.0 mg/dL), and his albumin was high at 251.5 (reference range 0.0-30.0 mg/gm). (*Id.* at 226-28, 774, 781.) Petitioner also had a liver function test, which revealed that petitioner's ALT was high at 95 IU/L (reference range of 9-33 IU/L) and his AST was high at 90 IU/L (reference range 12-41 IU/L). (Id. at 228, 779-80.)

Petitioner saw Dr. Rae A. King on September 26, 2017, for an eye exam. (Ex. 2, pp. 242-52.) Dr. King noted that petitioner's diagnoses included mild diabetic retinopathy, childhood left eye trauma, left eye aphakia, and a cataract in his right eye. (*Id.* at 247.) On September 29, 2017, petitioner underwent an ultrasound of his right upper quadrant that was determined to be unremarkable. (*Id.* at 820-21.) Petitioner returned to Dr. Michaels on October 3, 2017, for a follow up appointment for the ulcer on his left foot. (*Id.* at 256-62.) During this appointment, petitioner underwent another selective wound debridement to remove devitalized tissue on his left foot. (*Id.* at 259.)

On October 14, 2017, petitioner presented to urgent care with complaints of facial droop on the right side; however, the physician observed that petitioner's facial droop was on the left, rather than the right. (Ex. 2, pp. 266-88, 733-34.) Patient underwent an electrocardiogram ("EKG"), which was normal; a computed tomography ("CT") scan, which was unremarkable; and a magnetic resonance imaging ("MRI"), which showed no acute ischemic event, no enhancing intracranial mass, and mild non-enhancing white matter disease of the brain. (*Id.* at 270-73, 822-24.) At this appointment, petitioner's glucose was high at 129 mg/dL (reference range 70-99 mg/dL) and his ALT and AST were high at 82 IU/L (reference range 9-33 IU/L) and 77 IU/L (reference range 12-41 IU/L), respectively. (*Id.* at 271-72, 782, 784.) Petitioner's differential diagnosis at this appointment was between a cerebrovascular accident, transient ischemic attack, Bell's palsy, and electrolyte abnormalities. (*Id.* at 269.) Petitioner was prescribed 20 mg of prednisone and 400 mg of acyclovir. (*Id.* at 268.)

Petitioner saw Dr. Head on October 17, 2017, for a follow up evaluation for Bell's palsy. (Ex. 2, pp. 292-306.) Petitioner described his symptoms as including light sensitivity in his left eye, an inability to close/bling his left eye, and a protrusion of the left eye, as well as slurred speech and twitching/paralysis of the left side of the face. (*Id.* at 293.) Dr. Head decreased petitioner's dosage of prednisone to 10 mg. (*Id.* at 297.) During this appointment, petitioner's glucose was high at 155 mg/dL (reference range 70-140 mg/dL) and his hemoglobin A1C was high at 8.90% (reference range => 6.99%). (*Id.* at 300-02, 786-89.)

On October 24, 2017, petitioner had a follow up appointment with his ophthalmologist, Dr. King. (Ex. 2, pp. 320-29.) Dr. King noted that petitioner was experiencing left eye foreign bodies, redness, pain, and light sensitivity with some tearing. (*Id.* at 321.) Petitioner saw his podiatrist, Dr. Michaels, on October 31, 2017, for a follow up regarding the ulcer on his left foot. (*Id.* at 333-40.) Petitioner underwent another selective wound debridement to remove devitalized tissue on his foot. (*Id.* at 336.)

On November 2, 2017, petitioner saw Dr. Shafa for a follow up regarding his eye pain. (Ex. 2, pp. 344-54.) He reported feeling a foreign body sensation and upper lid swelling. (*Id.* at 345.) Dr. Shafa noted that petitioner had been diagnosed with Bell's palsy in his left eye and has been unable to fully close his left eye. (*Id.*) Petitioner's left eye exam revealed a "capped glands upper lid, swollen lid, internal chalazion," an exposed cornea when blinking, a scar over his cornea, lens fragments attached to iris, vitreous syneresis, and hazy view. (*Id.* at 348-49.) Petitioner saw Dr. Shafa again on November 9, 2017, for a "follow up on chalazion." (*Id.* at 359.) Dr. Shafa again noted that petitioner had recently been diagnosed with Bell's palsy in his left eye. (*Id.*) At this encounter, petitioner was assessed as suffering from Bell's palsy with exposure keratopathy in his left eye. (*Id.* at 362-63.) He was instructed to continue using Systane drops and Genteal nighttime ointment. (*Id.*)

Petitioner began seeing neurologist Dr. Joseph A. Wapenski on November 13, 2017, "for left facial paralysis" and Bell's palsy. (Ex. 2, pp. 371-78.) Dr. Wapenski notes that petitioner's facial paralysis began on October 14, 2017. (*Id.* at 371.) He describes petitioner's paralysis as "moderate" with "some drooling left corner of mouth and left eye irritation and altered taste." (*Id.*) He noted that petitioner underwent a CT scan, which was normal, and an MRI, which showed small vessel white matter disease. (*Id.* at 371-72.) Dr. Wapenski acknowledged the fact of petitioner's flu and hep B vaccinations on September 19, 2017, but also noted petitioner's history of uncontrolled diabetes. (*Id.*) He noted that petitioner was treated with prednisone and acyclovir. (*Id.* at 371.) Petitioner reported experiencing temporary paresthesia and weakness of his left leg, as well as elevated glucose levels after starting steroids. (*Id.* at 372.) During petitioner's examination, Dr. Wapenski noted that petitioner's face showed "asymmetry with weakness of the face drooling left corner of mouth," and that petitioner could "voluntarily close and cover entire left eye with eyelid." (*Id.* at 375.) Petitioner was assessed as having left-side Bell's palsy and a resolved transient lower left leg radiculopathy. (*Id*.)

On November 14, 2017, petitioner saw Dr. Michaels for another follow up for the ulcer on his left foot. (*Id.* at 382-88.) At this appointment, petitioner's diagnosis was ulcer of the left toe and type 2 diabetes with neurological manifestation. (*Id.* at 385.) Petitioner underwent another selective wound debridement and the devitalized tissue was removed. (*Id.*)

On November 17, 2017, petitioner began seeing an acupuncturist to treat his Bell's palsy. (Ex. 3, p. 1.) The acupuncturist noted that petitioner reported symptoms "2 weeks after flu shot" and noted that petitioner experienced light sensitivity and dry eyes.

(*Id.*) The acupuncturist recommended attending appointments two times a week for four weeks. (*Id.*) Petitioner thereafter attended additional acupuncture appointments on November 18, November 20, and December 11, 2017. (*Id.* at 1-2.)

Petitioner had another follow up appointment with Dr. Michaels on November 28, 2017. (Ex. 2, pp. 392-98.) Petitioner underwent another selective wound debridement to remove devitalized tissue. (*Id.* at 394-95.) Petitioner's primary care physician, Dr. Head, saw him for a routine follow up the next day on November 29, 2017. (*Id.* at 402-17.) At this appointment, petitioner's glucose was within the normal range at 124 mg/dL (reference range 70-140 mg/dL); however, his hemoglobin A1C was high at 9.30% (reference range <=6.99%). (*Id.* at 410-11, 790-91.) Dr. Head directed petitioner to follow up with his neurologist and ophthalmologist for his left Bell's palsy. (*Id.* at 407.)

Petitioner followed up with his optometrist, Dr. Shafa, on December 7, 2017, for his keratitis and Bell's palsy. (Ex. 2, pp. 421-32.) Dr. Shafa instructed petitioner to continue using Systane drops and Genteal nighttime ointment. (*Id.* at 427.) Additionally, Dr. Shafa noted that petitioner was experiencing "mild iritis" in his left eye, and she was unsure of whether it was related to his Bell's palsy. (*Id.*) Petitioner was prescribed Pred Forte eye drops. (*Id.*) On December 12, 2017, petitioner saw Dr. Michaels for another follow up regarding the ulcer on his left foot. (*Id.* at 436-41.) At this appointment, Dr. Michaels determined that petitioner's ulcer was healed. (*Id.* at 438.)

On January 5, 2018, petitioner continued his care with Dr. Shafa. (Ex. 2, pp. 445-56.) Dr. Shafa noted that petitioner's iritis had resolved, but continued to recommend petitioner use Systane drops and Genteal nighttime ointment to treat Bell's palsy. (*Id.* at 451.) Petitioner's left eye exam revealed an exposed cornea, a scar across the cornea, lens fragments, and a hazy view. (*Id.* at 449-50.)

Petitioner saw his neurologist, Dr. Wapenski, on January 8, 2018 for another follow up regarding his Bell's palsy. (Ex. 2, pp. 460-66.) Dr. Wapenski described petitioner's "mild left face residua" as "much better," and noted that he continued to suffer from "left eye exposure keratitis." (*Id.* at 461.) Dr. Wapenski's impression described petitioner's condition as "Neuropathy diabetic," "Resolving Bell's Left," and "Old left eye injury." (*Id.* at 462.) Additionally, petitioner noted distorted hearing in his left ear. (*Id.*) On January 10, 2018, petitioner saw Dr. Head for a routine follow up. (*Id.* at 470-85.) At this appointment, petitioner's hemoglobin A1C was high at 8.80% (reference range <=6.99%), and his ALT and AST were high at 49 IU/L (reference range 9-33 IU/L) and 51 IU/L (reference range 12-41 IU/L), respectively. (*Id.* at 476-77, 794, 796-97.) Petitioner saw Dr. Michaels on January 29, 2018, for a follow up regarding the ulcer on his left foot. (*Id.* at 489-95.) Petitioner's ulcer remained healed. (*Id.* at 492.)

On February 5, 2018, petitioner saw Dr. Wapenski again. (Ex. 2, pp. 496-508.) Dr. Wapenski noted that petitioner's Bell's palsy "is showing very good recovery" and "[h]e can almost completely cover his left eye." (*Id.* at 500.) Petitioner was still

experiencing "mild weakness of the left face," "left ear hearing distortion," and "paresthesia of the chest," left abdomen, and left leg. (*Id.*)

Petitioner saw his primary care physician on February 12, 2018, for a routine follow up. (Ex. 2, pp. 512-23.) During this visit, petitioner's glucose was high at 194 mg/dL (reference range 70-140 mg/dL), and his hemoglobin A1C was high at 8.80% (reference range <=6.99%). (*Id.* at 517-19, 798-99.)

On February 16, 2018, petitioner saw Dr. Dennis L. Frew regarding "hearing issues in [his] left ear." (Ex. 2, pp. 527-32.) Dr. Frew noted that petitioner "presents for left sided Bell's palsy with onset October 2017 and treated with steroids and has a past medical history of [uncontrolled type 2 diabetes, hypertension, hyperlipidemia, left aphakia, left Bell's palsy, and left eye trauma]." (*Id.* at 527-28.) Petitioner underwent a CT, which came back as normal. (*Id.* at 529-30, 825-26.) Roughly three weeks later, on March 8, 2018, petitioner saw Dr. Frew again for a follow up on his audio exams. (*Id.* at 536-44.) Dr. Frew noted that petitioner had "left moderate sensorineural hearing loss." (*Id.* at 536.) Thereafter, petitioner underwent a brain MRI that revealed "[n]o significant abnormality . . . in either facial nerve," and "[n]o areas of abnormal enhancement in either IAC." (*Id.* at 540, 827-28.)

On March 12, 2018, petitioner had an appointment with ophthalmologist Dr. Seongmu Lee for an eyelid evaluation. (Ex. 2, pp. 548-58.) Dr. Lee found that petitioner's facial function was improving and recommended "aggressive ocular surface lubrication." (*Id.* at 554.) Additionally, Dr. Lee discussed implanting gold weights or tarsorrhaphy surgery to close petitioner's eyelid if his symptoms got worse. (*Id.*) Petitioner had a follow up appointment with Dr. Michaels for the ulcer on his left foot. (*Id.* at 562-69.) Dr. Michaels noted that petitioner's ulcer remained healed. (*Id.* at 563.)

On April 3, 2018, petitioner had a follow up appointment with Dr. Head. (Ex. 2, pp. 573-86.) Petitioner's glucose was slightly elevated at 144 mg/dL (reference range 70-140 mg/dL), his hemoglobin A1C was high at 8.80% (reference range <=6.99%), his cholesterol was high at 206 mg/dL (reference range 0-199 mg/dL), and his ALT and AST were high at 45 IU/L (reference range 9-33 IU/L) and 55 IU/L (reference range 12-41 IU/L), respectively. (*Id.* at 579-82, 803-10.) On April 16, 2018, petitioner presented to Dr. Michaels for a follow up regarding the ulcer on his left foot. (*Id.* at 590-97.) There were no significant changes during this appointment. (*Id.*)

On May 21, 2018, petitioner had a follow up appointment with Dr. Lee regarding his eyelid. (Ex. 2, pp. 601-10.) Petitioner reported "dryness and tearing on/off." (*Id.* at 602.) Dr. Lee again noted that petitioner should continue "aggressive ocular surface lubrication," and discussed the surgical options to help close petitioner's eyelid. (*Id.* at 606.) Petitioner "elect[ed] [the] nonsurgical plan." (*Id.*) On May 24, 2018, petitioner had a follow up appointment with his neurologist, Dr. Wapenski, for paresthesia in his chest and left facial palsy. (*Id.* at 614-27.) After reviewing petitioner's old MRI and CT scan, Dr. Wapenski ordered an MRI of petitioner's neck, as well as an electromyogram and nerve conduction study (EMG/NCV) to documents neuropathy in petitioner's left

extremities. (*Id.* at 619-27.) On June 15, 2018, petitioner underwent an MRI of the cervical spine that showed "left paracentral disc protrusion with minimal effacement of the left ventral aspect of the spinal cord contributing to his mild spinal canal stenosis," and "[d]isc osteophyte complexes which appear predominately osteophytic . . . contributing to mild spinal canal stenosis." (*Id.* at 623-25, 829-30.) Petitioner had a follow up appointment with Dr. Michaels on May 29, 2018, during which it was determined that the ulcer on his left foot remained healed. (*Id.* at 631-38.)

Petitioner saw Dr. Frew on June 22, 2018, for a follow up regarding his Bell's palsy and hearing loss. (Ex. 2, pp. 642-46.) Petitioner noted that he had "no improvement from initial exam." (*Id.* at 642.) Dr. Frew ordered a repeat audio exam and requested that petitioner follow up after the exam. (*Id.*) On June 28, 2018, petitioner saw Dr. Lee for a follow up regarding his left eye. (*Id.* at 650-59.) During this appointment, petitioner's left eye was "very red and irritated." (*Id.* at 651.) Petitioner reported using Genteal gel, which had been working well until that week. (*Id.*) Dr. Lee recommended continuing "aggressive ocular surface lubrication." (*Id.* at 656.) Petitioner was prescribed Maxitrol for his irritation. (*Id.* at 658.) At his follow up appointment on July 9, 2018, petitioner reported that his eye was "[n]o longer irritated." (*Id.* at 664.) On July 11, 2018, petitioner underwent an EMG/NCV that showed "electrophysiological evidence for severe, ongoing and chronic, generalized polyneuropathy with axonal>demyelinating features," as well as "a superimposed left S1 radiculopathy and a mild median neuropathy at the left wrist." (*Id.* at 676-98.)

On July 20, 2018, petitioner saw an ophthalmologist Dr. Essence C. Bell for a follow up regarding his glaucoma. (Ex. 2, pp. 702-13.) Petitioner complained of right eye pain and swelling. (*Id.* at 703.) Petitioner noted that he had been applying Maxitrol, which helped. (*Id.*) Petitioner was diagnosed with an age related cataract and proliferative retinopathy in his right eye. (*Id.* at 707.) Petitioner had a follow up appointment with ophthalmologist Azad Mansouri, M.D., on August 3, 2018, for a diabetic retinal evaluation. (*Id.* at 717-29; Ex. 6, pp. 4-20.) Petitioner was diagnosed with severe nonproliferative diabetic retinopathy. (Ex. 2, p. 723; Ex. 6, p. 14, 46-47.) Petitioner underwent an intravitreal injection of Avastin into his right eye and cataract surgery in his left eye. (Ex. 2, p. 724; Ex. 6, pp. 16, 18.) Additionally, petitioner underwent an ocular coherence tomographic study, which confirmed petitioner's diagnosis. (Ex. 6, pp. 24-26.)

On August 19, 2018, petitioner saw Dr. Head for a diabetic screening. (Ex. 6, pp. 29-31.) Petitioner had a follow up appointment with Dr. Lee on August 20, 2018. (*Id.* at 34-36.) Petitioner had another appointment with Dr. Lee on October 8, 2018. (*Id.* at 83-98.) Petitioner reported feeling a foreign object sensation and dryness in his left eye. (*Id.* at 85.) Dr. Lee recommended petitioner "continue aggressive ocular surface lubrication." (*Id.* at 89.)

Petitioner began participating in a diabetes and hypertension panel management program on October 15, 2018. (Ex 6, pp. 107-10.) At that time, petitioner was overdue

for a hemoglobin A1C test, a foot exam, flu and hep B immunizations, and a microalbumin test. (*Id.* at 109.)

On November 12, 2018, petitioner followed up with Dr. Lee regarding his Bell's palsy. (Ex. 6, pp. 129-44.) Petitioner reported that he noticed numbness and tightness in his face. (*Id.* at 131.) There were no significant changes during this appointment. (*Id.* at 127-44.) Petitioner had a follow up neurology appointment with Dr. Sonal R. Hazariwala on November 15, 2018. (*Id.* at 159-79.) Dr. Hazariwala noted that petitioner had previously been diagnosed and treated for facial tingling and paresthesias and has since developed "numbness along the inside of the jaw/cheek." (*Id.* at 160.) During this appointment, petitioner underwent a magnetic resonance angiography ("MRA") of his head and neck, which showed no acute intracranial abnormality and no cervical abnormality; however, there was evidence of an intracranial aneurysm. (*Id.* at 170.) Dr. Hazariwala diagnosed petitioner with "[l]eft facial numbness" of "unclear etiology." (*Id.* at 167.) He noted that, although petitioner's tightness may be related to his Bell's palsy, the "numbness is concerning." (*Id.*) Additionally, he discussed petitioner's EMG/NCS results and diagnosed petitioner with polyneuropathy. (*Id.*) Petitioner's labs during this appointment showed high microalbumin at 46.0 mg/dL (reference range 0.0-2.0 mg/dL) and high albumin at 384.9 mg/gm (reference range 0.0-30.0 mg/gm). (*Id.* at 189.)

On February 11, 2019, petitioner called to schedule a podiatry appointment regarding a potential ulcer on his left foot. (Ex. 6, pp. 290-92.) He saw Dr. Michaels on February 12, 2019, who confirmed that petitioner had an ulcer on his left foot. (*Id.* at 308-11.) During this appointment, petitioner underwent an x-ray that showed no evidence of osteomyelitis and an excisional wound debridement. (*Id.* at 311-15, 328-29.) At this appointment, petitioner's hemoglobin A1C was high at 10.3% (reference range <=7.0%). (*Id.* at 341-42.) On March 5, 2019, petitioner called his doctor's office and noted that he had an allergic reaction to medication. (*Id.* at 374-81.) Petitioner saw Dr. Michaels the next day to address these side effects. (*Id.* at 385-96.) During this appointment, petitioner underwent a selective wound debridement. (*Id.* at 389.) Petitioner's labs showed that his red blood cell count was low at 3.92 10*6 uL (reference range 4.06-5.69 10*6 uL), his HGB was low at 11.6 g/dL (reference range 13.0-17.0 g/dL), and his hematocrit was low at 35.9% (reference range 38.8-50.0%). (*Id.* at 420.) Based on these labs, petitioner's doctor expressed concern that he was developing an infection. (*Id.* at 406.) Petitioner underwent an MRI of his foot that found no evidence of osteomyelitis or abscess; however, it did find cellulitis. (*Id.* at 426-27, 436-38.) Petitioner saw Dr. Michaels for another follow up appointment on March 15, 2019. (*Id.* at 464-73.) Dr. Michaels noted that petitioner's ulcer had healed. (*Id.* at 467.) Petitioner had two more follow up appointments regarding his foot: one on March 22, 2019, and the other on March 29, 2019. (*Id.* at 502-11, 525-34.) Petitioner's foot remained healed. (*Id.*)

b. As reflected in petitioner's testimony

In addition to submitting two written statements, petitioner testified at a fact hearing on August 25, 2022. (ECF Nos. 59-60; Exs. 1, 4.) He noted that prior to

September 2017, he had never experienced facial paralysis. (Tr. 8.) Though he experienced a gap in treatment from 2014 to 2017, he explained that prior to his vaccination, he had been seeking treatment for diabetes and peripheral neuropathy and saw a doctor regularly. (*Id.* at 9, 34.) Additionally, petitioner testified that he injured his left eye when he was six years old. (*Id.* at 9-10, 52.) This injury has affected his vision in that eye. (*Id.* at 10.) Petitioner also had a toe infection, which petitioner claimed was due to running, but is also a symptom of diabetes. (*Id.* at 33, 47-48, 53-56.) In 2017, petitioner was also diagnosed with hypertension. (*Id.* at 35.)

Petitioner explained that he did not seek treatment from September 2014 through July of 2017. (Tr. 11.) He was unemployed during those years and he could not afford to see a doctor. (*Id.*) During this time, petitioner explained that he managed his diabetes through diet and exercise and monitored his blood sugar using a monitor and urinary strips. (*Id.* at 12, 39-41.) However, when we went to the doctor, his A1C was still high and noted that his diabetes was not under control until he began seeing a doctor in 2017. (*Id.* at 12, 39.)

Petitioner testified that he received the flu and hep B vaccines on September 19, 2017. (Tr. 13.) He testified that he did not notice anything different until around October 19, 2017, when he "woke up that morning and [his] face was twisted." (*Id.* at 13, 50.) He explained that, at that time, he believed he was having a stroke and went to the emergency room. (*Id.* at 13.) In the emergency room, petitioner was diagnosed with Bell's palsy and was prescribed prednisone and acyclovir. (*Id.* at 14-15.) However, his primary care physician decreased his prednisone because it impacted his blood pressure and blood sugar. (*Id.*) At this time, petitioner was taking Lisinopril to help with his blood pressure and kidneys and was only monitoring his glucose levels "to a degree." (*Id.* at 15, 51.)

Petitioner noted that even when he was able to take prednisone and acyclovir, he was still experiencing symptoms of his Bell's palsy. (Tr. 15.) He explained that his symptoms included problems speaking, twisted facial features, light sensitivity, hearing issues, and a tingling sensation on the left side of his face. (*Id.* at 16.) Additionally, petitioner explained that his eye does not close all the way and he often slurs his words and drools. (*Id.* at 17.) He noted that his doctor had recommended surgery on his left eye. (*Id.*) He explained that the Bell's palsy primarily impacted the left side of his face. (*Id.*) Petitioner also testified that he experienced severe pain in his abdomen which was diagnosed as a "neuropathy episode." (*Id.* at 20.)

Petitioner testified that the last time he sought treatment for his Bell's palsy was July 2018. (Tr. 21-22.) He explained that he had not sought treatment since then because he was functioning well and the pandemic made it more difficult to seek care. (*Id.* at 22.) He testified that he wanted to follow up with a neurologist but the soonest appointment was for February 2023. (*Id.*) He explained that he continued to do facial exercises, use ointment on his eye, and massage his face. (*Id.* at 23.)

Petitioner testified that his current symptoms include tearing, facial twitching, sensitivity to sunlight, hearing issues, and drooling. (Tr. 23.) Petitioner testified that he has to wear sunglasses when he is outside due to his eye sensitivity and goggles in the shower because he cannot close his eye all the way. (*Id.* 19-20.) He explained that his ophthalmologist recommended surgery options to help him close his eye. (*Id.* at 21.) Additionally, he testified to how his condition has impacted him psychologically. He explained that his confidence is low and that it has impacted his personal life as a mentor through his church. (*Id.* at 23-24, 27-29.)

Petitioner testified that he discussed the possible causes of his Bell's palsy with his treating physicians. (Tr. 25.) He noted that none of his doctors "said exactly what caused it;" however, he describes three times that his doctors mentioned potential causes. (*Id.*) First, he described how the doctors in the emergency room asked if petitioner had been out of the country or "around any viruses or anyone with a virus." (*Id.*) He explained that he told the doctor that he had not been out of the country, but had been vaccinated and the doctor had said it was possible those vaccinations could have caused his Bell's palsy. (*Id.*) Second, he described how his first neurologist explained to him that the type of vaccine he had received was associated with Bell's palsy. (*Id.*) And finally, his second neurologist told him that Bell's palsy is usually caused by a virus and tested him for HIV, which came back negative. (*Id.* at 25-26.) He noted that his doctors never told him that his diabetes could have caused his Bell's palsy. (*Id.* at 26-27.)

IV. Expert Opinions

a. Petitioner's Expert, Dr. M. Eric Gershwin, M.D.[3]

Petitioner's expert, Dr. M. Eric Gershwin, submitted three expert reports in this case. (Exs. 7, 63, 69.) In his first report, Dr. Gershwin begins by noting that the molecular mechanisms of Bell's palsy "have not been dissected." (Ex. 7, p. 2.) He goes on to explain the current state of the mechanistic research and notes that "[t]he mechanism of Bell's palsy is thought to be due to an abnormal immune response and likely has more than [one] mechanism." (*Id.* at 5.) One such mechanism, which according to Dr. Gershwin is most relevant to this case, is a "T cell mediated inflammatory process, including a localized inflammatory reaction within the facial nerve." (*Id.*) In support of this conclusion, Dr. Gershwin cites two studies that determined that:

[3] Dr. Gershwin received his medical degree from Stanford University. (Ex. 62, p. 1.) He is board certified in Internal Medicine, Internal Medicine with a subspeciality of Rheumatology, and Allergy and Clinical Immunology. (*Id.* at 2.) He is currently a Professor of Medicine in the Division of Rheumatology/Allergy and Clinical Immunology and Director of the Allergy-Clinical Immunology Program at the University of California School of Medicine in Davis, California. (*Id.* at 1.) He has written 69 books and monographs, 938 experimental papers, and 208 book chapters, and he has participated in 38 guest editorials and book reviews. (*Id.* at 8-125.)

> [H]istologic changes in the facial nerve can be summarized as follows: 1) The nerve was infiltrated by small, round inflammatory cells from the internal acoustic meatus to the stylomastoid foramen. 2) There was a breakdown of the neuron myelin sheaths that involved the macrophages. 3) There was an increase in the spaces between the neurons, which was interpreted as edema. 4) The bony fallopian canal was normal, and there was no sign of facial nerve compression by the bone of the fallopian canal. The small round cells of lymphocytic nature and the breakdown of myelin sheaths probably are the histologic expression of an autoimmune response.

(*Id.* (quoting A. Greco et al., *Bell's Palsy and Autoimmunity*, 12 AUTOIMMUNITY REVS. 323, 325 (2012) (Ex. 38, p. 3) (discus Stephen L. Liston & M. Stephen Kleid, *Histopathology of Bell's Palsy*, 99 LARYNGOSCOPE 23, 24 (1989) (Ex. 39, p. 3))).)

Dr. Gershwin cites several studies that suggest "[a] cell-mediated autoimmune mechanism has been suggested as the pathogenesis of Bell's palsy." (Ex. 7, p. 5 (citing O. Abramsky et al., *Cellular Immune Response to Peripheral Nerve Basic Protein in Idiopathic Facial Paralysis (Bell's Palsy)*, 26 J. NEUROLOGICAL SCIS. 13 (1975) (Ex. 40); Francis H. McGovern et al., *Immunological Concept for Bell's Palsy: Further Experimental Study*, 86 ANNALS OTOLOGY RHINOLOGY & LARYNGOLOGY 300 (1977) (Ex. 41); P. S. J. Z. Mulkens et al., *Acute Facial Paralysis: A Virological Study*, 5 CLINICAL OTOLARYNGOLOGY 303 (1980) (Ex. 42)).) Specifically, Dr. Gershwin notes that a clinical study found "some alterations in the lymphocyte subsets of the peripheral blood during the acute stage of" Bell's palsy. (*Id.* (citing Aharon Aviel et al., *Peripheral Blood T and B Lymphocyte Subpopulations in Bell's Palsy*, 92 ANNALS OTOLOGY RHINOLOGY & LARYNGOLOGY 187 (1983) (Ex. 43)).) Yet another study found "cellular and humoral immunologic alterations" in patients with Bell's palsy. (*Id.* (citing M. Mañós-Pujol et al., *Cellular Immunity Abnormalities in Patients with Recurrent Bell's Palsy*, 12 CLINICAL OTOLARYNGOLOGY 283 (1987) (Ex. 44)).) Additionally, Dr. Gershwin notes that "[d]ecreased percentages of total T cells (CD3) and T helper/inducer cells (CD4) have been documented in the acute phase of disease compared with control patients." (*Id.* (citing Clara Gorodezky et al., *The HLA System and T-Cell Subsets in Bell's Palsy*, 111 ACTA OTO-LARYNGOLOGICA (STOCKHOLM) 1070 (1991) (Ex. 45)).)

Dr. Gershwin also notes that there is an association between facial paralysis and Guillain-Barré syndrome (GBS), a "cell mediated, autoimmune neuritis," which continues to support the theory that the mechanism that causes Bell's palsy is immune mediated. (Ex. 7, p. 6 (citing Donald I. Charous & Bruce I. Saxe, *The Landry-Guillain-Barré Syndrome*, 267 NEW ENG. J. MED. 1334 (1962) (Ex. 46); Oded Abramsky et al., *Cell-Mediated Immunity to Neural Antigens in Idiopathic Polyneuritis and Myeloradiculitis: Clinical-Immunologic Classification of Several Autoimmune Demyelinating Disorders*, 25 NEUROLOGY 1154 (1975) (Ex. 47)).) Dr. Gershwin explains that patients with Bell's palsy and GBS have been shown to respond to a human basic protein (P1L) of peripheral nerve myelin. (*Id.* (citing Abramsky et al., *supra*, at Ex. 40; Christian A. Vedeler et al., *Antibodies to Peripheral Nerve Tissue in Sera from Patients with Acute Guillain-Barré Syndrome Demonstrated by a Mixed Haemagglutination*

Technique, 2 J. NEUROIMMUNOLOGY 209 (1982) (Ex. 48)).) Additionally, Dr. Gershwin notes that patients with Bell's palsy and GBS had decreased T lymphocytes, specifically T suppressor cells. (*Id.* (citing Christian A. Vedeler et al., *Immunoglobulins, Complement Components and Lymphocyte Subpopulations in Bell's Palsy*, 25 EUR. NEUROLOGY 177 (1986) (Ex. 49); H. Nyland & A. Næss, *Lymphocyte Subpopulations in Blood and Cerebrospinal Fluid from Patients with Acute Guillain-Barré Syndrome*, 17 EUR. NEUROLOGY 247 (1978) (Ex. 50); R. A. C. Hughes et al., *Lymphocyte Subpopulations and Suppressor Cell Activity in Acute Polyradiculoneuritis (Guillain-Barré Syndrome)*, 51 CLINICAL EXPERIMENTAL IMMUNOLOGY 448 (1983) (Ex. 51); Aviel et al., *supra*, at Ex. 43)).) Dr. Gershwin also notes that "similar changes in peripheral blood lymphocyte subpopulations were also described in the course of several demyelinating diseases, such as in acute exacerbations of multiple sclerosis and during the acute stage of [GBS]." (*Id.* (citing Mulkens et al., *supra*, at Ex. 42; Nyland & Næss, *supra*, at Ex. 50; R. P. Lisak et al., *T and B Lymphocytes in Multiple Sclerosis*, 22 CLINICAL EXPERIMENTAL IMMUNOLOGY 30 (1975) (Ex. 52)).) Based on these similarities, Dr. Gershwin concludes that GBS and Bell's palsy "may share a similar etiology and pathogenesis," and even notes that, in some cases, Bell's palsy is considered "a mononeuritic variant of [GBS]," or alternatively a polyneuropathy specific to the cranial nerves. (*Id.* (citing Aviel et al., *supra*, at Ex. 43; J. Chaco, *Subclinical Peripheral Nerve Involvement in Unilateral Bell's Palsy*, 52 AM. J. PHYSICAL MED. 195 (1973) (Ex. 53); Kedar K. Adour et al., *The True Nature of Bell's Palsy: Analysis of 1,000 Consecutive Patients*, 88 LARYNGOSCOPE 787 (1978) (Ex. 12); Leslie P. Weiner et al., *Medical Progress: Viral Infections and Demyelinating Diseases*, 288 NEW ENG. J. MED. 1103 (1973) (Ex. 54); Peter W. Lampert, *Autoimmune and Virus-Induced Demyelinating Diseases: A Review*, 91 AM. J. PATHOLOGY 176 (1978) (Ex. 55)).) Nonetheless, Dr. Gershwin acknowledges that "Bell's palsy is a clinical syndrome, and it is entirely possible that more than one disease entity can produce an idiopathic facial palsy." (*Id.* at 5.)

In addition to the similarities between Bell's palsy and GBS, Dr. Gershwin also relies on the fact that "viruses and immune mechanisms involvement have also been proposed in Bell's palsy," which he notes supports the theory that it is caused by "an autoimmune postviral disease." (Ex. 7, p. 6 (citing Abramsky et al., *supra*, at Ex. 40).) Dr. Gershwin explains that the mechanism leading to this potential causal association is not clear. (*Id.* (citing Aviel et al., *supra*, at Ex. 43).) However, he then explains some potential theories that have been considered. (*Id.* at 6-7.) Specifically, Dr. Gershwin notes that patients with Bell's palsy have "elevated concentrations of the cytokines interleukin-1 (IL-1), IL-6, and tumor necrosis factor-alpha (TNF-alpha)," which, according to Dr. Gershwin, is evidence of an activation of cell-mediated effectors. (*Id.* at 6 (citing Mustafa Yilmaz et al., *Serum Cytokine Levels in Bell's Palsy*, 197 J. NEUROLOGICAL SCIS. 69 (2002) (Ex. 57)).) Dr. Gershwin supports this conclusion by noting that some studies have "proposed that a breakdown of peripheral tolerance occurred in patients with hepatitis C virus infection as a consequence of interferon (IFN)-alpha therapy." (*Id.* at 6-7 (citing Matthew Hoare et al., *Bell's Palsy Associated with IFN-α and Ribavirin Therapy for Hepatitis C Virus Infection*, 25 J. INTERFERON & CYTOKINE RSCH. 174 (2005) (Ex. 58)).) Dr. Gershwin suggests these issues exist in the

myelin sheath because most patients with Bell's palsy do recover quickly. (*Id.* at 7 (citing Abramsky et al., *supra*, Ex. 40).)

Dr. Gershwin posits that the evidence that Bell's palsy is cell-mediated and its association with viruses is also evidence that Bell's palsy can be caused by vaccines. (Ex. 7, pp. 2-3, 7.) Specifically, Dr. Gershwin notes that "[i]ntranasal administration of influenza vaccines may reduce the transmission of influenza more efficiently than parenteral administration because it stimulates both mucosal and systemic immune responses." (*Id.* at 7 (citing Edel A. McNeela & Kingston H.G. Mills, *Manipulating the Immune System: Humoral Versus Cell-Mediated Immunity*, 51 ADVANCED DRUG DELIVERY REVS. 43 (2001) (Ex. 61)).) In support of this conclusion, Dr. Gershwin submitted several case studies, reports, and research studies. (*Id.* at 2-3 (citing Barbara Rath et al., *"All that Palsies is not Bell's [1]" – The Need to Define Bell's Palsy as an Adverse Event Following Immunization*, 26 VACCINE 1 (2007) (Ex. 13) (comprehensive literature review regarding classifying Bell's palsy as an adverse event following immunization, which specifically notes that Bell's palsy has been studied following various vaccines); 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) (Ex. 15) (case-control study and a case-series analysis finding that there is a "strong association between the inactivated intranasal influenza vaccine used in Switzerland and Bell's palsy," however, this vaccine is no longer used); 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) (Ex. 16) (study revealing a "possible association between influenza vaccines and an increased risk of Bell's palsy")).)[4]

Dr. Gershwin also acknowledges the association between diabetes and Bell's palsy. (Ex. 7, pp. 3-4.) He notes that "approximately 5-10% of patients" with Bell's palsy also have diabetes. (*Id.* at 2 (citing Raymond L. Hilsinger, Jr. & Kedar Karim Adour, *Idiopathic Facial Paralysis, Pregnancy, and the Menstrual Cycle*, 84 ANNALS OF OTOLOGY RHINOLOGY & LARYNGOLOGY 433 (1975) (Ex. 8); Mark May & Susan R. Klein,

[4] However, Dr. Gershwin acknowledges that some studies have found no association between vaccination and Bell's palsy. (Ex. 7, pp. 2-3 (citing Leonoor Wijnans et al., *Bell's Palsy and Influenza (H1N1)pdm09 Containing Vaccines: A Self-Controlled Case Series*, 12 PLOS ONE 1 (2017) (EX. 20) (finding "no evidence of increased incidence of Bell's palsy following seasonal influenza vaccination overall, nor for monovalent pandemic influenza vaccine in 2009); Ali Rowhani-Rahbar et al., *Immunization and Bell's Palsy in Children: A Case-Centered Analysis*, 175 AM. J. EPIDEMIOLOGY 878 (2012) (EX. 14) (finding no association between immunization and Bell's palsy in children); Sharon K. Greene et al., *Near Real-Time Surveillance for Influenza Vaccine Safety: Proof-of-Concept in the Vaccine Safety Datalink Project*, 171 AM. J. EPIDEMIOLOGY 177 (2010) (EX. 17) (study looking at several adverse events associated with vaccination and not finding a statistically significant relationship between seasonal trivalent inactivated influenza vaccine and Bell's palsy); Julia Stowe et al., *Bell's Palsy and Parental Inactivate Influenza Vaccine*, 2 HUM. VACCINES 110 (2006) (Ex. 18) (study found "no evidence of an increased risk of Bell's palsy in the three months following parenteral inactivated influenza vaccine"). *But see* Samuel Shapiro, *Invited Commentary: Immunization and Bell's Palsy in Children*, 175 AM. J. EPIDEMIOLOGY 886 (2012) (Ex. 19) (identifying some issues with how the study in Ex. 18 was conducted)).)

Differential Diagnosis of Facial Nerve Palsy, 24 OTOLARYNGOLOGIC CLINICS N. AM. 613 (1991) (Ex. 9); Erik Peitersen, *The Natural History of Bell's Palsy*, 4 AM. J. OTOLOGY 107 (1982) (Ex. 10); R.E. Mountain et al., *The Edinburgh Facial Palsy Clinic: A Review of Three Years' Activity*, 39 J. ROYAL COLL. SURGEONS EDINBURGH 275 (1994) (Ex. 11); Adour et al., *supra*, at Ex. 12).) However, Dr. Gershwin also notes that the magnitude of the association between diabetes and Bell's palsy is controversial. (*Id.* at 3 (citing Ru-Lan Hsieh et al., *Correlates of Degree of Nerve Involvement in Early Bell's Palsy*, 9 BMC NEUROLOGY 1 (2009) (Ex. 21); I.M. Smith et al., *Idiopathic Facial (Bell's) Palsy: A clinical Survey of Prognostic Factors*, 13 CLINICAL OTOLARYNGOLOGY 17 (1988) (Ex. 22); Luzy Abraham-Inpijn & Pieter P. Devriese, *Hypertension and Diabetes Mellitus in Bell's Palsy*, *in* DISORDERS OF THE FACIAL NERVE: ANATOMY, DIAGNOSIS, AND MANAGEMENT 251 (Malcomn D. Graham & William F House eds., 1982) (Ex. 23); Akira Takahashi & Itsuro Sobue, *Concurrence of Facial Paralysis and Diabetes Mellitus: Prevalence, Clinical Features and Prednisolone Treatment*, *in* DIABETIC NEUROPATHY: CLINICAL MANAGEMENT 173 (Aristidis Veves & Rayaz Malik eds., 2nd ed. 1982) (Ex. 24)).) Specifically, he quotes a paragraph from a study critical of research that found an association between Bell's palsy and diabetes because the literature is older and the studies used tests not common in clinical practice. (*Id.* at 3-4 (quoting Maria Riga et al., *The Role of Diabetes Mellitus in the Clinical Presentation and Prognosis of Bell Palsy*, 25 J AM. BOARD FAM. MED. 819, 821-24 (2012) (Ex. 25, pp. 3-6)).)

Instead, Dr. Gershwin opines that petitioner's diabetes merely left petitioner more susceptible to Bell's palsy because "diabetics already have some degree of nerve ischemia." (Ex. 7, p. 4 (citing Riga et al., *supra*, at Ex. 25).) Dr. Gershwin opines that after petitioner received his vaccination, he "develop[ed] immune response as expected from any vaccination," and that, "[i]n the absence of any other etiological events that can be associated with Bell's palsy, [petitioner] would have a localized T cell mediated inflammatory response based upon a molecular mimicry with either the influenza or the hepatitis B vaccine." (*Id.* at 7.) He concludes that, instead of causing his Bell's palsy, petitioner's diabetes instead "suggests that his facial nerve was already damaged from his diabetic neuropathy and therefore any further inflammatory response would compromise the nerve further." (*Id.*)

In his second report, Dr. Gershwin addresses the etiology of Bell's palsy and goes into further detail regarding the T cell reaction caused by petitioner's vaccination and the temporal association between petitioner's vaccination and development of Bell's palsy. (Ex. 63.) First, Dr. Gershwin notes that the etiology of Bell's palsy has been summarized as: (1) anatomical structure, (2) viral infections, (3) ischemia, (4) immune inflammatory, and (5) acute cold exposure. (*Id.* at 1 (citing Wenjuan Zhang et al., *The Etiology of Bell's Palsy: A Review*, 267 J. NEUROLOGY 1896 (2020) (Ex. 64)).) He notes that the authors of that study additionally point out that the clinical and immunological data "'suggests activation of cell-mediated effectors and the involvement of immune mechanisms in Bell's palsy.'" (*Id.* (quoting Zhang et al., *supra*, at Ex. 64, p. 6).) Additionally, Dr. Gershwin notes that petitioner developed Bell's palsy "within a month of vaccination," which "is consistent with the production of T cell immunity after influenza vaccination." (*Id.* at 2 (citing Tina Schmidt et al., *CD4⁺ T-Cell Immunity After Pandemic*

Influenza Vaccination Cross-Reacts with Seasonal Antigens and Functionally Differs from Active Influenza Infection, 42 EUR. J. IMMUNOLOGY 1755 (2012) (Ex. 66) (finding increases in T cell production up to 10 weeks post-vaccination with influenza); Ulrik Stervbo et al., *Age Dependent Differences in the Kinetics of γδ T Cells After Influenza Vaccination*, 12 PLOS ONE 1 (2017) (Ex. 67)).) Dr. Gershwin notes that there is a similar timeline for an immunological response to the hep B vaccine. (*Id.* (citing I. Desombere et al., *Characterization of the T Cell Recognition of Hepatitis B Surface Antigen (HBsAg) by Good and Poor Responders to Hepatitis B Vaccines*, 122 CLINICAL & EXPERIMENTAL IMMUNOLOGY 390 (2000) (Ex. 68)).) Dr. Gershwin acknowledges that petitioner "has had diabetes for more than a decade;" however, he notes that petitioner did not develop Bell's palsy until after he was vaccinated. (*Id.*) Dr. Gershwin concluded that "[t]he acute T cell-mediated immunity expected after his seasonal influenza and second Hepatitis B vaccination was the final insult that led to further ischemic changes and his development of Bell's palsy." (*Id.*)

In his third report, Dr. Gershwin addresses whether, in his opinion, petitioner's Bell's palsy was a result of his diabetes alone. (Ex. 69.) For this report, Dr. Gershwin reviewed a study, which found that "'[a]ll the clinical and immunological data . . . suggests activation of cell-mediated effectors and the involvement of immune mechanisms in Bell's palsy.'" (*Id.* (quoting Zhang et al., *supra*, at Ex. 64, p. 6).) Additionally, Dr. Gershwin notes that "[n]ot all studies have demonstrated an association of diabetes and Bell's palsy," and "[t]he relationship between Bell's [p]alsy and diabetes is not clearly related to diabetes control (hyperglycemia) and they suggest further studies are warranted." (*Id.* (citing Riga et al., *supra*, at Ex. 25).)

b. Respondent's Expert, Dr. Thomas P. Leist, M.D., Ph.D.[5]

Respondent's expert, Dr. Thomas P. Leist, submitted two reports in this case. (Exs. A; C.) In his first report, Dr. Leist opined that "diabetes related microvascular injury is the likely cause of [petitioner's] Bell's palsy." (Ex. A, p. 5.) In support of this opinion, Dr. Leist cites a study done by Pecket and Schattner, which found that "'in a series of 126 patients with Bell's palsy, chemical or overt diabetes was found in 39% of cases.'" (*Id.* (quoting P. Pecket & A. Schattner, *Concurrent Bell's Palsy and Diabetes Mellitus: A Diabetic Mononeuropathy?*, 45 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 652 (1982) (Ex. A, Tab 2)).) In another study of 684 patients with Bell's palsy, 11.4% had diabetes. (*Id.* (citing Kedar K. Adour et al., *Prevalence of Concurrent Diabetes Mellitus and Idiopathic Facial Paralysis (Bell's Palsy)*, 24 DIABETES 449 (1975) (Ex. A, Tab 1)).) Additionally, relying on one of the studies cited by Dr. Gershwin, Dr. Leist explains that "'Bell's palsy is considered to be an entrapment neuropathy resulting from inflammation, edema, and strangulation. Diabetes, hypertension, and

[5] Dr. Leist is board certified in Psychiatry and Neurology. (Ex. B, p. 1.) He received his medical degree from the University of Miami and his Ph.D. in Biochemistry from the University of Zurich. (*Id.*) He currently works as a Professor of Neurology at Thomas Jefferson University, Chief of the Division of Clinical Neuroimmunology and Director of the Comprehensive Multiple Sclerosis Center, a neurology consultant at the Inglis Foundation, and Director of Hospital-Based Neurology Infusion Service. (*Id.*) He has published 61 peer-reviewed papers, 12 review and invited publications, editorials, commentaries, and book chapters, and participated in 47 clinical trials. (*Id.* at 5-14.)

hypercholesterolemia have all been related to microangiopathies.'" (*Id.* (quoting Riga et al., *supra*, at Ex. 25, p. 1).)

Dr. Leist notes that petitioner's diabetes was "poorly controlled from at least 2007." (Ex. A, p. 4.) Additionally, Dr. Leist opines that "[t]here was clinical evidence microvascular/microcirculatory compromise in [petitioner's] case prior to the administration of vaccines on September 19, 2017." (*Id.* at 5.) Dr. Leist opines that these "preexisting microvascular/microcirculatory compromise included diabetic neuropathy indicative of microvascular compromise/microvascular injury of the peripheral nerves, diabetic retinopathy and macular edema suggestive of microvascular changes/injury in the eyes, microalbuminuria – suggesting microvascular injury in the kidneys, and skin ulcers indicating peripheral vascular compromise." (*Id.*) Additionally, Dr. Leist noted that "elevated hemoglobin A1c values correlated significantly with severe Bell's palsy." (*Id.* (citing Riga et al., *supra*, at Ex. 25).) Therefore, Dr. Leist opines "that association between Bell's palsy and diabetes and severity of Bell's palsy and elevated hemoglobin A1c is well established." (*Id.*)

Finally, Dr. Leist rejects the opinion that vaccines can cause Bell's palsy. (Ex. A, p. 5.) He noted that "[t]he Institute of Medicine has reviewed the question of an association of inactivated influenza vaccine and Bell's palsy and concluded that the evidence favored rejection of a causal relationship." (*Id.* (citing IOM Report 2012 (Ex. A, Tab 4)).)

In his second report, Dr. Leist reiterates his opinion that "[t]he association of diabetes and Bell's palsy is well documented." (Ex. C, p. 1 (citing Pecket & Schattner, *supra*, at Ex. A, Tab 2; Adour et al., *supra*, at Ex. A, Tab 1).) Dr. Liest further quotes an article by Zhang et al, which states that "in certain clinical conditions, such as diabetes mellitus, primary ischemic neuropathy is likely to occur," and "after transient ischemia and reperfusion, acute inflammation of the diabetic nerve is seen." (*Id.* (quoting Zhang et al., *supra*, Ex. 64, p. 5.) Dr. Leist notes that Dr. Gershwin acknowledged this when he admitted that petitioner's diabetes left him more susceptible to Bell's palsy. (*Id.* at 2 (citing Ex. 63, p. 2).)

Additionally, Dr. Leist opines that "Dr. Gershwin does not provide any evidence that influenza vaccine causes inflammation essentially restricted to the facial nerve and does not explain why the two-step process of injury he proposes as having occurred is the more likely cause of Bell's palsy in [petitioner's] case than poorly controlled diabetes alone." (Ex. C, p. 2.) Dr. Leist acknowledges that diabetes can lead to inflammation in the facial nerve; however, he noted that "Dr. Gershwin does not provide any insights how to distinguish reactive inflammation of the facial [nerve] following transient ischemia due to diabetes . . . from vaccine induced inflammation of the facial nerve which he alleges occurred but for which he has not provided any evidence." (*Id.* (citing Zhang et al., *supra*, at Ex. 64).)

V. Discussion

a. *Althen* prongs one and three

Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (quoting *Pafford v. Sec'y of Health & Human Servs.*, No. 01-0165V, 2004WL 171359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006)). Scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Andreu ex rel. Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1380 (Fed. Cir. 2009). In this case, petitioner relies on Dr. Gershwin's assertion that his Bell's palsy was caused by "a localized T cell mediated inflammatory response based upon a molecular mimicry with either the influenza or the hepatitis B vaccine." (ECF No. 64, p. 6 (citing Ex. 7, p. 7); ECF No. 66, p. 1.) According to Dr. Gershwin, this immune reaction caused inflammation in the facial nerve, which led to paralysis of the facial muscles. (ECF No. 64, pp. 6-7 (citing Ex. 7, pp. 1-2, 5).)

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1278. A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. Petitioner contends that he "developed Bell's palsy within a month of vaccination," which "is consistent with the production of T cell immunity after influenza vaccination." (ECF No. 64, p. 10 (citing Ex. 63, p. 2 (relying on Schmidt et al., *supra*, at Ex. 66)).) Petitioner relied on Dr. Gershwin's assertion that "T cells are activated after a flu vaccination within 3.5 weeks," and that similar data exists for the hep B vaccine. (*Id.* (citing Ex. 63 (relying on Exs. 66-68).) Therefore, petitioner notes that he developed Bell's palsy within 3.5 weeks following vaccination, "which is within the period expected given the proposed mechanism." (*Id.*)

Petitioner argues that respondent has not actually challenged his showing with respect to either *Althen* prongs one or three. (ECF No. 66, pp. 1-2, 4.) This is not accurate. (*See* ECF No. 65.) However, the analysis below does confirm that petitioner's claim fails under *Althen* prong two regardless of the outcome of prongs one or three. Accordingly, in the interest of brevity, this decision will assume, but not decide, that petitioner has satisfied *Althen* prongs one and three in order to reach the dispositive issues in this case relating to whether there is a logical sequence of cause and effect implicating petitioner's vaccination in *his own* course of Bell's palsy.[6] *Accord Winkler v.*

[6] Though not resolving *Althen* prongs one and three in this case, I do note that another special master has determined that there are reputable medical theories supporting the conclusion that the flu vaccine and the hep B vaccine can cause Bell's palsy up to 40 days post-vaccination depending on the theory proffered. *E.g.*, *Sturdevant v. Sec'y of Health & Human Servs.*, No. 17-172V, 2022 WL 3369716 (Fed. Cl. Spec. Mstr. July 19, 2022); *Beraki v. Sec'y of Health & Human Servs.*, No. 17-243V, 2021 WL 4891119 (Fed. Cl. Spec. Mstr. Sept. 20, 2021); *E.A. v. Sec'y of Health & Human Servs*., No. 18-1587V, 2023 WL

Sec'y of Health & Human Servs., No. 18-203V, 2021 WL 6276203, at *23 (Fed. Cl. Spec. Mstr. Dec. 10, 2021) (citing *Vaughan ex rel. A.H. v. Sec'y of Health & Human Servs.*, 107 Fed. Cl. 212, 221-22 (2012); *Hibbard v. Sec'y of Health & Human Servs.*, 698 F.3d 1355, 1364 (Fed. Cir. 2012)), *aff'd*, 88 F.4th 958 (Fed. Cir. 2023) (finding no abuse of discretion where the special master "assumed" *Althen* prong one was met before concluding *Althen* prong two was dispositive).

b. *Althen* prong two

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375-77; *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1326 (Fed. Cir. 2006); *Grant*, 956 F.2d at 1148. Medical records are generally viewed as particularly trustworthy evidence. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, medical records and/or statements of a treating physician's views do not *per se* bind the special master. *See* § 300aa-13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) ("[T]here is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted."). A petitioner may support a cause-in-fact claim through either medical records or expert medical opinion. § 300aa-13(a). The special master is required to consider all the relevant evidence of record, draw plausible inferences, and articulate a rational basis for the decision. *Winkler*, 88 F.4th at 963 (citing *Hines*, 940 F.2d at 1528). Petitioner need not prove his vaccination was the sole cause of his injury, but he must show that it was a substantial contributing factor and a "but for" cause. *Shyface*, 165 F.3d at 1352. Although petitioners do not bear a burden of eliminating other causes of injury, evidence of other possible sources of injury can be relevant to determining whether a prima facie showing has been made as to vaccine causation. *Winkler*, 88 F.4th at 963 (citing *Stone v. Sec'y of Health & Human Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012).)

On petitioner's behalf, Dr. Gershwin presents a "cell-mediated immune response following vaccination" as the "final insult" that "further compromised [petitioner]'s ischemia" and led to his Bell's palsy. (Ex. 63, p. 2; Ex. 69, p. 1.) Dr. Gershwin's search for an inciting immune event may be a natural extension of his own proposed theory of causation, however, he acknowledges that his theory is only one possible etiologic explanation for Bell's palsy. He explains that studies are lacking that "would help define the mechanisms" of injury in Bell's palsy and that it "likely has more than [one] mechanism." (Ex. 7, pp. 2, 5.) He specifically states that "Bell's palsy is a clinical syndrome, and it is entirely possible that more than one disease entity can produce an idiopathic facial palsy." (*Id*. at 5.) He explains that while the "key evidence" for the etiology of Bell's palsy includes immune inflammation, it also includes anatomical structures, viral infections, acute cold exposure, and, importantly, ischemia. (Ex. 63, p.

2640710 (Fed. Cl. Spec. Mstr. Jan. 24, 2023); *Arredondo v. Sec'y of Health & Human Servs.*, No. 18-1782V, 2023 WL 8181138 (Fed. Cl. Spec. Mstr. Oct. 31, 2023).

1.) Dr. Gershwin posits that there are no other "etiological events" that could explain petitioner's condition. (Ex. 7, p. 7.) However, he contradicts himself by acknowledging that "facial nerves get their vascular supply by a multitude of vessels and when they are obstructed, it will lead to ischemia. Since patients with diabetes may already have nerve ischemia, the pre-existing microangiopathy may facilitate the natural history of Bell's palsy." (*Id.* at 4.) Further to this, Dr. Gershwin agrees that petitioner "has chronic insulin-dependent Type II diabetes, and has a previous history of polyneuropathies secondary to the vascular/ischemia associated with diabetes." (Ex. 69, p. 1.) Dr. Gershwin explicitly opines that petitioner "was already compromised based upon his underlying ischemia from the diabetes." (Ex. 63, p. 2.)

Thus, both parties' experts opine that petitioner's diabetes is *at minimum* a significant part of the explanation for petitioner's Bell's palsy. Further to which, respondent's expert, Dr. Leist, is persuasive in observing that Dr. Gershwin has little to no basis for additionally invoking an immune mechanism of injury.[7] (Ex. A, p. 5; Ex. C, p. 2.) Dr. Leist correctly explains that on this record "Dr. Gershwin does not provide any insights how to distinguish reactive inflammation of the facial [nerve] following transient ischemia due to diabetes, which he opines was present, from vaccine induced inflammation of the facial nerve which he alleges occurred but for which he has not provided any evidence." (Ex. C, p. 2.) In contrast to the lack of any clinical indicators of injurious post-vaccination inflammation, the medical records very clearly establish not merely that petitioner had coincidental diabetes, but that his diabetes was not under control at the time of his Bell's palsy and that it was otherwise manifesting similar complications pre-dating the vaccinations at issue. Specifically, Dr. Leist explains that

> [t]here was clinical evidence [of] microvascular/microcirculatory compromise in [petitioner]'s case prior to the administration of vaccines on September 19, 2017. Preexisting findings that indicated preexisting microvascular/microcirculatory compromise included diabetic neuropathy indicative of microvascular compromise/microvascular injury of the peripheral nerves, diabetic retinopathy and macular edema suggestive of microvascular changes/injury in the eyes, microalbuminuria – suggesting

[7] Petitioner contends that respondent's expert "does not dispute that [petitioner]'s vaccines caused harm." (ECF No. 66, p. 2.) But this is not accurate. Petitioner focuses on the phrasing of the conclusion to Dr. Leist's two reports in which he stated that petitioner "did not suffer an enduring injury" as a consequence of his vaccinations. (Ex. A, p. 6; Ex. C, p. 2.) Petitioner interprets Dr. Leist's reports as implying that petitioner may have suffered a short-term harm. (ECF No. 66, pp. 2-3.) While Dr. Leist's use of the word "enduring" is a bit confusing, it is apparent when examining his opinion as a whole that Dr. Leist does, in fact, challenge petitioner's contention that petitioner suffered any vaccine caused injury at all. In his first report, Dr. Leist stressed that petitioner "did not report any immediate or delayed side effects of the vaccinations in the minutes, hours, and days following administration." (Ex. A, p. 4.) Instead, he stated, "It is my opinion that diabetes related microvascular injury is the likely cause of [petitioner]'s Bell's palsy." (*Id*. at 5.) In his second report, Dr. Leist further indicated that "Dr. Gershwin does not provide any evidence that influenza vaccine causes inflammation essentially restricted to the facial nerve and does not explain why the two-step process of injury he proposes as having occurred is the more likely cause of Bell's palsy in [petitioner]'s case than poorly controlled diabetes alone." (Ex. C, p. 2.)

> microvascular injury in the kidneys, and skin ulcers indicating peripheral vascular compromise.

(Ex. A, p. 5.) Dr. Gershwin has not challenged this assessment. In fact, notwithstanding his search for an additional final straw, Dr. Gershwin agrees not only that petitioner was suffering diabetes-related ischemia sufficient to induce neurologic damage but further that the relevant facial nerve was likely already damaged by petitioner's diabetes prior to vaccination. (Ex. 7, p. 7 (stating that "[t]he presence of diabetes in [petitioner] suggests that his facial nerve was already damaged from his diabetic neuropathy"); Ex. 63, p. 2 (submitting "that he was already compromised based upon his underlying ischemia from diabetes").)

Dr. Gershwin opines that all of this left petitioner merely susceptible to Bell's palsy and stresses that petitioner's history of diabetic ischemic polyneuropathy "does not make [him] immune from a secondary insult." (Ex. 69, p. 1.) The latter point may be true as far as it goes, but it is not evidence that a secondary insult is implicated. Apart from a post-vaccination onset, petitioner has not pointed to any clinical evidence that supports Dr. Gershwin's opinion. Even while assuming the onset of petitioner's Bell's palsy could be compatible with Dr. Gershwin's theory, Dr. Leist is correct to stress that petitioner did not report any immediate or delayed side effects of the vaccinations in the days following vaccination. (Ex. A, p. 4.) Especially in the context of petitioner's chronic, ongoing, and uncontrolled diabetes, the three-and-a-half week latency in this case, even if generally plausible under petitioner's theory, is not compelling on its own as any kind of clear indicator of vaccine causation.[8] Nor does petitioner point to any medical opinion by any treating physician favoring his assertion of vaccine causation.[9] (ECF No. 64, pp. 8-10; ECF No. 66, pp. 2-4.) Moreover, Dr. Leist correctly observes that Dr. Gershwin "does not explain why the two-step process of injury he proposes as having occurred is the more likely cause of Bell's palsy in [petitioner]'s case than poorly controlled diabetes alone."[10] (Ex. C, p. 2.)

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[8] In some cases, post-vaccination Bell's palsy has been alleged based on an innate immune response that created an onset occurring within days of vaccination. *See, e.g., Sturdevant*, 2022 WL 3369716; *Beraki*, 2021 WL 4891119. In such cases, it is easier to see how the timing of onset could potentially be viewed as suspicious for a sequence of cause and effect whereby an acute event led directly, and more or less immediately, to the Bell's palsy. But, in any event, the Federal Circuit has explained that "[a]lthough probative, neither a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation." *Althen*, 418 F.3d at 1278 (citing *Grant*, 956 F.2d at 1149).

[9] Though petitioner testified that his doctors discussed the possibility of vaccine causation with him (Tr. 25-27), this is not reflected in any of his medical records. In any event, petitioner confirmed in his testimony that none of his physicians actually concluded his Bell's palsy was vaccine caused, acknowledging that none of his doctors "said exactly what caused it." (*Id.* at 25.)

[10] Though the idea of an immune trigger may be consistent with Dr. Gershwin's theory, his assertion of that trigger in this specific case, based on no clinical evidence apart from petitioner's very manifestation of the allegedly vaccine-caused injury, amounts to a circular logic that has been rejected in prior cases. *Accord Dodd v. Sec'y of Health & Human Servs.*, No. 09-0585V, 2013 WL 3233210, at *14 (Fed. Cl. Spec. Mstr. June 5, 2013) (explaining that "Dr. Kinsbourne's circular logic, that one event was caused by another simply because the second event occurred, is also unavailing"), *aff'd*, 114 Fed. Cl. 43 (2013);

Even granting petitioner the assumption that he has satisfied *Althen* prongs one and three, the Federal Circuit has cautioned that the second *Althen* prong "is not without meaning." *Capizzano*, 440 F.3d at 1327. Satisfying *Althen* prongs one and three generally serves largely as a threshold demonstration that a petitioner's claim is even possible. The Court explained that

> [t]here may well be a circumstance where it is found that a vaccine *can* cause the injury at issue and where the injury was temporally proximate to the vaccination, but it is illogical to conclude that the injury was actually caused by the vaccine. A claimant could satisfy the first and third prongs without satisfying the second prong when medical records and medical opinions do not suggest that the vaccine caused the injury, or where the probability of coincidence or another cause prevents the claimant from proving that the vaccine caused the injury by preponderant evidence.

Id.

That is precisely the scenario presented in this case. Given the lack of either treating physician support or meaningful clinical evidence and the undisputed presence of uncontrolled diabetes, Dr. Gershwin's mere *ipse dixit* assertion that petitioner's vaccination was a factor additional to his diabetes in bringing about his own Bell's palsy does not carry petitioner's burden of proof under *Althen* prong two. Moreover, Dr. Gershwin's own concessions regarding the important role of diabetes in bringing about petitioner's own injury dramatically undercut the assertion. This is not a question of petitioner disproving any role for his diabetes in causing his injury. As explained above, petitioner need not show his vaccination to have been the sole cause of his injury. *Shyface*, 165 F.3d at 1352. Instead, Dr. Gershwin's opinion is inadequate to preponderantly demonstrate that petitioner's vaccinations played any role, as a substantial contributing factor or a but for cause additional to his diabetes, in the development of his Bell's palsy.

c. Factor unrelated to vaccination

In this case, both parties addressed their arguments to the question of whether petitioner has met his burden of proof under the *Althen* test in the face of respondent's opposition, which is based primarily on diabetes as the cause of petitioner's condition. (ECF Nos. 64-65.) Importantly, however, as noted above, petitioner is not obligated as part of his prima facie burden of proof to eliminate alternative causes of his condition. *de Bazan*, 539 F.3d at 1352-53; *Winkler*, 88 F.4th at 962-63. Instead, once a petitioner has satisfied his own burden pursuant to the *Althen* test, the burden shifts to respondent

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Morgan v. Sec'y of Health & Human Servs., No. 12-77V, 2017 WL 6893079, at *22 (Fed. Cl. Spec. Mstr. Dec. 6, 2017) (concluding, "Petitioner's experts *assumed* that the very fact she experienced a rare injury at all was circumstantial proof of her 'unique genetic repertoire.' This kind of circular logic (the injury is itself proof of causation) does not meet the preponderant evidentiary standard set for a vaccine injury claim." (internal citation omitted)).

to demonstrate that his injury was caused by factors unrelated to vaccination. § 300aa-13(a)(1)(B); *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013).

Consistent with the parties' framing of the issue, I have concluded that petitioner did not meet his burden of proof under *Althen* for the reasons discussed above. *Accord Winkler*, 88 F.4th at 962 (quoting *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1356-58 (Fed. Cir. 2010), for the proposition that "a 'petitioner's failure to meet his burden of proof as to the cause of an injury or condition is different from a requirement that he affirmatively disprove an alternative cause"). However, in the interest of completeness, I additionally note that, even assuming *arguendo* that petitioner did meet his initial burden under *Althen*, I would still conclude that respondent has put forward sufficient evidence to preponderantly demonstrate that petitioner's Bell's palsy was more likely than not caused by his diabetes, rather than his vaccination(s). Although respondent has argued that the burden of proof never shifted to him, he is clear in contending that his expert has established diabetes as a more likely cause of petitioner's Bell's palsy to the exclusion of his vaccinations. (ECF No. 65, p. 32; ECF No. 38, p. 14.)

In order to meet his shifted burden of proof, respondent must demonstrate by preponderant evidence "that a particular agent or condition (or multiple agents/conditions) unrelated to the vaccine was in fact the sole cause (thus excluding the vaccine as a substantial factor)." *de Bazan*, 539 F.3d at 1354 (emphasis omitted). As with petitioner's burden under *Althen*, respondent must show a logical sequence of cause and effect linking the injury to the proposed factor unrelated. *Deribeaux*, 717 F.3d at 1368-69. It need not be scientifically certain but must be legally probable. *Id*. Conditions or other factors that are "idiopathic, unexplained, unknown, hypothetical, or undocumentable" cannot defeat a petitioner's claim. § 300aa-13(a)(2); *Knudsen ex rel. Knudsen*, 35 F.3d 543, 548 (Fed. Cir. 1994).

First, there is no meaningful dispute in this case that diabetes can cause Bell's palsy. Both parties' experts agree that the medical literature supports an association between diabetes and Bell's palsy, even as petitioner's expert, Dr. Gershwin, cautions that the magnitude of the association is unsettled. (Ex. A, p. 5; Ex .7, pp. 3-4.) Despite cautioning that not all available studies have confirmed an association between diabetes and Bell's palsy (Ex. 69, p. 1), Dr. Gershwin agrees on petitioner's behalf that "the role of diabetes in the development of Bell's palsy is well known" (Ex. 7, p. 3). Respondent's expert, Dr. Leist, further explains that Bell's palsy is an entrapment neuropathy that can result from, *inter alia*, primary ischemic neuropathy due to diabetes. (Ex. A, p. 5; Ex. C, p. 1.) Dr. Gershwin does not disagree. (Ex. 7, p. 4; Ex. 63, p. 1.) Although Dr. Gershwin asserts on petitioner's behalf that Bell's palsy can also have an immune etiology, he acknowledges that there are likely multiple pathophysiologic pathways and multiple disease entities that lead to Bell's palsy and/or facial paralysis. (Ex. 7, p. 5.) Consistent with Dr. Leist's opinion, he further agrees that diabetes-related ischemia can cause nerve damage, including damage to the nerve implicated in Bell's palsy. (Ex. 7, p. 4; Ex. 63, p. 1.)

Regarding a logical sequence of cause and effect, Dr. Leist is persuasive in contending that petitioner's diabetes alone is a sufficient explanation for his condition. (Ex. A, p. 5.) There is no question that petitioner had a longstanding history of uncontrolled diabetes. (*E.g.*, Ex. 63, p. 2 (Dr. Gershwin acknowledging petitioner "had diabetes for more than a decade, preceding his September 2017 influenza vaccination. His diabetes has been poorly controlled throughout much of that time.") Additionally, Dr. Leist cites literature reflecting a correlation between elevated hemoglobin A1c (as seen in uncontrolled diabetes) and severity of Bell's palsy (though not ultimate prognosis).[11] (Ex. A, p. 5 (citing Riga et al., *supra*, at Ex. 25, p. 6).) And, although petitioner asserts that he was attempting to manage his diabetes, he acknowledged in his testimony that his diabetes was not controlled around the time of the onset of his Bell's palsy.[12] (Tr. 11-13, 39-40, 45-46.) This is confirmed by his medical treatment records as discussed in the factual summary above, which show his ongoing elevations in blood glucose and hemoglobin A1C. Moreover, as Dr. Leist observes, there is no question that petitioner experienced preexisting microvascular compromise leading to neurologic complications attributable to his diabetes. (Ex. A, p. 5.) Petitioner has offered no argument that these complications were due to anything other than his diabetes. In fact, petitioner's own expert concedes that his Bell's palsy is at least partly explained by diabetic nerve damage. (Ex. 7, p. 7 (stating that "[t]he presence of diabetes in [petitioner] suggests that his facial nerve was already damaged from his diabetic neuropathy"); Ex. 63, p. 2 (submitting that petitioner "was already compromised based upon his underlying ischemia from diabetes").) Dr. Leist is also persuasive in contending that Dr. Gershwin has no basis for distinguishing inflammatory damage purportedly due to vaccination from the otherwise undisputed ischemic nerve damages resulting from petitioner's diabetes. (Ex. C, p. 2.) Thus, based on all of this, Dr. Leist is persuasive in opining "that diabetes related microvascular injury is *the* likely cause of [petitioner]'s Bell's palsy." (Ex. A, p. 5 (emphasis added).)

While Dr. Gershwin is well qualified to discuss the potential immunologic causes of a clinical syndrome such as Bell's palsy, Bell's palsy itself is a form of neuropathy, which places the condition more firmly in Dr. Leist's area of clinical expertise with respect to the spectrum of patient presentations and etiologies for the condition. Neither expert's curriculum vitae discloses any specific prior experience with either Bell's palsy or diabetes in particular. (Ex. 62; Ex. B.) However, Dr. Leist does represent that his clinical experience as a board-certified neurologist has included treatment of

[11] Dr. Gershwin urges caution regarding the clarity of the relationship observed by Riga et al., given the limitations noted by the authors (Ex. 69, p. 1; Ex. 7, p. 4), but does not seek to refute the study. In fact, Dr. Gershwin cited the study approvingly in his first report. (Ex. 7, pp. 3-4.)

[12] Specifically, petitioner testified that his diabetes was uncontrolled at the time he first saw Dr. Head, but that "I would say I began controlling it once I got under her care." (Tr. 39-40.) Petitioner's first encounter with Dr. Head on September 19, 2017, is the same encounter at which he received the subject vaccinations. (Ex. 2, pp. 218-38; Ex. 5, p. 6.) Petitioner separately testified, however, that he only subsequently (around 2021) learned of the need to reduce his carbohydrate intake to control his diabetes, suggesting his earlier efforts to control his diabetes were not fully effective. (Tr. 12-14, 46.)

patients with Bell's palsy. (Ex. A, p. 1.) Dr. Gershwin makes no similar representation and it is not readily apparent that his experience as a rheumatologist/immunologist would include any experience with patients presenting for Bell's palsy. Accordingly, even if all else were equal, Dr. Leist's opinion would still be entitled to greater weight with respect to his assessment of petitioner's clinical history vis-à-vis the underlying cause of his Bell's palsy. Where both parties offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1362 (Fed. Cir. 2000)). In determining whether a particular expert's testimony was reliable or credible, a special master may consider whether the expert is offering an opinion that exceeds the expert's training or competence. *E.g.*, *Walton v. Sec'y of Health & Human Servs.*, No. 04-503V, 2007 WL 1467307, at *17-18 (Fed. Cl. Spec. Mstr. Apr. 30, 2007) (finding that an otolaryngologist was not well suited to testify about disciplines other than his own specialty).

VI. Conclusion

Petitioner has clearly suffered and he has my sympathy. However, for all the reasons discussed herein, petitioner has not preponderantly demonstrated that he actually suffered a vaccine-caused injury and is therefore not entitled to compensation. Accordingly, this case is dismissed.[13]

IT IS SO ORDERED.

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

[13] In the absence of a timely-filed motion for review of this Decision, the Clerk of the Court shall enter judgment accordingly.