In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: January 8, 2025

RODNEY KOEHL,	*	PUBLISHED
Petitioner,	*	No. 20-0190V
v.	*	Special Master Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Hepatitis B ("Hep B") Vaccine; Epidermolysis Bullosa Acquisita ("EBA").
Respondent.	*	

Isaiah Kalinowski, Bosson Legal Group, P.C., Fairfax, VA, for Petitioner.
Tyler King, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

On February 24, 2020, Rodney Koehl ("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. (2018),[2] alleging that he suffered injuries, including epidermolysis bullosa acquisita ("EBA"), as a result of a hepatitis B ("Hep B") vaccination administered to him

[1] Because this Decision 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 and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision 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 (2018) ("Vaccine Act" or "the Act"). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

AUTHENTICATED U.S. GOVERNMENT INFORMATION GPO

on December 22, 2016.[3] Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating "this case [was] not appropriate for compensation under the terms of the [Vaccine] Act." Respondent's Report ("Resp. Rept.") at 2 (ECF No. 23).

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards, the undersigned finds Petitioner has failed to provide preponderant evidence that his Hep B vaccinations caused him to develop EBA. Thus, Petitioner has failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, the petition must be dismissed.

I. ISSUES TO BE DECIDED

Diagnosis is not at issue. The parties agree that Petitioner suffers from EBA. Pretrial Joint Submission, filed Dec. 6, 2023, at 1 (ECF No. 64). However, the parties dispute onset of Petitioner's EBA. Id. at 2. They also dispute whether there were preexisting conditions or intercurrent infections "that bear upon causation." Id.

Next, the parties dispute causation and whether Petitioner has provided preponderant evidence for all three Althen prongs. Pretrial Joint Submission at 2.

At the hearing, additional issues were identified that required clarification. See Order dated Jan. 17, 2024 (ECF No. 82). These issues were addressed by the parties in a post-hearing joint submission. Posthearing Joint Submission, filed Apr. 17, 2024 (ECF No. 101). Petitioner confirmed his position that all three Hep B vaccinations "were casual factors in bringing about, and/or significantly aggravating Petitioner's EBA." Id. at 2.

[3] Although Petitioner's petition only alleged injury due to his first Hep B vaccine administered on December 22, 2016, he received a three-dose series of Hep B vaccinations, with the second dose administered on January 23, 2017 and the third dose administered on July 24, 2017. Petitioner's Exhibit ("Pet. Ex.") 9 at 1-2. On March 5, 2020, the Court issued an order to show cause why Petitioner's claim should not be dismissed based on the three-year statute of limitations set forth in the Vaccine Act. Order to Show Cause dated Mar. 5, 2020 (ECF No. 8); see § 16(a)(2). Petitioner responded to the order by asserting that his vaccination occurred in three doses, beginning December 22, 2016 and completing July 24, 2017. Pet. Response to Order to Show Cause ("Pet. Decl."), filed Apr. 29, 2020, at 1 (ECF No. 12-1). At the hearing, Petitioner's expert, Dr. Alan N. Moshell, opined that all three of the Hep B vaccinations given to Petitioner were part of a three-dose series that stimulated Petitioner's immune system and played a causal role in his development of EBA. Tr. 230-34. Petitioner subsequently filed an amended petition, alleging that his EBA was caused by or significantly aggravated by the series of Hep B vaccinations. Amended ("Am.") Petition, filed Jan. 15, 2024, at 9 (ECF No. 79). Therefore, the undersigned evaluates the causal role of not only the Hep B vaccine administered on December 22, 2016, but also those administered on January 23, 2017 and July 24, 2017. And given the undersigned's findings as to onset, significant aggravation is not implicated or addressed in this Decision. See infra note 4.

The parties also confirmed that they disagreed about whether Petitioner's claims are time-barred. Posthearing Joint Submission at 2-4. However, the statute of limitations issue is moot due to the undersigned's finding herein that the onset of Petitioner's EBA was late August 2017 or early September 2017. See infra Section IV.C. Based on this finding, and the fact that the petition was filed on February 24, 2020, Petitioner filed his claim within the three-year statute of limitations under the Vaccine Act. Therefore, the petition was timely filed.

Lastly, in the post-hearing joint submission, Respondent asserted that Petitioner had failed to prove all three Althen prongs by reliable and persuasive evidence, regardless of whether Petitioner pursued a causation-in-fact claim or significant aggravation claim.[4] Posthearing Joint Submission at 4.

II. BACKGROUND

A. Procedural History

Petitioner filed his petition, pro se, on April 24, 2020 along with an expert report from Dr. Alan N. Moshell and medical records.[5] Petition; Pet. Exs. 1-8. The case was reassigned to the undersigned in July 2020. Notice of Reassignment dated July 31, 2020 (ECF No. 17). Respondent filed his Rule 4(c) report on November 25, 2020, arguing against compensation. Resp. Rept. at 2.

On July 19, 2021, Respondent filed an expert report from Dr. Emanual Maverakis. Resp. Ex. A. Petitioner filed a supplemental expert report from Dr. Moshell on October 1, 2021 and Respondent filed a supplemental expert report from Dr. Maverakis on April 26, 2022. Pet. Ex. 15(A); Resp. Ex. B.

Thereafter, a status conference was held to discuss next steps. Order dated May 19, 2022 (ECF No. 51). Respondent was not interested in settlement, and Petitioner wished to resolve entitlement through a hearing. Id. at 1. An entitlement hearing was set for January 2024. Prehearing Order dated June 3, 2022 (ECF No. 52). Thereafter, Mr. Kalinowski filed a

[4] Significant aggravation was alleged in Petitioner's amended petition but was not referenced in the parties' pre-hearing Joint Submission. See Am. Petition; Pretrial Joint Submission. The undersigned requested clarification from the parties. See Order dated Jan. 17, 2024. In their post-hearing joint submission, Petitioner asserts he "has pled significant aggravation . . . contingent on the Court's factual findings. . . . If the Court finds that the onset of EBA did not take place until Petitioner suffered from widespread, scarring blisters, then the three doses of [Hep B] were [alleged as] causal for that initial onset, and no significant aggravation is implicated." Posthearing Joint Submission at 3. The undersigned agrees with Petitioner's analysis. Based on the finding that onset of Petitioner's EBA did not occur until he had blisters on his hands, feet, and mouth, significant aggravation is not implicated or addressed in this Decision. See infra Section IV.C.

[5] Medical records were filed throughout litigation.

consented motion to substitute as Petitioner's counsel. Consented Motion to Substitute Attorney of Record, filed June 8, 2022 (ECF No. 53).

An entitlement hearing was held on January 10-11, 2024. Transcript ("Tr.") 1, 158. Petitioner, Cynthia Koehl, Dr. Moshell, and Dr. Maverakis testified at the hearing. Tr. 3, 160. A status conference was held on January 17, 2024 to address issues that arose in the hearing. Order dated Jan. 17, 2024. Both parties filed post-hearing exhibits. Pet. Exs. 60-88; Resp. Ex. D. A post-hearing joint submission was filed on April 17, 2024, following by a post-hearing brief from Petitioner on April 18, 2024 and a post-hearing brief from Respondent on May 16, 2024. Posthearing Joint Submission; Pet. Posthearing Memorandum in Support of Pet. Entitlement to Compensation ("Pet. Posthearing Memo."), filed Apr. 18, 2024 (ECF No. 102); Resp. Post Hearing Brief ("Br."), filed May 16, 2024 (ECF No. 105).

This matter is now ripe for adjudication.

B. Factual History

1. Summary of Medical Records[6]

Petitioner was born on November 7, 1967. Pet. Ex. 1 at 2. At the time of his vaccinations, Petitioner was employed by OSF Medical Systems ("OSF") in Illinois, where he worked in the building maintenance department. Pet. Ex. 2 at 49, 89. His past medical history was significant for atrial fibrillation, gastroesophageal reflux disease, anxiety, alcohol use, and smoking. Pet. Ex. 10-1 at 65, 154, 171.

On December 22, 2016, Petitioner presented to OSF Center for Occupational Health ("OH") and reported that he was cleaning out a sewer drain when his hand was "stuck by a needle." Pet. Ex. 2 at 88-89. He was wearing gloves, and when he took off the gloves, his finger was bleeding. Id. OSF OH ordered a blood borne pathogen exposure ("BBPE") evaluation. Id. at 90. The BBPE was negative for human immunodeficiency virus ("HIV") and hepatitis C and showed that Petitioner was not immune to hepatitis B. Id. at 93-95. OH gave Petitioner the first dose of the Hep B vaccine. Id. at 49; Pet. Ex. 9 at 1, 4.

On January 23, 2017, Petitioner received his second dose of the Hep B vaccine. Pet. Ex. 2 at 49; Pet. Ex. 9 at 1, 5.

Approximately one month later, on February 27, 2017, Petitioner presented to Dr. Philip Fifield at OSF. Pet. Ex. 3 at 1. Petitioner reported irritation of the skin above his right eye that

[6] This summary is taken, in part, from Respondent's Rule 4(c) report, as the undersigned finds it to be an accurate accounting of the medical records. See Resp. Rept. at 2-7. It has been edited to include additional relevant information from the records. For an additional factual summary, see Pet. Memo. in Support of Pet. Entitlement to Compensation ("Pet. Prehearing Memo."), filed Nov. 15, 2023, at 2-12 (ECF No. 63).

began two weeks earlier.[7] Id. Initially there was raised and irritated skin, and then some skin sloughing. Id. Petitioner did not specifically recall a blister. Id. The area healed and then he had a recurrence "lateral to the original area." Id. Physical examination showed a patch of skin (3/4 x 1 cm) "superior and nasal to the right eye" that was "erythematous and slightly raised." Id. at 2. "Toward the nasal aspect" there was "a linear area with some scabbing." Id. "There [were] no vesicles[,] open areas[,] or ulcers." Id. Dr. Fifield suspected either herpes zoster (shingles) or impetigo (infection) and prescribed anti-zoster therapy (famciclovir) and Bactroban topical ointment. Id. Petitioner was to follow up as needed. Id. at 3.

Petitioner returned to OSF and saw Physician Assistant ("PA") Korte Knoblauch on April 6, 2017 for an "erythematous rash above the right eye." Pet. Ex. 3 at 5. Petitioner reported that his previous rash had resolved with treatment but returned again after he completed the antiviral medication for suspected herpes zoster, and the Bactroban did not provide relief. Id. He reported that the rash had blistered, burned, had improved, but then returned. Id. Physical examination revealed a "[p]apular erythematous rash [] on the right superior eyelid" without vesicles or pustules. Id. at 6. PA Knoblauch referred Petitioner to Illinois Eye Center for further evaluation that same day. Id. at 7.

Ophthalmologist Dr. Thomas Wyman examined Petitioner on April 6, 2017. Pet. Ex. 4 at 2. He documented that the onset of Petitioner's rash was four to five weeks prior, and that it began with "burning, peeling skin on [his] upper lid." Id. Petitioner now had three "bumps" on his eyelid. Id. Dr. Wyman diagnosed "allergic dermatitis of the right upper eyelid." Id. at 5. He prescribed triamcinolone topical cream and instructed Petitioner to discontinue treatment with the antibiotic ointment. Id. Petitioner followed up with Dr. Wyman on April 14, 2017. Id. at 7. He was using the prescribed ointment two to three times a day and seemed to be improving. Id. Petitioner was to follow up as needed. Id. at 10.

Next, on May 23, 2017, Petitioner saw dermatologist Dr. Christopher Kroodsma at Central Illinois Dermatology for consultation. Pet. Ex. 5 at 2. Petitioner reported improvement with the cream prescribed by Dr. Wyman, but the rash had returned and was not healing. Id. Inspection showed a "very shallow erosion" (2-3 mm) of the right upper eyelid. Id. Petitioner also had brown macules (1-2 mm) on the anterior and posterior trunk and skin-colored papule(s) (3-4 mm) on the right posterior shoulder and mid-upper back. Id. No lesions on the hands or arms were noted. Id. Dr. Kroodsma diagnosed "[n]eoplasm of uncertain behavior, right upper eyelid." Id. He performed a shave biopsy which "did not show skin cancer, nor [herpes simplex virus ("HSV")] infection" but was consistent with an angiofibroma.[8] Id. Specifically, the eye lesion biopsy showed a "[p]apular skin lesion with fibrosis of the papillary dermis, vascular proliferation[,] and enlargement of some fibroblasts. The surface of the specimen [was] focally eroded and covered by scale-crust." Id. at 9. "Deeper levels" of the specimen did not show any

[7] Two weeks earlier would have been approximately February 13, 2017.

[8] Angiofibroma is "a lesion characterized by fibrous tissue and vascular proliferation; it often occurs as one or more small, flesh-colored papules, particularly on the face." Angiofibroma, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=2804 (last visited Dec. 6, 2024).

additional pathology. Id. Stains were negative for evidence of fungal or bacterial etiology. Id. Pathological diagnosis was "excoriated angiofibroma." Id. Dr. Kroodsma recommended treatment with topical Vaseline as needed. Id. at 2.

On July 24, 2017, Petitioner received his third Hep B vaccine. Pet. Ex. 2 at 49; Pet. Ex. 9 at 1-2.

On July 27, 2017, Petitioner saw Anthony Cutinello, D.O., at the Veterans Affairs ("VA") outpatient clinic for an annual checkup. Pet. Ex. 10-1 at 152. Petitioner reported that "within the last year he had a 'breakout' on [his] right upper eyelid," was seen by a dermatologist who performed a biopsy, and told him it was not cancer. Id. More recently, he had an episode beginning on May 18, 2017 and another episode that started on June 19, 2017. Id. Petitioner reported "a total of [eight] episodes." Id. They started as a pimple that would swell and develop scabs. Id. He also reported a "splash of sewage into his eyes" in December 2016. Id. He also described the needle stick injury of his right pinky finger. Id. Petitioner was currently smoking a pack of cigarettes daily. Id. at 153. Physical examination noted no skin abnormalities. Id. "The right upper lid appear[ed] normal" without swelling or redness. Id. Assessment was "[l]ikely blepharitis,[9] right upper lid." Id. Dr. Cutinello reviewed pictures on Petitioner's phone and it "appear[ed] that he had pustular swelling of the right eye." Id. Petitioner was to follow up in one year for a checkup and follow up sooner if he had a recurrence of blepharitis. Id. at 154. Petitioner received a pneumococcal 23 vaccination at this visit.[10] Id.

Petitioner saw his dentist, Mark D. Zaayenga, D.D.S., on January 18, 2018 and had an oral examination and X-rays. Pet. Ex. 87 at 2.

On January 26, 2018, Petitioner returned to Dr. Cutinello for a consultation regarding "blisters." Pet. Ex. 10-1 at 136-37. Petitioner stated his dentist advised him to seek medical treatment due to concern that Petitioner could have a "systemic" condition. Id. at 137. Petitioner had a "bad infection" in his jaw the prior week, requiring antibiotics. Id. He also reported that he would "get out of the shower and rip a layer of skin off of his eyelids and the bridge of his nose." Id. He had blisters in his mouth and lesions on his hands that cleared up while he was taking penicillin for his dental issue. Id. Petitioner explained that his skin was exposed to raw sewage at OSF where he worked. Id. He could go for "two weeks without any symptoms." Id. Petitioner was under a lot of stress and was treating with alprazolam prescribed by his cardiologist. Id. Physical examination did not reveal any periorbital (around the eye) rash or blistering. Id. at 138. However, Petitioner had erythematous areas on his left hand and foot dorsally without blistering or ulceration. Id. The left hand had "resolved areas" of circular redness with no "open areas." Id. Dr. Cutinello's assessment included "[h]emorrhagic blisters." Id. Plan included bloodwork and if the bloodwork was negative, Dr. Cutinello recommended Petitioner speak to his cardiologist about discontinuing his propafenone (antiarrhythmic). Id.

---

[9] Blepharitis is "inflammation of the eyelids." Blepharitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=6509 (last visited Dec. 6, 2024).

[10] The pneumococcal 23 vaccine is not a covered vaccine under the Vaccine Act. 42 C.F.R. § 100.3(a). Petitioner has not alleged that he suffered any injury as a result of this vaccination.

Dr. Cutinello also recommended Petitioner consider a dermatology consultation via telemedicine, continue his current medications, and follow up in six months. Id.

Moving forward to March 28, 2018, Petitioner followed up with OSF OH regarding his worker's compensation ("WC") claim related to "irritation of the skin on the dorsal surfaces of bilateral hands with thinning of skin" since he began at OSF in September 2016. Pet. Ex. 9 at 13. He reported that that morning, he "tore off a large layer of his skin" when removing a band-aid. Id. Physical examination showed evidence of scarring on the dorsal surfaces of the hands bilaterally; multiple scattered open areas on the dorsal surfaces of the hands and fingers bilaterally (seven areas on the left hand and six on the right hand), which were weeping a scant amount of serous fluid and measuring 1 cm or less, except for one large area measuring 3 x 2 cm on the dorsal surface of the right hand proximally; and his sensation was grossly intact. Id. Assessment was "[u]nspecified open wound of [the] hand." Id. at 14. Adaptic was applied, along with gauze and tape. Id. Petitioner was instructed to keep open areas clean and covered and to stop using nitrile gloves. Id.

On April 4, 2018, Petitioner returned to OSF OH regarding his WC claim related to "bilateral hand open wounds due to thinning skin and possible irritation to nitrile gloves, which seemed to start after beginning work at OSF in September 2016." Pet. Ex. 2 at 56. Petitioner "[r]eport[ed] overall improvement of wounds with avoidance of nitrile gloves and application of Adaptic and gauze dressings with some scab formation." Id. Physical examination was largely consistent with his March 28 examination, except for evidence of areas that had begun to scab, but continued to weep serous fluid, and a new blister was noted on the left dorsal hand measuring approximately 11 x 7.5 mm. Id. at 56-57. Assessment was "[u]nspecified open wound of [the] hand" and "[h]ealing bilateral hand wounds with one new area of blistering." Id. at 57. Petitioner was advised to continue covering open areas with Adaptic, gauze, and Coban bandage dressing; avoid nitrile gloves; use gentle soap and water to cleanse hands as needed; and try wearing cotton gloves under any other gloves used. Id.

Due to concerns about his blisters, Petitioner returned to his dermatologist Dr. Kroodsma on May 1, 2018. Pet. Ex. 5 at 3. Physical examination revealed "[m]ultiple excoriations and some erosions, as well as some patchy erythema on the hands, arms, feet[,] and legs, with some mild background erythema on the face, but no active lesions on the neck, chest, abdomen[,] or back." Id. Dr. Kroodsma documented that this "issue seemed to begin about [one-and-one-half] years ago." Id. A shave biopsy was performed "to assess for potential bullous pemphigoid porphyria[11] or drug reaction or other such process." Id. Petitioner returned to see Dr. Kroodsma on May 3, 2018 for a patch test, which was negative. Id. Dr. Kroodsma noted that Petitioner had "a fair amount of Raynaud type of symptoms involving the hands and feet, with cold temperatures of the skin and some blanching and violaceous appearing areas" that "may be causing some of his problems." Id. The plan was to see what the previous biopsies showed. Id. On May 10, 2018, the biopsy results were reported to Petitioner by Dr. Kroodsma via phone. Id.

[11] Bullous pemphigoid is "a usually mild, self-limited, subepidermal blistering skin disease, sometimes with oral involvement, predominantly affecting the elderly." Bullous Pemphigoid, Dorland's Online Med. Dictionary, https://www.dorlandsonline.com/dorland/definition?id=97045 (last visited Dec. 6, 2024).

The biopsies were consistent with "bullous pemphigoid or possibly [EBA]." Id. at 4, 12. Prednisone 20 mg daily was prescribed, and Petitioner was instructed to return to the office on May 24. Id. at 3.

Petitioner returned to see Dr. Kroodsma on May 24, 2018, reporting that prednisone seemed to have helped. Pet. Ex. 5 at 4. Dr. Kroodsma decreased Petitioner's prednisone dose and recommended Petitioner see a consultant at the University of Iowa ("UI") Dermatology Program for further evaluation and treatment. Id. After Petitioner obtained approval from his insurance, Dr. Kroodsma referred him to Mayo Clinic. Id.

On July 24, 2018, Petitioner saw dermatologist Dr. Rokea El-Azhary at Mayo Clinic. Pet. Ex. 6 at 8. Petitioner reported that since he was diagnosed with bullous pemphigoid, he had not had many blisters, but prednisone did not seem to help. Id. A physical examination revealed scars of bullous lesions. Id. Dr. El-Azhary also examined a "[l]arge file of photographs" that Petitioner brought with him, which showed tense blisters on the dorsum of the hands; lesions on the dorsum of the hands, fingers, ankles, and feet; and dorsum of all areas of the feet could be traumatized by work, shoes, walking, etc.; no evidence of anything in the mouth or on the eyes; and no blisters on the torso. Id. The biopsies taken by Dr. Kroodsma on May 1, were reviewed by pathologists at the Mayo Clinic, and the pathological diagnosis was "[s]ubepidermal separation with superficial dermal neutrophilic inflammation." Id. at 2-5. Additional studies were done (serum for indirect immunofluorescence[12] and ELISA antibody testing),[13] including a punch biopsy from the left finger. Id. at 9-32. The biopsy was suboptimal to rule out an "autoimmune mucocutaneous blistering disorder." Id. at 10-11. Serum for cutaneous immunofluorescence antibodies were negative for an autoimmune mucocutaneous blistering disorder. Id. at 23. However, after testing was complete, in a letter to Dr. Kroodsma dated August 14, 2018, Dr. El-Azhary wrote that "[a]ll of [Petitioner's] test results point to [EBA] and not to bullous pemphigoid." Id. at 38. Treatment with Dapsone and "strict avoidance of trauma" was recommended. Id.

---

[12] Immunofluorescence is "any immunohistochemical method that uses antibody labeled with a fluorescent dye." Immunofluorescence, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24888 (last visited Dec. 6, 2024). Indirect immunofluorescence is "immunofluorescence that involves a fluorochrome attached to an antiglobulin, with a tissue constituent that is stained using an unlabeled specific antibody and the labeled antiglobulin, which binds the unlabeled antibody." Indirect Immunofluorescence, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=82029 (last visited Dec. 6, 2024).

[13] ELISA is "any enzyme immunoassay utilizing an enzyme-labeled immunoreactant (antigen or antibody) and an immunosorbent (antigen or antibody bound to a solid support). A variety of methods (e.g., competitive binding between the labeled reactant and unlabeled unknown, or a sandwich technique in which the unknown binds both the immunosorbent and labeled antibody) may be used to measure the unknown concentration." ELISA, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=15958 (last visited Dec. 6, 2024).

Petitioner treated with Dapsone under Dr. Kroodsma until September 2018, when he discontinued treatment due to an apparent adverse reaction. Pet. Ex. 5 at 5-6. Petitioner's wife called Dr. Kroodsma and reported that Petitioner was feeling ill but had still been working because his employer would not give him light duty. Id. at 6. Dr. Kroodsma reverted to treatment with triamcinolone and added Plaquenil. Id. at 7.

Dr. Kroodsma referred Petitioner to UI Hospitals & Clinics for another consultation, and on February 12, 2019, Petitioner saw dermatologist Dr. Janet Fairley. Pet. Ex. 7 at 2. Physical examination showed "healing erosions with overlying crust on the hands, arms, and feet;" "milia on the dorsal hands;" "erythema of the hard palate;" and no other skin lesions of concern. Id. at 4. Dr. Fairley's assessment was EBA. Id. She noted Petitioner's condition was not well controlled on steroid therapy and Dapsone treatment was contraindicated due to a previous adverse reaction. Id. Rituximab treatment was recommended. Id. In an addendum dated February 22, Dr. Fairley noted Petitioner's Anti-Type VII Collagen Antibodies test[14] was within the normal limits. Id. at 4-5. She opined Petitioner's condition remained consistent with EBA. Id. at 5. Petitioner was treated with weekly infusions of rituximab from March 19, 2019 to April 10, 2019, and he tolerated the treatment well. Id. at 6-11, 15.

Medical records from 2023 show that Petitioner was seeking a dermatologist in Florida to treat his EBA. See, e.g., Pet. Ex. 37 at 142-43, 313-14. On March 29, 2023, Petitioner saw dermatologist Dr. Navid Farahbakhsh. Pet. Ex. 37 at 737-39. Physical examination did not reveal any active blisters although Petitioner provided photos of prior "erosions and bulla via cellphone." Id. at 738. Petitioner was advised to start Rituxan infusions and to follow up in four months. Id.

No further relevant records have been filed.

2. Testimony and Declarations

a. Petitioner

Petitioner submitted a declaration, dated April 29, 2020, describing the rash over his right eye lid and averring that he first noticed it on February 25, 2017. Pet. Decl. at 1. He sought treatment for it on February 27, 2017 and was diagnosed with shingles. Id. at 1.

Regarding his EBA, Petitioner stated that it began in March of 2018, "after [his] skin began to tear off [his] hands due to slight bumps and bruises." Pet. Decl. at 2. He referenced his medical records to support his statement that his EBA started in 2018. Id. He further confirmed that he was seeking compensation for his EBA condition. Id.

[14] Type VII collagen is the autoantigen in EBA. Pet. Ex. 43 at 12 (Sidonia Mihai & Cassian Sitaru, Immunopathology and Molecular Diagnosis of Autoimmune Bullous Diseases, 11 J. Cellular & Molecular Med. 462 (2007)). For more information on Type VII collagen, see infra Section II.C.1.b.i.

In addition to this declaration, Petitioner testified at his entitlement hearing on January 10, 2024. Tr. 3. He explained that he was generally in good health prior to the vaccinations at issue. Tr. 8. In 2016, he was self-employed by his corporation, Triple D Plumbing. Tr. 9. He sought outside employment in August 2016, and was hired by OSF Healthcare as a plumber in the maintenance department of the hospital. Id. He began that job in September 2016. Id. On December 22, 2016, he was cleaning out a sewer pipe when he was stuck by a suture needle in his hand. Tr. 11. He received treatment for the wound at the OSF OH and was given antibiotics and Hep B vaccinations. Tr. 11-12.

At some point, he developed a problem with his eye, but due to the passage of time, Petitioner was unable to recall when his eye rash began, although it may have started in February 2017. Tr. 15-16, 22. He testified that no one ever told him that his eye symptoms were related to his EBA. Tr. 106.

Regarding the blisters on his hands, feet, elbows, and mouth, subsequently diagnosed as EBA, Petitioner testified that those blisters began sometime from August 2017 through the fall of 2017. Tr. 47-48. He sought treatment because his dentist, Dr. Zaayenga, noted blisters in Petitioner's mouth during a dental appointment and suggested Petitioner seek medical treatment.[15] Tr. 51. Petitioner sought treatment for his blisters from Dr. Cutinello, his primary care physician at the VA, on January 26, 2018. Id. Petitioner agreed with Dr. Cutinello's history and description of the events documented in the note from that visit. Tr. 51-53. When questioned again about onset, Petitioner explained that his wife looked at family photographs, and based on these, Petitioner believed his EBA began in August 2017. Tr. 105-06.

When asked how long it took for his blisters to heal, Petitioner answered that it could take as long as a month, especially those on his feet. Tr. 70. After the blisters healed, the skin would appear purple or pink, or there would be dark colored spots. Id.

b. Cynthia Koehl, Petitioner's Wife

Petitioner's wife, Cynthia Koehl, also testified at the hearing on January 10, 2024. Tr. 3. She and her husband have been married for 30 years. Tr. 120. In 2016, Mrs. Koehl was employed by Better Banks, where she had worked for over 30 years; she retired in May 2017. Id.

In December 2016, Mrs. Koehl testified that her husband was in good health. Tr. 122. He had no skin issues at that time. Tr. 122-23. He developed an eye rash at the end of February 2017. Tr. 126. She recalled that the eye rash began a couple of days before he saw a physician for the problem. Id. At that time, her husband did not have any skin problems on his hands. Id.

The first time that Mrs. Koehl recalls seeing blisters on her husband's hands was September 9, 2017. Tr. 128. She based this testimony on a photograph taken that day that

[15] The dental records had not been filed in the record at the time of the hearing. Tr. 108. The records were filed after the hearing and show that Petitioner's dental appointment was January 18, 2018. See Pet. Ex. 87 at 2.

showed a couple of blisters on his hand. Id. (citing Pet. Ex. 68). After reviewing the photograph, and preparing for the hearing, she believed her husband first experienced blisters on his hands around the end of August 2017 or beginning of September 2017. Tr. 132. A second photograph showing blisters was taken October 28, 2017. Tr. 135-36 (citing Pet. Ex. 71). Mrs. Koehl confirmed that her husband first sought treatment for his skin blisters on his hands and feet in January 2018. Tr. 138-39.

After the hearing, Mrs. Koehl submitted a declaration, addressing the production of photographs from her mobile phone which were introduced at the hearing, and filed by Petitioner as exhibits 60-83. Pet. Ex. 88. She averred as to the authenticity of the photographs. Id. at 3.

C. Expert Reports

1. Petitioner's Expert, Dr. Alan Moshell[16]

a. Background and Qualifications

Dr. Moshell is a board-certified dermatologist with expertise in EBA. Pet. Ex. 8 at 2. He obtained his M.D. from New York University School of Medicine in 1971 and then completed an internship, residency, and fellowship in dermatology. Pet. Ex. 25(C) at 1. Throughout his career, he held various positions at the National Institute of Health ("NIH") until his retirement from NIH in 2006. Id. at 1-2; Tr. 169. Thereafter, he began to work in the dermatology department at Georgetown University School of Medicine where he is currently a Clinical Associate Professor. Pet. Ex. 25(C) at 2. He spends about 80% of his time treating patients and has seen and treated patients with EBA Tr. 170, 192-93. Dr. Moshell has authored or co-authored over 50 publications. Pet. Ex. 25(C) at 5-9.

b. Opinion

Dr. Moshell agreed that Petitioner's diagnosis of EBA was appropriate. Tr. 174; Pet. Ex. 15(A) at 1. EBA is an "autoimmune blistering disease." Pet. Ex. 43 at 2. In these conditions, "autoantibodies induce blisters on skin or mucous membranes." Pet. Ex. 46 at 2.[17] There are four major groups of autoimmune blistering diseases, including "pemphigus diseases and pemphigoid diseases, [EBA], and dermatitis herpetiformis." Pet. Ex. 43 at 2. These groups are categorized based on clinical presentation, histopathological features, and immunopathological involvement, including structures and autoantigens. Id. at 2, 2 tbl.1. "EBA is very rare," reported in less than 0.2 to 0.5 per million persons, whereas bullous pemphigoid (a pemphigoid disease) is "a hundred to a thousand times more common." Tr. 191; Pet. Ex. 15(A) at 1. EBA usually is diagnosed due to recurring blisters on the hand and feet that occur at sites of

[16] Dr. Moshell provided a letter and one expert report and testified at the hearing. Pet. Exs. 8, 15; Tr. 160.

[17] Hiroaki Iwata et al., B Cells, Dendritic Cells, and Macrophages Are Required to Induce an Autoreactive CD4 Helper T Cell Response in Experimental Epidermolysis Bullosa Acquisita, 191 J. Immunology 2978 (2013).

mechanical trauma and rubbing of the skin. Tr. 264. Multiple episodes of blistering that occur with less trauma than expected often leads patients to seek medical treatment. Tr. 265.

At the hearing, Dr. Moshell reviewed two photographs. Tr. 221-25. The first was taken September 9, 2017, showing Petitioner's hands. Tr. 223. Dr. Moshell opined that it showed broken blisters on the back of Petitioner's right hand and fifth finger, and a healing lesion near the elbow, all consistent with EBA. Id. The second photograph, taken October 28, 2017, showed a lesion on the right eye, but it could not be seen well and Dr. Moshell was unable to reach any conclusion based on it. Tr. 223-24. In the same photograph, Petitioner's hand had a red lesion "consistent with a broken blister from EBA," although Dr. Moshell could not be certain. Tr. 224. Dr. Moshell agreed that both photographs were consistent with those he had previously reviewed prior to the hearing. Tr. 225. Based on the photographs and medical records, Dr. Moshell agreed there was sufficient evidence to confirm Petitioner's diagnosis of EBA reached by his treating physicians. Id.

i. Althen Prong One

Dr. Moshell opined that although EBA has been shown to be caused by the development of autoantibodies, what triggers the development of these autoantibodies is not known. Pet. Ex. 8 at 3. He explained that while the autoantibodies are known, and their effect on type VII collagen is understood, the question of what induces the autoantibodies is "medically unknown." Id. at 2-3.

According to Dr. Moshell, "[t]he cause of all of the autoimmune blistering skin diseases is the development of [] autoantibodies."[18] Pet. Ex. 8 at 3. Specific to EBA, the autoantibodies "attack and disrupt" type VII collagen,[19] a component of the skin that attaches the epidermis (outer layer of skin) to the dermis (inner layer of skin) known as the basement membrane zone. Id. at 2; Tr. 185. Antibodies are directed against type VII collagen or other molecules that physically exist where type VII collagen exists in the basement membrane zone (the thin layer between the epidermis and dermis). Tr. 185, 269; Pet. Ex. 8 at 2. There is "targeting of molecules at the lower edge of the basement membrane zone [] and/or the upper parts of the dermis." Tr. 174.

He explained in more detail the pathogenesis of EBA. "Collagen VII is the molecule that makes up the structure of the skin called anchoring fibrils." Tr. 182. Collagen VII molecules line up "side by side" in the lowest level of the basement membrane zone and the upper level of the dermis to create these anchoring fibrils. Id. The anchoring fibrils are structures in the basement membrane zone that are "intimately involved in connecting the epidermis to the

[18] Dr. Moshell's statement is supported by medical literature. See, e.g., Pet. Ex. 43 at 1 (noting "the pathogenicity of autoantibodies . . . has been conclusively demonstrated experimentally").

[19] EBA is "distinguished from other autoimmune blistering diseases by the production of antibodies against type VII collagen." Pet. Ex. 49 at 1 (Erik A. Kumetz et al., Epidermolysis Bullosa Acquisita: A Case Report, 21 Am. J. Case Reps. e919432-1 (2020)).

dermis, overall tacking the epidermis down to the dermis." Tr. 183. In the autoimmune form of EBA, anchoring fibrils are damaged by autoantibodies that attack and cause inflammation, rendering them unable to function. Tr. 183, 279. When there is a blister on the skin, the dermis is the pink skin at the bottom of the blister and the epidermis sits on top of the blister. Tr. 185.

Another autoimmune blistering condition is bullous pemphigoid, which is "much more common," and often the first diagnosis when a patient presents with recurrent blisters. Tr. 187. However, Dr. Moshell acknowledged that the two differ in "clinical characteristics and [] immunologic testing."[20] Id.

Once stimulated, Dr. Moshell posited that some patients, "whose immune system is capable of developing these antibodies[,] will develop the ones that lead to disease." Pet. Ex. 8 at 3. Dr. Moshell suggested that the "medical reasoning" is that some patients have the "ability to produce autoantibodies," but not at the level that causes disease until something happens to "turn on the production of the autoantibodies to the level that they produce[] clinical disease."[21] Tr. 197.

After explaining the pathophysiology of EBA, Dr. Moshell proffered his causal theory related to vaccination. He proposed there is a "broad immune activation in those patients who are able to produce the antibodies, type VII collagen or the other molecules in the section of the basement membrane zone," that induces disease.[22] Tr. 205-06. He asserted that this general activation of the immune system increases the production of all antibodies. Tr. 280. Once the process begins, Dr. Moshell testified that it is like an avalanche. Tr. 207-08. Hep B stimulates the immune system, and once stimulated, the process continues for the rest of a patient's life, unless treatment is given to "tamp down" the immune system. Tr. 208.

---

[20] The majority of bullous pemphigoid cases are diagnosed with ELISA testing for antibodies to type XVII collagen, which disrupts attachment or creates a "localized inflammatory reaction." Tr. 188. The other bullous pemphigoid cases are diagnosed by immunofluorescence testing that shows human immunoglobulin ("Ig") in the superficial level of the basement membrane zone. Id. For a detailed discussion of pemphigoid diseases, including bullous pemphigoid, see Pet. Ex. 43 at 4-12.

[21] For further discussion, see Pet. Ex. 44 at 7-8 (Cassian Sitaru, Experimental Models of Epidermolysis Bullosa Acquisita, 16 Experimental Dermatology 520 (2007)).

[22] Dr. Moshell agreed that there may also be some bystander destruction, but he did not opine that bystander activation was the principal theory of causation. Tr. 198. "In bystander activation, there is a robust or exaggerated immune response to an exogenous agent that induces local tissue inflammation and stimulation of otherwise normal unaffected cells. This inflammation can result in the release of normally sequestered self-antigens. The inflammation can result in nonspecific activation of previously dormant autoreactive Th1 cells that then react against the newly released self-antigens." Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57, 75 (Kathleen Stratton et al. eds., 2012).

Relevant to his theory, Dr. Moshell assumed there is "an activation of the immune system in general," and that the "process of vaccination stimulates the immune system," activating autoreactive antibodies to attack and damage collagen VII. Tr. 198. Dr. Moshell suggested that the vaccination was the "preceding event" and were it not for vaccination, "the disease would not have occurred," although he was unable to "describe the specific steps involved" in "the disease development." Pet. Ex. 15(A) at 1-2.

Dr. Moshell viewed all three Hep B vaccinations as a three-dose series that constitutes the "vaccination." Tr. 234-35. He testified that the three Hep B vaccines are given in a series to induce the best immunity against development of Hep B infection. Id.

Dr. Moshell did not provide supportive evidence, studies, medical articles, or other foundational evidence to support his opinion that general or broad activation of the immune system by Hep B vaccination can induce or trigger the specific autoantibodies that cause EBA.[23] He conceded there is no medical literature that describes the triggers of immune activation for EBA, although he asserts there are such studies related to bullous pemphigoid. Tr. 199-200. Dr. Moshell attributed the lack of EBA studies to the fact that it is a rare disease which makes it difficult to study. Tr. 200, 209; Pet. Ex. 8 at 2; Pet. Ex. 15(A) at 1. Dr. Moshell referenced other illnesses as examples of how skin conditions can be caused by either infection or medications, including a form of psoriasis triggered by streptococcal infection, known as post-streptococcal guttate psoriasis,[24] and vancomycin[25]-induced linear IgA bullous dermatosis. Pet. Ex. 15(A) at 1. None of Dr. Moshell's literature described an association between the Hep B vaccine and EBA. He agreed that none of the case reports filed reported EBA as a diagnosis. Tr. 256.

---

[23] Many articles referenced by Dr. Moshell do not discuss Hep B vaccination or autoimmune blistering skin diseases, and are therefore not discussed as they are either not relevant, or less relevant, to the issues herein.

[24] Psoriasis refers to "any of a group of common chronic, squamous dermatoses with variable symptoms and courses; some are inherited. Principal histologic findings are Munro microabscesses and spongiform pustules; also seen are rounded, circumscribed, erythematous, dry, scaling patches of various sizes, covered by gray, silvery, or white, umbilicated, lamellar scales." Psoriasis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=41881 (last visited Dec. 6, 2024). Guttate psoriasis is "psoriasis characterized by the abrupt appearance of small droplike lesions over much of the skin surface, especially on the trunk and proximal limbs; seen primarily in children and young adults, especially following streptococcal infections." Psoriasis Guttata, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100775 (last visited Dec. 6, 2024).

[25] Vancomycin is "an antibiotic [that] is highly effective against cocci, especially staphylococci, and other gram-positive bacteria." Vancomycin Hydrochloride, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=52529 (last visited Dec. 6, 2024).

Koga et al.[26] describes EBA, its clinical presentation, different forms of the illness, diagnostic studies, and therapy. See Pet. Ex. 16(A1). The authors confirm that the cause of EBA is not known. Id. at 11. While the cause is unknown, the authors suggested it involves a genetic predisposition, accompanied by "environmental factors or infections." Id. Animal models suggest that certain bacteria may be implicated. Id. at 11-12. But there is "little human data" to explain the pathogenesis of this complex disease. Id. at 12. The authors also discussed the development of antibody production. Id. at 12. They proposed that EBA is caused by tolerance loss due to genetic factors and the skin microbiome. Id. at 13 fig.5. Bacterial infection was identified as a causal factor in the animal studies described, however, vaccination was not mentioned. See id. at 1-20. Similarly, Sitaru stated, "the genetic and/or environmental events triggering autoimmunity against type VII collagen[] . . . remain to be elucidated." Pet. Ex. 44 at 8.

Another reason Dr. Moshell offered for the lack of a published association between Hep B vaccination and EBA is the lack of research "to confirm post immunization effects." Pet. Ex. 15(A) at 1. Dr. Moshell suggested that only a "small subset . . . could get EBA from the vaccination," presumably meaning there must be a genetic predisposition. Id. He stated "[t]here always must be a first report, which may only be the first time the relationship is recognized even if it happened in other people previously." Id.

Other articles cited by Dr. Moshell include case reports of bullous pemphigoid, not EBA, after vaccination. Tr. 254. Dr. Moshell agreed that bullous pemphigoid and EBA are different diseases.[27] Tr. 255-56. He cited a case report by Berkun et al.,[28] who wrote that while the etiology of pemphigus is unknown, it is occasionally associated with an antecedent factor. Pet. Ex. 17(A2) at 1. In Berkun et al., the patient received the first dose of a Hep B vaccination three months prior to onset. Id. The authors acknowledged that that association between vaccination and illness was "temporal only." Id. at 3. In their discussion, they mention several possible causal mechanisms, including a non-specific activation of the immune system, molecular mimicry,[29] the mechanism that causes infections to cause autoimmune diseases, activation and expansion of autoreactive T cells, adjuvant-induced response, and activation of lymphocytes and antigen presenting cells. Id. None of these mechanisms were identified as a leading theory. See id. Further, the authors did not reach any conclusions as to any of the causal mechanisms identified. See id.

---

[26] Hiroshi Koga et al., Epidermolysis Bullosa Acquisita: The 2019 Update, 5 Frontiers Med. 1 (2019).

[27] For an analysis of the different autoimmune blistering diseases and their immunopathological characteristics, see Pet. Ex. 43 at 2 tbl.1.

[28] Yackov Berkun et al., Pemphigus Following Hepatitis B Vaccination—Coincidence or Causality?, 38 Autoimmunity 117 (2005).

[29] Dr. Moshell testified that molecular mimicry is not the proposed causal theory at issue here. Tr. 197, 269.

Baroero et al.[30] reviewed three cases of post-vaccination and post-viral bullous pemphigoid and offered a list of possible causal theories, including "nonspecific immune reactivation," "intrauterine transmitted maternal IgG antibodies" (in childhood cases), Th17 cell activation leading to increased IL-17 and release of pro-inflammatory cytokines, and CD25 deficiency. Pet. Ex. 18(A3) at 3. The authors concluded the "mechanism of induction [was] unclear," although there was a temporal relationship. Id. at 4. Similarly, de la Fuente et al.[31] discussed three cases of bullous pemphigoid in infants following vaccination and offered "nonspecific immune reactivation" and "intrauterine transmitted maternal IgG antibodies" as causal theories. Pet. Ex. 40 at 1-2. Like Baroero et al., the authors noted they were "uncertain" about the etiology of their cases but noted the temporal relationship between vaccination and onset of lesions. Id. at 2. Other causal theories suggested in bullous pemphigoid cases include "[s]pecific immune stimulation of hepatitis B surface antigen . . . to induce immune complex formation" and "T lymphocyte activation." Pet. Ex. 39 at 4.[32]

Another paper referenced by Dr. Moshell described a systematic review of literature related to vaccination and its association with autoimmune bullous diseases. See Pet. Ex. 38.[33] Twenty-six papers, published from 1995 to 2021, with 28 patients, were reviewed. Id. at 1. Most were case reports of bullous pemphigoid diseases. Id. Hep B vaccination was identified in three cases, none of which involved EBA. Id. at 1. The authors concluded that there was insufficient information to suggest that vaccinations increase the risk of autoimmune bullous diseases. Id. at 5.

Articles about the Covid-19 vaccination were also referenced by Dr. Moshell, including one case report of an EBA-like reaction after administration of the Pfizer Covid-19 vaccine. See Pet. Ex. 41.[34] The patient developed "mild itchiness, burning sensation, and mild redness over her hands and face" the evening that she received her vaccination. Id. at 2. The next day, she

---

[30] Luca Baroero et al., Three Case Reports of Post Immunization and Post Viral Bullous Pemphigoid: Looking for the Right Trigger, 17 BMC Pediatrics 1 (2017).

[31] Sonia de la Fuente et al., Postvaccination Bullous Pemphigoid in Infancy: Report of Three New Cases and Literature Review, 30 Pediatric Dermatology 741 (2013).

[32] Zülal Erbagci, Childhood Bullous Pemphigoid Following Hepatitis B Immunization, 29 J. Dermatology 781 (2002).

[33] Michael Kasperkiewicz & David T. Woodley, Association Between Vaccination and Autoimmune Bullous Diseases: A Systematic Review, 86 J. Am. Acad. Dermatology 1160 (2022).

[34] Ahlam Al-Zahrani et al., Epidermolysis Bullosa Acquisita-Like Cutaneous Reaction Reported After the 2nd Dose of Pfizer-Biontech Covid-19 Vaccination: A Case Report, 8 Indo Am. J. Pharm. Scis. 137 (2021). Of note, the Covid-19 vaccines were messenger RNA ("mRNA") based, unlike the Hep B vaccine. See Pet. Ex. 41 at 1; Pet. Ex. 19(A4) (Hep B package insert).

developed large blisters and she was subsequently diagnosed with an EBA-like disease. Id. at 2-6. Possible differential diagnoses included bullous pemphigoid, pemphigus, pityriasis lichenoid, and linear IgA bullous dermatosis. Id. at 5. The authors did not reach any conclusions about causation. See id. at 1-7.

Another article discussed disease exacerbations in some patients with autoimmune bullous disease after Covid-19 vaccinations. See Pet. Ex. 42.[35] Of the 446 patients studied, only two had EBA, and neither of these patients had an exacerbation of illness after vaccination. Id. at 5. The authors stated that "[t]he mechanisms of triggering disease activity after vaccination are unclear." Id. at 7. Several theories were suggested, including "excessive generation of type I interferons and proinflammatory cytokine[s]," "regulatory T cell[] dysfunction" (specifically as to pemphigus and pemphigoid disorders), and "[c]ytotoxic T cell activation . . . especially in [the] pemphigoid group." Id. No causal mechanisms were suggested specific to EBA. See id. at 1-8.

Overall, the medical literature referenced by Dr. Moshell supported his opinion that EBA is an autoantibody driven disease, "characterized by autoantibodies to structural proteins of the skin." Pet. Ex. 46 at 2. However, the literature also confirmed that what induces the development of the relevant autoantibodies is not known. Further, although a number of different possible causal mechanisms were identified in the articles, there was no consensus reached as to the most likely medical theory to explain disease induction.

ii. Althen Prong Two

Dr. Moshell agreed that Petitioner's diagnosis was EBA. Tr. 174; Pet. Ex. 15(A) at 1. Usually, a patient with EBA will test positive for antibodies to type VII collagen on ELISA testing. Tr. 190. Although Petitioner did not test positive for autoantibodies against type VII collagen, Dr. Moshell explained that their presence is not required to reach a diagnosis of EBA. Tr. 190, 248-49. Even if an ELISA test is not positive, there may be antibodies deposited in the basement membrane zone, and these antibodies may not be picked up by ELISA testing. Tr. 190. Further, immunofluorescent studies are more sensitive, although less specific, than ELISA tests because they look for deposits of human Ig in the deepest area of the basement membrane zone. Tr. 189-91; see also Pet. Ex. 8 at 2. Petitioner had immunofluorescent studies that showed IgG and complement at the dermoepidermal junction, confirming his EBA diagnosis. Tr. 248.

Regarding a logical sequence of cause and effect, Dr. Moshell testified that all three Hep B vaccines stimulated Petitioner's immune system to the point that he developed "full-blown and persistent EBA." Tr. 234-35. He further opined that Petitioner's clinical course and symptoms were consistent with the posited theoretical mechanism. Tr. 211. Petitioner's clinical course was progressive and rituximab had a positive effect and improved symptoms, which he opined was supportive of the causal theory. Tr. 211-12. He opined that a positive effect to treatment

[35] Nika Kianfar et al., Exacerbation of Autoimmune Bullous Diseases After Severe Acute Respiratory Syndrome Coronavirus 2 Vaccination: Is There Any Association?, 9 Frontiers Med. 1 (2022).

with rituximab supports the theory of immune stimulation because it suppresses the immune system, and specifically the B cell production of antibodies. Tr. 266-68.

Next, Dr. Moshell testified that Petitioner did not have any alternative cause for his EBA. Tr. 210-11; Pet. Ex. 15(A) at 2. There was no congenital condition that explained the development of his EBA. Tr. 210. Petitioner did not have any signs or symptoms of an antecedent or intercurrent infection or illness prior to the onset of his EBA. Tr. 210-11. Dr. Moshell specifically disagreed that Petitioner had a herpes zoster infection or that diagnosis of zoster in Petitioner (as related to the eye lesion) was accurate since no testing was done to confirm such a diagnosis. Pet. Ex. 15(A) at 2. Additionally, Dr. Moshell explained that Petitioner's only pre-existing condition was atrial fibrillation, for which he took medication. Id. And he stated there was no conclusive testing to implicate a drug or medication interaction as the cause of Petitioner's EBA. Id.

iii. Althen Prong Three

Dr. Moshell offered two different opinions about onset. In his expert reports, Dr. Moshell opined that the onset of Petitioner's EBA began within a few months after he received the first Hep B vaccine on December 22, 2016, when Petitioner developed "areas of skin sloughing, first above his right eye in Feb[ruary] of 2017." Pet. Ex. 8 at 1; see also Tr. 252.

However, at the hearing, Dr. Moshell opined that onset occurred when Petitioner developed widespread blisters, including those on his hands, in the summer of 2017. Tr. 179, 241. He changed his opinion after reviewing and considering the expert opinions of Respondent's expert, Dr. Maverakis. Tr. 176-77, 241. Dr. Maverakis opined that Petitioner's eye lesion in February 2017 was unlikely to be EBA because it was a "unilateral" in presentation and "nonscarring." Tr. 176-77 (quoting Resp. Ex A at 10). Although Dr. Moshell initially thought Petitioner's eye lesions were part of his EBA, after he considered Dr. Maverakis' opinions, Dr. Moshell agreed that the eye lesions were "very limited and nonscarring," and therefore, "not typical of EBA." Tr. 178-79. The area involved was very limited and only on the face, inconsistent with the classic EBA presentation. Tr. 176, 179. Additionally, the biopsy of the eye lesion did not show EBA. Tr. 280-81. Ultimately, Dr. Moshell concluded that more likely than not, Petitioner's eye lesions were not EBA, especially given that a biopsy was performed. Id.

Dr. Moshell further asserted that EBA was never considered as a diagnosis relative to Petitioner's eye lesions. Tr. 181. There were differences of opinions by the treating physicians as to the diagnosis of Petitioner's eye rash but EBA was not among the diagnoses considered. Id. However, there was a consensus reached as to the diagnosis of Petitioner's blistering of his hands and feet; the treating physicians agreed he had an autoimmune blistering disease. Tr. 181-82. And after testing, the treating physicians reached a consensus that Petitioner's diagnosis was EBA. Tr. 182.

Dr. Moshell testified that after the Hep B vaccine series was complete, he would expect that "as quickly as ten days to two weeks, more commonly about six weeks, but it could take months . . . before the antibody levels were high enough to produce clinical disease." Tr. 209.

According to Dr. Moshell, the most likely time frame for onset was two to six weeks after the third vaccination. Tr. 282-83. He added that the EBA was due to "the combined effect of all three [Hep B vaccines]" but "it was the third that put it over the top and increased the autoantibody production to the point that it produced disease." Tr. 283.

2. Respondent's Expert, Dr. Emanual Maverakis[36]

a. Background and Qualifications

Dr. Maverakis is a board-certified dermatologist and a tenured professor at the University of California, Davis. Resp. Ex. A at 1. He obtained his B.S. in Molecular Genetics from University of California, Los Angeles and his M.D. from Harvard Medical School. Resp. Ex. C at 1. Thereafter, he completed an internship in internal medicine at the Beth Israel Deaconess Medical Center at Harvard Medical School and a residency in dermatology at University of California, Davis. Id. at 5. Throughout his career, he has won various awards and honors and has authored or co-authored over 200 publications. Resp. Ex. A at 1-2; Resp. Ex. C at 1-28. Dr. Maverakis "specialize[s] in treating patients with immune-mediated diseases." Resp. Ex. A at 2. He treats patients with immunobullous diseases, including EBA. Tr. 293.

b. Opinion

Dr. Maverakis agreed that Petitioner's diagnosis is EBA. Tr. 295. But he opined there is no reliable scientific explanation to support vaccine causation. Tr. 299.

i. Althen Prong One

Dr. Maverakis opined that Petitioner's expert did not provide a reliable scientific explanation of how Hep B vaccination can cause EBA. Tr. 299. Dr. Maverakis testified that Dr. Moshell did not propose "much of a theory" other than to say that "something [] stimulated [the] immune system." Tr. 327.

Next, Dr. Maverakis explained that EBA has never been associated with Hep B infection. Tr. 300-01; Resp. Ex. A at 12. He testified that there are "a hundred million people in the world who have hepatitis B" and it is a "very common viral disease." Tr. 302. And therefore, Dr. Maverakis opined that if it were associated with EBA, evidence of this purported risk would be available. Tr. 301-02.

Moreover, he agreed with Dr. Moshell that there has never been a report of "a vaccine associated cause of EBA." Tr. 301. When he drafted his first expert report in July 2021, Dr.

[36] Dr. Maverakis provided two expert reports and testified at the hearing. Resp. Exs. A-B; Tr. 160.

Maverakis performed a PubMed search[37] using EBA and vaccine as the search terms, and there were no results. Resp. Ex. A at 11.

Regarding vaccines in general, he opined that in adults, there is "no credible evidence [that] immunobullous diseases [are] induced by vaccination." Resp. Ex. A at 11. Using the most common autoimmune bullous condition, bullous pemphigoid, as an example, he noted that given the 150 million influenza vaccinations given each year, if vaccination were causal, one would expect cases of bullous pemphigoid to be reported. Id. However, only a handful of such cases have been reported. Id. Additionally, he stated there is no evidence that giving vaccines to patients with immunobullous conditions worsens the diseases. Id.

Specific to Hep B vaccination, Dr. Maverakis opined that approximately 35% of adults have received the vaccination, constituting "tens of millions of individuals." Resp. Ex. B at 2. Given this number, some causes of autoimmune diseases after vaccination would be expected by chance. Id. But he reiterated that "there is no credible medical or scientific evidence that immunobullous diseases are caused by [Hep B] vaccinations in adults." Id.

Dr. Maverakis criticized Dr. Moshell's comparisons of EBA with other skin conditions with different causal mechanisms. For example, Dr. Moshell opined that vaccines can cause severe allergic reactions, and in response, Dr. Maverakis opined that EBA is not caused by an allergic reaction. Resp. Ex. B at 7 (citing Pet. Ex. 15(A) at 2). He criticized Dr. Moshell's example of poison ivy because it is caused by a "delayed-type hypersensitivity reaction" and it is not an autoantibody driven illness. Id. at 4 (citing Pet. Ex. 15(A) at 1). EBA and poison ivy are "totally different types of diseases caused by different immune cells and different antigens." Id. Dr. Moshell also compared EBA to psoriasis associated with streptococcal infection (guttate psoriasis). Pet. Ex. 15(A) at 1. Although Dr. Maverakis agreed there is an association between guttate psoriasis and infections, he opined that this association does not explain the cause of EBA. Resp. Ex. B at 6. He explained guttate psoriasis is caused by infection which results in "a perivascular, predominantly lymphocytic infiltrate to the skin." Id.

Next, Dr. Moshell discussed vancomycin-associated linear IgA bullous dermatosis, an "extremely rare autoimmune subepidermal immunobullous disease." Resp. Ex. B at 6 (citing Pet. Ex. 15(A) at 1). Although Dr. Maverakis agreed there is an association between vancomycin and linear IgA bullous dermatosis, he disagreed that this example provided evidence of vaccine associated EBA. Id. Lastly, Dr. Maverakis acknowledged the causal relationship between Hep B vaccines and lichen planus, but explained that lichen planus is not an immunobullous disease. Id. at 7.

Dr. Maverakis also disagreed there was credible evidence of an association between bullous pemphigoid and vaccination. Resp. Ex. B at 7-8. Even if there were, he opined that

[37] "PubMed is a free resource supporting the search and retrieval of biomedical and life sciences literature . . . . The PubMed database contains more than 37 million citations and abstracts of biomedical literature." Nat'l Libr. Med., Nat'l Ctr. for Biotechnology Info., PubMed Overview, https://pubmed.ncbi.nlm.nih.gov/about/ (last visited Dec. 6, 2024).

bullous pemphigoid and EBA involve different structural proteins, "which means that the disease process is caused by different stimuli." Tr. 298. For EBA, it is collagen VII, whereas in bullous pemphigoid, the antigens are BP-1 and BP-2. Id. While he agreed that a virus may be associated with bullous pemphigoid, Dr. Maverakis disagreed that there was evidence of vaccine associated EBA. Tr. 299.

Regarding medical literature cited by Dr. Moshell, Dr. Maverakis offered specific criticisms. Baroero et al. reported three cases of bullous pemphigoid in children, two after vaccination and one after viral infection. Tr. 304-05 (citing Pet. Ex. 18(A3) at 1). The first case (three month old male) had onset of his bullous rash two days after vaccination, which Dr. Maverakis opined was too soon to conclude that it was an autoimmune post-vaccination illness. Tr. 307; Pet. Ex. 18(A3) at 1. The second child had a history of gastrointestinal problems, and onset was five days after vaccination. Pet. Ex. 18(A3) at 2-3. Dr. Maverakis questioned causation due to the child's history and the fact that onset was five days, again too soon for an autoimmune reaction to occur. Tr. 305-07. The third child had herpetic stomatitis ten days prior to onset of her lesions and did not receive a vaccine. Tr. 305, 307; Pet. Ex. 18(A3) at 3. Of note, the authors stated the "mechanism of induction of [bullous pemphigoid] is still unclear" but due to the temporal relationship, there was a "possible association" between vaccination/infection and bullous pemphigoid. Pet. Ex. 18(A3) at 1.

Kasperkiewicz and Woodley presented a systematic review of the literature related to post-vaccination autoimmune bullous diseases and reported on 24 case reports/series. Pet. Ex. 38 at 1. The time frame between vaccination and the first symptom ranged from one day to three months. Id. at 2-5 tbl.I. Dr. Maverakis explained that cases with an onset of only one day or up to five days were not vaccine-related since antibodies would not be present so soon after vaccination. Tr. 310. He opined that these cases with early onset do not represent autoimmunity after vaccination. Id. Further, he noted that none of the 24 cases were patients with EBA. Id. Because EBA is different than other autoimmune bullous diseases, he opined that these cases do not prove that EBA can occur after vaccination. Tr. 310-11.

Erbagci described a 12-year-old female who developed bullous pemphigoid one week after a Hep B vaccination. Pet. Ex. 39 at 1-3. Dr. Maverakis testified that this article was "probably" included in Kasperkiewicz and Woodley, although the literature reviewed in Kasperkiewicz and Woodley was not cited. Tr. 314 (citing Pet. Ex. 38). Dr. Maverakis concluded that an association with vaccination was coincidental because when the child received additional Hep B vaccinations, she did not have a recurrence of bullous pemphigoid.[38] Tr. 314-15; see Pet. Ex. 39 at 4. Like other articles, the author stated that "the causes of childhood [bullous pemphigoid] remain unknown." Pet. Ex. 39 at 1.

[38] Dr. Maverakis' interpretation of this statement in the article is questionable. The referenced statement appears to be a general observation unrelated to the case report, since the child's reported medical history did not reference additional injections of the vaccine. See Pet. Ex. 39 at 4 ("Some authors have suggested that the onset of [bullous pemphigoid] after vaccination may be coincidental, because they observed no recurrence of [bullous pemphigoid] following further injections of the same vaccine.").

At the hearing, Dr. Maverakis cited a statement issued by the International Pemphigus and Pemphigoid Foundation stating that "there have only been a relatively small number of reported cases of [bullous pemphigoid] presenting after vaccination." Tr. 312 (quoting Resp. Ex. D at 5).[39] "[C]arefully conducted studies" have not supported the conclusion that bullous pemphigoid is caused by vaccination. Id. (quoting Resp. Ex. D at 5). If a patient does develop bullous pemphigoid after vaccination, "it is most likely a chance occurrence." Id. (quoting Resp. Ex. D at 5). Moreover, patients with immunobullous disease are encouraged to get vaccinated. See Tr. 314; Resp. Ex. D at 4.

Lastly, Dr. Maverakis testified that literature cited by Petitioner describing animal studies/models do not support the position that EBA can be caused by vaccination, and instead demonstrate "how difficult it is to induce EBA by vaccination." Tr. 318-22, 384-85 (citing Pet. Ex. 44). In the paper by Sitaru, induction of EBA in mice required a strong adjuvant, complete Freud's adjuvant (mycobacterium), resulting in an "emulsion [] cloudy with bacteria . . . full of immune-activated substances." Tr. 320. A concentrated dose of antigen was injected in the mouse over the course of five days followed by a slight lower dose of the antigenic emulsion repeated every three weeks. Tr. 320-22. Similarly, in another study, a very aggressive synthetic antigen was used to induce autoimmunity. Tr. 323 (citing Pet. Ex. 45).[40] Dr. Maverakis emphasized that vaccines do not contain strong adjuvants, increased levels of cross-reactive material, or extraordinary amounts of antigen. Tr. 384-85.

ii. Althen Prong Two

Dr. Maverakis opined that Petitioner's Hep B vaccines did not cause his EBA. Tr. 299. In addition to his opinions about onset, discussed below, Dr. Maverakis offered several reasons in support of his opinion. First, he opined that none of Petitioner's treating physicians attributed his EBA to his vaccinations. Tr. 329-30.

Next, Dr. Maverakis observed that Petitioner was tested for an autoimmune reactions to type VII collagen and he did not have the autoantibodies. Resp. Ex. B at 3, 5. Thus, Dr. Maverakis opined that "Petitioner's disease was not likely caused by autoantibodies to type VII collagen." Id. at 5. However, he did not explain why this fact was important or failed to support Petitioner's causal theory.

Third, Dr. Maverakis noted that Petitioner smoked, and "[s]moking damages the epithelial barrier." Tr. 386. Petitioner likely had multiple infections like colds that activate the

[39] Information for Pemphigus and Pemphigoid Patients Related to COVID-19, Int'l Pemphigus & Pemphigoid Found., https://www.pemphigus.org/information-for-pemphigus-and-pemphigoid-patients-related-to-coronavirus-disease-covid-19/ (last updated Feb. 14, 2023).

[40] Ana Gabriela Sitaru et al., T Cells Are Required for the Production of Blister-Inducing Autoantibodies in Experimental Epidermolysis Bullosa Acquisita, 184 J. Immunology 1596 (2010).

immune system. Id. And Dr. Maverakis also assumed Petitioner had been sunburned during his life. Id. He opined that all of these may be triggers of EBA. Id.

iii. Althen Prong Three

Regarding onset, Dr. Maverakis opined that it was "difficult to know when [] Petitioner's disease actually [] developed." Resp. Ex. B at 8. Like Dr. Moshell, he offered several different opinions about the onset of Petitioner's EBA.

As for Petitioner's eye lesions, Dr. Maverakis initially opined that they were not a manifestation of Petitioner's EBA. Resp. Ex. A at 10; Resp. Ex. B at 7. In his first expert report, Dr. Maverakis opined "[t]he unilateral presentation of this [eye] lesion and its non-scarring nature would suggest that it is less likely to have been related to the Petitioner's EBA-like disease." Resp. Ex. A at 10. In his second expert report, Dr. Maverakis further opined that it was "difficult to determine the etiology of the eye lesion[s]." Resp. Ex. B at 7.

At the hearing, Dr. Maverakis offered inconsistent opinions about Petitioner's eye lesions. First, he confirmed that he had previously opined that Petitioner's eye lesions were most likely not associated with EBA. Tr. 330. However, he was "influenced" by Dr. Moshell's report and testified there was "a possibility" or it was "possibly more likely than not" that Petitioner's eye lesions were the first manifestation of EBA. Id. In addition to being influenced by Dr. Moshell's opinions, he also attributed the change in his opinion to records describing the eye lesions as blisters, which occurred later in Petitioner's clinical course. Tr. 330-31. Also, Dr. Maverakis explained that in his first expert report, he did not opine that the eye lesions were "less likely than not" part of Petitioner's EBA, but that EBA was "a less likely diagnosis." Tr. 342.

During the hearing, he testified that he did not "have a very strong opinion" about whether the eye lesions were associated with EBA. Tr. 342. But when questioned the third time during the hearing (during redirect), Dr. Maverakis "changed [his] mind." Tr. 393. He opined that Petitioner's eye irritation was a manifestation of EBA. Id. He attributed his changed opinion to Petitioner's description of blisters, the influence of Dr. Moshell's expert report, and because a photograph showed some lesions on Petitioner's face. Id.

Using the eye lesions as the manifestation of EBA, Dr. Maverakis opined that onset of Petitioner's EBA was February 13, 2017. Tr. 390. He based this opinion on the medical record dated February 27, 2017, documenting that Petitioner reported his eye irritation "started a couple of weeks ago." Tr. 393 (quoting Pet. Ex. 3 at 4). Dr. Maverakis subtracted 14 days from the date of that medical record, placing onset on February 13, 2017. Id.

Relative to the eye lesions, Dr. Maverakis also explained that the prodromal phase of EBA can be a unilateral process and there may not be blistering. Tr. 331. This "prodromal period could last [] from a couple of months up to . . . over a year." Id. And he opined that there is "no set criteria" for defining onset of the illness. Id.

In addition to offering an onset opinion based on the eye lesions, Dr. Maverakis also offered an opinion about onset of Petitioner's hand lesions. Tr. 331-32. He opined that "EBA classically occurs on the back of the hands." Tr. 331. When Petitioner was seen at OSF OH on March 28, 2018, the records "clearly state[] . . . that he's been having problems with the back of his hands bilaterally with thinning of the skin . . . and that those problems began since the beginning of work" in September of 2016. Tr. 332 (citing Pet. Ex. 9 at 13-14). Based on this record, Dr. Maverakis testified that Petitioner's EBA started in September 2016, which would place onset prior to vaccination. Id.

However, in his first expert report, Dr. Maverakis observed that the "limited records available in 2016 do not document these symptoms, or lesions[,] or scarring of the hands at that time. Scarring on the dorsal surfaces of the hands is first noted in the records on March 28, 2018." Resp. Ex. A at 10 (citing Pet. Ex. 9 at 13-14).

Further, Dr. Maverakis explained that in EBA, like other immunobullous diseases, "symptoms present well before diagnosis occurs." Tr. 296-97. For example, in Iranzo et al.,[41] the "mean delay to diagnosis was 20-75 months" in 12 patients with EBA. Resp. Ex. A, Tab 2 at 1.

Dr. Maverakis agreed with Dr. Moshell that it takes a couple of days to a month or more for blisters to form and then heal. Tr. 334.

Dr. Maverakis also agreed with Dr. Moshell that the optimal time for onset of vaccine associated autoimmunity is two to six weeks after the third vaccine. Tr. 306. In immunobullous diseases, antibodies bind at the "dermoepidermal junction," which would not occur until seven to nine days after vaccination. Id.

III. DISCUSSION

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). Petitioner need not make a specific type of

[41] P. Iranzo et al., Epidermolysis Bullosa Acquisita: A Retrospective Analysis of 12 Patients Evaluated in Four Tertiary Hospitals in Spain, 171 Brit. J. Dermatology 1022 (2014).

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, a 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 [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner 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 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 [P]etitioner's evidence on a requisite element of the [P]etitioner'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. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

Medical records, specifically contemporaneous medical records, are presumed to be accurate and generally "warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a

condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. 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. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received actually 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. 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 special master 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).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

IV. 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, 569 F.3d at 1375; 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, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (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 at 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 failed to provide preponderant evidence of a sound and reliable theory to explain how Hep B vaccinations can cause EBA. In a nutshell, Dr. Moshell posits that the Hep B vaccination series results in broad immune activation in those persons who can produce autoantibodies against type VII collagen, or another molecule in the basement membrane zone, to induce EBA. He further opined that vaccination causes such broad activation of the immune system which increases the level of all antibodies. However, he did not provide any supporting foundational evidence to show that the Hep B vaccine (1) causes a broad immune activation, or (2) induces the production of antibodies which either did not exist prior to illness or were not present in sufficient quantify as to induce disease. Thus, he did not provide any evidence that broad immune activation triggers production of the autoantibodies that cause EBA. Further, he did not provide evidence that the theory of broad immune activation is recognized as the probable causal mechanism of EBA.

At the outset, the undersigned appreciates Dr. Moshell's candor in acknowledging that this case represented a "first report."[42] Pet. Ex. 15(A) at 1. Dr. Moshell's testimony about the anatomy and physiology of EBA was elegant[43] and scholarly. However, he conceded that what induces autoantibodies in EBA is "medically unknown." Pet. Ex. 8 at 3. And he cited literature in support of this proposition. See, e.g., Pet. Ex. 16(A1) at 11 ("[T]he exact cause of [EBA] is unknown."); Pet. Ex. 44 at 8 (acknowledging that the "genetic and/or environmental events triggering autoimmunity against type VII collagen . . . remain to be elucidated").

Thus, while Dr. Moshell offers a theory, it is unavoidably speculative. When evaluating whether petitioners have carried their burden of proof, theories that are speculative and/or conclusory in nature are consistently rejected because the "expert statements [] are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315).

[42] Dr. Moshell noted that there is always "a first report" although he suggested that a first report may simply reflect the "first time" an illness is recognized in association with a preceding event. Pet. Ex. 15(A) at 1.

[43] Dr. Maverakis testified that "Dr. Moshell was very elegant in his testimony on causation." Tr. 330.

At best, it is only one possible theory among other possible theories, as evidenced by the medical literature. And allowances for possibilities is not the legal standard for a finding of entitlement to compensation under the Vaccine Act. Possibilities are not sufficient to establish causation. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence).

Even given the lack of medical knowledge, however, Dr. Moshell's theory of broad immune stimulation is overly simplistic and not developed. He posits there is broad immune activation in patients with a predisposition to develop antibodies that attack type VII collagen or other molecules in the basement membrane zone. But he does not explain how broad immune activation is caused by Hep B vaccines or provide evidence that such vaccination does cause broad immune activation. He does not describe how broad immune stimulation triggers the antibody production that causes EBA. Although several articles mention non-specific immune activation, the articles do not provide an explanation of how non-specific immune activation induces EBA. See, e.g., Pet. Ex. 17(A2) at 3.

In his prehearing brief, Petitioner cites medical articles and offers a host of theories about how Hep B can cause EBA. See Pet. Prehearing Memo. at 15-24. However, these theories were not advanced by Dr. Moshell at the hearing. Based on the medical literature, numerous other theories, including molecular mimicry,[44] are identified as possible causal mechanisms; however, none have been identified as a leading theory and there is no consensus as to the most likely theory to explain disease induction. See supra Section II.C.1.b.i. Thus, Dr. Moshell's assertion,

[44] In Respondent's prehearing brief, he suggests that Petitioner's theory of causation is based on molecular mimicry. Resp. Br. Regarding Entitlement, filed Dec. 6, 2023, at 18 (ECF No. 66). However, at the hearing, Dr. Moshell focused on the theory of broad immune activation and testified that molecular mimicry is not the proposed causal theory at issue here. Tr. 197, 269. Even if Dr. Moshell did offer the theory of molecular mimicry, it fails for all of the reasons discussed herein. Further, simply arguing the theory of molecular mimicry, without more, is insufficient. See, e.g., McKown v. Sec'y of Health & Hum. Servs., No. 15-1451V, 2019 WL 4072113, at *50 (Fed. Cl. Spec. Mstr. July 15, 2019) (explaining that "merely chanting the magic words 'molecular mimicry' in a Vaccine Act case does not render a causation theory scientifically reliable, absent additional evidence specifically tying the mechanism to the injury and/or vaccine in question" (emphasis omitted)); Johnson v. Sec'y of Health & Hum. Servs., No. 14-254V, 2018 WL 2051760, at *26 (Fed. Cl. Spec. Mstr. Mar. 23, 2018) ("Petitioners cannot simply invoke the concept of molecular mimicry and call it a day. Rather, they need to offer reliable and persuasive medical or scientific evidence of some kind (whether expert testimony or literature) . . . . (internal citations omitted) (emphasis omitted)); Mattus-Long v. Sec'y of Health & Hum. Servs., No. 15-113V, 2022 WL 4242140, at *27 (Fed. Cl. Spec. Mstr. Aug. 31, 2022) (noting "the mere mention of molecular mimicry is not a 'get out of jail free card' in the Program, entitling claimants to compensation, merely because it has scientific reliability as a general matter"); Sheets v. Sec'y of Health & Hum. Servs., No. 16-1173V, 2019 WL 2296212, at *17 (Fed. Cl. Spec. Mstr. Apr. 30, 2019) (determining Petitioner had not satisfied Althen prong one when he did not relate molecular mimicry "to either the vaccines in question or Petitioner's own specific condition").

without more, does not constitute evidence of causation. Petitioner alternatively seems to argue that "[v]accination [] can theoretically unmask subclinical [bullous pemphigoid] by enhancing an autoimmune response or nonspecific immune mechanisms." Pet. Prehearing Memo. at 19 (quoting Pet. Ex. 39 at 1). Again, this is a conclusory statement and it does not explain how a nonspecific immune response induces autoantibodies that attack specific molecules, either collagen VII or some other molecules, in the basement membrane zone, so as to cause EBA.

Petitioner acknowledges that while he has not filed articles that establish a link between Hep B vaccination and EBA, he "has filed articles associating [Hep B] with other, closely related conditions, as well as articles associating other vaccinations with EBA." Pet. Prehearing Memo. at 18-19. However, the medical literature and expert testimony also do not establish that comparisons between EBA and other bullous diseases provide evidence of a causal mechanism applicable to EBA. Like EBA, the triggers of other bullous diseases are not known. Although the literature includes case reports of antecedent events, including vaccination, the authors do not reach conclusions about vaccine causation. Moreover, Dr. Moshell makes it clear that EBA and other bullous conditions are different; they involve different antibodies, different antigens, and different structures are attacked in each. Without evidence that the underlying causal mechanisms of different bullous diseases are the same, case reports about other illnesses do not offer proof of a causal association between Hep B vaccination and EBA.

"An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of Health & Human Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280). Here, Dr. Moshell failed to explain how this literature on other illness could be extrapolated to EBA. See id. at *12 (finding the case reports offered by the petitioner had even less value than case reports do generally because they reported a sequence in which a vaccine, but not the vaccine at issue, preceded the onset of the injury at issue (citing Campbell v. Sec'y of Health & Hum. Servs., 97 Fed. Cl. 650, 668 (2011))).

Petitioner need not make a specific type of evidentiary showing to prove that a theory is sound and reliable by preponderant evidence. Given the state of current scientific knowledge, there is no way that a petitioner could satisfy such a requirement. 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"). However, the evidence shows that how EBA is induced is not known. And Petitioner did not provide preponderant evidence to show that Hep B vaccination can induce EBA.

Further, the medical literature filed herein does not provide support for Petitioner's theory. There are no case reports of EBA following Hep B. There are no case reports of EBA following vaccination, except for one "EBA-like diagnosis" following a Covid-19 vaccine. See Pet. Ex. 41 at 1-6. While the undersigned generally finds case studies may provide some evidence of causation, a single case report involving another vaccine is not sufficient, without more, to constitute sufficient evidence upon which to conclude that Hep B vaccination can cause EBA, especially in light of the other deficiencies discussed above. The other case reports do not

involve the same disease or same vaccine. Therefore, the relevance of these case reports is not clear.

While case reports merit some evidentiary weight, they do not constitute strong supportive evidence of vaccine causation, and alone cannot meet the preponderance of the evidence standard. See Campbell, 97 Fed. Cl. at 668; Caves v. Sec'y of Health & Hum. Servs., No. 07-443V, 2010 WL 5557542, at *14 (Fed. Cl. Spec. Mstr. Nov. 29, 2010) (finding a petitioner's "reference to case reports [did] not help her meet her burden of demonstrating a persuasive and reliable theory causally connecting the [] vaccine to [the injury]"), mot. for review den'd, 100 Fed. Cl. 119 (2011), aff'd, 463 F. App'x 932 (Fed. Cir. 2012); Muchnik ex rel. Muchnik v. Sec'y of Health & Hum. Servs., No. 90-703V, 1991 WL 217673, at *4 (Fed. Cl. Spec. Mstr. Oct. 10, 1991) ("For the petitioner to establish causation in fact by a preponderance of the evidence in any given case requires something more than case reports . . . .").

Moreover, case reports about one vaccine cannot automatically be imputed to a different vaccine, particularly when the mechanism offered has not been suggested as to the vaccine at issue. See, e.g., K.O., 2016 WL 7634491, at *12 ("An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive."); Crosby v. Sec'y of Health & Hum. Servs., No. 18-1478V, 2021 WL 3464125, at *9 (Fed. Cl. Spec. Mstr. July 22, 2021) (declining to give substantial weight to an article because it was on a different vaccine than the one at issue making reasoning difficult); see also Deshler v. Sec'y of Health & Hum. Servs., No. 16-1070V, 2020 WL 4593162, at *19-21 (Fed. Cl. Spec. Mstr. July 1, 2020) (declining to attribute case reports on the flu vaccine to pneumococcal vaccines); McDonald v. Sec'y of Health & Hum. Servs., No. 15-612V, 2023 WL 2387844, at *23 (Fed. Cl. Spec. Mstr. Mar. 7, 2023).

Finally, there does not appear to be any other Vaccine Program decision involving EBA. The lack of other cases may reflect the rarity of the condition but also illustrates that EBA has not been associated with vaccination or the Hep B vaccination in the Program.

In summary, for all the reasons described, Petitioner has failed to offer a sound and reliable medical theory in support of his claim. Thus, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to the first Althen prong.

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, 993 F.2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); Snyder, 88 Fed. Cl. at 746 n.67. "As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases." Welch v. Sec'y of Health & Hum. Servs., No. 18-494V, 2019 WL 3494360, at *8 (Fed. Cl. Spec. Mstr. July 2, 2019).

Since Petitioner failed to prove Althen prong one, it follows that he cannot prove Althen prong two. However, even if Petitioner had proven Althen prong one, the undersigned finds Petitioner has failed to show by preponderant evidence that there is a logical sequence of cause and effect showing Petitioner's Hep B vaccines caused his EBA.

Further, the undersigned finds that none of Petitioner's treating physicians documented or notated an association between his Hep B vaccines and his illness. After reviewing the medical records, the undersigned finds that none of Petitioner's treating physicians opined that his Hep B vaccinations caused his EBA. This is especially notable since Petitioner was seen by specialists, including two dermatologists at academic institutions (Dr. El-Azhary at Mayo Clinic and Dr. Fairley at the UI), and they did not attribute his EBA to his Hep B vaccinations.

Generally, treating physician statements are typically "favored" 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.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280). Although no treating physician's views bind the special master, per se, their views are carefully considered and evaluated. § 13(b)(1); Snyder, 88 Fed. Cl. at 746 n.67. An opinion by a treating physician that is not supported by a factual basis or other evidence is conclusory in nature. See Robertson v. Sec'y of Health & Hum. Servs., No. 18-554V, 2022 WL 17484980, at *17 (Fed. Cl. Spec. Mstr. Dec. 7, 2022); Cedillo v. Sec'y of Health & Hum. Servs., 617 F.3d 1328, 1347 (Fed. Cir. 2010). Furthermore, "[a] treating physician's recognition of a temporal relationship does not advance the analysis of causation." Isaac v. Sec'y of Health & Hum. Servs., No. 08-601V, 2012 WL 3609993, at *26 (Fed. Cl. Spec. Mstr. July 30, 2012).

Accordingly, the undersigned finds that Petitioner failed to satisfy his burden under 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 phrase has been defined as a "medically acceptable temporal relationship." Id. 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. The explanation for what is a medically acceptable time frame must also be consistent 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 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542; see also Pafford, 451 F.3d at 1358. Thus, prong three contains two parts. First, Petitioner must establish the "timeframe for which it is medically acceptable to infer causation" and second, Petitioner must demonstrate that the onset of the disease occurred in this period. Shapiro, 101 Fed. Cl. at 542-43.

Here, the parties dispute onset. Determining onset is challenging due to Petitioner's clinical course and the question of whether his eye lesions were part of his EBA. Further, both experts changed their opinions about onset during the hearing, complicating the issue further. After consideration of all the evidence, the undersigned finds that Petitioner's eye lesions were not a manifestation of his EBA. Thus, onset is based on the initial manifestation of Petitioner's blistering lesions on his hands and feet.

There are several reasons that the undersigned finds that Petitioner's eye lesions were not part of his EBA. First, he was evaluated and treated for his eye rash by Dr. Fifield (February 27, 2017), PA Knoblauch (April 6, 2017), ophthalmologist Dr. Wyman (April 6, 2017), dermatologist Dr. Kroodsma (May 23, 2017), and VA physician Dr. Cutinello (July 27, 2017). While they gave him several different diagnoses, none of them diagnosed an autoimmune blistering disorder. Second, a biopsy was done of the eye lesion, and it showed an angiofibroma, and not pathological findings consistent with EBA. Third, both experts agreed that Petitioner's eye lesions were atypical of EBA because they were not widespread or scarring in nature. In contrast, the experts agreed that Petitioner's hand and feet lesions were classic for EBA. Diagnostic studies and biopsy of such lesions then confirmed EBA.

Petitioner's blistering condition was first described by Dr. Cutinello in the medical records on January 26, 2018. Petitioner had blisters in his mouth and lesions on his hands that were healing. However, when Petitioner was seen at OSF OH on March 28, 2018, he reported that he had been having problems with his hands since September 2016. Despite that history, the limited records in 2016 do not document any blisters or scarring on Petitioner's hands or feet. Dr. Maverakis conceded this point. See Resp. Ex. A at 10 ("Petitioner notes that irritation and 'thinning' of the skin began in 2016, however, the limited records available in 2016 do not document these symptoms, or lesions or scarring of the hands at that time.").

Moreover, Petitioner was seen numerous times from February 2017 to July 2017 (see dates above) for his eye rash, and the records from those visits do not document that Petitioner had blisters or lesions on the hands, feet, or mouth. In fact, a physical examination in July 2017 did not note any skin abnormalities. See Pet. Ex. 10-1 at 153.

The first evidence of hand blisters is a photograph taken by Mrs. Koehl of Petitioner on September 9, 2017, showing broken blisters on the back of his hand and a healing lesion on his arm. The experts agreed that this photograph showed findings consistent with EBA. They also agreed that it takes a couple of days to a month or more for blisters to form and heal. Mrs. Koehl testified that based on this photograph, she believes that the onset of her husband's blistering skin condition was at the end of August or beginning of September 2017.

In summary, a photograph taken of Petitioner on September 9, 2017 showing blisters and a scar consistent with a healing lesion seen with EBA. The presence of a healing lesion indicates the condition had been present for a period of a day up to a month. Thus, onset occurred sometime after Petitioner saw Dr. Cutinello on July 27, 2017, and weeks to a month before the photograph dated September 9, 2017.

Petitioner received his last Hep B vaccination on July 24, 2017. Both experts agreed that the optimal time for onset of a vaccine-associated autoimmune illness is two to six weeks after the third vaccination. Given agreement on this point, the undersigned finds that an appropriate onset would be two to six weeks after July 24, 2017, a time frame from August 7 to September 4, 2017.

The photograph taken September 9, 2017, Ms. Koehl's testimony that onset of Petitioner's blisters associated with EBA occurred in late August or early September 2017, and the experts' agreement on the time frame that it takes for blisters to begin to heal, all point to onset in late August or early September 2017. This time frame is within the two to six week time frame appropriate given vaccine-associated autoimmunity.

However, a temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Thus, even though Petitioner has provided preponderant evidence satisfying Althen prong three, Petitioner is not entitled to compensation.

V. ALTERNATIVE CAUSATION

If the undersigned had concluded that Petitioner established a prima facie case, Petitioner would have been entitled to compensation unless Respondent put forth preponderant evidence "that [Petitioner's] injury was in fact caused by factors unrelated to the vaccine." Whitecotton v. Sec'y of Health & Hum. Servs., 17 F.3d 374, 376 (Fed. Cir. 1994), rev'd on other grounds sub nom., Shalala v. Whitecotton, 514 U.S. 268 (1995); see also Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1151 (Fed. Cir. 2007). Since Petitioner did not prove Althen prongs one and two, the burden did not shift to Respondent to prove alternative causation.

However, for completeness sake, the undersigned addresses alternative causation because the parties disputed whether there were preexisting conditions or intercurrent infections that caused Petitioner's EBA. Respondent's expert, Dr. Maverakis, testified that Petitioner had risk factors for EBA, including smoking, multiple infections, and sunburns. However, Dr. Maverakis did not opine that any of these risk factors independently, or combined, caused Petitioner's EBA. Thus, the undersigned does not find Dr. Maverakis and Respondent established by a preponderance of evidence that Petitioner's injury was "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B).

VI. CONCLUSION

Petitioner has suffered a serious and chronic debilitating illness as he described in detail at the hearing, in the medical records, and by the parties' experts. The undersigned extends her sympathy for the suffering Petitioner has experienced due to his illness. However, the undersigned's Decision cannot be based on her sympathy, but must be based on the evidence and the law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to provide preponderant evidence of causation, and therefore, the Petition must be dismissed.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master