In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: September 2, 2025

THEO ROGAN,	*	PUBLISHED
Petitioner,	*	No. 17-1916V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Ruling on Entitlement; Human Papillomavirus ("HPV") Vaccine; Alopecia Areata ("AA").
Respondent.	*	

Mark Sadaka, The Law Offices of Sadaka Associates, LLC, Englewood, NJ, for Petitioner.
Tyler King, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

On December 8, 2017, Theo Rogan[2] ("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),[3] alleging that as a result of a human papillomavirus ("HPV")

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website 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 Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The petition was initially filed by Jonathan Rogan on behalf of his minor child, Theo Rogan; however, when Theo Rogan reached the age of majority during the pendency of this case, the case caption was amended. Order dated Sept. 29, 2021 (ECF No. 77).

[3] 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 Ruling to individual sections of the Vaccine Act are to 42 U.S.C.A. § 300aa.

vaccine administered on October 17, 2016, he developed alopecia. Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating "this case is not appropriate for compensation under the Vaccine Act." Respondent's Report ("Resp. Rept.") at 2 (ECF No. 13).

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards,[4] the undersigned finds Petitioner has provided preponderant evidence that the HPV vaccine he received on October 17, 2016 caused him to develop alopecia, satisfying Petitioner's burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is entitled to compensation.

I. ISSUES TO BE DECIDED

The parties agree that Petitioner received his first dose of the HPV vaccine on October 17, 2016 and was first noted to have lost his eyelashes in November 2016. Joint Submission, filed Sept. 16, 2024, at 1 (ECF No. 161). Further, the parties do not dispute Petitioner's February 2017 diagnosis of alopecia areata ("AA").[5] Id.

As to causation, the parties dispute all three Althen prongs. Joint Submission at 1. Respondent disputes that Petitioner provided "reliable evidence that HPV vaccine can cause AA" and disputes that Petitioner produced "reliable evidence that the HPV vaccine did cause [Petitioner's] AA." Resp. Response to Petitioner's Motion for a Ruling on the Record ("Resp. Response"), filed Nov. 14, 2024, at 8, 12 (ECF No. 167).

II. BACKGROUND

A. Procedural History

On December 8, 2017, Petitioner filed a petition requesting compensation. Petition. The case was assigned to now-Chief Special Master Corcoran. Notice of Assignment dated Dec. 11, 2017 (ECF No. 4). Between December 2017 and February 2018, Petitioner filed medical records and photographs.[6] Petitioner's Exhibits ("Pet. Exs.") 1-6. Respondent filed a Rule 4(c) report on May 4, 2018, arguing against compensation. Resp. Rept. at 2.

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[4] While the undersigned has reviewed all of the information filed in this case, only those filings and records that are most relevant will be discussed. See Moriarty v. Sec'y of Health & Hum. Servs., 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision."); see also Paterek v. Sec'y of Health & Hum. Servs., 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[5] Alopecia areata "is an autoimmune disease characterized by hair loss due to inflammatory responses that target the hair follicles." Resp. Ex. E, Tab 2 at 1 (Teontor Simakou et al., Alopecia Areata: A Multifactorial Autoimmune Condition, 98 J. Autoimmunity 74 (2019)).

[6] Petitioner continued to file medical records throughout litigation.

On October 16, 2018, Petitioner filed an expert report from Dr. M. Eric Gershwin. Pet. Ex. 7. On May 31 and June 3, 2019, Respondent filed expert reports from Dr. Maryanne Makredes Senna and Dr. Stephen Mark Tompkins. Resp. Exs. A, C. The case was then reassigned to the undersigned. Notice of Reassignment dated Oct. 7, 2019 (ECF No. 35). On January 13, 2020, Petitioner filed a supplemental expert report from Dr. Gershwin. Pet. Ex. 91.

The undersigned held a Rule 5 conference on April 14, 2020. Order dated Apr. 14, 2020 (ECF No. 54). She preliminarily concluded that Petitioner "may be able to satisfy all three Althen prongs." Id. at 1. If the parties were unable to settle the case, the undersigned would schedule an entitlement hearing. Id. at 2. On July 30, 2020, an entitlement hearing was set for December 2021. Prehearing Order dated July 30, 2020 (ECF No. 64).

On November 10, 2021, the December 2021 entitlement hearing was postponed per request of the Petitioner to allow Petitioner to obtain a dermatological expert. Order dated Nov. 10, 2021, at 1 (ECF No. 88). On February 1, 2022, Petitioner filed an expert report from Dr. Jill Javahery. Pet. Ex. 106. The entitlement hearing was then rescheduled for February 2023. Prehearing Order dated May 19, 2022 (ECF No. 97). At Petitioner's request, the February 2023 hearing was postponed and later rescheduled for September 2024. Order dated Jan. 13, 2023 (ECF No. 121); Order dated Mar. 13, 2023 (ECF No. 129).

On December 14, 2023, Respondent filed an expert report from Dr. Andrew Krakowski. Resp. Ex. E. Petitioner declined to file additional expert reports. Joint Status Rept., filed May 28, 2024 (ECF No. 149).

On May 28, 2024, Petitioner filed a joint status report requesting a ruling on the record in lieu of the September 2024 entitlement hearing. Joint Status Rept., filed May 28, 2024 (ECF No. 149). The September 2024 entitlement hearing was cancelled. Order dated May 28, 2024 (ECF No. 150). Petitioner filed his motion for a ruling on the record on September 12, 2024. Pet. Motion for a Ruling on the Record ("Pet. Mot."), filed Sept. 12, 2024 (ECF No. 160). Respondent filed his responsive brief on November 14, 2024 and Petitioner filed a reply on December 2, 2024. Resp. Response; Pet. Reply to Resp. Response ("Pet. Reply"), filed Dec. 2, 2024 (ECF No. 168).

This matter is now ripe for adjudication.

B. Summary of Medical Records[7]

Petitioner was fourteen years old when he received his first HPV vaccination[8] on October 17, 2016 at his pediatrician's office. Pet. Ex. 2 at 6, 34. That day, he complained of moderate back pain, which appeared to the pediatrician to be myofascial in nature. Id. at 4-5. Petitioner's physical examination was otherwise normal. Id. at 4.

Prior to vaccination, Petitioner had a history of eczema, atopic dermatitis, allergies, seborrhea, psoriasis, and hip and joint problems. See Pet. Ex. 2 at 4, 7-9, 21-38; Pet. Ex. 3 at 5; Pet. Ex. 4. On April 30, 2015, he was diagnosed with psoriasis by his pediatrician. Pet. Ex. 2 at 29. He was being followed by an allergist, Dr. Steven M. Meltzer, who diagnosed allergic rhinitis and severe atopic dermatitis in August and September 2016, two months prior to his HPV vaccination. Pet. Ex. 4 at 3-4; see also Pet. Ex. 2 at 9.

Approximately three weeks after vaccination, on November 9, 2016, Petitioner presented to his pediatrician for a preventative examination. Pet. Ex. 2 at 10. At that visit, Petitioner reported that his "right lateral upper eyelid lashes fell out." Id. Petitioner's mother also indicated that he had "developed vitiligo[9] around the same time, which [was] stable." Id. On examination, the pediatrician did not note any hair loss over Petitioner's scalp. Id. at 11. Petitioner was instructed to continue to follow up with Dr. Meltzer for psoriasis and allergic rhinitis, and he was referred to dermatology for an abnormal mole. Id. at 12.

On December 14, 2016, Petitioner was evaluated by a dermatologist, Mavis Billips, M.D., for pruritic scaly skin eruptions on his face and body. Pet. Ex. 5 at 1. Dr. Billips noted a history of dermatitis for several years, for which Petitioner was being treated with Fluocinolone, with slow improvement. Id. She also noted there was no family history of skin problems. Id. On examination, Dr. Billips observed "[l]ichenified eczematous red scaly papules and plaques over the flexural surfaces" of Petitioner's arms, back of ankles, and dorsal hands. Id. His skin was dry and excoriated from scratching. Id. Petitioner also had no right upper eyelashes and

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[7] This summary of medical records is taken in part from the parties' briefs, with edits from the undersigned, as the undersigned finds they provided an accurate representation of the records. Pet. Mot. at 2-4; Resp. Response at 3-5.

[8] Petitioner received the Gardasil HPV 9-valent vaccine, which is recombinant vaccine prepared from the purified virus-like particles of nine types of HPV (6, 11, 16, 18, 31, 33, 45, 52, and 58). About Gardasil 9, https://www.gardasil9.com/patient-pd/what-is-gardasil-9/about-gardasil-9/#isi-onpage (last visited Aug. 25, 2025); Gardasil 9, U.S. Food & Drug Admin., https://www.fda.gov/vaccines-blood-biologics/vaccines/gardasil-9 (last updated Mar. 26, 2025) (package insert).

[9] Vitiligo is "a chronic, usually progressive, type of hypomelanosis in which melanocytes are destroyed, resulting in white patches on the skin that may be surrounded by a hyperpigmented border; there is an autosomal dominant predisposition to the condition, and the etiology is thought to be an autoimmune mechanism." Vitiligo, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=53308 (last visited Aug. 25, 2025).

only sparse lower lashes. Id. Dr. Billips diagnosed atopic dermatitis and hypotrichosis[10] of the right eyelashes. Id. Dr. Billips did not document a history of psoriasis or diagnosis Petitioner with psoriasis. See id. Petitioner was prescribed Locoid,[11] Atarax,[12] Elidel,[13] Alevicyn,[14] and Latisse.[15] Id. at 1-2; Pet. Ex. 2 at 37.

A letter from Dr. Billips to Petitioner's pediatrician dated December 23, 2016 indicated that Petitioner's mother had called to report that Petitioner's scalp hair was falling out. Pet. Ex. 2 at 37. Dr. Billips suspected alopecia, which required more aggressive therapy, and recommended Petitioner be evaluated and treated by the pediatric dermatology department at University of Irvine. Id.

On February 14, 2017, Petitioner was evaluated by dermatologist, Jill Javahery, M.D., for chief complaints of hair loss on his scalp, eyebrows, and eyelashes. Pet. Ex. 1 at 1. Dr. Javahery indicated that Petitioner's hair loss was focal and moderate in nature with a sudden onset and that it had been present for approximately three months. Id. She noted that Petitioner had received the HPV vaccine two weeks before the onset of hair loss. Id. Dr. Javahery also noted a history of scalp bumps and redness. Id. She did not document a history of psoriasis or diagnosis Petitioner with psoriasis. See id. Dr. Javahery diagnosed Petitioner with AA with discrete non-scarring patches of hair loss and exclamation point hairs distributed over his eyes and scalp. Id.

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[10] Hypotrichosis is the "presence of less than the normal amount of hair." Hypotrichosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24440 (last visited Aug. 25, 2025).

[11] Locoid (hydrocortisone butyrate) is "used topically for the relief of inflammation and pruritus in corticosteroid-responsive dermatoses." Hydrocortisone Butyrate, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=81260 (last visited Aug. 25, 2025).

[12] Atarax (hydroxyzine hydrochloride) is "used . . . in urticaria and other manifestations of allergic dermatoses." Hydroxyzine Hydrochloride, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=81323 (last visited Aug. 25, 2025).

[13] Elidel (pimecrolimus) is "a calcineurin inhibitorimmunosuppressant" that is "applied topically to treat moderate to severe atopic dermatitis." Pimecrolimus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39242 (last visited Aug. 25, 2025).

[14] Alevicyn (Levicyn) is used "to manage and relieve the burning, itching and pain experienced with various types of dermatoses." Hypochlorous Acid Topical (Rx), Medscape, https://reference.medscape.com/drug/alevicyn-hypochlorous-acid-topical-1000215#0 (last visited Aug. 25, 2025).

[15] Latisse "is an FDA-approved treatment to grow eyelashes for people with inadequate or not enough lashes." Latisse, www.latisse.com (last visited Aug. 25, 2025).

She prescribed a steroid cream and explained that AA is an autoimmune disease of patchy hair loss that responds well to treatment but can recur. Id.

On May 8, 2017, Petitioner's records were updated at his pediatrician's office by Dr. Elmo Agatep to include an allergic reaction of alopecia to the HPV vaccine. Pet. Ex. 101 at 50. At that visit, Dr. Agatep noted that Petitioner had "hair loss, from vaccine." Id. at 51. On June 12, 2017, Petitioner was seen by his pediatrician who noted that Petitioner did not receive additional doses of the HPV vaccine as planned due to the onset of alopecia. Id. at 53.

Petitioner had six follow-up appointments with Dr. Javahery between March and July 2017. Pet. Ex. 1 at 4-21. At those visits, he was treated with intralesional Kenalog injections with improvement and hair regrowth observed in mid-April. See id. At each of this visits, Petitioner's diagnosis remained AA. See id. Dr. Javahery did not diagnosis Petitioner with psoriasis. See id. Petitioner continued to have follow-up appointments with Dr. Javahery for the remainder of 2017 through 2021. See Pet. Ex. 103 at 25-95; Pet. Ex. 104; Pet. Ex. 108.

Petitioner has not filed any medical records since January 2023.

C. Expert Reports[16]

1. Petitioner's Expert, Dr. M. Eric Gershwin[17]

a. Background and Qualifications

Dr. Gershwin is a Distinguished Professor of Medicine in the Division of Rheumatology, Allergy, and Clinical Immunology at the University of California, Davis School of Medicine. Pet. Ex. 8 at 1. He is board certified in internal medicine, rheumatology, and allergy and clinical immunology. Id. at 2. He completed his M.D. at Stanford University after which he completed an internship and residency in internal medicine at Tufts New England Medical Center and trained in immunology at the National Institutes of Health in Maryland. Id. at 1-2. Dr. Gershwin has held various editor and reviewer positions on medical journals, and he has authored or co-authored over 1,000 publications during his career. Id. at 5-143. He has authored on the subject of alopecia and autoimmunity. Id. at 123; Pet. Ex. 9.[18]

[16] Although the undersigned has reviewed all of the expert reports and medical literature, for the sake of brevity this Ruling does not include all details of the experts' opinions. Instead, the undersigned focuses on the experts' material opinions, as they relate to the relevant issues.

[17] Dr. Gershwin submitted two expert reports. Pet. Exs. 7, 91.

[18] Naseeha Islam et al., The Autoimmune Basis of Alopecia Areata: A Comprehensive Review, 14 Autoimmunity Revs. 81 (2015).

b. Opinion

i. Althen Prongs One and Two

Dr. Gershwin opined that the HPV vaccine caused Petitioner's AA via the mechanism of molecular mimicry. Pet. Ex. 7 at 5-7. Molecular mimicry occurs when a foreign antigen in the vaccine resembles one in the host (referred to as autoantigen, host-antigen, or self-antigen) and the immune response to the vaccine inadvertently targets the autoantigen. Id. Pathogenesis of AA also involves the activation of cytotoxic T cells[19] ("generation of cytotoxic CD8+ cells;" "generation of a CD8 response").[20] Id. at 5, 7; see also Resp. Ex. C at 6.[21] Exposure of autoantigens in hair follicles attracts T cells to the "hair bulb area."[22] Pet. Ex. 7 at 4. There is a collapse of the hair follicle immune privilege, resulting in changes to the "cytokine/chemokine profiles," leading to this infiltration of destructive cytotoxic T cells directed at hair follicles. Id. Dr. Gershwin noted that once cytotoxic T cells are generated, there is "immunological programing of [] cytotoxic T cell expansion" so that the signals required for the initial proliferation of these cells are no longer required. Id. at 5.

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[19] T cells, or T lymphocytes, are "cells primarily responsible for cell-mediated immunity." T Lymphocytes, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87562 (last visited Aug. 25, 2025). "They are characterized by specific surface antigens . . . . T cell antigen receptors are triggered by antigen only when associated with self [major histocompatibility complex ("MHC")] antigens, e.g., by antigens processed and presented by macrophages, viral antigens on the surface of host cells, and tumor neoantigens. When activated by antigen, T lymphocytes proliferate and differentiate into T memory cells and the various types of regulatory and effector T cells," including cytotoxic T cells. Id. Cytotoxic T cells are "differentiated T lymphocytes that can recognize and lyse target cells bearing specific antigens recognized by their antigen receptors. Recognition is MHC restricted; the foreign antigen is recognized only in association with self MHC antigens. The cytotoxic activity requires firm binding of the lymphocyte to the target cell to produce holes in the plasma membrane of the target cell, loss of its cell content, and osmotic lysis." Cytotoxic T Lymphocytes, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87555 (last visited Aug. 25, 2025). Dr. Gershwin uses "lymphocytes" in his reports, but for consistency and simplicity, the undersigned uses "cells."

[20] CD8 cells are "T lymphocytes that carry the CD8 antigen; major subtype[] [is] the cytotoxic T lymphocytes." CD8 Cells, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64001 (last visited Aug. 25, 2025).

[21] Respondent's expert, Dr. Tompkins' explanation of molecular mimicry in the context of AA is concise and understandable, and the undersigned has cited it to explain Dr. Gershwin's theory. See Resp. Ex. C at 6.

[22] Dr. Gershwin identified additional cytokines and chemokines which may be involved. See Pet. Ex. 7 at 4-5. Chemokines, specifically Th1 chemokines, "are more prominent in alopecia patients than healthy individuals." Id. at 4. For additional discussion of cytokines, chemokines, and NK cells, see Pet. Ex. 7 at 4.

In support, Dr. Gershwin cited medical literature,[23] including several articles by Taisuke Ito. Ito describes the hair follicle as a "'miniorgan' with unique immune and hormone microenvironments." Pet. Ex. 27 at 4.[24] Ito explains that AA is caused by a collapse of the immune privilege of the hair follicle and an "autoimmune reaction[] against hair follicle autoantigens." Id. at 1. In AA, histopathology of hair bulbs reveals an "accumulation of mononuclear cells . . . composed of both CD4+ and CD8+ cells."[25] Id. at 2, 3 fig.1.

Ito provides the following illustration to explain the pathogenesis of AA.

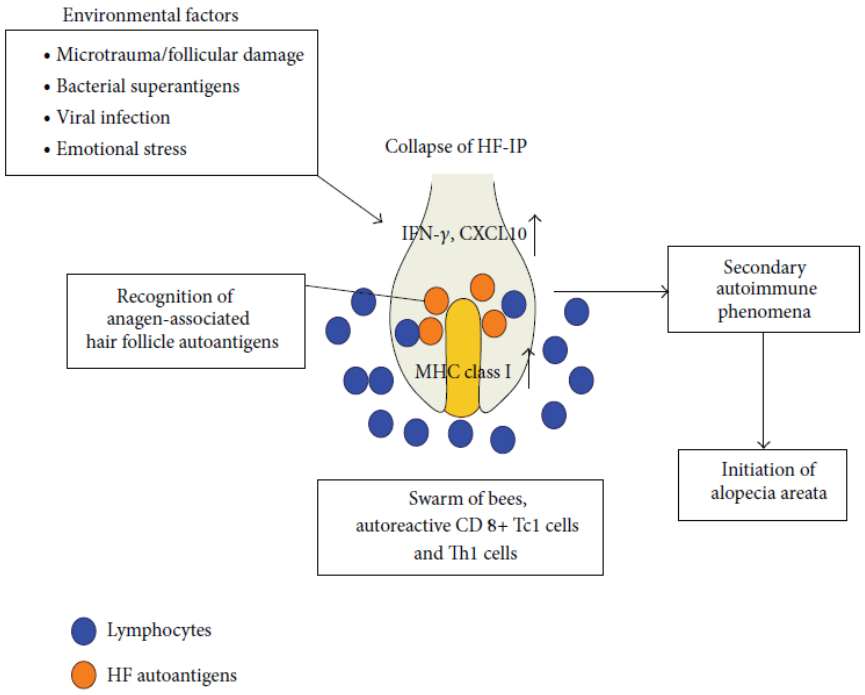

FIGURE 1: The pathogenesis of alopecia areata and treatment strategies. Environmental factors such as viral infections and bacterial superantigens may induce IFN-γ and CXCL10 expressions in the hair bulbs. Subsequently, autoreactive Th1 and Tc1 cells (blue circles) accumulate in and around hair bulbs—the so-called "swarm of bees." Anagen-associated hair follicle (HF) autoantigens (orange circles) are recognized by Th1 and Tc1 cells, which lead to a secondary autoimmune phenomenon and resultant hair loss.

Pet. Ex. 27 at 3, fig.1.

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[23] The parties filed many medical articles, and while the undersigned has reviewed all of them, only those most relevant are discussed for the sake of brevity.

[24] Taisuke Ito, Recent Advances in the Pathogenesis of Autoimmune Hair Loss Disease Alopecia Areata, 2013 Clinical & Developmental Immunology 1.

[25] Pathogenesis of AA, as explained by Ito, is complex, involving many moving parts, and the description by the undersigned is very simplified. For a complete description by Ito, see, for example, Pet. Ex. 27; Pet. Ex. 52 (Taisuke Ito et al., Maintenance of Hair Follicle Immune Privilege Is Linked to Prevention of NK Cell Attack, 128 J. Investigative Dermatology 1196 (2007) (also cited as Resp. Ex. C, Tab 9).

Support for the notion that AA is an autoimmune condition derives, in part, from the fact that AA patients often have other autoimmune conditions, including, for example, vitiligo, lupus, and thyroiditis. Pet. Ex. 7 at 3. Further support that AA is an autoimmune illness is based on positive response to treatment with steroids and immunotherapeutic agents. Id. Moreover, there is "increased expression of specific [human leukocyte antigens ("HLAs")[26]] [found] in alopecia patients . . . rarely seen in healthy individuals." Id.

Dr. Gershwin acknowledged that although AA "appears to be a T cell disease[,] [] it has been a major challenge to identify T cell epitopes."[27] Pet. Ex. 7 at 6. The inability to identify or predict these epitopes has made it impossible to understand "individual immune reactions" or the reasons behind responses to vaccines. Id. at 6-7. And although he advances the theory of molecular mimicry and cytotoxic T cells, Dr. Gershwin acknowledged there may be "multiple other immune pathways involved in the [cause] of [AA]." Id. at 7. He described the role of HLAs in alopecia, the strong association with the MHC class 1 alleles, the "repertoire of cells at the [hair follicle]," including natural killer cells, the cytokine IFN-γ (interferon gamma), chemokines, tumor necrosis factor, and histological findings. Id. at 4-5. He cited a number of articles that discuss what is known about the pathogenesis of AA, supported by relevant studies. See, e.g., Pet. Ex. 9 at 4-6; Pet. Ex. 10 at 6-10;[28] Pet. Ex. 14 at 4-6;[29] Pet. Ex. 16 at 1-12.[30]

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[26] HLAs are "histocompatibility antigens governed by genes of the HLA complex (the human major histocompatibility complex), a region on the short arm of chromosome 6 containing several genetic loci, each having multiple alleles." Human Leukocyte Antigens, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56923 (last visited Aug. 25, 2025). "As with most autoimmune diseases, AA exhibits HLA gene associations." Pet. Ex. 48 at 2 (discussing HLA associations in AA pathogenesis) (Amos Gilhar et al., Lymphocytes, Neuropeptides, and Genes Involved in Alopecia Areata, 117 J. Clinical Investigation 2019 (2007)).

[27] Epitopes, or antigenic determinants, are "a site on the surface of an antigen molecule to which a single antibody molecule binds; generally an antigen has several or many different antigenic determinants and reacts with antibodies of many different specificities." Antigenic Determinants, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=69449 (last visited Aug. 25, 2025).

[28] Abdullah Alkhalifah et al., Alopecia Areata Update: Part I. Clinical Picture, Histopathology, and Pathogenesis, J. Am. Acad. Dermatology 177 (2010).

[29] Andrew F. Alexis et al., Alopecia Areata: Autoimmune Basis of Hair Loss, 14 Eur. J. Dermatology 364 (2004).

[30] K.J. McElwee et al., What Causes Alopecia Areata?, 22 Experimental Dermatology 609 (2013).

According to Dr. Gershwin, Petitioner was genetically susceptible to developing AA and had risk factors for the condition due to his "history of atopy."[31] Pet. Ex. 7 at 1; Pet. Ex. 91 at 1. Regarding genetic susceptibility in AA, Dr. Gershwin explained there is "observed heritability" in first degree relatives, verified by studies of twins and families. Pet. Ex. 7 at 2. He reviewed the association of HLA and alopecia, noting the specific genetic alleles involved, as well as other genetic associations. Id.

As for predisposing risk factors, in his first report, Dr. Gershwin reported that eczema, seborrhea, and psoriasis were not associated with alopecia. Pet. Ex. 7 at 1. However, the medical literature cited by Dr. Gershwin in his first report noted a relationship between AA and atopic diseases. See, e.g., Pet. Ex. 39 at 4 ("The relationship between atopy and AA is well established.").[32] Thus, in his second report, Dr. Gershwin agreed that "patients with alopecia may have a predisposing history of atopy," although the "predisposing history" of psoriasis and alopecia was "less clear." Pet. Ex. 91 at 1 (citing Pet. Ex. 92).[33] An article he cited stated that "patients with psoriasis have a 2.5-fold higher risk of AA development." Pet. Ex. 92 at 1. To the extent that atopy and psoriasis are predisposing factors for AA, Dr. Gershwin asserted this strengthens his position that Petitioner had a genetic predisposition to develop AA. Pet. Ex. 91 at 1-2. He explained that while atopy and/or psoriasis may make an individual more susceptible to AA, these conditions "do not themselves cause [AA]." Id. at 2.

In addition to genetic susceptibility and risk factors, Dr. Gershwin noted that environmental insults, hormones, infections, ultraviolet light exposure, injuries, and emotional distress also contribute to the etiology of AA. Pet. Ex. 7 at 3. Stress hormones, such as corticotropin-releasing hormone, are implicated, as are viral infections, including from cytomegalovirus ("CMV"), hepatitis B and C, Epstein Barr virus ("EBV"), and swine flu virus. Id.

In support, he cited a paper by Rodriguez and Duvic,[34] a retrospective analysis of self-reported environmental exposures in patients on the National AA Registry. Pet. Ex. 38 at 1. They identified 12 previously healthy young patients with sudden onset AA within one week to six months of EBV infectious mononucleosis. Id. at 2. While the authors noted that the

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[31] Atopy is "a genetic predisposition toward the development of immediate (type I) hypersensitivity reactions against common environmental antigens (atopic allergy)." Atopy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4738 (last visited Aug. 25, 2025). "The most common clinical manifestation is allergic rhinitis; bronchial asthma, atopic dermatitis, and food allergy occur less frequently." Id.

[32] William V.R. Shellow et al., Profile of Alopecia Areata: A Questionnaire Analysis of Patient and Family, 31 Int'l J. Dermatology 186 (1992).

[33] Francesco Tassone et al., Clinico-Dermoscopic Features of Alopecia Areata in Patients with Psoriasis, 4 JAAD Case Reps. 665 (2018).

[34] Thomas A. Rodriguez & Madeleine Duvic, Onset of Alopecia Areata After Epstein-Barr Virus Infectious Mononucleosis, 59 J. Am. Acad. Dermatology 137 (2008).

pathogenesis of AA is "incompletely known," they posited that positive family history points to genetic predisposition and the "discordance of the disease in identical twins" suggests an environmental trigger. Id. at 1. Rodriguez and Duvic concluded that infection, specifically EBV, was a possible trigger and recommended further study. Id. at 2-3. "Other viruses, including hepatitis B, hepatitis C, [] and swine flu have also been suggested to trigger alopecia." Pet. Ex. 7 at 3.

HPV infection has been shown to be associated with AA. Tu et al.[35] reported on a large cohort study (30,001 patients) in Taiwan from 2000 and 2012, finding that "patients with HPV infections had a significantly greater risk [155%] of [AA] for both genders[] [and] all subgroups" as "compared with the matched controls." Pet. Ex. 110 at 1, 6. The authors noted that "[t]he underlying mechanism . . . remains unclear" but proposed it probably involved "elevated IFN-γ induced by host immune response against HPV infection, causing immune cell infiltration and cytokine release that [] damage[d] the immune privilege of hair follicles." Id. at 6. In discussing the limitations of the study, the authors explained that the study group was mono-country, and considering "possible ethnic and geographical differences in the incidence and serotypes of HPV," the findings "may not be applicable to non-Asian ethnic groups." Id. at 8. The undersigned also notes that the study did not include ages younger than 18. See id. at 6. However, in this study group, there was strong epidemiological evidence that HPV infections are associated with AA.

Vaccinations are also associated with AA. Pet. Ex. 7 at 5. Dr. Gershwin cited to Wise et al.,[36] a 1997 paper about the association between vaccination and hair loss. Pet. Ex. 80. The authors examined 60 reports of hair loss from data obtained from the Vaccine Adverse Event Reporting System ("VAERS"), Food and Drug Administrations ("FDA"), the Centers for Disease Control and Prevention ("CDC"), and drug manufacturers dating back to 1969. Pet. Ex. 80 at 1. The majority of cases occurred following hepatitis B vaccinations (46), with other vaccines accounting for 14 cases. Id. None of the cases involved the HPV vaccine as it was not administered during the study period.[37] Four cases exhibited clear positive rechallenge ("hair loss more than once after vaccinations" with "the first episode [] resolved before onset of the second") and another 12 were possible rechallenge ("exacerbation or continuation of hair loss after second immunization but lacked indication of prior recovery"). Id. at 2. Ages ranged from two months to 67 years of age, with 16 of the 60 cases reported in children. Id. Onset was reported in 50 of the 60 cases, with 84% occurring within one month of vaccination, and five occurring within one day of vaccination. Id.

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[35] Ting-Yu Tu et al., Human Papillomavirus Symptomatic Infection Associated with Increased Risk of New-Onset Alopecia Areata: A Nationwide Population-Based Cohort Study, 119 J. Autoimmunity 1 (2021).

[36] Robert P. Wise et al., Hair Loss After Routine Immunizations, 278 JAMA 1176 (1997).

[37] FDA Approves Licensure of First U.S. HPV Vaccine, AAP News, June 1, 2006, https://publications.aap.org/aapnews/article-abstract/27/6/2006214/80730/FDA-approves-licensure-of-first-U-S-HPV-vaccine? (noting the first HPV vaccine was approved by the FDA in 2006).

More recently, an UpToDate article[38] stated that "vaccinations[] have been proposed as contributors to episodes of [AA]." Pet. Ex. 91 at 2 (quoting Pet. Ex. 97 at 3).

COVID-19 vaccinations have also been associated with AA. See Pet. Ex. 114 at 7;[39] Pet. Ex. 115.[40] Shakoei et al. noted reports of AA associated with all three COVID-19 vaccines (Pfizer, AstraZeneca, and Moderna), suggesting that vaccine antigens could "trigger T cell-mediated responses, which could lead to AA in genetically susceptible individuals." Id. They reported on a 74-year-old male who developed AA after receiving the Sinopharm vaccine, an inactivated whole-virus COVID-19 vaccine. Id. at 2, 7 fig 3. Birkett et al. conducted a literature search and identified 18 cases of AA associated with the COVID-19 vaccination. Pet. Ex. 115 at 1, 2 tbl.1. Half of the patients had a history of AA. Id. at 1. Onset of AA occurred within several days to three weeks after vaccination. Id. at 1-2, 2 tbl.1.

Notably, Dr. Gershwin cited an article by Tuccori et al.,[41] describing case reports of telegen effluvium[42] in two 11-year-old children after HPV vaccination, with worsening alopecia after subsequent doses of the vaccine. Pet. Ex. 98 at 1. In case number one, the child received the first dose of the HPV vaccine in May 2008, the second in June 2008, and the third in November 2008. Id. Onset of hair loss began in July, after the second dose, and worsened three weeks after the third dose. Id. at 1-2. The patient described in case number two developed hair loss approximately one month after receiving the first dose of the HPV vaccination, which worsened after the second and third vaccinations. Id. at 2. No alternative causes were identified. Id. at 2-3. Citing Wise et al., the authors explained that the immune mechanism involved suggested "the existence of antigenic molecular similarities between vaccines and hair follicles . . . in susceptible patients." Id. at 2 (citing Pet. Ex. 80). The authors concluded there was a "probable relationship" between the HPV vaccinations and alopecia, especially given the worsening of alopecia following subsequent vaccine doses. Id. at 3. Of note, the HPV vaccine

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[38] Andrew G. Messenger, Alopecia Areata: Clinical Manifestations and Diagnosis, UpToDate, https://www.uptodate.com/contents/alopecia-areata-clinical-manifestations-and-diagnosis (last updated May 22, 2019).

[39] Safoura Shakoei et al., Cutaneous Manifestations Following COVID-19 Vaccination: A Report of 25 Cases, 25 Dermatologic Therapy 1 (2022).

[40] Liam Birkett et al., Possible Associations Between Alopecia Areata and COVID-19 Vaccination and Infection, 42 Aesthetic Surgery J. 1 (2022).

[41] Marco Tuccori et al., Telogen Effuvium Following Bivalent Human Papillomavirus Vaccine Administration: A Report of Two Cases, 224 Dermatology 212 (2012).

[42] As described by Respondent's expert, Dr. Krakowski, telogen effluvium is "excessive hair shedding during the telogen phase of the hair cycle." Resp. Ex. E at 10. The telogen phase is the "resting phase" of the hair cycle. Id. Dr. Krakowski stated that, in contrast, hair shedding that occurs during the growing phase is called anagen effluvium. Id. at 11. Dr. Krakowski explained that AA is a form of anagen effluvium. Id.

given to the children described in these case reports was a bivalent vaccine with HPV types 16 and 18 made from virus-like particles. Id. at 1. Petitioner received the HPV 9-valent vaccine, which included nine types of the HPV virus, including types 16 and 18, and was also made from virus-like particles. See supra note 8.

ii. Althen Prong Three

Dr. Gershwin opined that onset within 14 days was consistent with his mechanistic theory. Pet. Ex. 7 at 5. The vaccine was administered October 17, 2016. Id. at 1. According to Petitioner's parents, he began to lose his eyelashes approximately two and a half weeks later. Id.

2. Petitioner's Expert, Dr. Jill Javahery[43]

a. Background and Qualifications

Dr. Javahery is Petitioner's treating dermatologist. Pet. Ex. 106 at 1. She is a board-certified dermatologist and is currently in private practice at Comprehensive Dermatology of Long Beach and El Segundo Dermatology. Pet. Ex. 107 at 1. Additionally, she serves as the resident rotation director for pediatric dermatology at the University of California, Irvine. Id. Dr. Javahery received her M.D. from the University of Miami in Florida. Id. She completed an internship in internal medicine at Beth Isreal Medical Center followed by a residency in dermatology at SUNY Downstate Medical Center. Id. Since completing her residency in 2007, Dr. Javahery has been in private practice. Id.

b. Opinion

Dr. Javahery has treated Petitioner for AA since February 2017. Pet. Ex. 106 at 1. She reviewed the expert reports of Dr. Gershwin, Dr. Senna, and Dr. Tompkins, and provided a one-page letter addressing Petitioner's medical history and causation. Id.

Dr. Javahery opined that Petitioner's April 30, 2015 psoriasis diagnosis was "made in error." Pet. Ex. 106 at 1; see also Pet. Ex. 2 at 29. First, she noted that the psoriasis diagnosis was not made by a dermatologist and explained that "most non dermatologists cannot distinguish one type of skin lesion from the next." Pet. Ex. 106 at 1. Next, there was no biopsy to substantiate a psoriasis diagnosis. Id. Further, Dr. Javahery has examined and interviewed Petitioner "many times over the years" and has found "no evidence that he has or ever has had psoriasis." Id.

As the 2015 psoriasis diagnosis was incorrect, Dr. Javahery opined that Petitioner had never been diagnosed with an autoimmune disease prior to his AA diagnosis. Pet. Ex. 106 at 1. Addressing the significance of the incorrect psoriasis diagnosis, Dr. Javahery explained that Respondent's expert, Dr. Senna, used "this error in [Petitioner's medical] history to incorrectly assess his baseline risk of developing [AA]." Id.

[43] Dr. Javahery provided one expert report. Pet. Ex. 106.

Moving to causation, Dr. Javahery opined that onset of autoimmune disease is often "related to some preceding insult or immune trigger." Pet. Ex. 106 at 1. She explained that "the phenomenon of vaccine induced autoimmune disease" can be seen in the administration of Covid vaccines. Id. She noted that these vaccines "contain black box warnings regarding the induction of auto-immune disease post vaccination ([immune thrombocytopenic purpura], myocarditis, etc.)." Id. Dr. Javahery used Covid vaccines as an example as "it is easier to notice patterns when vaccines are given out en masse." Id.

Dr. Javahery concluded that Petitioner,

> who had never experienced hair loss before and who never had a history of autoimmune disease, developed rapid onset of aggressive [AA] directly following an immunological event (injection of the HPV vaccine). Trying to imagine that there is no association between the two events is not only a stretch but is incredibly unfair to a young man who has suffered terribly as a result of this insult.

Pet. Ex. 106 at 1.

3. Respondent's Expert, Dr. Maryanne Makredes Senna[44]

a. Background and Qualifications

Dr. Senna is an assistant professor of dermatology at Harvard Medical School and attending dermatologist at Massachusetts General Hospital in Boston. Resp. Ex. A at 1; Resp. Ex. B at 1-2. Dr. Senna is board certified in dermatology. Resp. Ex. A at 1. She received her M.D. from Tufts University and subsequently completed her residency in dermatology at University of Massachusetts Medical Center. Resp. Ex. B at 1. Dr. Senna is the co-director of the Massachusetts General Hospital's Hair Loss Clinic. Resp. Ex. A at 1. In this capacity, she sees approximately 50 new alopecia patients each month. Id. Dr. Senna's clinical research has focused on inflammatory hair loss disorders such as AA. Id. She also directs a clinical trial unit dedicated to hair loss disorders. Id. Dr. Senna serves as an ad hoc reviewer for several journals. Id. at 2; Resp. Ex. B at 3. She has authored, or co-authored, numerous publications on the topics of alopecia and other forms of hair loss. Resp. Ex. B at 10-11.

b. Opinion

Dr. Senna opined that Petitioner's AA was not caused by the HPV vaccine. Resp. Ex. A at 6, 16.

Addressing the pathogenesis of AA, Dr. Senna explained that immune privilege protects the hair follicle from "immunological attack[s]." Resp. Ex. A at 8-9. The first step of AA pathogenesis is the breakdown of hair follicle immune privilege due to "stress or other exogenous factors in a genetically susceptible individual." Id. at 8. The breakdown of immune

[44] Dr. Senna submitted one expert report. Resp. Ex. A.

privilege "allows CD8+ cytotoxic T cells to penetrate" the growth phase hair bulb and "initiate an autoimmune attack in response to a still unrecognized antigen." Id. "This sets off a chain of events that eventually leads to inflammation of the hair follicle bulb region and resultant alopecia." Id. While earlier studies "focused on the role of IFN-γ produced by CD8+ T cells as the main cytokine driver of AA," recent studies show that the Th2 pathway is also involved. Id. Like Dr. Gershwin, Dr. Senna noted "[t]he likelihood that several different immunologic pathways are involved in AA pathogenesis." Id. at 9.

Dr. Senna did not dispute that AA is an autoimmune illness or disagree with Dr. Gershwin's theory of molecular mimicry/CD8+ T cell activation. Resp. Ex. A at 8. Instead, she opined there was no evidence that the HPV vaccination can cause the illness, and therefore, the only association between the HPV vaccination and AA was temporal. Id. at 10-11.

She explained that AA has a baseline rate of three percent in the general population. Resp. Ex. A at 10. Additionally, autoimmune disorders, such as AA, "peak during adolescence and early adulthood" which is the same timeframe that individuals receive the HPV vaccine series. Id. (citing Resp. Ex. A, Tab 54).[45] While this temporal association "leads some to attribute the development of autoimmune conditions to vaccines," this causal association is not supported by epidemiological studies. Id.

She cited epidemiological studies addressing the association of the HPV vaccination with autoimmune conditions; however, none addressed alopecia.[46] Chao et al.[47] identified potential new onset autoimmune conditions in HPV vaccine recipients by monitoring 189,620 women for 180 days after each HPV dose for new diagnosis of 16 prespecified autoimmune conditions. Resp. Ex. A, Tab 58 at 1. The authors found "no autoimmune safety signal" in women who received the HPV vaccine. Id. A large cohort study of HPV vaccinations in Denmark and Sweden found "no evidence supporting associations between exposure to []HPV vaccine and autoimmune, neurological, and venous thromboembolic adverse events." Resp. Ex. A, Tab 56 at

[45] S. Macleod & R.E. Appleton, Neurological Disorders Presenting Mainly in Adolescence, 92 Archives Disease Childhood 170 (2007). This article addresses neurological conditions and does not discuss alopecia.

[46] Dr. Senna also cited studies that addressed other adverse events. For example, the study by Gee et al. used Vaccine Safety Datalink to examine an association between HPV vaccination and GBS, stroke, venous thromboembolism, appendicitis, seizures, syncope, allergic reactions, and anaphylaxis. Resp. Ex. A, Tab 55 at 1 (Julianne Gee et al., Monitoring the Safety of Quadrivalent Human Papillomavirus Vaccine: Findings from the Vaccine Safety Datalink, 29 Vaccine 8279 (2011)).

[47] C. Chao et al., Surveillance of Autoimmune Conditions Following Routine Use of Quadrivalent Human Papillomavirus Vaccine, 271 J. Internal Med. 193 (2012).

1.[48] A cohort study by Scheller et al.[49] found no increased risk of multiple sclerosis or other demyelinating conditions following HPV vaccination. Resp. Ex. A, Tab 59 at 1.

Dr. Senna explained that Genovese et al.[50] performed a large meta-analysis of 243,289 patients who received either bivalent or quadrivalent HPV vaccines and found no correlation between autoimmune disease and HPV vaccination. Resp. Ex. A at 11 (citing Resp. Ex. A, Tab 50). However, it is not clear that the studies which formed the basis of the analysis identified AA as a captured adverse event. Thus, it is not clear whether the findings are relevant.

The other studies cited by Dr. Senna do not appear to have investigated alopecia as a designated outcome. For example, a Finnish study of 134,615 adolescent patients found no increased risk for the selected autoimmune conditions following bivalent HPV vaccinations. Resp. Ex. A, Tab 51 at 1.[51] Data was obtained from the national hospital discharge register. Id. Based on the medical literature, medical records of Petitioner, and the expert reports, it does not appear that hospitalization is required or routine in AA patients, for either diagnosis or treatment purposes. Thus, the methodology of this study may not have been tailored to capture cases of AA.

The same methodology problem exists in the study by Grimaldi et al.,[52] cited by Dr. Senna. Resp. Ex. A at 11 (citing Resp. Ex. A, Tab 53). Case definitions included certain prescribed autoimmune diseases, and alopecia was not included. Resp. Ex. A, Tab 53 at 2. Arana et al.[53] also studied pre-specified conditions and alopecia was not included. Resp. Ex. A at 11 (citing Resp. Ex. A, Tab 52 at 2, 7 app.). Finally, Dr. Senna asserted that her own search of medical literature "revealed no case reports of [AA] developing after HPV immunization." Id. at 12.

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[48] Lisen Arnheim-Dahlström et al., Autoimmune, Neurological, and Venous Thromboembolic Adverse Events After Immunisation of Adolescent Girls with Quadrivalent Human Papillomavirus Vaccine in Denmark and Sweden: Cohort Study, 347 BMJ f5906 (2013).

[49] Nikolai Madrid Scheller et al., Quadrivalent HPV Vaccination and Risk of Multiple Sclerosis and Other Demyelinating Diseases of the Central Nervous System, 313 JAMA 54 (2015).

[50] C. Genovese et al., HPV Vaccine and Autoimmune Diseases: Systematic Review and Meta-Analysis of the Literature, 59 J. Preventive Med. & Hygiene E194 (2018).

[51] Jozica Skufca et al., The Association of Adverse Events with Bivalent Human Papilloma Virus Vaccination: A Nationwide Register-Based Cohort Study in Finland, 36 Vaccine 5926 (2018).

[52] Lamiae Grimaldi-Bensouda et al., Risk of Autoimmune Diseases and Human Papilloma Virus (HPV) Vaccines: Six Years of Case-Referent Surveillance, 79 J. Autoimmunity 84 (2017).

[53] Jorge E. Arana et al., Post-Licensure Safety Monitoring of Quadrivalent Human Papillomavirus Vaccine in the Vaccine Adverse Event Reporting System (VAERS), 2009-2015, 36 Vaccine 1781 (2018).

Next, Dr. Senna disagreed that there is "always an identifiable inciting event" in the development of AA. Resp. Ex. A at 16. Instead, she explained that Petitioner "exhibited several independent predisposing factors." Id. Specifically, Petitioner had a "strong family history of autoimmune disease" and a personal history of "extensive, treatment resistant atopic dermatitis, allergy, and psoriasis." Id. Dr. Senna explained that patients with psoriasis and atopic dermatitis are at an increased risk of developing AA. Id. at 12-13. Relying on a study by Wu et al.,[54] Dr. Senna explained that patients with psoriasis have a "2.5-fold higher risk of developing [AA] than the general population." Id. at 12 (citing Resp. Ex. A, Tab 36). And patients with atopic dermatitis were at an "increased risk of AA compared to patients without [atopic dermatitis], with reported odds ratios ranging from 2.6 to 26.3." Id. at 13 (citing Resp. Ex. A, Tab 3;[55] Resp. Ex. A, Tab 40).[56]

Lastly, Dr. Senna offered an alternative cause and theory for Petitioner's AA. She opined that atopic dermatitis and allergies "have been shown to deplete [T regulatory cell ("Treg")[57]] numbers and cause Treg dysfunction." Resp. Ex. A at 14. She relied on an animal model that showed a decrease of Tregs in mice with "significant food allergy." Id. (citing Resp. Ex. A, Tab 46).[58] Similarly, she cited a study reporting that mouse models of atopic dermatitis showed Treg dysfunction. Id. (citing Resp. Ex. A, Tab 45).[59] Dr. Senna explained that Tregs are required for the "proliferation and differentiation of normal hair follicle stem cells." Id. at 9 (citing Resp. Ex. A, Tab 20).[60] Dr. Senna asserted that depleted Tregs are "significantly decreased in number in [AA], contributing to AA pathogenesis." Id. However, the studies she cited do not conclude that

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[54] Jashin J. Wu et al., The Association of Psoriasis with Autoimmune Diseases, 67 J. Am. Acad. Dermatology 924 (2012).

[55] C Goh et al., Profile pf 513 Patients with Alopecia Areata: Associations of Disease Subtypes with Atopy, Autoimmune Disease and Positive Family History, 20 J. Eur. Acad. Dermatology & Venereology 1055 (2006).

[56] Tadayo Ikeda, A New Classification of Alopecia Areata, 131 Dermatologica 421 (1965).

[57] Tregs are "a subset of $CD4^+$ T cells that can suppress activity of effector cells such as helper cells and suppressor cells, and inhibit autoimmune diseases." Regulatory T Cells, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64383 (last visited Aug. 25, 2025).

[58] Magali Noval Rivas et al., Regulatory T Cell Reprogramming Toward a Th2-Cell-Like Lineage Impairs Oral Tolerance and Promotes Food Allergy, 42 Immunity 512 (2015).

[59] Verena Moosbrugger-Martinz et al., Atopic Dermatitis Induces the Expansion of Thymus-Derived Regulatory T Cells Exhibiting a Th2-Like Phenotype in Mice, 20 J. Cellular & Molecular Med. 930 (2016).

[60] Niwa Ali et al., Regulatory T Cells in Skin Facilitate Epithelial Stem Cell Differentiation, 169 Cell 1119 (2017).

allergies or atopic dermatitis results in depletion of Tregs which leads to the induction of AA. See Resp. Ex. A, Tabs 20, 45-46.

Dr. Senna laid out her theory in five steps:

- Patients with allergy and atopic dermatitis have increased Th2 skewing, immunologically.
- Th2 skewing decreases Treg numbers and causes Tregs to be less effective at promoting immune tolerance.
- Tregs are required for normal telogen to anagen transition of the hair follicle.
- Treg numbers are reduced in [AA].
- The Th2 pathway has been implicated as part of AA pathogenesis.

Resp. Ex. A at 14-15. She concluded that Petitioner's "persistent, severe, widespread atopic dermatitis would have deleterious effects on Treg function and number and be enough by itself to cause [AA]." Id. at 15.

Although Dr. Senna opined that Petitioner had an increased risk of developing AA due to his other conditions, and that these conditions contributed to AA pathogenesis due to the resulting depletion of Tregs, she did not state that she held this opinion to a preponderant or "more likely than not" standard.

As to Althen prong three, Dr. Senna did not provide an opinion rebutting Dr. Gershwin's opinion that Petitioner's AA onset was temporally appropriate under his proposed causal mechanism.

4. Respondent's Expert, Dr. Stephen Mark Tompkins[61]

a. Background and Qualifications

Dr. Tompkins is a Professor of Infectious Diseases at the Center for Vaccines and Immunology at the University of Georgia, College of Veterinary Medicine. Resp. Ex. C at 1; Resp. Ex. D at 2. He received a Ph.D. in the field of immunology and molecular pathogenesis from Emory University. Resp. Ex. C at 1; Resp. Ex. D at 1. Thereafter, he completed postdoctoral fellowships in immunology and virology at Northwestern University and at the Center for Biologic Evaluation and Research. Resp. Ex. D at 1. His postdoctoral training focused on "immunological mechanisms of induction of autoimmune disease" and "immune response to influenza infection and vaccination." Resp. Ex. C at 1. Dr. Tompkins' research is dedicated to "understanding the interactions of the influenza virus and influenza vaccines with the host." Id. While aspects of his research focus on zoonotic influenza, the "core of [his] research" is devoted to "understanding the immune response to viral infection and vaccination." Id. Dr. Tompkins serves as an ad hoc reviewer and editor for several journals. Id.; Resp. Ex. D at 4-6. He has authored, or co-authored, numerous publications on the topics of immunology and

[61] Dr. Tompkins submitted one expert report. Resp. Ex. C.

virology. Resp. Ex. D at 23-33. Dr. Tompkins is not a medical doctor, and his opinions are limited to his areas of expertise.

b. Opinion

i. Althen Prongs One and Two

Dr. Tompkins opined that there was "no evidence" to support a "direct association between the HPV vaccination" and Petitioner's AA. Resp. Ex. C at 9.

Dr. Tomkins does not dispute molecular mimicry per se but criticized the lack of evidence about the cross reactive epitopes and T cell activation in the context of AA. Resp. Ex. C at 5-7. Dr. Tompkins opined there is no data showing "that T cells or B cells specific for [] proposed cross-reactive epitopes are present, activated, or involved in [AA]." Id. at 6. Further, while Dr. Tomkins agreed that CD8+ T cells play a role in AA, studies do not "demonstrate[] *de novo* priming of CD8+ T cells." Id. at 5-6. He stated that the "events triggering CD8+ T cell activation remain undefined." Id. at 6. "Specifically for this case, there is no human data defining the T cell responses initiating AA. Rather, there are genetic associations with AA and T cell responses described emerging secondary to autoimmune disease." Id.

Further, Dr. Tompkins opined it was unlikely that an immune response to the HPV vaccine could "cause non-specific activation of autoreactive T cells." Resp. Ex. C at 7. He identified IFN-γ as a "key cytokine in the induction of AA," noting that it and tumor necrosis factor alpha ("TNF-α") are the "primary cytokines produced by follicle-infiltrating NK and CD8+ T cells." Id. He cited a study by Herrin et al.[62] for the proposition that these cytokines are not elevated after the HPV vaccination. Id. (citing Resp. Ex. C, Tab 19).

Herrin et al. studied the innate and adaptive immune responses of two HPV vaccines, including the two valent (HPV 16 and 18) and four valent (HPV 6, 11, 16, and 18) vaccines. Resp. Ex. C, Tab 19 at 2. The study showed that both vaccines increased the "frequency of IFN-γ producing cells" in peripheral blood mononuclear cells.[63] Id. at 4. In contrast, there were no "significant trends" in these cytokines found in plasma. Id. at 5. CD8+T cell cytokine responses were not analyzed after vaccination. Id. at 4. CD4+ responses were tested 12 months after vaccination, but this would be too late to provide meaningful information about vaccine

[62] Douglas M. Herrin et al., Comparison of Adaptive and Innate Immune Responses Induced by Licensed Vaccines for Human Papillomavirus, 10 Hum. Vaccines & Immunotherapeutics 3446 (2014).

[63] Peripheral blood mononuclear cells ("PBMCs") consist of "lymphocytes and monocytes in the peripheral blood" and "are the key drivers of the immune responses." Julius M. Cruse & Robert E. Lewis, Illustrated Dictionary of Immunology 565 (3rd ed. 2009); Partho Sen et al., Perspectives on Systems Modeling of Human Peripheral Blood Mononuclear Cells, 4 Frontiers Molecular Bioscis. 1 (2018), https://www.frontiersin.org/journals/molecular-biosciences/articles/10.3389/fmolb.2017.00096/full.

causation here. Id. Thus, Herrin et al. supports the proposition that the HPV vaccine increases the production of the key cytokine IFN-γ, which is relevant to AA, and as such, the paper does not appear to support Dr. Tompkins' opinions.

After questioning whether the HPV can trigger AA, Dr. Tompkins took issue with Dr. Gershwin's opinion about the unpredictable nature of the immune system. Resp. Ex. C at 7. Dr. Tompkins agreed that the "generation of T cell receptor diversity is mostly random," however, the resulting repertoire is "heavily reduced by selection events during the thymic maturation of T cells." Id. This debate does not appear to be determinative as to whether the HPV vaccine can cause AA.

Regarding Dr. Gershwin's reliance on Wise et al., Dr. Tompkins argued the findings are not reliable because the study relied on VAERS data, which is "not indicative of causation." Resp. Ex. C at 6 (citing Pet. Ex. 80). However, the undersigned notes that Wise et al. also used data from the FDA, CDC, and product manufacturers. See Pet. Ex. 80 at 1. And in 60% of the cases, additional details were obtained by conducting interviews with the patient, parents, or treating physicians. See id.

Dr. Tompkins offered an opinion as to an alternative cause for Petitioner's AA. Resp. Ex. C at 4-5. He explained that based on Petitioner's medical history of eczema, seasonal allergies, atopic dermatitis, sinusitis, and psoriasis, Petitioner may have had "chronic inflammation of the dermis and associated tissues," which was "a plausible source of immune stimulation that could, over time, activate T cells associated with [] AA." Id. He surmised there was no "specific trigger" but instead, "constant inflammation provid[ed] an ongoing activation for T cell activation." Id. at 5. Dr. Tompkins did not state that he held this opinion to a more likely than not standard.

In conclusion, Dr. Tompkins opined there is "no evidence" in the exhibits filed to support "a direct association between the HPV vaccination and [Petitioner's] onset of hair loss." Resp. Ex. C at 8. Instead, he proposed that the "non-specific inflammation of the psoriasis, eczema[,] and general allergic rhinitis are more plausible culprits, as chronic, non-specific inflammation of the hair follicle resulting in activation of innate and subsequently adaptive (CD8+ T cells), resulting in [Petitioner's] alopecia." Id.

ii. Althen Prong Three

Dr. Tompkins agreed that the timing of Petitioner's alopecia onset was appropriate under the theory proposed by Dr. Gershwin, stating "the vaccination event did occur within an immunologic time frame that would allow for T cell priming or reactivation." Resp. Ex. C at 8.

5. Respondent's Expert, Dr. Andrew C. Krakowski[64]

a. Background and Qualifications

Dr. Krakowski is the chair of dermatology at St. Luke's University Health Network. Resp. Ex. E at 1; Resp. Ex. F at 1. He is board certified in pediatric dermatology and dermatology. Resp. Ex. E at 1; Resp. Ex. F at 2-3. He received his M.D. from the University of Pennsylvania. Resp. Ex. F at 1. Dr. Krakowski then completed a pediatrics residency at Johns Hopkins followed by a dermatology residency at the University of California, San Deigo. Id. He also completed both a research fellowship and clinical fellowship in pediatric dermatology at Rady Children's Hospital of San Diego. Id. In his clinical practice, he sees around six to eight pediatric patients with hair loss each week. Resp. Ex. E at 1. Approximately two to four of these patients are then diagnosed with AA. Id. In addition to his clinical practice, Dr. Krakowski is the program director for St. Luke's residency in dermatology. Id. Dr. Krakowski serves as a reviewer and editor for several journals. Id.; Resp. Ex. F at 3-4. He has authored, or co-authored, numerous publications on the topic of dermatology. Resp. Ex. F at 6-15.

b. Opinion

Dr. Krakowski opined that there is "no evidence" that Petitioner's HPV vaccine "was the 'more likely than not' cause of his [AA]." Resp. Ex. E at 17.

Dr. Krakowski agreed that Petitioner's diagnosis was alopecia. Resp. Ex. E at 13. Dr. Krakowski opined that Petitioner's presentation was "clinically consistent" with AA but could also be considered alopecia totalis.[65] Id. He disagreed, however, that Petitioner's alopecia onset was "rapid" as his hair loss occurred over a period of "months to years (rather than weeks to months)." Id. at 10.

In addition to agreeing with the diagnosis, Dr. Krakowski opined that AA is a "multifactorial[] autoimmune condition of the hair follicle." Resp. Ex. E at 11. Although he does not use the phrase "molecular mimicry," Dr. Krakowski's description is that of molecular mimicry; he stated the mechanism is where "one's own immune system mistakes otherwise normal, healthy hair follicles as 'dangerous' and begins to attack them." Id. Dr. Krakowski further explained that AA is "known to be caused by multiple inciting triggers that may set-off an autoimmune cascade" which leads to hair loss. Id. at 12. However, in a majority of AA cases, no specific cause for the onset of AA can be "ever be clearly identified." Id. Thus, it does not appear that Dr. Krakowski takes issue with the causal mechanisms described by Dr. Gershwin.

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[64] Dr. Krakowski submitted one expert report. Resp. Ex. E.

[65] Alopecia totalis is "complete loss of hair from the entire scalp, resulting from progression of [AA]." Total Alopecia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=55496 (last visited Aug. 25, 2025).

Dr. Krakowski also acknowledged there is a "rare association" of AA with vaccinations, including Japanese encephalitis, hepatitis B, herpes zoster virus, and HPV vaccinations. Resp. Ex. E at 12 (citing Pet. Ex. 19;[66] Pet. Ex. 29;[67] Resp. Ex. E, Tab 5;[68] Resp. Ex. E, Tab 6).[69] However, Dr. Krakowski also explained that trials using mouse models of AA, in which diphtheria and tetanus toxoids were added as controls, suggested that AA associated with vaccination was in the normal, predicted incidence range. Id. (citing Resp. Ex. E, Tab 8).[70] The authors explained their finding "suggest[ed] that AA associated with vaccination may be within the normal background levels of the given population." Resp. Ex. E, Tab 8 at 1.

In contrast to the "rare association" of AA with vaccinations, Dr. Krakowski noted that swine flu virus infection has been reported to trigger or exacerbate AA. Resp. Ex. E at 12 (citing Resp. Ex. E, Tab 7).[71] In a letter to the editor, Ito and Tokura described seven patients who had an occurrence or reoccurrence of AA within one to four months of swine flu infection. Resp. Ex. E, Tab 7 at 2 tbl.1. The authors theorized that swine flu infection induced "Th1 immune responses . . . by overproduction of IFN-γ with a high fever." Id. at 2. The authors explained that "Th1 immune reactions may play an important role in the development of AA, as IFN-γ may induce the collapse of [hair follicle-immune privilege] . . . by autoreactive CD8+ T cells." Id.

In addition to an association with swine flu and EBV infections, Dr. Krakowski noted a "well-known association of upper respiratory infections often preceding onset of [AA]." Resp. Ex. E at 16. He argued that the existence of an association between upper respiratory infection

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[66] Dagny Jagielska et al. Follow-Up Study of the First Genome-Wide Association Scan in Alopecia Areata: IL13 and KIAA0350 as Susceptibility Loci Supported with Genome-Wide Significance, 132 J. Investigative Dermatology 2192 (2012). Dr. Krakowski cited to Petitioner's exhibit 19 to support the proposition that AA has been reported after Japanese encephalitis vaccination; however, Petitioner's exhibit 19 does not address Japanese encephalitis vaccination.

[67] Petra Clara Arck et al., Stress Inhibits Hair Growth in Mice by Induction of Premature Catagen Development and Deleterious Perifollicular Inflammatory Events via Neuropeptide Substance P-Dependent Pathways, 162 Am. J. Pathology 803 (2003). Dr. Krakowski cited to Petitioner's exhibit 29 to support the proposition that AA has been reported after hepatitis B vaccination; however, Petitioner's exhibit 29 does not address hepatitis B vaccination.

[68] Yi Chun Lai & Yik Weng Yew, Severe Autoimmune Adverse Events Post Herpes Zoster Vaccine: A Case-Control Study of Adverse Events in a National Database, 14 J. Drugs Dermatology 68 (2015).

[69] David Geier & Mark Geier, A Case-Control Study of Quadrivalent Human Papillomavirus Vaccine-Associated Autoimmune Adverse Events, 34 Clinical Rheumatology 122 (2015).

[70] John P. Sundberg et al., Recombinant Human Hepatitis B Vaccine Initiating Alopecia Areata: Testing the Hypothesis Using the C3H/Hej Mouse Model, 20 Veterinary Dermatology 99 (2009).

[71] Taisuke Ito & Yoshiki Tokura, Alopecia Areata Triggered or Exacerbated by Swine Flu Virus Infection, 39 J. Dermatology 863 (2012).

and AA has been "reinforced with a surge of [AA] cases in association with COVID." Id. (citing Resp. Ex. E, Tab 15 (discussing seven case reports of new onset AA following COVID infection)).[72] Dr. Krakowski explained that Petitioner had a "long-standing history of upper respiratory infections" prior to receipt of the HPV vaccine. Id. He opined this history makes "the association with infection as strong a consideration as a potential inciting etiology for [Petitioner's] [AA] as anything else." Id.

The balance of Dr. Krakowski's report focused on association between AA and other conditions that Petitioner suffered—namely, atopic dermatitis, eczema, rhinitis, vitiligo and psoriasis. Resp. Ex. E at 14-17. Addressing atopy, Dr. Krakowski noted that Petitioner's atopic dermatitis was diagnosed before his hair loss. Id. at 15. He opined that Petitioner's clinical course "correlate[s] to what [] would [be] expect[ed] for [AA] developing the setting of atopic dermatitis." Id. Dr. Krakowski emphasized that atopy is "one of the most-well known associations with [AA] and a risk factor for more severe progression of disease." Id. He opined that Petitioner's "underlying atopic dermatitis must be considered as a possible inciting trigger for his [AA]." Id.

Further, Dr. Krakowski opined that Petitioner "likely has an underlying diagnosis of psoriasis and/or seborrheic dermatitis." Resp. Ex. E at 15. He explained that "patients with psoriasis are at increased risk for other autoimmune conditions – including [AA]." Id. Dr. Krakowski acknowledged that the link was "controversial . . . because of a possible 'shared genetic predisposition'" or because of medication used to treat psoriasis. Id. However, he explained that a meta-analysis by Jung et al.[73] of case-control, cross-sectional, and cohort studies on the prevalence of AA in patients with psoriasis and the prevalence of psoriasis in patients with AA, "suggested an overall bidirectional association between psoriasis and [AA]." Id. at 16 (citing Resp. Ex. E, Tab 13). However, Dr. Krakowski stated this opinion as a "possibility" and not more likely than not: "the possibility of an association with those conditions and [Petitioner's] [AA] must [] be considered." Id. at 15.

Finally, Dr. Krakowski noted an association between AA and other autoimmune conditions such as vitiligo, diabetes mellitus, and thyroiditis. Resp. Ex. E at 16. Dr. Krakowski noted that Petitioner reported developing vitiligo at his November 2016 visit. Id. at 14 (citing Pet. Ex. 2 at 10). This same visit noted a family history of diabetes mellitus and thyroid issues. Id. Dr. Krakowski asserted that these conditions should have been "more thoroughly investigated." Id.

In concluding his opinions as to causation, Dr. Krakowski opined Petitioner's history of atopy alone was "the most powerful risk factor for his development of [AA]." Resp. Ex. E at 17. He further opined that given Petitioner's "history of psoriasis and/or seborrheic dermatitis, his

[72] Rachel E. Christensen & Mohammad Jafferany, Association Between Alopecia Areata and COVID-19: A Systematic Review, 7 J. Am. Acad. Dermatology Int'l 57 (2022).

[73] Joon Min Jung et al., Association Between Psoriasis and Alopecia Areata: A Systematic Review and Meta-Analysis, 49 J. Dermatology 912 (2022).

unexplored and unexplained history of vitiligo, and his history of upper respiratory infections and sinusitis," it was now "clear that any one of these conditions are more likely[74] to have been the inciting culprit of [Petitioner's] [AA]." Id. In his expert report, Dr. Krakowski first used the word "possible" to describe the association between conditions noted in Petitioner's history, but in the conclusion of his report, he opined this association was "more likely than not."

Regarding a temporal association, Dr. Krakowski stated he had not "seen or heard of a characteristic 'timing' that would allow a person to back-date" the onset of hair loss to accurately identify a "specific trigger . . . with absolutely certainty." Resp. Ex. E at 12. He did not, however, refute the onset here as inconsistent with an immune-mediated mechanism.

III. LEGAL FRAMEWORK

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 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).

[74] But see Resp. Ex. E at 15 (opining atopic dermatitis and psoriasis were "possible" triggers, inconsistent with "more likely than not").

"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 a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [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. den'd, 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 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, 618 F.3d at 1347 ("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 that Petitioner has provided preponderant evidence of a sound and reliable causal mechanism as required under Althen prong one for the following reasons.

First, Dr. Gershwin's theory is well supported by the medical literature. His theory is referenced in numerous articles filed by both parties. Related to molecular mimicry, Ito provides that the pathogenesis of AA involves an "autoimmune reaction[] against hair follicle autoantigens." Pet. Ex. 27 at 1. Another example is set forth in Tuccori et al., who, citing Wise et al., state the "immune-mediated mechanism [] suggest[s] the existence of antigenic molecular similarities between vaccines and hair follicles . . . in susceptible patients." Pet. Ex. 98 at 2

(citing Pet. Ex. 80). And Guo et al.[75] explain that the "putative hair follicle may . . . be broken by activating in the immune system against hair follicle autoantigens." Resp. Ex. C, Tab 6 at 3.

Likewise, the literature includes references about activation of CD4+ and CD8+ T cells, as described by Dr. Gershwin. Guo et al. explain that scalp histopathology "reveals [] activated CD4+ and CD8+ cells . . . accumulate around . . . hair follicles" and cause "disruption and destruction." Resp. Ex. C, Tab 6 at 4-5. CD8+ T cells, after differentiation, are required for disease "induction and perpetuation." Id. at 5.

Second, Respondent's experts do not refute that Dr. Gershwin's theory and discussion of the pathogenesis of AA is sound and reliable. Dr. Senna acknowledges that the pathogenesis of AA involves "exogenous factors in a genetically susceptible" person. Resp. Ex. A at 8. She agrees the process involves CD8+ cytotoxic T cells and "an autoimmune attack to a[n] [] unrecognized antigen." Id. She, like Dr. Gershwin note there may be "several different immunologic pathways" leading to the condition. Id. at 9. She does not, however, disagree with the opinions of Dr. Gershwin as to the causal mechanisms.

Dr. Krakowski also agrees with the theory of molecular mimicry, explaining that "one's own immune system mistakes otherwise normal, healthy hair follicles as 'dangerous' and begins to attack them." Resp. Ex. E at 11. Regarding vaccinations, he agrees a "rare association" of AA has been reported after several vaccinations, including HPV. Id. at 12.

Only Dr. Tompkins offers criticism related to Dr. Gershwin's casual mechanism. And his criticism is not specific to the theory itself but is based on a lack of evidence supporting a "direct association." For molecular mimicry, Dr. Tompkins seeks evidence of the cross reactive epitopes, and here, there is no such evidence. Dr. Gershwin acknowledges that the T cell epitopes are not known.

Petitioner need not make a specific type of evidentiary showing or require identification of a specific antigenic trigger or here, proof of direct association, for an immune-mediated pathology to prove a theory is sound and reliable by preponderant evidence. Given the state of current scientific knowledge, there is no way to satisfy such a requirement. Requiring proof of the identify of a specific antigen to prove causation would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program").

Next, Dr. Tompkins opines it is unlikely that the HPV vaccine could induce an immune response to lead to "non-specific activation of autoreactive T cells." Resp. Ex. C at 7. He cites Herrin et al. in support of this criticism. However, Herrin et al. did not analyze CD8+T cell cytokine responses after vaccination. CD4+ responses were tested 12 months after vaccination, but this would be too late to provide meaningful information about vaccine causation here. Thus, Dr. Tompkins's reliance on Herrin et al. is misplaced.

[75] Hongwei Guo et al., The Role of Lymphocytes in the Development and Treatment of Alopecia Areata, 11 Expert Rev. Clinical Immunology 1335 (2015).

The same problem arises out of Dr. Tompkins' opinion that cytokines IFN-γ and/or TNF-α are not present after HPV vaccination. Herrin et al. showed that the HPV vaccination did increase IFN-γ producing cells in peripheral blood mononuclear cells as compared to pre-vaccination levels in response to HPV types 16 and 18. The vaccine at issue here also included HPV types 16 and 18. Herrin et al. did state there was no increase in IFN-γ or TNF-α in plasma, but this was distinct from the findings in the peripheral blood mononuclear cells post-vaccination. Dr. Tompkins did not make this distinction in his expert report. And if this distinction was relevant, he did not explain why.

In summary, of Respondent's experts, only Dr. Tompkins offered opinions critical to Dr. Gershwin's mechanistic theory, but his reliance on Herrin et al. appears misplaced. Thus, the undersigned does not find Dr. Tompkins' opinions persuasive.

Third, Dr. Gershwin's opinions about vaccine association are supported by several articles that suggest an association between vaccination and AA, relying on the same theoretical framework. Wise et al. described 60 instances of alopecia following vaccinations. While the paper was published before introduction of the HPV vaccination, it is relevant here to establish that vaccine causation of alopecia has been contemplated by the medical community since the paper was published in 1997. And more recently, COVID-19 vaccinations have been associated with vaccination.

Fourth, specific support for HPV vaccination as a causal agent of AA is based on two sources. First, HPV infection is associated with a "significantly greater" risk of AA based on the study by Tu et al., a large cohort of 30,000 patients. Although the authors cautioned that the findings may not apply to non-Asian ethnic groups, this caveat does not diminish the validity of the results as to the group studied.

Further, the case reports by Tuccori et al. support HPV vaccine associated AA. Both children had onset within approximately one month of vaccination, and both worsened after subsequent doses of the vaccine. Alternative causes were investigated and ruled out. The immune mechanism offered was the same mechanism advanced by Dr. Gershwin here, "the existence of antigenic molecular similarities between vaccines and hair follicles . . . in susceptible patients." Pet. Ex. 98 at 2. The authors concluded there was a "probable relationship" between the HPV vaccinations and alopecia, especially given the worsening of alopecia following subsequent vaccine doses. Id. at 3. In his expert report, Dr. Krakowski explains that AA is a form of anagen effluvium. The two children described by Tuccori et al. were described as having telogen effluvium. Id. at 11. However, neither Dr. Krakowski nor Respondent's other experts offered criticism of or rebuttal to the conclusions reached by the authors of Tuccori et al., that the HPV vaccine played a causal role in causing alopecia in these two cases.

The undersigned acknowledges that other special masters have reached a different outcome in three HPV/AA cases. See Rose v. Sec'y of Health & Hum. Servs., No. 17-1770V, 2025 WL 1218279 (Fed. Cl. Spec. Mstr. Mar. 20, 2025). Farag v. Sec'y of Health & Hum. Servs., No. 17-714V, 2023 WL 7203034 (Fed. Cl. Spec. Mstr. Sept. 29, 2023); Cordova v. Sec'y

of Health & Hum. Servs., No. 17-1282V, 2021 WL 3285367 (Fed. Cl. Spec. Mstr. June 23, 2021). These cases are distinguishable based on clinical presentations, risk factors, onset, diagnosis, potential alternative causes, and expert opinions. While Dr. Gershwin offered opinions in all three cases, the experts offered by Respondents differed (although Dr. Senna did offer an opinion in one case). Each case must be assessed on its own merit, based upon the facts and circumstances, and the expert opinions offered. Moreover, prior decisions are not binding on the undersigned. See Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); Boatmon, 941 F.3d at 1358.

And in another reasoned ruling on entitlement, Petitioner was awarded compensation for AA after the Hepatitis B vaccine. In Delozier, the Chief Special Master found Petitioner entitled to compensation noting that AA is an autoimmune disease, which may occur following infection, stress, and vaccination. Delozier ex rel. L.T. v. Sec'y of Health & Hum. Servs., No. 15-124V, 2019 WL 7556051, at *15, *19 (Fed. Cl. Spec. Mstr. Dec. 10, 2019). The medical literature offered by Petitioner in that case included Wise et al., which was credited as offering support as it showed instances of challenge-rechallenge. Id. at *16. Here, Petitioner has offered evidence (Tuccori et al.) that the HPV vaccination was associated with two cases of alopecia, with evidence of worsening (challenge-rechallenge) after a second and/or third dose of the HPV vaccination. The undersigned finds the evidence of challenge-rechallenge in the Tuccori et al. case reports strong evidence of causation here.

For all of these reasons, the undersigned finds that the weight of the evidence as to Althen prong one preponderates in Petitioner's favor.

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").

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and

effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds that Petitioner has proven Althen prong two by preponderant evidence for several reasons.

First, Petitioner's clinical course was consistent with vaccine causation via an autoimmune reaction consistent with that offered by Dr. Gershwin. Petitioner had risk factors for AA, and as explained by Dr. Gershwin, these risk factors made him more susceptible to developing the condition. And he was exposed to an antigenic trigger, the HPV vaccination.

Second, the undersigned finds that Respondent's experts' opinions suggesting alternate causes are not persuasive. Dr. Senna offers the Treg dysfunction theory based on Petitioner's allergies and history of atopic dermatitis. But her opinion in this regard was not stated to a standard of preponderance. The same is true of Dr. Krakowski's opinion that the Petitioner's underlying atopic dermatitis was a trigger for his AA. Dr. Krakowski initially stated this was a "possible" trigger for Petitioner's AA. Then, in the last paragraph of his report, he used the phrase "more likely than not" that Petitioner's atopic dermatitis caused his AA. This inconsistency renders Dr. Krakowski's opinion less persuasive. Similarly, Dr. Krakowski opined that Petitioner had psoriasis which increased his risk, but again, he stated that this alternative cause was only a "possibility."

Opinions expressed as possibilities are not sufficient to establish causation as they do not rise to the level of preponderance. See, e.g., Waterman, 123 Fed. Cl. at 573-74; Moberly, 592 F.3d at 1322 (emphasizing that possibilities does not equate to proof of causation by a preponderance of the evidence); de Bazan, 539 F.3d at 1351.

Moreover, Petitioner's treating physician convincingly opined that Petitioner did not have psoriasis.

Regarding Respondent's alternative cause assertions, the undersigned finds on the whole, that they are not persuasive. Dr. Senna did not offer her opinions to a preponderant standard, and Dr. Krakowski was inconsistent in his application of the appropriate standard. For this reason, the undersigned finds that Petitioner's history of atopic conditions increased his risk for AA, consistent with the opinions of Dr. Gershwin, but did not constitute an alternative cause of his AA.

Third, Petitioner's treating dermatologist, Dr. Javahery offered support in favor of causation. She opined that onset of autoimmune disease is often related to a preceding immune trigger. She explained that Petitioner never experienced hair loss before and had no history of autoimmune disease. He developed rapid onset of aggressive AA directly following HPV vaccination. She opined that it was "a stretch" to assume there was "no association between the two events." Pet. Ex. 106 at 1. While this statement may not reach the applicable standard of more likely than not, it provides some weight in favor of vaccine causation.

In adjudicating Althen prong two, 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).

Lastly, Petitioner's medical records document Dr. Agatep noted an allergic reaction of alopecia to the HPV vaccine and stated, "hair loss, from vaccine." Pet. Ex. 101 at 50-51.

"A treating doctor's recommendation to withhold a certain vaccination can provide probative evidence of a causal link between the vaccination and an injury a claimant has sustained." Andreu, 569 F.3d at 1376; see also Kelley v. Sec'y of Health & Hum. Servs., 68 Fed. Cl. 84, 98, 100 (2005) (determining that the petitioner's treating physicians' reluctance to authorize the petitioner with further tetanus vaccinations was "robust" medical evidence of vaccine causation). A treating physician's recommendation against future vaccination is supportive of a petitioner's prong two burden and "helps satisfy the second Althen prong." Michie v. Sec'y of Health & Hum. Servs., No. 19-453V, 2023 WL 10410004, at *7 (Fed. Cl. Spec. Mstr. Dec. 4, 2023).

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. Although the statements and opinions offered by Petitioner's treating physicians alone do not constitute preponderant evidence, when combined with the other evidence herein, they support a finding in favor of Petitioner.

Thus, the undersigned finds Petitioner has proven Althen prong two by preponderant evidence.

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 coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542.

The parties agree that Petitioner received his first HPV vaccine on October 17, 2016, and was first noted to have lost his eyelashes in November 2016. Joint Submission at 1.

Both parties' experts agree with this timeline. Dr. Gershwin opined that onset within 14 days was consistent with his mechanistic theory. Dr. Senna and Dr. Krakowski did not refute onset as inconsistent with an immune-mediated mechanism. And Dr. Tompkins agreed that the timing of Petitioner's AA onset was appropriate under the theory proposed by Dr. Gershwin.

Therefore, undersigned finds that Petitioner has met his burden of proof as to Althen prong three.

V. CONCLUSION

Based on the record, and for the reasons discussed above, the undersigned finds there is preponderant evidence to satisfy all three Althen prongs and to establish that Petitioner's HPV vaccination caused him to develop AA. Thus, the undersigned finds that Petitioner is entitled to compensation.

A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master