In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 17-1282V

(to be published)

* *

ADRIAN CORDOVA, IV,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

* *

Filed: June 23, 2021

Amy Senerth, Muller Brazil, LLP, Dresher, PA, for Petitioner.

Camille Collett, U.S. Dep't of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On September 18, 2017, Erika Hicks, as parent and natural guardian of A.C., a minor, filed a petition seeking compensation under the National Vaccine and Injury Compensation Program (the "Vaccine Program").[2] (ECF No. 1) ("Petition"). The Petition alleged that Mr. Adrian Cordova, IV suffered from alopecia areata ("AA") attributable to a human papillomavirus ("HPV") vaccine he received on July 22, 2016, or was significantly aggravated by a second HPV dose received on

[1] This Decision shall be posted on the Court of Federal Claims' website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012)). **This means that the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the Decision's inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Decision will be available to the public. *Id*.

[2] The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended at 42 U.S.C. §§ 300aa-10 through 34 (2012) [hereinafter "Vaccine Act" or "the Act"]. Individual section references hereafter will be to § 300aa of the Act (but will omit that statutory prefix).

October 12, 2016. Petition at 1. The caption was changed upon my order after the Petitioner became 18 years old. Order, dated June 1, 2021 (ECF No. 81).

I have determined this matter could be most efficiently resolved via ruling on the record. Based on the record and the parties' other written submissions, I find that Petitioner has not carried his evidentiary burden. Insufficient evidence supports the conclusion that the HPV vaccine can cause AA, or did so to Petitioner. And although Petitioner's significant aggravation claim is satisfied in some respects, the failure to show the vaccine can cause AA in the first place negatively impacts the argument that it could worsen it as well.

I. Factual Background

On July 22, 2016, Mr. Cordova (who was at the time thirteen years old) received his first dose of HPV vaccine in Aurora, Colorado. Ex. 1 at 2. Prior to vaccination, he had no history of anything like the AA he later experienced. *See e.g.* Pet. Ex. 2, p. 13; Pet. Ex. 10, pp. 1-6. There is also nothing from this initial record or not long thereafter suggesting he experienced any reaction to the vaccine, and his prior medical conditions do not bear on the matters at issue in this case.

Almost three months passed without any additional visits to a medical treater. Then, on October 12, 2016, Petitioner was taken back to his pediatrician with reports that the month before (meaning September 2016), his mother (Erika Hicks) had noticed[3] a 1x1 cm bald spot on the posterior of his head. Ex. 2 at 13-14; Ex. 10 at 9-13 (photographs of October 11, 2016). Within a few days thereafter, Petitioner's family had observed the spot increased to 2 x 2 cm. *Id*. Two weeks later, more bald spots began occurring on the front and side of his head, although Petitioner's hair loss had not otherwise progressed. *Id.*

Petitioner's pediatrician observed patchy, non-scarring bald spots throughout his scalp and head, although hair roots appeared intact, and referred him to a pediatric dermatologist, due to his rapid hair loss progression (although it appears from the record that this did not later occur due to financial issues with obtaining such additional treatment). Ex. 2 at 14. He also diagnosed Petitioner at this time with alopecia. *Id.* at 3, 13. That same day, Petitioner received his second HPV vaccine dose. *Id.* at 14.

Approximately two weeks later, on October 28, 2016, Mr. Cordova returned to his pediatrician, now complaining that more patches of his hair had fallen out. Ex. 2 at 12. In reaction, Petitioner had been shaving his head. *Id*. Treaters proposed that a fungal infection might explain

[3] In support of his claim, Petitioner offered affidavits from family members like Ms. Hicks, who elaborated on onset as well as Petitioner's experience suffering with AA. *See generally* Affidavit, filed June 3, 2019 as Ex. 13-1 (ECF No. 49-2) ("Adrian Cordova Aff."); Affidavit, filed June 3, 2019 as Ex. 14-1 (ECF No. 14-1) ("Vicki Hicks Aff."); Affidavit, filed July 11, 2019 as Ex. 15-1 (ECF No. 53-2) ("Adrienne Alexander Aff."); Affidavit, filed July 11, 2019 as Ex. 16-1 (ECF No. 53-3) ("Ebony Neal Aff."). But the witness statements are largely consistent with the medical record, which places uncontested issues such as onset and date of aggravation as occurring after the relevant vaccine doses, or otherwise pertain to matters not bearing on causation, like the degree of suffering Petitioner experienced from his AA. I accordingly do not discuss these items of evidence in further detail.

the hair loss, and prescribed oral antifungals, along with a cessation of head shaving. *Id.* No AA was observed at this visit, and Petitioner's diagnosis was changed from alopecia to trichotillomania (a fungal infection). *Id.* at 3, 12.

Petitioner next went back to his pediatrician on December 13, 2016, for further evaluation of his hair loss problem. Ex. 2 at 11. After his last visit, he had shaved the remainder of his head, and also completed the course of antifungal medication. *Id*; see also Ex. 10 at 14-15. Exam revealed AA plus broken hairs, but the fungal infection diagnosis was not abandoned, and it appeared some of the hair loss patches were displaying regrowth. Ex. 2 at 3, 11. Petitioner now received a cortisone shot into his scalp, and was advised to follow up with his treaters a few weeks later. *Id.* at 11.

On January 10, 2017, Petitioner returned for the scheduled follow-up visit. Ex. 2 at 10. He now reported that the steroid shots had been helpful, and that he had seen significant hair regrowth. *Id*. He received a second cortisone injection at this visit, with another at the end of January. *Id.* at 9, 10. Despite regrowth, however, Petitioner continued to experience persistent bald spots into 2017, and anxiety attributable to his hair loss began to plague him as well. *Id.* at 6, 8. His diagnosis remained a fungal infection, and Petitioner received additional cortisone injections. *Id.* at 3, 6.

By May 2017, Petitioner was demonstrating marked improvement in his hair growth, and the fungal infection diagnosis was maintained. Ex. 2 at 3-5. On September 30, 2017, Petitioner was seen at Advanced Dermatology with a chief complaint of hair loss of moderate severity located on his scalp which was claimed to be temporally associated with the HPV vaccine. Ex. 5 at 7. Petitioner was diagnosed now with widespread alopecia areata on about 80 percent of his scalp, prescribed an oral steroid, and informed about the autoimmune nature of AA. *Id.* Petitioner went back to the same dermatologic treater in December 2017, at which time it was noted that Petitioner was having trouble treating his condition.

As of January 2018, Petitioner's AA had spread to his eyelashes and legs. Ex. 5 at 4. Ongoing issues with financing his care, however, continued to get in the way of obtaining treatment. Petitioner thereafter experienced additional psychologic harms associated with his hair loss. No medical records since this time were filed in the case.

II. Expert Reports

A. *Petitioner's expert: M. Eric Gershwin, M.D.*

Dr. Gershwin, an immunologist, filed three reports. *See* Report, dated March 20, 2018, filed as Ex. 7-1 (ECF No. 23-1) ("First Gershwin Rep."); Report, dated August 8, 2019, filed as Ex. 17-2 (ECF No. 58-2) ("Second Gershwin Rep."); Report, dated November 23, 2019, filed as Ex. 18-1 (ECF No. 62-2) ("Third Gershwin Rep."). Dr. Gershwin opined that Mr. Cordova's AA was attributable to the HPV vaccine, and/or that the second dose exacerbated it.

Dr. Gershwin received his bachelor's degree from Syracuse University in Syracuse, New York, followed by his medical degree at Stanford University. Dr. Gershwin Curriculum Vitae, filed as Ex. 19 on 1 (ECF No. 82-2) ("Gershwin CV"). He then completed his internship and residency at Tufts–New England Medical Center in Boston, Massachusetts. *Id.* at 2. After completing a fellowship in immunology with the National Institute of Health, Dr. Gershwin became an assistant Professor in Rheumatology and Allergy at the University of California, School of Medicine in Davis, California. *Id.* Dr. Gershwin is now semi-retired—though he continues to work on a "callback" basis at the University of California, School of Medicine in Davis providing consultations for rheumatology and immunology patients. Gershwin CV at 1–2. Throughout his career, Dr. Gershwin has evaluated both pediatric and adult patients, though he now sees fewer pediatric patients than he did earlier in his career. *Id.* at 2. He currently serves as the editor-in-chief of the Journal of Autoimmunity as well as several other publications focusing on autoimmunity. Gershwin CV at 5-6.

First Report

Dr. Gershwin's first report began with an overview of AA. Its presentation is characterized by "one or more circular bald patches around the scalp," and has variants that involve more extreme hair loss, such as alopecia totalis. First Gershwin Rep. at 1. Nearly two percent of all individuals will experience some form of AA (making it somewhat common from a disease prevalence perspective), and it evolves or presents unpredictably. *Id.* at 1-2. Dr. Gershwin specifically emphasized that "[t]he genetic basis of alopecia is strongly supported" by a number of reliable items of scientific or medical literature. *Id*. at 2.

As Dr. Gershwin explained, AA is generally thought by medical science to be autoimmune-mediated. First Gershwin Rep. at 3. A number of factors support this contention, including (a) the association of AA with other known autoimmune illnesses, like lupus or rheumatoid arthritis, (b) the fact that immunosuppressive treatments are effective in arresting AA, and (c) the fact that certain human leukocyte antigens ("HLA") associated with AA can (when present in a person) also reflect a genetic propensity for the condition. *Id.* at 3-5.

The loss of hair that characterizes AA is in fact the product of a specific kind of immune-oriented attack. Hair follicles are understood to enjoy "immune privilege"—meaning they are especially resistant to environmental attacks, propagated by infection or other outside stimuli, that might otherwise harm them. First Gershwin Rep. at 3-4; L. Petukhova, et al., *Genome-wide Associate Study in Alopecia Areata Implicates Both Innate and Adaptive Immunity*, 466:7302 Nature 113-117 (2010), filed as Ex. 17.1 on Aug. 23, 2019 (ECF No. 58-3) ("Petukhjova"); McElwee, et al., *Alopecia Areata: An Autoimmune Disease?*, 8 Exp. Dermatol. 371-379 (1999), filed as Ex. 17.2 on Aug. 23, 2019 (ECF No. 58-4) ("McElwee"); F. Rajabi, et al., *Alopecia Areata: A Review of Disease Pathogenesis*, 179 Br. J. Dermatol. 1033-48 (2018), filed as Ex. 17.3 on Aug.

23, 2019 (ECF No. 58-5) ("Rajabi"). To some degree this immune privilege reflects the fact that the hair follicles, being on the scalp (and thus exposed externally to more outside environmental triggers than other parts of the body), need to be more resistant to infection. But in certain circumstances, privilege can "break," leading to hair follicle destruction. First Gershwin Rep. at 4.

The driver of the autoimmune attack causing AA is a specific immune cell, the T cell. Scientific research has revealed the identity of a "repertoire of cells" in the hair follicles of individuals suffering from AA, and those cells are largely a variety of T cells—mostly CD4+, or "helper" T cells, which indirectly aid the adaptive immune process, but also CD8+, or cytotoxic "killer" T cells, which are responsible for the direct autoimmune attack on the follicles. First Gershwin Rep. at 4. The attack by these cells only occurs, however, *after* immune tolerance has been broken, at which point the T cell lymphocytes infiltrate the follicle and attack. Gershwin Rep. at 4; Petukhova.

Dr. Gershwin next addressed the likelihood that the HPV vaccine could have triggered Mr. Cordova's AA. He began by discussing the propensity of other vaccines to stimulate uncommon adverse events. Measles vaccines (like Measles-Mumps-Rubella ("MMR")) have been shown to upregulate a wide variety of genes, some of which impact immunity or specific cell function, and even apoptosis, or destruction. First Gershwin Rep. at 5. Because "the multitude of genetic diversity in response to a vaccine is truly extraordinary," it is likely such diversity could result in "rare events" that, while not detectible in large-scale epidemiologic studies, could nevertheless be harmful, especially for susceptible individuals. First Gershwin Rep. at 5. In addition, Dr. Gershwin stressed the extent to which environmental factors were significant in impacting an individual's immune response. *Id.* at 6. And articles existed that provided a reliable basis for concluding that certain vaccines could be related to AA specifically. First Gershwin Rep. at 5; R. Wise et al., *Hair Loss After Routine Immunizations*, 278 JAMA 1176 (1997).[4]

Relying on the above, Dr. Gershwin proposed that the HPV vaccine instigated for Mr. Cordova the production of cytotoxic T cells. First Gershwin Rep. at 5. The T cells would be stimulated to attack due to homologic similarity between their hair follicle targets and "an epitope or region of the [HPV] vaccine." *Id.* The mechanism by which this would occur is molecular mimicry, adding that an animal model studying AA corroborates the applicability of the mechanism. *Id.* Here, the mimicry would be driven not by an autoantibody cross-attack, however, but T cells, which Dr. Gershwin noted were extremely difficult to study in such a context. First

[4] Dr. Gershwin's original report had referenced 81 items of medical literature. However, the former Special Master in charge of this case struck these 81 items as excessive, and ordered Petitioner to refile a selection of those articles deemed most relevant or helpful. *See* Order, dated Sept. 6, 2019 (ECF No. 59). Petitioner subsequently filed a total of 14 items of literature, but this subset does not include articles like Wise. *See* Notice of Filing, dated Oct. 11, 2018 (ECF No. 35). For this reason, not all of the articles referenced in Dr. Gershwin's earlier reports to support his assertions are cited herein.

Gershwin Rep. at 7 ("it is thus far impossible to individually predict T cell epitopes unique to a specific individual in response to a vaccine").

Regarding the timeframe for the theorized autoimmune attack, Dr. Gershwin proposed that the cytotoxic T cells required to damage the follicles could generate within days of the initial propagating signal. First Gershwin Rep. at 5. As a result, a 14-day onset "would certainly be consistent" with the time needed to generate such immune cells. First Gershwin Rep. at 5.

Second Gershwin Report

Dr. Gershwin's second report was substantially shorter, and attempted to respond both to some aspects of the report submitted by Respondent's expert (discussed below) and questions raised by the prior special master presiding over this matter about the degree to which the HPV vaccine might instigate a T cell response sufficiently pathologic to cause AA.

First, Dr. Gershwin cited the evidence he believed constituted credible support for Petitioner's purported AA worsening after receiving a second HPV vaccine dose. He observed that several individuals, including Petitioner's mother, father, and grandmother, had offered witness statements detailing their observations of worsening, and these statements plus other photographic evidence was documentary proof that his AA had progressed. Second Gershwin Rep. at 1-2. He also cited evidence of problems at school and with academic performance that Petitioner encountered after the second dose. *Id.* at 2.

Second, Dr. Gershwin attempted to bulwark his argument about the vaccine's alleged T cell-stimulative impact. He highlighted literature support for the conclusion that AA involved autoimmune destruction of hair follicles mediated by CD8+ T cells, adding that "innate and adaptive immunity are responsible for [AA]." Second Gershwin Rep. at 2; Petukhova at 114. He agreed that AA was not mediated by autoantibodies, but stressed it was still an autoimmune condition in nature, adding that T and B cells would likely work cooperatively in any autoimmune attack. Second Gershwin Rep. at 3. He did not, however, offer any additional literature or statements relating to the association between the vaccine and a pathologic increase in T cells sufficient for AA to occur (beyond the articles mentioned above, like Petukhova).

Third Gershwin Report

Dr. Gershwin submitted a final, two-page report in response to a question about an item of literature filed as a court exhibit in this case.[5] *See* M. Yokomine et al., *Enhancement of Humoral and Cell Mediated Immune Response to HPV16 L1-Derived Peptides Subsequent to Vaccination with Prophylactic Bivalent HPV L1 Virus-Like Particle Vaccine in Healthy Females,* 13 Exp. Therap. Medicine 1500 (2017), DOI: 10.3892/etm.2017.4150, filed as Court Exhibit 1 (ECF No. 60-1) ("Yokomine"). In Yokomine, ten women ages 23-33 received three doses of the Cervarix

[5] *See* Scheduling Order, dated November 8, 2019 (ECF No. 60).

formulation of the HPV vaccine, and blood samples were taken from each subject five times after the first dose was administered, over an eighteen-month period. Yokomine at 1501. Yokomine's authors observed a significant increase in T cell response within a month of receipt of the first dose (when compared to the subject's pre-vaccination status), suggesting that the vaccine could simultaneously induce a B *and* T cell response. *Id.* at 1503. The discussion of this increase does not specify, however, whether the T cells in question were helper or cytotoxic T cells. *Id.* Yokomine concluded that its findings helped better understand the efficacy of this version of the HPV vaccine—and in particular what biomarkers should be looked for when assessing the vaccine's success in stimulating immunity. *Id.* at 1501, 1505.

Dr. Gershwin deemed Yokomine's findings "not surprising," stressing that even a vaccine (like the HPV vaccine) intended to elicit a B cell response (in order to produce a specific antibody) will also necessarily have to prompt CD4+ helper T cells into action as well. Third Gershwin Rep. at 1. But he seemed to understand that one of Yokomine's primary goals was to identify a specific biomarker to measure the vaccine's immunogenicity—and thus not to measure if T cell reaction was abnormal. He added that the relevant biomarker might vary depending on an individual's personal genetic makeup. *Id.* Moreover (and admitting that his comments were adding "further confusion to the 'pot'"), Dr. Gershwin acknowledged that existing medical science could not easily distinguish (by epitopes) between CD4+ and CD8+ T cells, especially when compared to its ability to detect the presence of specific antibodies, and as a result Yokomine's authors "were only able to focus on continuous epitopes because that is the easy approach." *Id.* at 2. He concluded by emphasizing the difficulty in demonstrating an identity between the HPV vaccine components and hair follicle proteins sufficient to establish a potential for cross-reactivity, suggesting ultimately that genetic susceptibility was the key factor in the vaccine causing harm. *Id.*

B. *Respondent's expert: Andrew MacGinnitie, M.D., PhD.*

Dr. MacGinnitie—like Dr. Gershwin is an immunologist, although with more of a pediatric focus—offered two expert reports. Report, dated February 1, 2019, filed as Ex. C (ECF No. 44-1) ("First MacGinnitie Rep."); Report, dated March 16, 2020, filed as Ex. E (ECF No. 67-1) ("Second MacGinnitie Rep.").[6] He disputed Dr. Gershwin's contentions about an HPV-AA association.

Dr. MacGinnitie is an attending physician and the Clinical Director for the Division of Immunology at Boston Children's Hospital in Boston, Massachusetts. Dr. MacGinnitie Curriculum Vitae, filed as Ex. D on Feb. 8, 2019 (ECF No. 44-27) ("MacGinnitie CV"). He is also an Associate Professor of Pediatrics at Harvard Medical School. MacGinnitie CV at 1-2. Dr. MacGinnitie

[6] Dr. Francis Lobo, an immunologist, prepared Respondent's initial report, disputing Petitioner's contention that the HPV vaccine can cause AA. Report, dated May 29, 2018, filed as Ex. A (ECF No. 30-1) ("Lobo Rep."). Dr. Lobo died in 2018, however, and Respondent subsequently represented to the special master previously presiding over this case that he would no longer be relying upon that opinion (and Dr. MacGinnitie himself indicated in his first report that he had neither reviewed nor relied on Dr. Lobo's written report). First MacGinnitie Rep. at 2. For this reason, I do not discuss Dr. Lobo's opinion herein, and have not evaluated it in deciding entitlement.

received his undergraduate degree from Yale University, followed by both a medical degree and Ph.D. from the University of Chicago. *Id.* at 1. He thereafter completed his residency, followed by a fellowship in allergy and immunology at Boston Children's. *Id.* He is board certified in pediatrics and allergy and immunology, and has been in practice as an allergist/immunologist since 2004. MacGinnitie CV at 10. Dr. MacGinnitie's research focuses on food allergies, and he serves on the editorial board of the journal Annals of Allergy, Asthma and Clinical Immunology. He also maintains an active clinical practice seeing more than 1600 patients annually and has extensive experience caring for patients with a variety of immunologic diseases included reactions to vaccines.

First MacGinnitie Report

Dr. MacGinnitie's first report began with an overview of Mr. Cordova's relevant medical history, before moving on to a summary of Dr. Gershwin's opinion—and his individualized reactions and objections to it. First MacGinnitie Rep. at 2-3.

First, Dr. MacGinnitie contended that Dr. Gershwin's theory was neither logical nor testable. Rather, Dr. Gershwin "simply asserts" that the vaccine, via a molecular mimicry mechanism, triggered an autoimmune response—but without offering reliable evidence to show homology between the vaccine's antigens and the hair follicle proteins that are attacked in AA. First McGinnitie Rep. at 3. Molecular mimicry is in fact not understood to be a potential mechanism for AA. *Id.* at 4.

Next, Dr. MacGinnitie challenged the overall contention that any vaccine, let alone the HPV vaccine, could initiate AA. He noted that there were many proposed environmental triggers for AA, including infection, but little known about how any would set AA into motion. First MacGinnitie Rep. at 6 (*citing* Rajabi). Case reports, moreover, offered to show individual instances in which a vaccine's administration preceded onset of AA needed to be considered against the likely high background/baseline rate for AA (which Dr. Gershwin acknowledged was experienced by two to three percent of the total population, making it not particularly rare). First MacGinnitie Rep. at 6. In addition, Dr. MacGinnitie highlighted the fact that the HPV vaccine's immune-stimulative impact was known to be quite minimal. *Id.* at 8; J. Kelso, et al., *Adverse Reactions to Vaccines Practice Parameter 2012 Update*, (July) J. Allergy Clin. Immunol. 1-43 (2012), filed as Ex. C, Tab 15 on Feb. 8, 2019 (ECF No. 44-16). Vaccination generally was in his view less likely to produce illness than other immune challenges individuals face on a daily basis. First MacGinnitie Rep. at 8-9.

Articles offered by Dr. Gershwin about the general association between vaccination and AA, Dr. MacGinnitie maintained, were unpersuasive support for Petitioner's theory when evaluated closely. Some, for example, relied on data from passive surveillance systems, were specific to *other* vaccines, or ultimately showed few instances that could suggest an association. First MacGinnitie Rep. at 7. And the mouse AA model referenced By Dr. Gershwin as supporting

his theory about mechanism undercut the conclusion that one particular vaccine (hepatitis B) was associated with AA. First MacGinnitie Rep. at 7.

At the same time, Dr. MacGinnitie highlighted the fact that reliable, larger-scale epidemiologic studies did not associate the HPV vaccine generally with *any* autoimmune conditions. First MacGinnitie Rep. at 7-8; C. Chao, et al., *Surveillance of Autoimmune Conditions Following Routine Use of Quadrivalent Human Papillomavirus Vaccine*, 271 J. Intern. Med. 193-203 (2012), filed as Ex. C Tab 11 (ECF No. 44-12) ("Chao"); J. Skufca, et al., *The Associate of Adverse Events with Bivalent Human Papilloma Virus Vaccination: A Nationwide Register-Based Cohort Study in Finland*, 36 J. Intern. Med. 5962-5933 (2018), filed as Ex. C Tab 12 on Feb. 8, 2019 (ECF No. 44-1). Dr. MacGinnitie admitted, however, that he had seen no studies that specifically considered AA when evaluating whether other autoimmune illnesses were associated with the HPV Vaccine. First MacGinnitie Rep. at 7.

Dr. MacGinnitie further emphasized a weakness central to Petitioner's theory about the HPV vaccine's capacity to drive a pathologic process. He agreed with Dr. Gershwin that (as literature filed in the matter emphasized) AA "is mediated by CD8+ T Cells." First MacGinnitie Rep. at 4; M. Newport, *The Genetic Regulation of Infant Immune Responses to Vaccination*, 6(18) Font Immunol. 1-5 (2015), filed as Ex. C Tab 25 on Feb. 8, 2019 (ECF No. 44-26). But the HPV vaccine *itself* seeks to elicit an adaptive response in the immune system mediated by *B cell-created* antibodies. First MacGinnitie Rep. at 4; J. Schiller, et al., *Understanding and Learning From the Success of Prophylactic Human Papillomavirus Vaccines*, 10 Nat. Rev. Microbiol. 681-692 (2012), filed as Ex. C Tab 2 on Feb. 8, 2019 (ECF No. 44-3). Thus, the vaccine's intended purpose is *not* to create a T cell response of the sort understood to drive the hair follicle damage central to AA.

Dr. Gershwin's reports suggested that regardless of its intended design and immune impact, the HPV vaccine might nevertheless also stimulate production of the pathogenic T cells central to AA, but Dr. MacGinnitie observed numerous deficiencies in this argument. As a general matter, the fact that vaccine was not an effective treatment for an active HPV infection (like cervical cancer) was evidence that it did not inherently cause significant T cell upregulation—since that kind of immune cell would be "required to kill virally infected cells" that would be present during an ongoing wild infection. First MacGinnitie Rep. at 4. Dr. MacGinnitie also took issue with the literature Dr. Gershwin had offered to support this contention. *Id.* at 4-5. In particular, he noted the articles either confirmed the extent to which the vaccine instigated B cell production of antibodies, or only reliably established an increase in CD4+ helper cells (which would aid the B cell antibody production but not be pathogenic in causing AA). *See, e.g.*, L. Pinto, et al., *Cellular Immune Response to Human Papillomavirus (HPV)-16 L1 in Healthy Volunteers Immunized with Recombinant HPV-16 L1 Virus-Like Particles*, 188 J. Infect. Dis. 327-38 (2003), filed as Ex. C Tab 6 on Feb. 8, 2019 (ECF No. 44-7).

Dr. MacGinnitie deemed only one article directly relevant to Dr. Gershwin's T cells-due-to-HPV-vaccine-upregulation contention. First MacGinnitie Rep. at 5; D. Herrin et al., *Comparison of Adaptive and Innate Immune Responses Induced by Licensed Vaccines for Human Papillomavirus,* 10 Hum. Vaccines & Immunotherapeutics, 3446-54 (Dec. 2014), filed as Ex. 9.6-1 (ECF No. 35-6) ("Herrin"). Herrin was a study intended to evaluate the comparative immunogenic qualities of two different HPV vaccine formulations: Gardasil (the vaccine at issue in this case) and Cervarix. Herrin at 3446-47. To do so, the study's authors conducted a randomized trial of 27 women, with 15 of the total group receiving the Gardasil formulation. *Id.* at 3447. Although Herrin did attempt to measure post-vaccination CD4+ helper T cell levels in the study's subjects, and found increases, the study's authors ultimately did not choose to closely evaluate the extent to which the relevant kind of cytotoxic T cells were upregulated by the HPV vaccine—mainly because of "limited responses" (likely meaning not enough evidence of any measurable increase). From this, Dr. MacGinnitie concluded that the HPV vaccine did not in fact have the indirect effect of stimulating a significant CD8+ T cell response. First MacGinnitie Rep. at 5.

Dr. MacGinnitie also questioned the reliability and utility of another article. First MacGinnitie Rep. at 5; M. Mascolini, *Quadrivalent HPV Vaccine Elicits Antibody and Cell Responses in HIV+ Teens, Young Adults*, http://www.natap.org/2014/IAC/IAC_21.htm, filed as Ex. 9.7.1 (ECF No. 35-7) ("Mascolini"). The article—an abstract that contained only a summary of its findings rather than any of its underlying data—referenced a case control study matching 46 HIV-positive individuals against 47 uninfected individuals, all of whom were administered three doses of the HPV vaccine. Mascolini at 1. Mascolini concludes that the vaccine "stimulated cell-mediated immunity [meaning T cells]," observing increases in both CD4+ and CD8+ T cells over time (although it did not also observe AA, or any other autoimmune injury, as an adverse event). Mascolini at 2. Dr. MacGinnitie, however, noted that Mascolini did not "address the clinical significance" of these increased T cell levels observed, and also that it provided more information about the helper T cell increases. First MacGinnitie Rep. at 6.

Besides such arguments about the vaccine's capacity to increase pathologic T cells, Dr. MacGinnitie attacked Dr. Gershwin's contentions that molecular mimicry might be the relevant mechanism herein for the proposed vaccine cross-reaction. MacGinnitie Rep. at 9. Amino acid sequence homology was common in nature, he noted, meaning that it could in many cases be shown that a vaccine's antigenic components had similarity to amino acid sequences in hair follicle proteins. Yet if this were a reliable mechanistic explanation for pathology, the incidence of autoimmune injuries like AA should be significantly higher than what was actually observed. *Id.*

Dr. MacGinnitie also looked to Petitioner's specific circumstances to explain why he concluded the HPV vaccine could not have caused his AA. He saw no evidence in the record in the weeks before first onset that Mr. Cordova was experiencing an inflammatory process arguably attributable to vaccination. First MacGinnitie Rep. at 8. Dr. MacGinnitie also noted that none of Petitioner's treaters ever linked the HPV vaccine to his AA. *Id.* at 10. He did, however, concede

that a six-week post-vaccination onset after the first dose was medically acceptable, even though the timeframe was longer than what Dr. Gershwin proposed. *Id*. at 6.

Regarding the second HPV dose, Dr. MacGinnitie contested that the record supported the conclusion that Mr. Cordova's existing AA was worsened. Indeed, he noted that the medical record from the fall of 2016 suggested an arrest in Petitioner's hair loss as a result of treatment. First MacGinnitie Rep. at 6. Evidence of worsening was not present in the record until September 2017—almost a year after the second dose. *Id.* True advancement of the disease process was not evident then until January 2018—a course consistent with AA generally, but not with the second dose as having aggravated the condition. *Id.*

Second MacGinnitie Report

Dr. MacGinnitie prepared a four-page supplemental report in response to both of Dr. Gershwin's additional reports as well as the Yokomine court exhibit. First, he took issue with Dr. Gershwin's assertion (bulwarked by evidentiary references) that Mr. Cordova's AA had worsened after the second vaccine dose, maintaining that the actual record from October and December 2016 through January 2017 included no treater observation of worsening. Second MacGinnitie Rep. at 1-2; Ex. 2 at 10-12. He also reiterated his view (consistent with Dr. Gershwin's opinion) that AA was autoimmune and primarily mediated by CD8+ cytotoxic T cells. Second MacGinnitie Rep. at 2.

Dr. MacGinnitie next provided his reaction to Yokomine. He began by emphasizing that B and T cells did not differentiate into each other, but were the product of "distinct lineages." Second MacGinnitie Rep. at 2. Thus, contentions about the T cell-encouraging impact of the HPV vaccine could *not* be rooted in the determination that their existence flowed directly from the vaccine's intended immunologic effect (to stimulate the production of specific antibodies via B cells). CD4+ and CD8+ T cells are in fact themselves distinct, with different purposes—so evidence that one form of T cell increases post-vaccination does not imply the other also does.

Yokomine, Dr. MacGinnitie acknowledged, did "describe a modest T-cell response to HPV vaccination," but he noted that this response was only observed after stimulation of the T cells with HPV peptides, causing them to secrete a particular cytokine, or messenger cell. This in turn suggested to Dr. MacGinnitie that the observed T cell increases were merely an increase in CD4+ helper cells—not the cytotoxic CD8+ T cells specifically associated with AA hair follicle damage. Second MacGinnitie Rep. at 3. He therefore did not deem Yokomine as reliably supportive of Petitioner's theory.

III. Procedural History

After filing this action in September 2017, Petitioner continued to file relevant medical records, and Dr. Gershwin's first report by March 2018. Respondent then filed his Rule 4(c) Report on July 13, 2018 (ECF No. 31), contesting whether Petitioner had established an evidentiary basis

for entitlement. Over the next few years, the parties continued to litigate this case, responding to the special master's inquiries about causation contentions and filing additional expert reports, items of literature, and other documentary evidence. Some effort was also made toward settlement, beginning in the fall of 2019, but the parties could not agree to terms. The matter was reassigned to me in the summer of 2020, and after holding a status conference in August 2020, I informed the parties of my view that the case could be properly resolved on the record. *See* August 13, 2020 Order (ECF No. 74). Both sides filed their respective briefs, and the case is ripe for resolution.

IV. Applicable Law

A. *Standards for Vaccine Claims*

To receive compensation in the Vaccine Program, a petitioner must prove that: (1) they suffered an injury falling within the Vaccine Injury Table (i.e., a "Table Injury"); or (2) they suffered an injury actually caused by a vaccine (i.e., a "Non-Table Injury.) *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006). In this case, Petitioner does not assert a Table claim.

For both Table and Non–Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (explaining that mere conjecture or speculation is insufficient under a preponderance standard). On one hand, proof of medical certainty is not required. *Bunting v. Sec'y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). But on the other hand, a petitioner must demonstrate 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 & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Human Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non–Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health & Human Servs.,* 418 F.3d 1274 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278. Each *Althen*

prong requires a different showing and is discussed in turn along with the parties' arguments and my findings.

Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549. This standard was recently clarified by the Federal Circuit. *See Boatmon v. Sec'y of Health & Human Servs.*, 941 F.3d 1351, 1359–60 (Fed. Cir. 2019) (stating that the correct standard for *Althen* prong one is "reputable," and "sound and reliable" not a "lower reasonable standard" (internal quotations omitted)).

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. This is consistent with the petitioner's ultimate burden to establish his overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).[7]

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical 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 particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

[7] Although there has been some confusion in the past as to whether the first *Althen* prong is *itself* subject to a preponderant standard, ample controlling authority stands for the more straightforward proposition that the first *Althen* prong is subject to a preponderance standard. *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1350 (Fed. Cir. 2010).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Human Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Human Servs.*, No. 06–522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356–57 (2011), *aff'd without opinion*, 475 F. App'x. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 2013 WL 1896173 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Human Servs.*, No. 11–355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Significant Aggravation*

Besides arguing that the first dose of HPV vaccine caused Petitioner's AA, it is also alleged herein that the second dose aggravated his AA. Where a petitioner so alleges, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *See generally Loving v. Sec'y of Health & Hum. Servs*., 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Services*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether

> the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the Loving test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.,* 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, the Petitioner need not demonstrate an *expected* outcome, but merely that her current-post vaccination condition was worse than pre-vaccination. *Sharpe*, 964 F.3d at 1081. And a claimant may make out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish alternative cause, where a petitioner's showing is enough to make out a prima facie case and thereby shift the burden of proof to Respondent). *Id.* at 1083.

C. *Law Governing Analysis of Fact Evidence*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Human Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his

contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd*, *Rickett v. Sec'y of Health & Human Servs.*, 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras v. Sec'y of Health & Human Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms.").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Human Servs.*, No. 03–1585V, 2005 WL 6117475, at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy v. Sec'y of Health & Human Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, there are situations in which compelling oral 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 & Human Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like 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*, 2005 WL 6117475, at *19 ("[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*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Human Servs.*, No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document

everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Human Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

D. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Human Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

> (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

However, in the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings—e.g., the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of her own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing

Lampe, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 91997)); *see also Isaac v. Sec'y of Health & Human Servs.*, No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Human Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

E. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Human Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Human Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

F. *Consideration of Prior Vaccine Program Decisions*

In reaching a decision in this case, I have taken into account other decisions issued by special masters (including my own) involving similar injuries, vaccines, or circumstances. I also reference some of those cases in this Decision, in an effort to establish common themes, as well as demonstrate how such prior determinations impact my thinking on the present case.

There is no error in doing so. It is certainly correct that prior decisions from different cases do not control the outcome herein. *Boatmon*, 941 F.3d at 1358–59; *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). Thus, the fact that another special master reasonably determined elsewhere, on the basis of facts not in evidence in this case, that preponderant evidence supported the conclusion that vaccine X caused petitioner's injury Y does not compel me to reach the same conclusion in this case. Different actions present different background medical histories, different experts, and different items of medical literature, and therefore can reasonably result in contrary determinations.

However, it is equally the case that special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec'y of Health & Hum. Servs.*, 76 Fed. Cl. 328, 338–39 (2007) ("[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the expertise and experience to know the type of information that is most probative of a claim") (emphasis added). They would therefore be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions. This is especially so given that special masters not only routinely hear from the same experts in comparable cases, but are also repeatedly offered the same items of medical literature regarding certain common causation theories. It defies reason and logic to obligate special masters to "reinvent the wheel," so to speak, in each new case before them, paying no heed at all to how their colleagues past and present have addressed similar causation theories or fact patterns. It is for this reason that prior decisions can have high persuasive value—and why special masters often explain how a new determination relates to such past decisions. Even if the Federal Circuit does not require special masters to distinguish other relevant cases (*Boatmon*, 941 F.3d at 1358), it is still wise to do so.

G. *Standards for Ruling on the Record*

I am resolving Petitioner's claim on the papers rather than via hearing (and the parties have not objected in their filings that I do so). The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where (in the exercise of their discretion) they conclude that doing so will properly and fairly resolve the case. Section 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided case on the papers in lieu of hearing and that decision was upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that special master acted within his discretion in denying evidentiary hearing); *Burns*, 3 F.3d at 417; *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

ANALYSIS

I. Overview of Alopecia Areata and Treatment in Vaccine Program

As both testifying experts agreed, AA is widely understood to be an autoimmune disease characterized by hair loss and an unpredictable course. Rajabi at 1034. AA itself, however, is relatively common, with around two percent of people experiencing it at some point in their lives. First Gershwin Rep. at 1; Rajabi at 1033.

AA occurs when a mononuclear cell inflammatory infiltrate attacks the hair follicle bulb. Rajabi at 1037. The follicle is responsible for producing the hair shaft. Thereafter, T cell cytokines and cytotoxic T cells produce cytotoxic damage. *Id.* This disrupts the normal function of the hair follicle, resulting in thin, fragile hairs that easily detach or break off. *See* A. Tosti, et al., *Alopecia Areata: A Long Term Follow-Up Study of 191 Patients*, 55 J. Am. Acad. Dermatol. 438-441 (2006), filed as Ex. C Tab 8 on Feb. 8, 2019 (ECF No. 44-9). However, because immune damage is localized to the hair bulb, regrowth of the hair can occur after total hair loss, although the process can be slow. *Id.* 439. As Dr. Gershwin noted, AA is driven by a cellular immune response, i.e. T cell attacks, and not by humoral immunity, or antibody responses to specific antigens, whether instigated by the mechanism of molecular mimicry or something else. Gershwin Rep. at 4. AA also unquestionably has a genetic aspect. Rajabi at 1036.

The triggers for AA are not well understood. Rajabi at 1034. Potential triggers include environmental stress, neuropathic or endocrine disorders, infections, and vaccines. *Id.* at 1034, 1036; J. Sundberg, et al., *Recombinant Human Hepatitis B Vaccine Initiating Alopecia Areata: Testing the Hypothesis Using the C3H/HeJ Mouse Model*, 271 J. Intern. Med. 193-203 (2021), filed as Ex. C Tab 10 on Feb. 8, 2019 (ECF No. 44-11). As Dr. Gershwin explained, several potential pathogenic mechanisms by which AA might occur have been proposed, including molecular mimicry,[8] the induction of the cytokine interferon (type 1 IFN), or a "cytokine storm," in which cytokines upregulated after some instigating event greatly increase in number for a period of time, causing harm simply through their proliferation. First Gershwin Rep. at 5. Once AA is triggered, its clinical course is variable and not monophasic in progression. Some patients experience recurring loss and regrowth, other patients experience one episode, and some will experience everything in between.

AA has been an alleged vaccine injury in several prior cases, and petitioners have successfully established that a variety of vaccines could cause/trigger it. *See, e.g.*, *DeLozier v. Sec'y of Health & Hum. Servs.*, No. 15-124V, 2019 WL 7556051 (Fed. Cl. Spec. Mstr. Dec. 10, 2019) (Petitioner preponderantly established that the hepatitis B vaccine could trigger an autoimmune response resulting in a single AA occurrence), *mot. for rev. granted*, 152 Fed. Cl. 558 (Fed. Cl. 2020) (vaccine responsible for entirety of injured party's illness and not simply first instance). However, I have identified no reasoned decisions in which the HPV vaccine in any formulation was found to be causal of AA.

[8] Molecular mimicry is of course a commonly-invoked mechanism in the Vaccine Program for explaining how the immune system might aberrantly cause disease, and proposes that foreign antigens presenting to immune system cells might be confused with self-structures, causing the immune system to mistakenly attack *both* the antigens and self-structures (with the latter constituting the damaging autoimmune response). *See Garner v. Sec'y of Health & Hum. Servs.*, No. 15-063V, 2017 WL 1713184 at *5 (Fed. Cl. Spec. Mstr. Mar. 24, 2017).

II. Petitioner Has Not Carried Her *Althen* Burden

Although the record suggests that Petitioner's AA diagnosis at times competed with a fungal infection explanation for his hair loss, it does not appear Respondent ultimately questions AA as the relevant diagnostic classification for Petitioner's injury, and I find the evidence preponderantly supports it as well. This still leaves, however, determining whether the HPV vaccine could cause AA, or did so to Petitioner.[9]

A. *Althen* Prong One

Petitioner's theory proposes that the HPV vaccine could cause an upregulation of the kind of T cells that would be directly responsible for breaking a hair follicle's immune privilege, the primary driver of AA. While I find it undisputed that AA is autoimmune in character, and that its pathogenesis is likely driven by cytotoxic T cells, the causation theory offered in this case is otherwise not supported by sufficient reliable evidence to make a finding that the HPV vaccine "more likely than not" could instigate/trigger AA.

Generally, Petitioner lacks evidence[10] suggesting an association between the HPV vaccine and AA, whether in the form of literature proof of studies or testimony from Dr. Gershwin derived from his own research or treatment experience. At most, he can cite to a potential association with *other* vaccines, like the hepatitis B vaccine, or to case reports where AA was observed to follow vaccination. But neither kind of evidence is especially reliable. *See Al-Uffi vs. Sec'y of Health & Hum. Servs.*, No. 13-956V, 2017 WL 1713113 at *16 (Fed. Cl. Spec. Mstr. Feb. 22, 2017) ("Individual case studies are not themselves particularly probative in the context of establishing the first *Althen* prong (especially where they involve a totally different vaccine)").

There are also ample reasons to doubt that the HPV vaccine itself generally could cause AA, let alone any autoimmune injury. As Dr. MacGinnitie observed, the wild virus itself is not associated with AA, diminishing the likelihood the vaccine would also be. And Respondent has noted that some large-scale epidemiologic studies, like Chao,[11] do not support the conclusion that

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[9] Because I have found that Petitioner did not preponderantly establish the first two *Althen* prongs, I include no analysis of the third prong. Had I done so, however, I would have determined that (based on Dr. MacGinnitie's concession) the six-week post-vaccination onset of Petitioner's first manifestation of AA (in September 2016) was medically acceptable—and also that the claim the second dose could have initiated worsening in a two-week post-vaccination timeframe was preponderantly demonstrated.

[10] I fully recognize claimants in Program cases are not *required* to offer direct proof of causation—any more than they are required to offer *any* particular kind of evidence. But I raise the fact that this kind of proof is missing herein mainly to emphasize that the *secondary or circumstantial* proof offered by Petitioner in this case is itself either distinguishable or otherwise lacking in reliability or probative value. That is not always the case—petitioners can sometimes win with reliable and persuasive circumstantial evidence.

[11] I have in other cases noted Chao's reliability. *See, e.g.*, *Maciel v. Sec'y of Health & Human Servs.*, No. 15-362V, 2018 WL 6259230, at *27 (Fed. Cl. Spec. Mstr. Oct. 12, 2018), *mot. for review den'd*, slip op. No. 15-362V (Fed. Cl. Apr. 1, 2019); *Johnson v. Sec'y of Health & Hum. Servs.*, No. 14-254V, 2018 WL 2051760, at *25 (Fed. Cl. Spec.

the HPV vaccine is likely to produce autoimmune injury, by comparing the background incidence rate for a large number of autoimmune illnesses against what those who received the vaccine actually experienced. Admittedly, Chao should be given comparatively less weight here, since it did not explicitly include AA among the autoimmune conditions considered—but Petitioner has offered no counter-evidence to suggest that its findings are unreliable with respect to the HPV vaccine more broadly.

To get around such limitations, Petitioner has attempted to show how the HPV vaccine might play a role in the T cell attack specific to AA. He thus cites a few small-scale studies aimed at evaluating the immunogenicity of the HPV vaccine, noting that they suggest some increased T cell upregulation after receipt of the vaccine. It is definitely undisputed in this case that AA is likely mediated by a specific class of cytotoxic T cell – CD8+ cells. Accordingly, even though it also has been established that the HPV vaccine is designed principally to elicit a *B cell* antibody-producing response, Petitioner maintains that the evidence of a concurrent T cell increase allows for the conclusion that the vaccine could trigger AA's pathogenesis.

The articles offered to support this contention, like Yokomine or Herrin, are not fully persuasive on this point. Yokomine,[12] for example, does not cleanly stand for the proposition asserted, since (as Dr. MacGinnitie points out—and Dr. Gershwin seemed to allow, if eliptically) it is not fully evident its authors actually measured increased CD8+ T cells, as opposed to an increase in helper T cells. Herrin for its part did not ultimately attempt to measure the relevant T cells. And both articles involve fairly small sample sizes as well, diminishing the probative weight to be given to their conclusions.

But even if I give this evidence some probative weight, does it support the conclusion that the T cell increases post-vaccination are enough to be pathologic? Or do these articles simply measure the general *immunogenicity* of the vaccine—as their authors admit was their primary intent? *See, e.g.*, Yokomine at 1503; Herrin at 3446. I find the latter is more consistent with the actual content of both articles. At best, evidence of some T cell increase after receipt of the HPV vaccine has in this case been established to *plausibly* occur—*not* that this increase (a) is likely

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Mstr. Mar. 23, 2018); *Sullivan v. Sec'y of Health & Human Servs.*, No. 10-398V, 2015 WL 1404957, at *11-12 (Fed. Cl. Spec. Mstr. Feb. 13, 2015).

[12] I note also that Yokomine was filed not by Petitioner but as a court exhibit. Although it is now in the record, and I have considered it, the fact that it was deemed significant by the prior special master in this case (perhaps in the hope of encouraging settlement, by pointing out to Respondent potential strengths in Petitioner's claim) does not obligate me to give it the same weight, especially since no entitlement determination was made in this matter before the claim's transfer. Indeed—authority supports the conclusion that I could grant relief denied by a prior transferring special master, assuming the record supported my subsequent determination. *See McGowan v. Sec'y of Health & Hum. Servs.,* 31 Fed. Cl. 734, 737-38 (1994) (transferee special master not restricted by "law of the case" from granting renewed motion to dismiss that transferor special master had previously denied). Having reviewed Yokomine carefully, and against the backdrop of both experts' written reports, I do not find that it appreciably assists Petitioner, as discussed above. And relying on studies measuring the after-effects of vaccines on immune cell production, as Yokomine does, is only one step toward showing the HPV vaccine is "more likely than not" causal of injury.

causal of AA, or (b) even involves the specific kind of cytotoxic T cells that drive AA. *See Canuto v. Sec'y of Health & Human Servs.*, 660 Fed. Appx. 955, 957 (Fed. Cir. 2016) (citing *W.C. v. Sec'y of Health & Human Servs.*, 704 F.3d at 1356 (Fed. Cir. 2013) ("the petitioner must do more than demonstrate a 'plausible' or 'possible' causal link between the vaccination and the injury; he must prove his case by a preponderance of the evidence.")). Evidence that a vaccine causes an immune response—the intended function of any vaccine—does not amount to a showing that this response is, or can become, pathologic, absent *additional* proof linking to evidence of the vaccine's otherwise-intended response.

Thus, there is an overarching deficiency in Dr. Gershwin's contentions about the capacity of the HPV vaccine to cause an upregulation of the cytotoxic T cells central to hair follicle destruction. Although it is not disputed that CD8+ T cells are central to AA's progression, few reliable articles stand for the proposition that the HPV vaccine causes the upregulation of this T cell in sufficient amounts to be pathologic. Indeed, the articles that do evaluate this possibility, like Herrin, approach the issue not from the standpoint of AA, but rather considering the immunogenic efficacy of the vaccine. They also in some instances (Yokomine in particular) cannot meaningfully separate the extent to which they reveal upregulation of T helper cells versus the cytotoxic T cells that primarily lead to the follicle destruction characteristic of AA. And these articles are consistently based on very small samples as well, further limiting the weight they should receive (independent from the inconclusive nature of their findings). *See, e.g.*, Yokomine (10 subjects); Herrin (27 subjects).

Ultimately, it bears repeating: these articles are only observing relative increases in T cell counts post-vaccination—*not that these amounts are pathogenic*. Dr. Gershwin thus makes the same error other experts have, in conflating the expected and intended result of a vaccine (to stimulate a healthy immune response) with the argument that this increase is evidence of the same vaccine's potentially-harmful impact. That argument is only *plausible* support, of a particularly weak kind, for causation.

One consistent defense offered up by Dr. Gershwin to excuse this inability to offer more robust evidence warrants comment. Throughout his reports, Dr. Gershwin (perhaps anticipating arguments Respondent might make) takes pains to note the rarity of the alleged vaccine-caused injury. *See, e.g.*, First Gershwin Rep. at 6. In his apparent view, this rarity means not only that few individuals will experience the asserted injury, but also that epidemiologic studies (which might otherwise suggest the vaccine is highly unlikely to cause an injury) are useless since they cannot be gauged with enough sensitivity to draw conclusions about the possibility. *Id.* In addition, research regarding T cell homology to self-structures (here, the hair follicles) is extremely limited or difficult to perform, further explaining why more reliable evidence discussing the propensity of *any* vaccines to trigger upregulation of pathologic T cells specific to the follicle is absent.

Dr. Gershwin thus invokes rarity of injury and obscurity of mechanism to defend the evidentiary insufficiency of his opinion. *But this is not how the preponderant standard functions.* While preponderance does not require medical certainty (and indeed leads ample room for doubt even when petitioners *prevail*), it *does* require claimants to offer some items of reliable scientific or medical proof (e.g. expert opinions, research, studies, etc.) that, taken together, establish causation is "more likely than not." *I.J. v. Sec'y of Health & Hum. Servs.*, No. 16-864V, 2021 WL 1232733 at *30 (Fed. Cl. Spec. Mstr. Jan. 4, 2021). A claimant unable to do so cannot turn around and invoke rarity of injury to excuse the absence of sufficient proof. Were it otherwise, no non-Table vaccine claim would ever be denied, so long as the petitioner proved injury and a covered vaccine. This argument thus seeks to evade the "heavy lift" petitioners asserting causation-in-fact claims must perform. *See Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357 at 1360 (Fed. Cir. 2000).

At bottom, Petitioner has not offered sufficient reliable evidence suggesting that the HPV vaccine could likely initiate an autoimmune cross-reaction, driven by cytotoxic T cells and leading to the follicle destruction associated with the HPV vaccine.

B. *Althen* Prong Two

Petitioner's claim also founders on the second, "did cause" prong. Here, only a temporal association links his AA onset to the first HPV dose six weeks before. There is no evidence of any immediate reaction or measured autoimmune/inflammatory process that preceded onset. None of the articles filed in this case shed light on what a person might experience pre-onset of AA that can be compared to what happened in this case. And no treaters appear to have concluded that the HPV vaccine likely caused Petitioner's AA. The fact that his AA began post-vaccination is not itself enough to base a finding that Petitioner has met the second *Althen* prong. *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 2010) ("a proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury").

II. Petitioner's Significant Aggravation Claim is not Well-Founded

Since the Federal Circuit's liberalizing of the *Loving* standard in *Sharpe,* it has become far easier to meet many of the prongs for a significant aggravation claim. And indeed, that is true here. Petitioner has demonstrated that he had AA prior to the second dose of HPV vaccine (given to him at the first medical visit to treat the AA), and that its progression worsened after that date. The record, coupled with the witness statements filed herein, more preponderantly support that conclusion than Dr. MacGinnitie's counter-argument, derived from his review of some records) that Petitioner's hair loss was inconsistent or even temporarily reversed. Thus, I readily can find that *Loving*'s first three prongs have been satisfied.

Petitioner has not, however, satisfied the critical fourth prong, which parallels *Althen*'s first, that the HPV vaccine can cause AA to worsen. This is because Dr. Gershwin did not demonstrate that a second dose of the HPV vaccine could have this impact. As discussed above, it was not preponderantly established that the HPV vaccine (designed primarily to elicit a B cell response) could also cause the production of sufficient cytotoxic T cells to initiate AA—and if so it is difficult to conceive of how the same vaccine could worsen AA. Indeed, no showing was made at all that the vaccination would impact AA in any regard; even the articles that discuss the connection *other* vaccines might have with AA do not address the risk of vaccination once the autoimmune condition is underway.

At most, Petitioner can point to the fact that his AA progressed in a somewhat faster tempo post-second dose (although, since he sought treatment for the first time when he received the second dose, the record is equally consistent with the conclusion that his AA's progression was already in motion). But limited evidence here of challenge-rechallenge[13] flies in the face of my existing finding that the first dose had nothing to do with vaccination—for this record does not permit the conclusion that dose number one prompted any response at all.

CONCLUSION

Other vaccines have preponderantly been demonstrated in the Vaccine Program to be associated with the capacity to trigger a single AA occurrence, or even the entirety of the condition, and thus I do not deem this claim to have been unreasonably advanced—especially given the obvious general temporal relationship between vaccination and onset. But not nearly enough was demonstrated about the HPV vaccine's impact on the immune system to conclude it likely could initiate a T cell-driven autoimmune attack that mediates AA—or worsen an existing case. Accordingly, Petitioner cannot meet his burden of proof, and I am compelled to dismiss this claim.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the clerk of the court **SHALL ENTER JUDGMENT** in accordance with the terms of this decision.[14]

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[13] "Challenge-rechallenge happens when a person (1) is exposed to one antigen, (2) reacts to that antigen in a particular way, (3) is given that same antigen again, and (4) reacts to that antigen similarly. Typically the second reaction is faster and more severe." *Nussman v. Sec'y of Health & Hum. Servs.*, 83 Fed. Cl. 111, 119 (Fed. Cl. 2008) (internal citations omitted) (quoting *Nussman v. Sec'y of Health & Hum. Servs.*, No. 99-500V, 2008 WL 449656, at *9 (Fed. Cl. Spec. Mstr. Jan. 31, 2008).

[14] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.

IT IS SO ORDERED.

/s/ Brian H. Corcoran
Brian H. Corcoran
Chief Special Master