CORRECTED

In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 15-124V

(to be published)

* *	Chief Special Master Corcoran
CHRISTINE DELOZIER, * *parent and next friend of L.T., a minor*, * * Petitioner, * * v. * * SECRETARY OF HEALTH AND HUMAN SERVICES, * * Respondent. *	Filed: December 10, 2019 Alopecia Areata; Autoimmune Condition; Vaccine as Trigger; Chronic Condition; Althen Prong One; Genetic Basis for Condition

Richard Gage, Richard Gage, P.C., Cheyenne, WY, for Petitioner.

Jennifer L. Reynaud, U.S. Dep't of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

On February 9, 2015, Christine DeLozier,[2] as parent and next of friend of L.T., a minor, filed a petition seeking compensation under the National Vaccine and Injury Compensation Program (the "Vaccine Program").[3] (ECF No. 1) ("Petition"). Petitioner alleged that L.T. suffered

[1] This Ruling 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 Ruling 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 Ruling'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 Ruling will be available to the public. *Id*.

[2] Although the Petition was originally filed under the name "Christine Torres," Petitioner has since changed her last name to DeLozier, and therefore the case caption has been amended. (ECF No. 58).

[3] 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).

from alopecia areata ("AA") attributable to a hepatitis B vaccine ("HBV vaccine") L.T. received on November 6, 2012.

An entitlement hearing was held in this matter on February 4, 2019, and with post-trial briefing concluded in June the case is now ripe for resolution. Based on the evidence submitted, I find that Petitioner has preponderantly established that the HBV vaccine could trigger an autoimmune response resulting in a *single* AA occurrence—and therefore since the medical record supports the contention that this occurred in L.T.'s case, Petitioner is entitled to an award of damages associated with that first occurrence. I do *not* find, however, that preponderant evidence supports Petitioner's broader contention that all *subsequent* outbreaks of AA Petitioner has experienced (or may in the future) are also attributable to her 2012 receipt of the HBV vaccination—and therefore she is not entitled to damages associated with any such subsequent, unrelated AA occurrences.

I. Factual Background

L.T.'s family health history and pre-vaccination health

L.T. was born on June 29, 2009, largely healthy after a 32-week pregnancy. Ex. 1 at 3; *see also* Ex. 4 at 11, 84, 89–90. Significantly for this case, L.T. appears to have a family history of autoimmune diseases. Ms. DeLozier in particular (who testified at trial) has previously experienced an unspecified autoimmune connective disorder. *See* Ex. 1 at 4; Tr. at 26–28. This condition carried symptoms of diffuse hair loss, a positive ANA,[4] and occurred about seven to ten years before L.T.'s birth. Ex. 1 at 3; *see also* Ex. 4 at 11. Petitioner specifically reported that she developed this disorder after being out of the country—and recalled having received the HBV vaccine prior to travelling. Ex. 4 at 11.

L.T. was growing and developing normally before her receipt of a third HBV dose, although she did suffer from asthma and eczema. Tr. 19–20 (stating that L.T. had eczema before her third HBV vaccine); Ex. 3 at 314 (noting diagnosis of asthma in November 2011). She had received her first and second HBV vaccine doses at two and four months old, respectively, with no reported reaction. Ex. 4 at 36–37; Tr. at 31; *see generally* Ex. 5 (vaccination record). Thereafter there is a gap in medical records—and a deviation from the normal HBV vaccination schedule—because of a lapse in insurance coverage. Tr. at 30–31.

[4] An antinuclear antibody ("ANA") test is typically used to assess the presence of systemic lupus erythematosus ("SLE"), as well as other autoimmune diseases (e.g., mixed connective tissue disease, scleroderma, rheumatoid arthritis, Sjögren's syndrome, and polymyositis). However, because otherwise-healthy individuals often test positive for ANA, follow-up testing is necessary to corroborate the diagnosis, although a negative result generally excludes the diagnosis of some autoimmune diseases. *See* K. Pagana, et al., *Mosby's: Manual of Diagnostic and Laboratory Tests* 80, 82 (6th ed. 2018).

L.T.'s vaccination and health history to the end of 2013

L.T. was three years old when she received her third dose of the HBV vaccine on November 6, 2012. Not long after, Ms. DeLozier noticed problems with L.T.'s health. Specifically, she observed that "a couple days after vaccine there was increased hair coming out [of L.T.'s head]." Tr. at 11. Then, four days after vaccination, she noticed "two prominent bald spots"—about the size of a quarter—on L.T.'s head. *Id.* at 11. At the same time, L.T. complained of joint pain in her hip and wrists, had developed a rash, and appeared to be walking with a limp. *See id.* at 11–12.

Later that month, Ms. DeLozier took L.T. to a dermatologist, Dr. Elaine Gilmore.[5] Ex. 4 at 8–10; *see generally* Ex. 7 (Records from Dr. Gilmore's office). At that visit, Dr. Gilmore observed "widespread . . . alopecic patches on the scalp." Ex. 4 at 9. In her assessment, Dr. Gilmore noted that L.T.'s recent hair loss was "possibly stimulated by [her] recent [HBV] vaccine," although she expressed the need for further research to evaluate whether such a relationship had scientific support. *Id*. Dr. Gilmore also noted that L.T. had symptoms of eczema, and discussed the possible diagnosis of atopic dermatitis versus irritant contact dermatitis with Petitioner. *Id*.

After examination, Dr. Gilmore prescribed L.T. a topical steroid to treat her alopecia and an ointment to treat her eczema. Ex. 4 at 9. Dr. Gilmore recorded L.T.'s family history of eczema, but noted (erroneously) that L.T. appeared to lack a family history of alopecia or similar conditions. Ex. 4 at 8, 11 (observing a family history of connective tissue disease in mother characterized by hair loss during doctor's appointment a few months later); Tr. at 8–9 (describing family history in mother of autoimmune connective tissue disorder characterized by hair loss). In December 2012, L.T. had a series of lab tests done that revealed she had a positive ANA screen. Ex. 4 at 6.

On January 7, 2013, L.T. visited Dr. Gilmore's practice again and this time was treated by Kristen Ahern, M.D., a resident/fellow. Ex. 7 at 1. Petitioner reported that L.T.'s dermatitis was improving, although her bald spots were worsening. *Id.* Dr. Ahern (consistent with Dr. Gilmore) also noted that L.T.'s alopecia was "possibly stimulated by recent [HBV] vaccine," and instructed Petitioner to continue on the treatment plan previously discussed. *Id.* at 2. A physical exam showed a negative hair pull test and small black dots in some of the bald spots indicating hairs that were present (and thus potentially returning) but had "not yet erupted." *Id.* at 1.

On January 22, 2013, L.T. visited Bethany Marston, M.D., a pediatric rheumatologist. *See* Ex. 4 at 11. Dr. Marston noted that L.T. was suffering from AA and transient joint pain that had developed about four days after her third HBV vaccination. *Id.* Dr. Marston recorded that L.T.'s

[5] There is some discrepancy between when Petitioner alleges this visit occurred and what the records establish. Petitioner asserts that L.T. saw Dr. Gilmore on November 12, 2012. Pet'r's Post-Hearing Brief at 2 (citing Ex. 4, at 1). But Exhibit 4 at 1 is actually a note from a subsequent March 2013 doctor's visit at Sunrise Pediatrics, and thus references the November visit as part of L.T.'s medical history. *See* Ex. 4 at 1 ("date form completed 3/27/13"). The date "11/12" (on Ex. 4 at 1) seems to be a generic reference to November 2012. In any event, the evidence does establish that Petitioner sought *some* treatment for L.T. in November 2012, and the records reflect her contemporaneous assertion that L.T. experienced hair loss within a few days of vaccination.

joint pain was not associated with swelling, erythema, or functional disability and that L.T.'s symptoms were responding to the ointments and steroids she was earlier prescribed. *See id.* Other testing revealed negative inflammatory markers and rheumatoid factor, and, "despite a questionable family [history] of unspecified [mixed connective disease] in her mom and some other autoimmune-induced syndromes in her extended family," L.T.'s positive ANA was deemed by Dr. Marston most likely the result of her then-resolving AA. *Id.*

L.T. returned to Dr. Gilmore's practice again in March 2013. Ex. 7 at 4. L.T. was seen at this time by Kimberly Brady, M.D., a resident. Ex. 7 at 4. Dr. Brady recorded that L.T.'s parents had not noticed any improvement in her condition since their last visit. *Id.* She also noted that L.T.'s HBV vaccine appeared temporally related to her AA, but "because this is not a widely seen association, and [AA] is a relatively common disorder, it is difficult to say that the hepatitis vaccine was responsible for the onset of [AA]." *Id.* at 4–5. Dr. Brady's physical exam also revealed a negative hair pull test and small black dots in some bald areas. *Id.* at 4–5. To address the lack of improvement in L.T.'s AA symptoms, Dr. Brady changed L.T.'s steroid cream and ointment prescription. *Id.* Other than her AA, L.T. had a normal physical exam. *Id.*

L.T. visited Dr. Gilmore's practice three months later in June 2013 (now more than six months since the onset of her AA symptoms) and was seen by Rachel Garner, M.D., a resident. Ex. 7 at 7. Dr. Garner noted that L.T. still had some lingering AA symptoms, but there was evidence of recovery as well—some short hairs in the bald areas that appeared to be new. *Id.* at 7–8. L.T.'s rash had also returned, but it was found to be negative for fungus, leading Dr. Garner to characterize it as atopic dermatitis (eczema), treatable with ointments and creams. *Id.* at 8. Dr. Garner also performed a hair pull test which was negative and observed short hairs that appeared new. *Id.* at 8.

Later, in August 2013, Petitioner took L.T. to Rochester General Hospital. Ex. 11 at 1. There, L.T. was seen by Tracy Henderson, M.D. *Id.* at 4. Dr. Henderson observed that L.T. had patchy AA and had been diagnosed with AA in November 2012, four days after her third HBV vaccine. *Id.*at 4–5. Dr. Henderson also recorded a family history of an autoimmune disease in L.T.'s mother. *Id.* at 3. Notes from the visit also show that Petitioner brought several articles into the visit that mentioned an association between AA and the HBV vaccine. *Id.* at 4.

L.T.'s health history from 2014 to 2016

There are several subsequent gaps in the medical record. L.T. appears to have had her next doctor's visit in April 2014 for behavioral concerns at Rochester General Hospital. Ex. 11 at 12–17. During this visit Dr. Henderson did not note any AA symptoms in the physical exam section of the visit records. *Id*. at 16 ("Well-appearing, alert, very active."). No mention was made at this visit of L.T.'s AA, although it also appears the visit (besides behavioral concerns) was occasioned by Ms. DeLozier's wish to inquire about L.T.'s dietary issues.

In August 2014, L.T. had a five-year well-child visit at Rochester General Hospital. Ex. 10. at 1-10. There, Andrew Sherman, M.D., observed that L.T. suffered from chronic eczema. *Id.* at 4. Dr. Sherman also logged that L.T. had previously experienced AA because of an adverse reaction to the HBV vaccine in November 2012. *Id.* Regarding L.T.'s initial hair loss, Dr. Sherman recorded that L.T. lost her hair for "about a year," and also suffered from worsening eczema around the same time. *Id.* Dr. Sherman's objective physical examination did not note any AA symptoms, but did note some eczema behind L.T.'s knees. *See id.* at 5–6. Only eczema was listed in the current concerns section. *Id.* at 6. Accordingly, this record does not suggest that L.T. was at this point still experiencing sequelae from her November 2012 onset of AA.

L.T. returned to Rochester General Hospital nine months later, on May 22, 2015, because she developed chicken pox and a sore throat. Ex. 10 at 11–19. Dr. Sherman noted that she had run a fever for four days after likely exposure to the varicella virus from her brother and father. *Id.* at 17. At this visit, AA was recorded in the current problem list, but is not mentioned in her in her physical exam, assessment, and diagnoses (thus strongly suggesting she was not experiencing any active symptoms from it). *Id.* 11, 17–18. L.T. was treated for her sore throat and chicken pox and discharged. *Id.* at 18. A few months later, on July 1, 2015, L.T. had another visit at Rochester General Hospital because of possible insect bites and a swollen eye. *Id.* at 20. AA is mentioned nowhere but her medical history. *See id.*

The next filed record is from August 2015, when L.T. returned to Rochester General Hospital for a sore throat. Ex. 11 at 18–24 (visit on August 10, 2015). L.T. was tested for a sore throat, treated for her sore throat, and discharged. *Id.* She was seen by Julia Stein, M.D., who noted a medical history of AA but did not record any other information about AA at that time. *See generally id.* Later that same month, L.T. visited Rochester General Hospital for a routine child exam. Ex. 10 at 28 (visit on August 19, 2015). L.T.'s weight was noted as being low and weight management was discussed. *Id.* at 35. Dr. Sherman did not list any current concerns, active issues, or chronic issues. *Id.* No notes of AA were made except in the health history section. *See id.* (noting that L.T. was visiting the dermatologist later that day).

The same day, August 19, 2015, L.T. visited Universal Dermatology PLLC—Dr. Gilmore's practice. Ex. 9 at 1. Petitioner's reason for bringing L.T. in was for "hair loss that is multifocal and mild in severity," that had manifested two weeks prior. *Id.* A physical exam revealed some hair loss patches on L.T.'s central forehead and right inferior occipital scalp, along with thin eyebrows and "coin-like eczematous patches" on her arms and left thigh. *Id.* Dr. Gilmore recommended that her AA could be treated using topical steroids, and that Petitioner should contact his office if symptoms plateaued or worsened despite treatment. *Id.*

L.T. had a follow-up visit with Dr. Gilmore on September 25, 2015. Ex. 9 at 5. There, Dr. Gilmore noted that L.T.'s AA was improving with treatment and improved overall. *Id.* Dr. Gilmore observed "minimal alopecic patches on the anterior scalp and thin eyebrows throughout." *Id.* Dr. Gilmore advised Petitioner to reduce the amount of steroid being taken and that L.T. could apply

ointment to her face to stimulate eyebrow growth. *Id.* L.T. was scheduled to follow-up in one-month for a "focused visit." *Id.*

L.T.'s health history from 2016 to present

L.T. next returned to Dr. Gilmore more than six months later, in April 2016. Ex. 28 at 1. At this time, Dr. Gilmore observed discrete non-scarring patches of hair loss on the "right superior temple and left occipital scalp." *Id.* Dr. Gilmore discussed with Petitioner the natural history of AA, which can be associated with flares and remissions. *Id.* Dr. Gilmore also noted that L.T.'s hair loss, at that time, was not severe enough to warrant systemic therapy. *Id.*

In November 2016, L.T. presented to Dr. Gilmore again for further evaluation. Ex. 28 at 2. During the visit Dr. Gilmore observed AA on L.T.'s "right superior central forehead, left superior central forehead, and left lateral eyebrow." *Id.* This distribution of AA was notably different than what had been reported in August 2015. *Compare* Ex. 9 at 3 (showing AA patches in August 2015 behind the right ear and on the left superior forehead), *with* Ex. 28 at 3 (showing AA patches in November 2016 on the left eyebrow and on two locations on the forehead). Dr. Gilmore discussed new possible therapies for L.T.'s condition, but explained that it would be difficult to predict her future hair growth. *Id.* The next month, L.T. went into Rochester Regional Health for a well-child visit. Ex. 29 at 6. Dr. Sherman observed that L.T. had AA symptoms, and was also complaining of knee, wrist, and elbow pain at times, consistent with complaints from the previous month. *Id.*

L.T. went to the doctor again in December 16, 2016, for a well-child visit at Rochester Regional Health. Ex. 29 at 1. There, in the subjective notes section, Dr. Sherman recorded that L.T. "[s]till has [a] patch of alopecia behind right ear and near midline of mid occipital area." *Id.* at 6. The AA patch behind her right ear was also noted in the objective section of the visit record. *Id.* at 6–7. L.T. was scheduled for another well-child visit next year and informed Dr. Sherman that she would follow-up on her AA with a dermatologist and rheumatologist. *Id.* at 6–8.

L.T. presented to Rochester Regional again in late June 2017 for a cough and sore throat. Ex. 29 at 17. No notes on AA or eczema were made other than in the health history section. *See id.* Then, L.T. visited Rochester Regional again in August 2017 for her eight-year well-child visit. Ex. 29 at 27. In the subjective notes section of the visit Dr. Sherman recorded that L.T. had no current concerns and was "doing well." *Id.* at 32. Dr. Sherman also did not mention any AA in his objective examination of L.T. *Id.* at 33. L.T. declined immunizations citing a prior medical history of AA after vaccination. *Id.*

L.T. returned to Rochester Regional in October 2018 for her nine-year well-child visit. Ex. 29 at 42. In the subjective notes section Dr. Sherman recorded that there were no current concerns, L.T. was doing well, and there were "no current hair changes." *Id.* at 47–48 (assessing no active or chronic issues and stating that L.T. was doing well). In the objective physical assessment Dr. Sherman did not note any AA. *Id.* at 48. No records were filed for the period after the end of 2018 setting forth any concerns about new AA flares.

II. Witness Testimony

A. *Petitioner's expert: Dr. David Norris*

Dr. Norris filed a single report and testified at hearing. *See* Ex. 16, filed June 7, 2017 (ECF No. 40-1) ("Norris Report"). He opined largely that L.T.'s third HBV vaccine "was the trigger that caused [her] to develop [AA]." Norris Report at 2.

Dr. Norris obtained a B.A. from Johns Hopkins University in 1969. Ex. 17, filed on June 7, 2017 (ECF No. 40-2) ("Norris CV"). He earned his M.D. from Duke University Medical School in 1972. *Id.* at 1. After completing his formal education, Dr. Norris held a sub-fellowship in Hematology and Oncology during 1973 at Duke University; an internship in Internal Medicine during 1973–1974 at Ohio State University; and a Dermatology Residency during 1974–1977 at the University of Colorado School of Medicine. *Id.* Additionally, Dr. Norris is board certified in Dermatology, Dermatologic Immunology and Diagnostic and Laboratory Immunology. *Id.*

Dr. Norris is presently the chairman of the Department of Dermatology at the University of Colorado School of Medicine, where he has also been a professor. Norris CV at 1. Dr. Norris has received research funding for several topics relating to immunodermatology and the "Alopecia Areata Registry." *Id.* at 3. Dr. Norris is also published and has conducted research related to AA, dermatology, and immunodermatology. *See generally id.*

Dr. Norris provided an overall description of AA. Norris Report at 1. AA is a clinical hair loss disease that is "at its root an immunologic disease controlled by genes." Tr. 41–42. In particular, AA is a polygenic autoimmune disease, with "other autoimmune components that go along with it." *Id.* at 42. The most common of such comorbid conditions is hypothyroidism, "[b]ut another important immunologic disease that goes along with [AA] is atopy." Tr. at 42–43 (noting that L.T. has atopic dermatitis). He also described the waxing and waning nature of the disease, and how a patient's long-term prognosis is ultimately affected by her genetic profile. *Id.* at 51–52 ("the current thinking is that the patients that have the worst genetic profile will get the worst disease and early on and the . . . baldness on the scalp will remain").

From this, Dr. Norris proposed a theory of how vaccines generally (and more specifically, the HBV vaccine) could cause AA. Norris Report at 2. Dr. Norris submitted that because it is an autoimmune disease, AA's onset may follow events such as "infections, periods of stress, and immunostimulation, including vaccination." *Id.* (citing Y. Zafrir, et al., *Vaccines, Infections, and Alopecia Areata, in Vaccines & Autoimmunity* (Yehuda Shoenfeld et al. eds., 2015), filed as Ex. 27 on Jan. 30, 2019 (ECF No. 55-2) ("*Zafrir*")); Tr. at 43–44. Dr. Norris, referring to *Zafrir*, explained that that these events can cause a change in the "immune privilege" of hair follicles (meaning the capacity to tolerate antigen presentation without an inflammatory, immune-driven response), which in turn causes the hair loss associated with AA. Tr. at 44–45. Dr. Norris added that in his understanding, the dermatologic medical community accepts the existence of triggers as significant in causing AA, with 25 percent of AA patients able to identify a likely trigger after

symptoms occur. *Id.* at 48. However, as Respondent has correctly observed, *Zafrir* considered mouse studies that "suggest[ed] that there was *no association between vaccination and AA* development." *Zafrir* at 3 (emphasis added).[6]

Dr. Norris offered some other specific items of medical literature that he maintained supported the contention that the HBV vaccine could trigger AA. Tr. at 57; Norris Report at 2 (citing R. Wise, et al., *Hair Loss After Routine Immunizations*, 278 JAMA 1176 (1997), filed as Ex. 26 on Jan. 30, 2019 (ECF No. 55-1) ("*Wise*") (concluding that "we believe that immunizations warrant consideration among potential causes of hair loss"); Tr. at 49–51. *Wise* analyzed reports from the Vaccine Adverse Event Reporting System ("VAERS"), plus reports made to the FDA that predated VAERS, of instances in which AA following vaccination was alleged. *See Wise* at 1. The study yielded 60 evaluable reports of hair loss following immunization since 1984 and coded for alopecia, with 46 of the instant cases involving the HBV vaccine. *Id.* In addition, 16 of the 60 cases involved a positive rechallenge[7]—meaning the individual had previously been vaccinated, but saw new symptoms in a shorter timeframe after receipt of a follow-up dose. *Id.* Although only four of the sixteen were confirmed as definite rechallenges, three of the four rechallenges involved the administration of the HBV vaccine. *Id.* at 1–2.

Dr. Norris deemed such evidence to strongly support causation. Tr. at 51. But he did acknowledge some limitations with *Wise.* He conceded that *Wise* was published more than 20 years ago (and hence had not been followed up with any corroborative studies), and also that the study's authors had only discussed hair loss in general terms. *Id.* at 50. However, he maintained his opinion that the cases discussed in *Wise* mostly involved AA. *Id.* Overall, Dr. Norris deemed *Wise* his best evidence for the proposition that the HBV vaccine could cause AA. *See* Tr. at 89.

Petitioner also relied on a more recent article, although it was filed shortly after the hearing (and not otherwise mentioned by Dr. Norris in his testimony). *See* C. Richardson, et al., *Evaluation of the Relationship Between Alopecia Areata and Viral Antigen Exposure*, 9 Am. J. Clinical

[6] *Zafrir* has other issues that limit the weight it should receive. It was co-authored by Dr. Yehuda Shoenfeld, a frequently-seen medical expert in Vaccine Program cases who takes an expansive (but frequently unpersuasive and/or unreliable) view of the capacity of vaccines generally to cause autoimmunity. *See, e.g., Yalacki v. Sec'y of Health & Human Servs.*, No. 14-278V, 2019 WL 1061429, at *33 (Fed. Cl. Spec. Mstr. Jan. 31, 2019) (finding that Dr. Shoenfeld's blanket assertions regarding vaccines and autoimmunity "harm his overall credibility"). *Zafrir* also relied on another article not filed in this case that—as Dr. Norris conceded—says nothing about AA triggers. *Zafrir* at 2, citing D. Bogdanos, et al., *Tracing Environmental Markers if Autoimmunity: Introducing the Infectome*, 56 Immunologic Research 220 (2013). Respondent's Post-Hearing Brief at 6, filed on June 3, 2019, (ECF No. 65); Tr. at 97–98.

[7] Other special masters have described "rechallenge" as follows: "[c]hallenge-rechallenge happens when a person (1) is exposed to one antigen, (2) reacts to that antigen in a particular way, (3) is given the 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 & Human Servs.*, 83 Fed. Cl. 111, 119 (Fed. Cl. 2008) (internal citations omitted) (quoting *Nussman v. Sec'y of Health & Human Servs.*, No. 99-500V, 2008 WL 449656, at *9 (Fed. Cl. Spec. Mstr. Jan. 31, 2008)).

Dermatology 119 (2018), filed on February 28, 2019, as Ex. 30 (ECF No. 60) ("*Richardson*").[8] *Richardson*'s findings are based on a larger sample of patients—nearly 250,000 cases of HBV vaccination and 656 cases of AA—derived from the electronic medical record database at the University of Rochester Medical Center. *See Richardson* at 1. The query sought "to identify patients with AA, coexisting viral infections, vaccinations, or interferon therapy [ ] to determine if the presence of AA and these conditions was higher than in AA patients without the associated conditions or therapy." *Id.*

Accounting for different types of alopecia and hair loss, *Richardson's* authors observed an increased frequency of AA among those who had received the HBV surface antigen, whether from vaccination or wild virus infection. *See Richardson* at 4, 6 (finding support for the conclusion "that exposure to hepatitis B antigens, through either infection or vaccination, are associated with AA in a subset of patients"). Specifically, they observed AA to be two times more *prevalent* in individuals exposed to the HBV vaccine than the total population, and 2.73 times more likely to occur in a person exposed to the HBV vaccine. *Id.* at 4 (showing increased prevalence and odds ratio of AA after exposure to the HBV vaccine).[9] *Richardson* discussed two possible mechanisms that could explain how HBV vaccination could trigger AA: (1) the induction of interferon (a cytokine whose upregulation could be instigated by the immune process stimulated by vaccination);[10] or (2) molecular mimicry. *Id.* at 5. *Richardson* supports the plausibility of each of these causative theories, although it admits more research is needed to determine if either is the responsible biologic mechanism causing the increased prevalence and odds reported earlier. *See id.* at 5–7.

[8] *Richardson* was not identified or discussed during the hearing, despite it being published almost a full year prior. It was also co-authored by one of L.T.'s treaters, Dr. Elaine Gilmore. *Richardson* at 1. In addition, it focuses on only two cases—one of which is likely L.T. herself. *See Richardson* at 2 (describing "patient 1" as a "3-year-old female with hair loss of several weeks' duration []. [AA] began 4 days after receiving the third dose of the hepatitis B vaccine. The patient also developed transient joint pain of the wrists, arms, and right knee without associated swelling. She had a positive antinuclear antibody (ANA) (1:320 speckled/homogenous pattern) . . . . , and she had no significant medical history. Her family history included a mother with positive ANA and AA, and a sister with positive thyroid antibodies. Physical examination revealed widespread and well-demarcated alopecic patches on the scalp without scarring, erythema, or scale."). Such references actually *undercut* Petitioner's assertion that "nobody in [L.T.'s] family had ever been diagnosed with alopecia." *See* Petitioner's Post-hearing Brief at 1, filed on June 3, 2019, (ECF No. 64).

[9] The authors also observed that the enhanced possibility of AA after vaccination was statistically reliable. *See Richardson* at 4 fig. 2 (reporting a *p*-value of <0.0005 for HBV and AA association). A low *p*-value helps establish "statistical significance," because it shows "something other than chance must be involved." Federal Judicial Center & National Research Council, *Reference Manual on Scientific Evidence* 250 (3d ed. 2011). By contrast, a large *p*-value "indicate[s] that disparity can easily be explained by the play of chance." *Id.* Generally, a *p*-value of 5 percent or less ($p<0.05$) is considered the starting point—indicating that random error is not at work. *Id.* at 251–52; *see also id.* at 251 n.101 (citing *Castaneda v. Partilda*, 430 U.S. 482, 496 n.17 (1977); *Hazelwood School District v. United States*, 433 U.S. 299, 311 n.17 (1977)).

[10] Interferons are "any family of glycoproteins that exert virus non-specific but host specific antiviral activity by inducing the transcription of cellular genes coding for antiviral proteins that selectively inhibit the synthesis of viral RNA and proteins." *Dorland's* Illustrated Medical Dictionary 948 (32 ed. 2012) (hereinafter "*Dorland's*").

In addition to proposing that the HBV vaccine could trigger AA, Dr. Norris testified about L.T.'s individual circumstances, opining that the vaccine had likely done so in this case (and in a reasonable timeframe as well). Norris Report at 2; Tr. at 55–56. In support, he identified four cases from *Wise* in which the studied individual reported hair loss as early as one day after vaccination. Norris Report at 2; Tr. at 55–56 ("[i]n the case here, though, it's . . . . [onset is in] several days, and there are plenty of cases like that in the literature"); *Wise* at 1–2. He also emphasized that in the cases reported in *Wise*, onset of hair loss after vaccination was "usually more rapid after subsequent revaccination with HBV vaccine." Norris Report at 2. Thus, he reasoned, because L.T. had previously received the HBV vaccine, her immune response in November 2012 (and its purported damaging impact, in the form of AA) was likely faster than it would have been. Tr. at 84 (disagreeing with Dr. Tollefson's opinion).

On cross examination Dr. Norris defended *Wise* and its applicability to this case. *See* Tr. at 108–32. Respondent's counsel questioned Dr. Norris about several issues with *Wise*—e.g., the small sample size of clear rechallenge cases, the lack of specificity in *Wise* when describing the type of hair loss, and whether any of the clear rechallenge cases were similar enough to L.T.'s case to be helpful. *Id.* On each of these points Dr. Norris maintained that *Wise* was persuasive. *Id.*

B. *Respondent's expert: Dr. Megha Tollefson*

Dr. Tollefson served as Respondent's expert, and offered a single report as well as testimony at hearing. Ex. A, filed Sept. 15, 2017 (ECF No. 43-1) ("Tollefson Report"). Dr. Tollefson disputed Dr. Norris's opinion that L.T.'s injuries were caused by her receipt of the HBV vaccine in February 2012, maintaining instead that her genetic risk factors played more of a role in causing her symptoms. *See generally* Tollefson Report.

Dr. Tollefson graduated with a BA from Stanford University in 1999. Ex. B, filed on Jan. 28, 2019 (ECF No. 54-1) ("Tollefson CV"). She went on to earn her M.D. from Mayo Medical School in 2003. Tollefson CV at 1. She subsequently completed a pediatric residency at the Mayo Clinic College of Medicine from 2003–2006; a dermatology residency at the Mayo Clinic College of Medicine from 2007–2010; and a pediatric dermatology fellowship at Stanford University from 2010–2011. *Id.* Dr. Tollefson is board certified in dermatology and pediatric dermatology. *Id.* She has been an attending pediatric dermatologist at the Mayo Clinic since 2010. *Id.* at 2. She has also served as an assistant professor in pediatrics at the Mayo Clinic College of Medicine and Science. *Id.* Outside of her clinical practice Dr. Tollefson has a few publications and research projects on AA in children. *Id.* at 11, 19–20, 31.

Like Dr. Norris, Dr. Tollefson took some time at hearing to review AA (which she agreed L.T. experienced). Tollefson Report at 4; Tr. at 150. She accepted Dr. Norris's contention that AA is rightly understood to be an autoimmune disorder with a possible genetic basis. Tollefson Report at 2–3; Tr. at 150–51. However, in her view susceptibility to AA stems mostly from genetic

factors—particularly mutations in the filaggrin[11] gene. *See* Tollefson Report at 3 ("[p]atients with [AA] have a strong underlying genetic susceptibility profile . . . more likely to be associated with more severe disease and strong risk among family members.") (citing R. Betz, et al., *Loss-of-function Mutations in the filaggrin gene and Alopecia Areata: Strong Risk Factor for a Severe Course of Disease in Patients Comorbid for Atopic Disease*, 127(11) J. Investigative Dermatology 2539–43 (2007)); *see also* Tr. at 151. Dr. Tollefson also noted that certain gene mutations have been associated with a "more severe disease course, and also with atopic dermatitis (AD), or eczema." *Id.* Dr. Tollefson allowed, however, that some environmental triggers (for example, stressful events) could also produce AA, but that no one trigger has been identified as a primary cause. *Id.*

Based on review of the relevant medical records, Dr. Tollefson opined that L.T. possessed several of the risk factors necessary for the development of AA. Tollefson Report at 3. In particular, she identified L.T.'s family history, as well as her eczema and respiratory problems. *Id.* at 3–4. To support her assertion that L.T. had a strong family history of autoimmune disease, Dr. Tollefson referenced Ms. DeLozier's unspecified autoimmune connective tissue disorder, with hair loss and a positive ANA; L.T.'s third cousin having vitiligo; and L.T.'s older sister having positive thyroid antibodies. *Id.* This, Dr. Tollefson maintained, opened the door to the possibility that L.T. would have a positive ANA if tested (and in fact L.T. did so on some occasions). *Id.* (noting that a positive ANA can develop even in the absence of other symptoms). At a minimum, such facts allowed for the conclusion that L.T. was at an increased risk of autoimmune disease and AA. *Id.* (citing N. Barahmani, et al., *History of Atopy or Autoimmunity Increases Risk of Alopecia Areata*, 61 J. Am. Acad. Dermatology 581–91 (2009), filed as Ex. C (ECF No. 54-2) ("*Barahmani*")).

Dr. Tollefson also discussed some other risk factors, like L.T.'s eczema and "episodes of wheezing responsive to albuterol and steroids." Tollefson Report at 4. As she explained, eczema in many patients has been linked to mutations "in the filaggrin gene and other innate immune factors." *Id.* (citing T. Miyagaki, et al., *Lifetime Incidence Risk of Alopecia Areata Estimated at 2.1% by Rochester Epidemiology Project*, 1990–2009, 134(4) J. Investigative Dermatology1141–42 (2014), filed as Ex. G (ECF No. 54-6)) (noting also that "vaccines do not cause eczema). Respiratory problems are also common in patients who have a family history of eczema. *Id.* Based upon these factors, Dr. Tollefson proposed that L.T. might have "atopic diathesis," with a genetic risk for atopic disorders "none of which are caused by any vaccines." *Id.* The presence of such atopic diseases increased L.T.'s risk for AA. *Id.* (citing *Barahmani*).

Besides offering her own reading of the record, Dr. Tollefson disputed the reliability of some of the primary literature relied upon by Dr. Norris to associate the HBV vaccine specifically

[11] Filaggrin is "a protein that is synthesized in the granular level of the epidermis and aggregates intermediate filaments of keratin by promoting formation of disulfide bonds." *Dorland's* at 706.

with AA. *See* Tollefson Report at 3; Tr. 151–56. *Wise*, for example, did not appear to differentiate among the various types of alopecia. Tollefson Report at 3; Tr. at 155–56. This made it "impossible to say how many, if any of [those] cases, were associated with [AA]." Tollefson Report at 3. In addition, the four "clear examples" of hair loss from *Wise* offered to support proof of rechallenge after a subsequent HBV vaccination were either distinguishable from L.T.'s case or not probative of causation. Tr. at 153 (noting only a purely temporal relationship in patient 1 between vaccination and hair loss), 154–55 (stating that the facts given about patient 2 only show that patient was susceptible to hair loss and there was multiple triggers), 155 (stating that patient 3 likely suffered from telogen effluvium[12] rather than from AA). As a result, *Wise* at best established a pure temporal association between vaccination and AA. Tollefson Report at 3.

Dr. Tollefson similarly contested the degree to which the concept of challenge-rechallenge supported Petitioner's causation arguments in light of L.T.'s own history. Many of the studied individuals referenced in *Wise* had experienced hair loss in the process of receiving *multiple* doses of HBV. Tr. at 151–55; Tollefson Report at 3; *see also Wise* at 1–2 (Patient 1 developing hair loss after second and third HPV vaccine; Patient 2 developing hair loss after first, second, and third HPV vaccine; Patient 3 developing hair loss after first and second HPV vaccine). By contrast, although L.T. received HPV twice prior to the vaccination at issue, she *never* previously experienced any adverse effects. Tollefson Report at 3.

Dr. Tollefson also responded to questions on a number of related issues. *See generally* Tr. 166–72. Some of these questions were directed to the role of genetics in developing AA. Tr. at 168. She admitted that in some children, genetics played a more important role in the development of AA than a triggering event. Tr. at 168. Other questions were directed to a hypothetical: whether, in an individual who had AA, multiple relapses (separated by years) could be attributed to different triggers. Tr. at 171. To that, Dr. Tollefson answered yes. *Id*.

Finally, Dr. Tollefson questioned whether onset of L.T.'s AA symptoms occurred in a medically acceptable timeframe. Tollefson Report at 3. In so maintaining, she noted that L.T.'s onset did not in fact closely match the cases reported in *Wise*, who received the same vaccine but experienced symptoms each time. *Id.* Dr. Tollefson proposed that onset of AA after a trigger could occur as much as a few weeks to a couple of months thereafter, although she was unable to identify a minimum time that it would take for symptoms to manifest, without more evidence of an autoimmune reaction. Tr. at 158. (stating that she would need to know "as Dr. Norris said, if there was any subclinical inflammation there").

C. *Christine DeLozier*

[12] Telogen effluvium is "the early, excessive, temporary loss of club hairs from normal resting follicles in the scalp as a result of traumatization by some stimulus (e.g., after surgery or childbirth; with starvation, side effects of drugs, traction on hair, high fever, or certain diseases; or with psychogenic stress). The normal hair cycle is changed and the anagen phase ends prematurely, moving into the catagen and telogen phases." *Dorland's* at 595.

Ms. DeLozier testified at hearing about L.T.'s health history and family medical history. *See* Tr. at 5–31. She explained that she had a history of hair loss herself. Tr. at 8, 27. She had experienced such hair loss after a trip to Nicaragua, several years before L.T. was born, and recalled that she had received the HBV vaccine shortly before this trip. Tr. at 27–28. Ms. DeLozier's hair loss was diagnosed as a mixed connective tissue disorder, and testing revealed a positive ANA. Tr. at 8–9.

Petitioner also described L.T.'s experience with AA over time. Tr. at 12–22. As she admitted, L.T. had experienced a partial recovery from her AA about a year after onset. Tr. at 19–20 (describing L.T.'s hairline as receded and thinner). Thereafter, L.T. would experience recurrences of hair loss if she had an illness or infection. Tr. 20–22. Petitioner did not testify to another post-vaccination AA recurrence.

III. Procedural History

After filing this action, Petitioner continued to file relevant medical records. Respondent then filed his Rule 4(c) Report on February 28, 2017 (ECF No. 36), contesting whether Petitioner had established an evidentiary basis for entitlement. Rule 4(c) Report at 4–5. Over the next few years, the parties continued to litigate this case, and even engaged in a period of settlement discussions that were ultimately unfruitful. (ECF No. 44). They filed expert reports, pre-hearing briefs, and then presented evidence at an entitlement hearing held in February of 2019. Post-trial briefs were filed in June 2019. (ECF Nos. 64, 65).

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).[13]

[13] 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).

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).

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. *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.

C. *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").

D. *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").

ANALYSIS

I. Overview of Alopecia Areata

As both testifying experts agreed, AA is widely understood to be an autoimmune disease characterized by hair loss. Norris Report at 1; Tollefson Report at 2–3. Such hair loss appears in patches, typically on the scalp. S. Birela, et al., *Chapter 66: Non-bulbous Skin Diseases: Alopecia Areata, Vitiligo, Psoriasis, and Urticaria, in The Autoimmune Diseases* 971, 971–74 (N. Rose & I. Mackay Eds., 2014) (co-authored by Dr. Norris) ("*Autoimmune Diseases*").[14] AA is relatively common, with around two percent of people experiencing it at some point in their lives. *Id.* at 971; Norris Report at 1. This translates to roughly 5.3–6.5 million people in the United States. *See Autoimmune Diseases* at 971; Norris Report at 1.

AA is associated with a number of other diseases and conditions, such as vitiligo, atopic dermatitis (eczema), hyper and hypothryoidism, and, less commonly, other autoimmune diseases like connective tissue disease. *Autoimmune Diseases* at 972. Other conditions like nail loss or abnormalities may precede or follow AA. *See Zafrir* at 2. The disease also unquestionably has a genetic aspect. Norris Report at 1; Tollefson Report at 2–3. Indeed, literature suggests that "genetic control of innate and acquired immunity is *the most powerful factor* in determining the susceptibility to all variants of AA." *Autoimmune Diseases* at 973 (emphasis added).

AA occurs when a "mononuclear cell inflammatory infiltrate attacks the hair follicle (HF) bulb." *Autoimmune Diseases* at 971. The HF is responsible for producing the hair shaft. *See id.* Thereafter, T cell cytokines and cytotoxic T cells produce cytotoxic damage. *See id.* This disrupts the normal function of the HF, resulting in thin, fragile hairs that easily detach or break off. *See id.* 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.* at 972.

The triggers for AA are not well understood. *See Richardson* at 2. "Potential triggers include emotional stress, metabolic or endocrine disorders, infections, drugs, and vaccines." *Id.* Several potential pathogenic mechanisms by which AA might occur have been proposed, including molecular mimicry[15], the induction of the cytokine interferon (type 1 IFN), or a "cytokine storm,"

[14] AA is only one type of alopecia. Other types include alopecia totalis (loss of all hair on the scalp) and alopecia universialis (loss of all scalp and body hair). *See Zafrir* at 1–2. Additionally, there is another type of alopecia called telogen effluvium that is known to occur in response to stress on the body. Tollefson Report at 3.

[15] 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-

in which cytokines upregulated after some instigating event greatly increase in number for a period of time, causing harm simply through their proliferation. *Richardson* at 5–6; Tr. at 115; Pet'r's Brief at 7. Once AA is triggered, its clinical course is variable and not monophasic in progression. Tr. at 51–52; Resp.'s Post-Hearing Brief at 7. As Dr. Norris elaborated in his testimony, some patients experience recurring loss and regrowth, other patients experience one episode, and some will experience "everything in between." Tr. at 51–52.

II. Petitioner Has Carried Her *Althen* Burden With Respect to Her First Instance of AA in November 2012

Petitioner has *barely* met her preponderant burden with respect to L.T.'s initial outbreak of AA. The evidence is close, and almost in equipoise, on most of the *Althen* prongs in this case. But well-reasoned and controlling precedent in the Vaccine Program requires me in such close cases to decide the matter for the petitioner. *See Walther v. Sec'y of Health & Human Servs.*, 485 F.3d, 1146, 1150 (Fed. Cir. 2007) ("[u]nder our case law, 'close calls regarding causation are resolved in favor of injured claimants.'") (quoting *Althen*, 418 F.3d at 1280). I find that is appropriate here.

First, there is just enough evidence to support Petitioner's contention under the "can cause" prong that AA could be triggered by vaccination - and the HBV vaccine in particular. It was largely agreed-upon by the experts in this case that AA is likely an autoimmune process, thereby "opening the door" to a determination that something impacting the immune response could be implicated as causal. In addition, Petitioner offered some reasonable items of literature, like *Wise*, supporting the association between the HBV vaccine and hair loss generally, if not AA specifically. *See Wise* at 1–2.

Wise also demonstrated some instances of challenge–rechallenge. While this aspect of *Wise* did not aid Petitioner specifically on the second *Althen* prong (since the record in this case does *not* establish that L.T. ever experienced AA or hair loss following her earlier HBV doses),[16] it does lend some support for the contended HBV-AA association. Respondent is also correct about *Wise's* limitations as *strong* proof of the association (indeed, its authors acknowledge the tentative scope of its determinations (*Wise* at 1)), and this item of literature has not been updated with corroborative research since its publication twenty years before (although it has not been rebutted either). *Wise* nevertheless is deserving of some weight, and does go directly to the issue in contention.

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structures (with the latter constituting the damaging autoimmune response). Lauren Sompayrac, *How the Immune System Works* 122 (5th ed. 2016).

[16] Rechallenge is commonly understood to have this dichotomous effect when asserted by a Vaccine Act petitioner. Cases relying on rechallenge to prove *Althen* prong I have sometimes failed under *Althen* prong II. See *Nussman v. Sec'y of Health & Human Servs.*, 83 Fed. Cl. 111, 119 (Fed. Cl. 2008) ("[t]here can only be rechallenge if there was an initial challenge and associated adverse reaction").

Richardson also assists Petitioner's theory to a degree. *Richardson* involved a significantly larger sample size than *Wise*, and statistical analysis of this sample showed a reliable correlation of increased rates of AA in individuals who also had the HBV vaccine. *Richardson* by its own admission is not definitive on the point (especially to the extent it did not account for the possibility of a mere temporal association of AA to HBV vaccination). *See Richardson* at 5. In addition, *Richardson*'s post-hearing filing (and the fact that it was not discussed by Dr. Norris) causes me to give it a little less weight than I might otherwise. *See McClellan v. Sec'y of Health & Human Servs.*, No. 14-714V, 2019 WL 4072130, at *30 n.28 (Fed. Cl. Spec. Mstr. July 2019, 2019) ("I typically give late-filed items less weight where a party has not demonstrated a justification for their dilatoriness—for example, because the item in question was only published after hearing.").[17] It nevertheless was credible evidence favoring Petitioner's claim—and was not refuted by Respondent, let alone addressed in his post-hearing briefing.

Respondent's failure to rebut Petitioner's case went beyond not addressing the late-filed *Richardson*. Certainly he pointed out some deficiencies in Petitioner's causation theory, such as the lack of a reliable mechanistic explanation for how a vaccine would induce the breaking of immune tolerance (although it is well recognized in the Program that petitioners can prevail even in the absence of proof of mechanism). Dr. Tollefson was also a credible and competent expert witness, and her points about the genetic basis for AA were effectively established. But in this case (unlike others) Respondent was unable to offer evidence casting doubt on Petitioner's theory (e.g., an epidemiologic study that discounted the purported association between the HBV vaccine and AA).[18] I also found Dr. Norris to be a qualified and persuasive expert witness, and his embrace of Petitioner's theory gave it a little added heft.

Second, the record does support (again, just barely) the conclusion that the HBV vaccine in this case likely triggered L.T.'s AA onset in November 2012. Petitioner has offered treater support for this contention, such as Dr. Gilmore's notes from an appointment in January 2013. Ex. 7 at 1–2. While Respondent correctly observes that the record also includes some more ambiguous statements about an association (especially since not all treaters signed on to this reasoning), or may simply evidence overreliance on the temporal association (which is recognized as an insufficient basis to ascertain causation), I nevertheless discern sufficient, reliable treater support connecting L.T.'s AA to her HBV vaccination to deem it evidentiarily significant. Dr. Norris also

[17] The fact that one of L.T.'s own treaters co-authored *Richardson* (and even appears to discuss L.T.) is also somewhat problematic—although in other cases articles written about an injured party have been deemed deserving of evidentiary weight (and in Respondent's favor) if only for their discussion of a condition. *See McClellan*, 2019 WL 4072130, at *4 (discussing article addressing mutation in petitioner's daughter, the allegedly injured party).

[18] In many prior cases, I have found the existence of strong and reliable epidemiologic evidence to tip the scales in Respondent's favor—even though it is unquestionably the case that this kind of proof need not be offered by a petitioner. *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). The converse must also be true: where Respondent *does not* offer such evidence, arguments a Petitioner may make on causation will stand a little more unscathed.

provided a persuasive interpretation of the record, and what factors specific to L.T. would implicate the vaccine as triggering her genetically-associated AA. *See generally* Tr. at 66–69.[19]

Finally, the timeframe in which L.T.'s AA began—two to four days post-vaccination—was established to be a medically acceptable temporal relationship for an autoimmune response. Petitioner claims this timeframe is supported by *Wise* and Dr. Norris's expert opinion. Dr. Norris's opinion as to timing, in his report and testimony, relied mostly on *Wise*. Tr. at 56; Norris Report at 2. Although Respondent's criticisms of *Wise* are reasonable, I find overall that the concept that the autoimmune response to the HBV vaccine could begin in that timeframe is reasonable, especially given Dr. Norris's acceptance of it.

I again emphasize that Petitioner's showing was not particularly *robust.* Other evidence could have rebutted it. But the record in this case establishes that, at least with respect to the first occasion of L.T.'s AA, Petitioner carried her preponderant burden.

III. Petitioner Has Not Carried her Burden of Proof with Respect to Any Subsequent Occurrences of AA

Although Petitioner has established entitlement to damages stemming from her *first* occurrence of AA after the November 2012 vaccination, I do not find that she persuasively established that any subsequent recurrences (the first of which appears to have happened around the time of her August 2015 return visit to Dr. Gilmore, reporting new-onset AA symptoms) can *also* be attributed to that same initial vaccine event. The thinness of Petitioner's overall evidentiary showing may have been just enough to be preponderant in determining causation with respect to the first occurrence of AA, but that same slim showing shifts against Petitioner when the larger picture (including what the experts agreed about AA) is taken into account.

Determination of this aspect of Petitioner's claim turns almost wholly on the first *Althen* prong.[20] Although I have found that Petitioner was successful in establishing that a vaccine might be a sufficient environmental trigger to produce an occurrence of AA, the *context* of that occurrence is significant. Both experts agreed that AA is known to have a significant genetic component that serves as a baseline "requirement" for AA to manifest. Tr. at 41–42, 150–51; *see also* A. Alkhalifah, et al., *Alopecia Areata Update: Part I. Clinical Picture, Histopathology, and Pathogensis*, 62 J. Am. Acad. Dermatology, no. 2, 177, 184–85 (2010) , filed on July 18, 2017, as

[19] As already noted, however, this case does *not* support the conclusion that L.T.'s vaccine reaction was associated with her prior receipt of the HBV vaccine, under a challenge-rechallenge theory. The record does not establish she ever reacted to those prior vaccinations, nor did she experience AA after them. The general concept that a person who has received a vaccine before without incident will nevertheless inherently (or likely) have a faster response later, as reflected in the injury asserted in a Vaccine Act claim, is speculative and conclusory.

[20] I note as well that Petitioner's subsequent recurrences of AA (the first of which appears to have happened no earlier than August 2015) have not been shown to have happened post-vaccination. Accordingly, Petitioner would still in this case not be entitled to damages for those occurrences even if she had not maintained that the first vaccination explained all subsequent AA recurrences.

Ex. 18 (ECF No. 42) (family history of AA increases chances of developing the condition); A. Gilhar, et al., *Alopecia Areata*, 336:16 New Eng. J. Med. 1514, 1521–22 (2012), filed on July 18, 2017, as Ex. 19 (ECF No. 42-1) (patients with AA often have a family history of atopy and autoimmune disorders). Here, there was ample circumstantial evidence that L.T. was just such a person. Indeed, as Petitioner herself testified, *she* had experienced hair loss in association with a likely autoimmune event even before L.T. did. Tr. at 8–9.

Given the above, can it be concluded that the same initial trigger—here, the November 2012 vaccination—could reliably be deemed responsible for all *future* AA recurrences? The evidence offered in this case does not support that assertion. AA is clearly viewed as non-monophasic and inherently subject to recurrence. Moreover, vaccines are not the sole possible trigger. Ms. DeLozier admitted as much, when she recounted the fact that L.T. had experienced recurrences under *other* circumstances, such as when L.T. had an infection or fever. Tr. at 20–22.

The scientific and expert evidence filed in this case otherwise did not stand for the proposition that response to an initial vaccine trigger could explain every subsequent occurrence of AA in a susceptible individual. None of the literature offered by Petitioner so states, or establishes what a vaccine like HBV would do to alter a person's subsequent immune privilege in future triggering events. At best, Dr. Norris attempted to outline how a vaccine could theoretically create a "smoldering immune response," consistent with the condition's relapsing nature, and thus even separate subsequent triggers would be linked to the initial event, but such assertions were (unlike most of his testimony) conclusory and unpersuasive. Tr. at 175–77. At bottom, nothing that is known about AA (at least as reflected in the filings in this case) suggests that the first instance of AA in a person (likely to occur in childhood) is the linchpin explanation for all subsequent recurrences.

The capacity of a vaccine to trigger a pathologic response must be placed in the context of the overall disease's expected course. In other cases in the Vaccine Program, petitioners have demonstrated that the "trigger effect" of vaccination can not only cause an initial reaction but lead to permanent progressive harm, with the initial injury beginning a progressive process of worsening symptoms. For example, vaccines have in some cases been shown to cause direct, catastrophic injury to the brain, such as an encephalopathy that has significant follow-on health impacts. *See, e.g., Estep v. Sec'y of Health & Human Servs.*, 28 Fed. Cl. 664, 669 (Fed. Cl. 1993) (affirming special master's finding that a "DPT vaccination can cause an acute encephalopathy, and that anything that can cause an acute encephalopathy can [subsequently] cause permanent neurologic damage"). A comparable theory that has found success involves circumstances where petitioners demonstrate that a seizure triggered by vaccination (sometimes merely due to the fever associated with the body's innate immune response) will set up conditions for future, more harmful seizures in the future. *Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1375–76 (Fed. Cir. 2009) (reversing special master, and finding that petitioners had shown that the vaccine in question could trigger seizures).

The "trigger" effect of the vaccine in this case, by contrast, is different – more isolated and discrete in scope. Both sides' experts agreed that AA has a significant genetic underpinning, and thus a person who likely possesses that predisposition (a conclusion that can be made about L.T. based on preponderant evidence) is more susceptible to experiencing AA on a lifelong basis. While it may have been established in this case that the HBV vaccine could trigger one instance of AA, it has not been similarly shown that *any* trigger (vaccine or not) would so alter a person's immune response that all AA recurrences would invariably be associated with the first, made worse due to the first, etc. None of the medical literature filed in this case stands for this proposition, and in fact (in stressing the importance of the genetic susceptibility to AA) actually undermines it.

It is more likely that an individual's subsequent recurrences are attributable to the genetic susceptibility underlying AA. *See McClellan*, 2019 WL 4072130, at *35–36 (finding that petitioner "did not establish that a vaccine could, under the circumstances, trigger a non-febrile seizure sufficient to significantly worsen a preexisting seizure disorder with an unmistakable genetic origin"); *Sharpe v. Sec'y of Health & Human Servs.*, No. 14-65V, 2018 WL 7625360 (Fed. Cl. Spec. Mstr. Nov. 5, 2018) (finding that neither the record supported "[p]etitioner's contention that the vaccines [] received could, or did, injure [their daughter] as alleged," nor did petitioners establish a significant aggravation claim given their daughter's existing "DYNC [gene] mutation"), *aff'd*, 142 Fed. Cl. 630 (2019), *appeal docketed*, No. 19-1951 (Fed. Cir. May 31, 2019). And Dr. Norris did not otherwise credibly establish with reliable evidence that AA can be thought of as a "smoldering" condition, in which the instigating trigger for an outbreak is a spark that is never extinguished.

The implication of my ruling is that Petitioner's recoverable damages in this case are circumscribed in several respects. Clearly she is entitled to costs associated with L.T.'s treatment from November 2012 until any initial AA symptoms associated with the triggering abated (and a preliminary review of the record suggests that had occurred by no later than August 2014). She can also seek an award of pain and suffering associated with her initial November 2012 symptoms. But she cannot recover damages associated with any new, discrete AA recurrences that L.T. experienced *post-vaccination*, beginning no later than August 2015.

CONCLUSION

Ms. DeLozier has carried her burden in establishing that the HBV vaccine could trigger AA, and did so in L.T.'s case in November 2012. She should therefore receive a damages award reflecting the costs of treatment of that first occurrence, plus any other damages flowing therefrom. However, she has not established an entitlement to damages associated with any *subsequent* AA recurrences, which have not preponderantly been shown to be attributable to the earlier HBV vaccine.

In order to guide the parties through the damages phase of the action, a separate damages order will issue.

IT IS SO ORDERED.

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