In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 07-170V
Filed: March 4, 2011
Unpublished

SCOTT R. HAMMITT, as the Legal Representative of his Minor Daughter, RACHEL HAMMITT, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	Decision on Remand; Severe Myoclonic Epilepsy of Infancy (SMEI), Dravet Syndrome; SCN1A Gene Mutation; DTaP Vaccine; Factor unrelated to vaccine was the "sole cause" and "principally responsible" for injury; Prima facie case

Curtis R. Webb, Twin Falls, I.D., for Petitioner.
Althea Walker Davis, U.S. Department of Justice, Washington, D.C., for Respondent.

DECISION ON REMAND[1]

Golkiewicz, Special Master.

Petitioner sought review of the undersigned's Decision denying his claim on behalf of his daughter for compensation pursuant to the National Vaccine Injury Compensation Program.[2] Hammitt v. Sec'y of the Dept. of Health & Human Servs., No. 07-170V, 2010 WL 3735705 (Fed. Cl. Spec. Mstr. Aug. 31, 2010) (hereinafter "Hammitt I"). On December 22, 2010, Judge Wheeler issued an opinion finding that the undersigned applied the incorrect legal standard in finding that respondent successfully proved that Rachel's SCN1A gene mutation, and not her immunizations, was the cause of her condition, SMEI.[3] Citing the Federal Circuit's decision in

[1] The undersigned intends to post this decision on the website for the United States Court of Federal Claims, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002). **As provided by Vaccine Rule 18(b), each party has 14 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 entire decision will be available to the public. Id. Any motion for redaction must be filed by no later than fourteen (14) days after filing date of this filing.** Further, consistent with the statutory requirement, a motion for redaction must include a proposed redacted decision, order, ruling, etc. See also Langland v. Sec'y of the Dept. of Health & Human Servs., No 07-36V (Fed. Cl. Spec. Mstr. February 3, 2011)(Order granting in part and denying in part petitioners' request for redaction)(discussing pertinent law regarding redaction).

[2] This Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 et seq. (West 1991 & Supp. 2002) (hereinafter "Program," "Vaccine Act" or "the Act"). Hereafter, individual section references will be to 42 U.S.C. §§ 300aa of the Act.

[3] SMEI stands for Severe Myoclonic Epilepsy of Infancy. It is also referred to as Dravet Syndrome in this case. This serious seizure disorder "[b]egins in the first year of life in previously healthy children. Hemiclonic seizures, which may be long

De Bazan v. Sec'y of the Dept. of Health & Human Servs., 539 F.3d 1347 (Fed. Cir. 2008), Judge Wheeler determined that "Respondent faces a heavier burden than 'more likely than not' in showing that a 'factor unrelated' caused the injury, and not the vaccine." Hammitt v. Sec'y of the Dept. of Health & Human Servs., No. 07-170V, slip op. at 2 (Fed. Cl. Dec. 22, 2010)(Remand Order granting remand)(hereinafter "Remand Order"). Judge Wheeler further stated that the undersigned "did not articulate clearly whether Petitioner presented a prima facie case under the Althen test, and whether the burden of proof therefore shifted to Respondent." Id. Nor did the undersigned find, as required by De Bazan, "that the SCN1A gene mutation was the sole substantial factor in causing Rachel's SMEI." Id. The case was remanded for proceedings consistent with the Order. Id. The Remand Order indicated the remand shall not exceed ninety days.

The undersigned conducted a status conference with the parties on January 5, 2011, to discuss Judge Wheeler's Remand Order and to determine how to proceed.[4] Minute Entry, January 6, 2011. In response to the undersigned's inquiries, the parties indicated the desire to submit further briefing on remand. Order, filed January 6, 2011. Simultaneous briefs were filed on February 4, 2011.[5]

Petitioner filed his Brief on Remand ("P Brief") on February 4, 2011. In summary, petitioner argues that he has proven the prima facie case for vaccine causation and cites other Program cases wherein a similar sequence of events and theory proved successful for petitioners. P Brief at 1-4. Petitioner also argues that the "absence of proof of an alternate cause is not an element of petitioner's prima facie case." P Brief at 4-6. Further, petitioner contends the Federal Circuit's decision in Doe/11 v. Sec'y of the Dept. of Health & Human Servs., 601 F.3d 1349 (Fed. Cir. 2010), does not allow respondent to present evidence of alternative causation to generally rebut petitioner's prima facie case. P Brief at 6-7. Regarding Doe/11, petitioner attempts to strictly limit the holding. Petitioner continues, quoting portions of testimony, particularly a segment of Dr. Raymond's testimony, that petitioner believes concedes the role of the vaccination in causing Rachel's SMEI and therefore proves his case according to the Restatement (Second) of Torts. P Brief at 8-11.

Respondent's brief discusses the parties' respective burdens and argues that petitioner failed to establish a prima facie case or, alternatively, respondent proved the SCN1A mutation was principally responsible and, if it is required legally, the sole cause of Rachel's condition. Respondent argues that she is not prohibited from presenting evidence of alternative causation and the special master may consider respondent's evidence of alternative causation "in determining whether petitioner met his burden of establishing a prima facie case." R Memorandum in Response to Remand Order, filed February 4, 2011 (citing Doe/11, 601 F.3d at

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lasting, are characteristic and can be associated with fever. Myoclonic, absence, tonic-clonic, and partial seizures also occur. The epilepsy is refractory and developmental regression ensues." Hammitt I, 2010 WL 3735705, at *1 (internal citations omitted).

[4] During the January 5, 2011, status conference, petitioner orally moved to file additional evidence. This request was denied as beyond the scope of the Remand Order; however, the undersigned encouraged petitioner to make a formal motion to submit the desired evidence to the undersigned, as well as to Judge Wheeler in case the undersigned was incorrect in reading the scope of the Remand Order. Order, filed January 6, 2011. Petitioner filed two formal requests, one to the undersigned and one to Judge Wheeler, to file the additional evidence. P Motion for Leave to File Evidence on Remand, filed January 13, 2011. Respondent filed responses on January 26, 2011. Petitioner's Motion to file additional evidence was denied by the undersigned on February 3, 2011. Order Denying Petitioner's Motion for Leave to File Evidence on Remand, filed February 3, 2011.

[5] On February 7 and February 9, 2011, the undersigned's office confirmed that neither party desired responsive briefing.

1358)("R Memorandum"). Respondent argued that petitioner failed to meet his burden due to his inability to prove brain damage resulted from the first seizure and in the face of overwhelming evidence that his daughter's SCN1A gene mutation was solely responsible for her SMEI. R Memorandum at 5-9. Respondent alternatively argues that she has proven that a factor unrelated, the SCN1A mutation, is principally and solely responsible for the injury. R Memorandum at 9-15. Respondent further contends that the language in De Bazan v. Sec'y of the Dept. of Health & Human Servs., 539 F.3d 1357, 1354 (Fed. Cir. 2008), regarding the factor unrelated being the "sole substantial factor," should be "interpreted to mean that respondent must establish that the factor unrelated, **not** the vaccination, is the actual cause of the alleged." R Memorandum at 12 (emphasis in original). Respondent's position is that she must prove by a preponderance of the evidence that the factor unrelated is principally responsible for the alleged injury; "To require more would improperly subject respondent to a standard of medical or scientific certainty, which is clearly not contemplated under the Act." Id. at 12-15.

DISCUSSION[6]

The issues on remand are whether petitioner proved his prima facie case and if so, whether respondent proved a factor unrelated to the vaccine was the sole, substantial cause of Rachel's SMEI.[7] What appears to be a straightforward directive on remand is not so clear in practice as the issue of shifting burdens under the Vaccine Act continues to be a matter of considerable debate. Respondent argued throughout this case that her burden to prove a factor unrelated never arose, that the burden never shifted to respondent; rather, respondent argued that the SCN1A evidence was offered in rebuttal to petitioner's prima facie case. See, e.g., Hammitt I, 2010 WL 3735705, at *14, n. 12.

My colleague explored the seemingly discordant precedent regarding burden shifting in Heinzelman v. Sec'y of the Dept. of Health & Human Servs., No 07-01V, 2008 WL 5479123, at *4-16 (Fed. Cl. Spec. Mstr. Dec. 11, 2008)(resting ultimately upon the Federal Circuit assigning "the burden of ruling out other potential causes to the respondent"), appeal docketed, No. 07-01 (Fed. Cl. Jan. 6, 2011). Possibly complicating the discussion further is the Federal Circuit's opinion in Doe/11 v. Sec'y of the Dept. of Health & Human Servs., 301 F.3d 1349, 1358 (Fed. Cir. 2010), which was discussed by both parties in their briefs on remand. In Doe/11, it appears the Circuit found that neither the statute nor prior Circuit precedent precludes the government from presenting evidence of alternative causation to rebut petitioner's case-in-chief. Doe/11, 601 F.3d at 1358.[8]

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[6] This discussion presumes knowledge of Hammitt I. For ease of reference, pertinent citations will be to the Hammitt I decision, with citations to the record of Hammitt omitted.

[7] The issues discussed in this case on remand are similar to those discussed in the Remand Decision in the case of Stone v. Sec'y of the Dept. of Health & Human Servs., No. 04-1041, slip op. (Fed. Cl. Spec. Mstr. Jan. 20, 2011) appeal docketed, No. 04-1041V (Fed. Cl. Feb. 22, 2011). For expediency, when these cases were originally litigated, testimony was taken at the same time. The instant case and the Stone case presented the same issue regarding the relationship of the SCN1A gene mutation to SMEI. In the Stone Remand Decision, the undersigned found that the evidence established that the SCN1A gene mutation was the sole and principal cause of Amelia Stone's SMEI. Stone, No. 04-1041, slip op. at 4 (Fed. Cl. Spec. Mstr. Jan. 20, 2011). The same finding is made here for Rachel Hammitt's case.

[8] See also Walther v. Sec'y of the Dept. of Health & Human Servs., 485 F.3d 1146, 1151, n. 4 ("Where multiple causes act in concert to cause the injury, proof that the particular vaccine was a substantial cause may require the petitioner to establish that the other causes did not overwhelm the causative effect of the vaccine."); Shyface v. Sec'y of the Dept. of Health & Human Servs., 165 F.3d 1344, 1352 (adopting the Restatement (Second) of Torts rule for determining vaccine causation and acknowledging that contributing factors must be weighed when concurrent forces are alleged to bring about a single harm. "'Some other event which is a contributing factor in producing the harm may have such a predominant effect in bringing it about as to make the effect of the actor's negligence insignificant and, therefore, to prevent it from being a substantial factor. So too,

However, while this issue of burden shifting is subject to continuing debate, what is clear is that based upon the record as a whole, the SCN1A gene mutation is the sole, substantial cause of Rachel's SMEI, and therefore the cause that is principally responsible for her SMEI.[9] Therefore, the burden-shifting issue is not critical to resolving this case as the preponderant, indeed the overwhelming, weight of the evidence is that the gene mutation is the **principal and sole cause** of Rachel's condition. However, to address the Remand Order and for the sake of completeness, the undersigned addresses the evidence in three ways: petitioner's prima facie case without SCN1A rebuttal evidence; petitioner's prima facie case with the SCN1A rebuttal evidence; and lastly, assuming *arguendo* that petitioner proved his prima facie case, respondent's factor unrelated evidence. It is recognized that there is much overlap in the respective discussions.

For purposes of the following discussion, the undersigned adopts and affirms the entire discussion and findings from Hammitt I, with two exceptions: the statement that the undersigned "likely would have found for petitioner" in the absence of evidence on Rachel's SCN1A mutation and the finding that respondent had proven that the SCN1A gene mutation was a "but for" and "substantial factor" cause of Rachel's SMEI. E.g., Hammitt I, 2010 WL 3735705, at *14, *47. In lieu of these findings and after consideration of the record as a whole, the undersigned finds: 1) that petitioner failed to prove the prima facie case and, and 2) that even if petitioner proved his prima facie case, that respondent proved by a preponderance of the evidence that the SCN1A gene mutation was the sole cause and that it was principally responsible for Rachel's SMEI. As was done initially, Hammitt I, 2010 WL 3735705, at *9-10, *43-45, the undersigned relies heavily on the essentially unrebutted testimony of respondent's expert, Dr. Raymond, who is a clinical neurogeneticist. "In contrast to Dr. Raymond's cogent explanations, Dr. Kinsbourne[, petitioner's expert,] was unable to adequately address the issues presented in this case, specifically those relating to genetics." Id. at *44; see also id. at *7-8 (discussing Dr. Kinsbourne's credentials generally and as related to this case).

It is emphasized that while it was held that the incorrect legal standard was used in Hammitt I to express findings as to the weight accorded the parties' evidence, while poorly articulated, it was the undersigned's firm belief in resolving the case in the first instance, as it is my explicit finding now, that based upon the record as a whole, Rachel's gene mutation was the sole cause of her SMEI.

Petitioner's Prima Facie Case without Consideration of the SCN1A Rebuttal Evidence

Discussing the parties' respective burdens in the Remand Order, Judge Wheeler stated:

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although no one of the contributing factors may have such a predominant effect, their combined effect may . . . so dilute the effect of the actor's negligence as to prevent it from being a substantial factor.'" Restatement (Second) of Torts § 433 cmt. d).

It is noted that in this case petitioner's expert agreed that Rachel's SMEI has a genetic basis, indeed "a very powerful one." Hammitt I, 2010 WL 3735705, at *22. The experts agreed that the vaccine caused a fever which may have triggered the initial seizure. Dr. Kinsbourne agreed with the undersigned that the issue in this case "is the role of [Rachel's] initial seizure, this complex seizure[,] in altering whatever mutation we have." Id.

[9] Compare De Bazan 539 F.3d at 1354 (discussing respondent's burden as being "sole substantial factor in bringing about the injury."), with 42 U.S.C. § 300aa-13(a)(2)(B) (describing respondent's burden under the Vaccine Act as preponderant evidence that the factor unrelated was "the agent or agents principally responsible for causing petitioner's illness, disability, condition, or death.").

> As the Federal Circuit pointed out, if the petitioner has successfully proven a prima facie case through the three elements of the Althen test – (1) 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 – then there is a presumption that the vaccination is the "causation-in-fact **absent proof that some other factor was the actual cause."**

Remand Order at 2 (quoting De Bazan, 539 F.3d 1354)(emphasis in original and Remand Order). As noted by petitioner, P Brief at 10-11, the Federal Circuit adopted the Restatement (Second) of Torts rule "for purposes of determining vaccine injury, that an action is the 'legal cause' of harm if that action is a 'substantial factor' in bringing about the harm, and that the harm would not have occurred but for the action." Shyface, 165 F.3d 1344, 1352.

As stated in Hammitt I, "**in the absence of the evidence presented regarding Rachel's SCN1A mutation**, the undersigned likely would have found for petitioner." Hammitt I, 2010 WL 3735705, at *14 (emphasis in Hammitt I). However, upon further consideration, in analyzing only the evidence of petitioner's prima facie case, it is found that the lack of brain damage thwarts petitioner's prima facie case.

Primarily, petitioner has not shown evidence of any brain damage or effect that the initial fever and seizure had on Rachel's ultimate condition. Dr. Kinsbourne, petitioner's expert, recognized that SMEI has a "very powerful" genetic component. Hammitt I, 2010 WL 3735705, at *22. However, Dr. Kinsbourne argued that "the pertussis vaccination caused fever, the fever triggered the seizure, the seizure lasted a long time" and caused damage by lowering Rachel's seizure "threshold."[10] Id. Dr. Raymond and respondent's other expert in this case, Dr. Wiznitzer, conceded that the vaccine caused a fever in this case, which in turn may have triggered Rachel's initial complex febrile seizure.[11] Hammitt I, 2010 WL 3735705, at *41. However, neither doctor saw any evidence that the vaccination or the initial seizure "caused any brain damage or injury that contributed to her SMEI." Id. As Dr. Raymond explained, "while complex febrile seizures **can** injure the brain, 'you have to put that in context of these cases **where we have no evidence that the complex febrile seizures actually injure the brain**; that their course was in any, shape or form different than any other individual who [has] Dravet syndrome.'" Id. (emphasis in Hammitt I). There was simply no evidence of any role by the

[10] In petitioner's Brief on remand, petitioner argues that prior Vaccine Act cases have a controlling effect on the outcome here. P Brief at 2-4 (discussing cases that had a similar theory proposed, which was successful, but that were without evidence of the genetic mutation, Simon v. Sec'y of the Dept. of Health & Human Servs., No. 05-941V, 2007 WL 1772062 (Fed. Cl. Spec. Mstr. Jun. 1, 2007) and Mersburgh v. Sec'y of the Dept. of Health & Human Servs., No 05-5049V, 2007 WL 5160385 (Fed. Cl. Spec. Mstr. Jul. 9, 2007)). Decisions of special masters are not binding on special masters' subsequent cases. Guillory v. U.S., 59 Fed. Cl. 121, 124 (Fed. Cl. 2003). Simply put, the testimony presented in this case regarding the lack of evidence of harm following the immunization and initial seizure preponderates against petitioner's prima facie case. Also, as it relates to the next sections discussing respondent's rebuttal evidence, petitioner fails to acknowledge the evidence of the genetic mutation that was present in this case, but which was not presented in the Simon and Mersburgh cases. Hammitt I, 2010 WL 3735705, at *44. This argument was also discussed in the undersigned's original decision in this case. Id.

[11] Petitioner cites Hammitt I as stating, "[t]he March 15, 2004 seizure was the first symptom of Rachel Hammitt's Severe Myoclonic Epilepsy of Infants (SMEI)." P Brief at 8. However, the undersigned made no finding in Hammitt I that the March 15, 2004, seizure was indeed the first symptom of Rachel's SMEI.

vaccination in the development of her SMEI and Dr. Kinsbourne offered no persuasive testimony to counter this testimony.[12] Id. at 43.

Dr. Kinsbourne simply "inferred" damage from the initial seizure. Hammitt I, 2010 WL 3735705, at *41. This inference was in spite of Dr. Kinsbourne's agreement that "a trigger doesn't necessarily have to have a further deeper impact." Id. Dr. Kinsbourne also responded "no" to the undersigned's question of whether "there was any other clinical manifestation of the brain damage you maintain occurred." Id. Further, as discussed in Hammitt I:

> Dr. Kinsbourne stated the clinical evidence for his theory rests upon the fact "that the vaccine was given, that [her] temperature was elevated, and . . . the seizure occurred and how long it was . . ." and that further seizures followed. However, Dr. Kinsbourne stated "no" in response to the undersigned's inquiry of whether "there was any other clinical manifestation of the brain damage you maintain occurred." Further, Dr. Kinsbourne conceded Rachel experienced no developmental delay until after the first year of life. Then in response to the undersigned's question, "[d]oes your theory of lower seizure threshold account for developmental delay after the first year?" Dr. Kinsbourne responded, "Not necessarily, no. And there are two ways of looking at it one or both may be correct . . . ." In response to the undersigned's question regarding whether "the [first] seizures themselves contribute[d] to additional damage," Dr. Kinsbourne replied, "I don't remember it well enough." To which the undersigned noted, "[s]o the impairment of the sodium channels is a possible explanation for this for [her] subsequent condition . . . ?" Dr. Kinsbourne replied: "That's one way of looking at it."

Hammitt I, 2010 WL 3735705, at *41 (internal citations omitted).[13]

Dr. Raymond testified that the typical age of onset of SMEI is two months to nine months and onset [of the first seizure] is associated with a temperature elevation. Id. Dr. Raymond stated that the temperature elevation does not "play any sort of causal role in the disease." Id. at 42. Dr. Raymond was asked whether Rachel's temperature elevation and subsequent seizure played a role in her condition. Dr. Raymond explained, "in terms of her overall clinical course, no. She was going to have [SMEI]." Id. at *43. Dr. Raymond continued, "the substantial factor to her having [SMEI] is the mutation . . . [the seizure] occurred in the context of her having a mutation and [SMEI], and it is consistent with her having [SMEI] . . . . [The DTaP vaccination] had no significant role in the development of her having [SMEI]." Id.

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[12] See also Hanlon v. Sec'y of Dep't of Health & Human Servs., 191 F.3d 1344, 1349 (Fed. Cir. 1999)(affirming of the Special Master's decision and agreeing that "'where a [tuberous sclerosis, TS,] child receives DPT vaccine and remains perfectly normal (in temperature, eating, sleeping, affect, and activity) but has a[n] [afebrile] seizure within three days, TS, not DPT, is the cause in fact of that seizure.'")(quoting Barnes v. Sec'y of the Dept. of Health & Human Servs., 1997 WL 620115, at *33 (Fed. Cl. Spec. Mstr. Sept. 15, 1997)).

[13] "Dr. Kinsbourne's theory that Rachel's first complex febrile seizure experienced post-vaccination caused her to suffer brain damage appears to be in addition to, or a variation of, Dr. Kinsbourne's theory that Rachel's initial complex febrile seizure resulted in a lowering of her seizure threshold and led to further seizures resulting in her ultimate diagnosis of SMEI. See infra p. 14-15." Hammitt I, 2010 WL 3735705, at *41, n. 37.

In his Brief on remand, petitioner argues that Dr. Raymond, respondent's geneticist expert, "acknowledged that [the] vaccination played **a role** in the development of Rachel Hammitt's SMEI." P Brief at 9-11 (citing Transcript, May 14-15, 2009, filed June 16, 2009, pages 339-40 (hereinafter "Tr. at")). Petitioner quotes Dr. Raymond's testimony from the hearing and gleans a meaning the undersigned cannot perceive. Petitioner states, "please note that the critical testimony from Dr. Raymond acknowledged that [the vaccination] played **a role** in the development of Rachel Hammitt's SMEI." P Brief at 9. Petitioner then further reasons that "if the vaccination's role in the SMEI would 'lead reasonable men to regard it as a cause, using the word in the popular sense' then the vaccination was a substantial factor in the cause of the SMEI." P Brief at 11 (quoting Restatement (Second) of Torts § 431 cmt. a; see also § 431 cmt. b.). However, review of the cited portion of Dr. Raymond's testimony clearly evidences his opinion that the vaccination "had no significant role in the development of her having [SMEI] . . . [the genetic mutation is] the sole cause. The dysfunction in the channel secondary to the SCN1A [mutation] is the sole cause of [Rachel's] sever[e] myoclonic epilepsy of infancy." Hammitt I, 2010 WL 3735705, at *32 (citing Tr. at 340).

Reviewing the record, petitioner's prima facie case lacks any persuasive evidence of the vaccination causing Rachel's SMEI. Dr. Kinsbourne was singularly unpersuasive in his testimony, relying on an inference as opposed to proof of causation. Tr. at 476. As was the case throughout, the testimony of Drs. Wiznitzer and Raymond was powerful in explaining why such an inference of blame is faulty. Accordingly, it is found that petitioner has failed to establish his prima facie case of vaccine causation by preponderant evidence.

Petitioner's Prima Facie Case with the SCN1A Mutation Evidence as Rebuttal Evidence

As was found above, petitioner failed to prove a prima facie case without consideration of the SCN1A mutation as rebuttal evidence. Thus, this next exercise is unnecessary, but is done to facilitate any further review by Judge Wheeler.

Relevant to the case at hand, Doe/11 discussed "whether the special master committed legal error by considering evidence of a possible alternative cause . . . in deciding whether [petitioner] established a prima facie case." Doe/11 v. Sec'y of the Dept. of Health & Human Servs., 601 F.3d 1349, 1351, 1356 (Fed. Cir. 2010)

Petitioner argues that evidence of Rachel's gene mutation, the factor unrelated, may not be used to generally rebut petitioner's prima facie case without establishing a "factor unrelated" defense. P Brief at 6-7 (citing Doe/11, 601 F.3d 1349 (Fed. Cir. 2010)). Petitioner narrowly construes the holding in Doe/11 to approve of a special master's evaluation of evidence regarding a factor unrelated only when examining the reliability of a discrete portion in petitioner's case. P Brief at 7 ("In the Doe 11 context this means that the special master considered evidence of SIDS in evaluating whether the petitioners' daughter suffered moderate to severe edema (the lynchpin of the petitioners' causal sequence, and the pathological finding which the petitioners maintain implicated the Hepatitis B vaccine in her death).").

The undersigned disagrees with petitioner's narrow reading of Doe/11. Just as the special master did in Doe/11, the undersigned considered the evidence of Rachel's genetic mutation, "along with other testimony, medical records, and medical literature in evaluating whether [Rachel's] medical theory reflected a 'logical sequence of cause and effect,' as required by prong two of the Althen test." P Brief at 7 (quoting Doe/11, 601 F.3d at 1357). As was the case in

Doe/11, the undersigned acknowledges this genetic evidence may also be relevant to the factor unrelated defense had petitioner proven his prima facie case. Doe/11, 601 F.3d at 1357. However, this evidence may also be used to determine the reliability of petitioner's theory and his "proposed causal sequence of vaccine injury," id., in the presence of the genetic mutation. The Federal Circuit stated, "neither § 300aa-13 nor our cases limit what evidence the special master may consider in deciding whether a prima facie case has been established." Doe/11, 601 F.3d at 1358 (citing Walther, 485 F.3d at 1151). The opinion goes on, "§ 300aa-13(a) requires the special master's findings to be based 'on the record as a whole.'" Doe/11, 601 F.3d at 1358. Ultimately, the Federal Circuit held that "the special master did not commit legal error by considering evidence that [alleged injury] could have been caused by [a factor unrelated to the vaccination]." Id.

Considering the record as a whole, respondent's evidence regarding Rachel's SCN1A gene mutation clearly and convincingly eliminates the vaccine as a causal agent, and thereby prevents petitioner from establishing a prima facie case. Dr. Wiznitzer and Dr. Raymond cogently made the case for respondent that Rachel would have developed SMEI regardless of the vaccination and the initial seizure; and unfortunately for petitioner, Dr. Kinsbourne was ineffective in presenting petitioner's case and rebutting respondent's evidence.[14] Petitioner's expert recognized that SMEI has a "very powerful" genetic component, Hammitt I, at *22, and also agreed that the issue presented in the case is the initial seizure's role in altering the expression of mutation. Id.

As discussed under petitioner's prima facie case, Dr. Kinsbourne "inferred . . . complex febrile seizures, . . . like [Rachel's seizure], were apt to cause brain damage which, of course, would be superimposed on the propensity to a have the seizure disorder that might have been – to have the seizure disorder in some form represented by the SCN1A variant." Hammitt I, 2010 WL 3735705, at *41 (quoting Tr. at 476). As discussed previously, Dr. Kinsbourne was unable to evidence any brain damage or developmental delay following the initial seizure. Supra pp. 5-7.

Drs. Wiznitzer and Raymond rejected petitioner's theory of an inference of brain damage and that the vaccination contributed to Rachel's condition. At one point, Dr. Wiznitzer testified, "You have someone who is destined to develop SMEI, there's no doubt about that. The genetic mutation tells us, this is going to happen. . . . [The vaccine and febrile seizure] did not alter her clinical history. Her clinical history would evolve the same whether she's had a fever that day or some other time." Hammitt I, 2010 WL 3735705, at *13 (citing Tr. at 134-35). As noted several times herein, Dr. Raymond explained, "while complex febrile seizures **can** injure the brain, 'you have to put that in context of these cases **where we have no evidence that the complex febrile seizures actually injure the brain**; that their course was in any, shape or form different than any other individual who [has] Dravet syndrome.'" Hammitt I, 2010 WL 3735705, at *41 (emphasis added in Hammitt I). As will be discussed more fully in the factor unrelated section,[15] Dr.

[14] Petitioner argues that ruling out alternative causes is not his burden, citing Walther v. Sec'y of the Dept. of Health & Human Servs., 485 F.3d 1146 (Fed. Cir. 2007). P Brief at 4-6. Indeed, "the Vaccine Act does not require the petitioner to bear the burden of eliminating alternative causes **where the other evidence on causation is sufficient to establish a prima facie case.**" Walther v. Sec'y of the Dept. of Health & Human Servs., 485 F.3d 1146, 1149-50 (Fed. Cir. 2007)(emphasis added). However, as recognized in Doe/11, petitioner's failure to prove her prima facie case "is different from a requirement that he affirmatively disprove an alternative cause." Doe/11, 601 F.3d at 1358 (citing De Bazan, 539 F.3d at 1353-54).

[15] As was recognized in Doe/11, there appears to be overlap in the evidence being considered as rebuttal evidence to petitioner's prima facie case and respondent's factor unrelated defense. See Doe/11, 601 F.3d at 1357 ("While some of the government's evidence might also have been relevant to a claim of alternative causation (i.e., SIDS) had Doe proved their prima

Raymond, by analyzing the myriad characteristics of Rachel's SCN1A mutation and her clinical course, convincingly testified that the SCN1A mutation was the sole cause of her SMEI. Infra pp. 9-11.

Again, it cannot be overemphasized that Dr. Kinsbourne was simply "unable to adequately address the issues presented in this case, specifically those relating to genetics." Hammitt I, 2010 WL 3735705, at *44. Nor was petitioner's expert able to offer "any cogent explanation for how an environmental trigger, specifically a vaccine, significantly contributed to Rachel's SMEI." Id. At best, Dr. Kinsbourne's testimony, particularly that regarding Rachel's SCN1A mutation, was speculative, and speculative testimony does not equate to preponderant evidence. Id. (citing Hennessey v. Sec'y of the Dept. of Health & Human Servs., 91 Fed. Cl. 126, 133 (Fed. Cl. 2010); Doyle v. Sec'y of the Dept. of Health & Human Servs., 92 Fed. Cl. 1, 2010 WL 1135742, at *8 (Fed. Cl. 2010)("[P]roof of causation entails more than having a well-qualified expert proclaim that the vaccination caused a disease. Mere conclusory opinions-or ones that are nearly so as unaccompanied by elaboration of critical premises-will not suffice as proof of causation, no matter how vaunted or sincere the offeror. See, e.g., Moberly, 592 F.3d 1315, 1324 ("the special master is entitled to require some indicia of reliability to support the assertion of the expert witness")").

Considering the lack of evidence showing the initial seizure damaged her brain, coupled with Rachel's genetic mutation and the persuasive expert evidence regarding her inevitable development of SMEI regardless of the vaccination and initial seizure, there is a glaring gap in petitioner's case concerning the logical sequence of cause and effect, the second prong of Althen. Petitioner failed to prove a prima facie case with preponderant evidence.

Respondent's Evidence of the SCN1A Gene Mutation, A Factor Unrelated to the Vaccine

Finally, the undersigned discusses in an abbreviated fashion respondent's factor unrelated defense. Since the near entirety of Hammitt I was devoted to this topic, a summary discussion is provided herein.

The Federal Circuit in De Bazan, 539 F.3d at 1354, stated, "successfully proving the elements of the Althen test establishes that the medical evidence indicating that the vaccine **may** have caused the petitioner's injury is strong enough to **infer causation-in-fact *absent proof that some other factor was the actual cause."*** Thus, even if petitioner's evidence in this case had been sufficient to gain this **inference** of causation-in-fact, petitioner would still not be entitled to compensation as respondent offered preponderant evidence that the SCN1A gene mutation was the sole cause of and principally responsible for Rachel's condition.

Respondent's expert, Dr. Raymond was "relied upon heavily in deciding this case." Hammitt I, 2010 WL 3735705, at *10. Dr. Raymond is a board certified geneticist and neurologist with a specialty in child neurology. Hammitt I, 2010 WL 3735705, at *9, *44 (internal citations omitted). His testimony was found to be "well explained, cogent, based upon the knowledge and practices of a clinical geneticist, and supported by the medical literature."

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facie case, the special master only considered the evidence of SIDS 'for the limited purpose of evaluating carefully the reliability of the medical underpinnings of petitioners' proposed causal sequence for a vaccine injury.'"). Where the line is drawn between respondent's rebuttal evidence and respondent's factor unrelated defense is currently unclear to the undersigned. What is clear is that Doe/11 sanctions the use of evidence of alternative causes, even alternative causes that do not qualify legally as factors unrelated, as rebuttal evidence. See id. at 1351-52, 1357-58 (discussing SIDS as being oftentimes regarded as an idiopathic condition and thus not qualifying as a factor unrelated under § 300aa-13(a)(1), (2)).

Hammitt I, 2010 WL 3735705, at *44. In contrast, petitioner's expert, Marcel Kinsbourne, was found unreliable and unpersuasive. Hammitt I, 2010 WL 3735705, at *8, *44. Dr. Raymond opined in his written report and testified that Rachel's SCN1A gene mutation was the **sole** cause of her SMEI. R Ex A at 5; Transcript for May 15, 2009, Hearing at 336, 339-40; e.g., Hammitt I, 2010 WL 3735705, at *1, *16, *19, *20. Dr. Raymond's conclusion was based upon a comprehensive explication of the genetic information presented in this case, the medical literature concerning the relationship of the SCN1A gene mutation to SMEI, and Rachel's clinical findings. Based upon his consideration of the totality of information, Dr. Raymond testified, "if he was providing counseling to this family as a geneticist in his clinical practice, 'I would say that this[, the mutation,] is the sole cause of their child's illness.'" Hammitt I, 2010 WL 3735705, at *22.

As summarized in the Hammitt I Decision, the factors Dr. Raymond relied upon were:

- Rachel's mutation arose *de novo*;

- the mutation at issue results in a non-conservative amino acid change with the new amino acid having very different physical properties from what is found at the location in non-affected individuals;

- the mutation affects the beginning of the resultant protein, the N-terminus, a functionally important region, as evidenced by report of only SMEI resulting form mutations in this region;

- the mutation occurs in an area that is well-conserved across species, signaling significant ramifications when altered;

- there are reports evidencing similar or comparable mutations resulting in SMEI in or near the same location as Rachel's mutation; and

- there is an absence of the mutation in the normal population.

Hammitt I, 2010 WL 3735705, at *22, *46. Dr. Raymond emphasized in his testimony, and the undersigned so found, that it was the presence of these cumulative factors and the clinical presentation in this case that convinced him that Rachel's SMEI was **solely** caused by the SCN1A gene mutation. See Hammitt I, 2010 WL 3735705, at *20, *22, *46. Dr. Raymond's testimony regarding the genetic issues went essentially unrebutted. See Hammitt I, 2010 WL 3735705, at *23 ("Nor did petitioner offer the testimony of a geneticist to rebut the testimony of Dr. Raymond.").

As stated previously, Dr. Kinsbourne, petitioner's expert, recognized that SMEI has a "very powerful" genetic component. Hammitt I, 2010 WL 3735705, at *22. Also, as noted in the Hammitt I decision, Dr. Kinsbourne agreed with the undersigned that the issue presented in the case "is the role of [Rachel's] initial seizure, this complex seizure[,] in altering whatever mutation we have." Id. As Dr. Raymond explained, "while complex febrile seizures **can** injure the brain, 'you have to put that in context of these cases **where we have no evidence that the complex febrile seizures actually injure the brain**; that their course was in any, shape or form different than any other individual who [has] Dravet syndrome.'" Id. at *41 (emphasis added in Hammitt I). There was simply no evidence of any role by the vaccination in the development of

her SMEI and Dr. Kinsbourne offered no persuasive testimony to counter this testimony. Id. Again, petitioner failed to rebut the evidence that demonstrates Rachel would have developed SMEI regardless of the vaccination.

Dr. Kinsbourne was "unable to adequately address the issues presented in this case, specifically those relating to genetics." Hammitt I, 2010 WL 3735705, at *44. Nor was petitioner's expert able to offer "any cogent explanation for how . . . a vaccine significantly contributed to Rachel's SMEI." Id. At best, Dr. Kinsbourne's testimony, particularly that regarding Rachel's SCN1A mutation, was speculative and speculative testimony does not equate to preponderant evidence. Id. (citing Hennessey v. Sec'y of the Dept. of Health & Human Servs., 91 Fed. Cl. 126, 133 (Fed. Cl. 2010)). Petitioner did not "offer the testimony of a geneticist to rebut the testimony of Dr. Raymond. Rather, petitioner presented a series of arguments intended to undermine Dr. Raymond's conclusion." Hammitt I, 2010 WL 3735705, at *23. Those arguments were disposed of in Hammitt I, 2010 WL 3735705, at *22-43.

Based upon Dr. Raymond's expertise and vastly superior testimony, Dr. Kinsbourne's unfortunately very weak testimony, the presence of genetic factors that when considered cumulatively by a geneticist enable the geneticist to opine to a genetic cause, Hammitt I, 2010 WL 3735705, at *20, and the absence of evidence that the complex febrile seizure actually injured the brain, id. at *41, the undersigned is convinced beyond any doubt that respondent proved by a preponderance of the evidence that Rachel's SCN1A gene mutation was the sole, substantial cause, principally responsible for her SMEI.

Conclusion

Based upon the entire record, the undersigned finds petitioner failed to prove his prima facie case; further, even if petitioner established a prima facie case, respondent proved by a preponderance of the evidence that the genetic mutation was the sole cause, principally respondent for Rachel's SMEI. Petitioner is denied compensation. The Clerk of the Court is directed to enter judgment accordingly.

IT IS SO ORDERED.[16]

s/ Gary J. Golkiewicz
Gary J. Golkiewicz
Special Master

[16] This document constitutes a final "decision" in this case. Vaccine Rule 28.1. Unless a motion for review of this decision is filed within 30 days, the Clerk of the Court shall enter judgment in accord with this decision. Id.