# In the United States Court of Federal Claims
## OFFICE OF SPECIAL MASTERS

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| BERNADETTE SKORUPSKA, mother and natural guardian of N.S., a minor, | * | |
| | * | No. 16-1517V |
| | * | Special Master Christian J. Moran |
| Petitioner, | * | |
| v. | * | |
| | * | Filed: February 10, 2026 |
| SECRETARY OF HEALTH AND HUMAN SERVICES, | * | |
| Respondent. | * | |
| * * * * * * * * * * * * * * * * * * * * * * | | |

Phyllis Widman, Widman Law Firm LLC, Linwood, N.J., for petitioner;
Ryan D. Pyles, United States Dep't of Justice, Washington, D.C., for respondent.

## DECISION DENYING ENTITLEMENT TO COMPENSATION[1]

Bernadette Skorupska, on behalf of her minor son, N.S., claims that after receiving the Haemophilus influenzae type b ("Hib") vaccine on November 16, 2013, N.S. suffered infantile spasms and related seizures and neurodevelopmental impairments, as well as a significant aggravation of his "developmental problems characterized by motor difficulties and hypotonia." She seeks compensation pursuant to the National Childhood Vaccine Injury Act, 42 U.S.C. § 300aa–10 through 34. Ms. Skorupska supported her claim with reports from three experts: a rheumatologist, Dr. Brawer; a neurologist, Dr. Ghacibeh; and a geneticist and pediatric neurologist, Dr. Huq. She also argued her position through three briefs.

[1] The E-Government Act, 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services), requires that the Court post this decision on its website. This posting will make the decision available to anyone with the internet. Pursuant to Vaccine Rule 18(b), the parties have 14 days to file a motion proposing redaction of medical information or other information described in 42 U.S.C. § 300aa-12(d)(4). Any redactions ordered by the special master will appear in the document posted on the website.

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The Secretary maintains that Ms. Skorupska is not entitled to compensation. The Secretary relied upon opinions expressed by an expert in genetic medicine and neurology he had retained, Dr. Raymond. The Secretary submitted a single brief regarding entitlement.

Ms. Skorupska is not entitled to compensation. The main flaw in her case is that she failed to present persuasive evidence that N.S. responded to the vaccine in a way consistent with her expert's theory. Ms. Skorupska has not shown with preponderant evidence that the Hib vaccine harmed N.S. Moreover, N.S. was born with a genetic mutation that made his neurologic problems nearly inevitable.

## I. Facts

### A. Birth and Pre-Vaccination History

#### 1. Medical Records

N.S. was born on March 7, 2013. Exhibit 28 at 20. He did not display any problems as a neonate. Id.

However, at the time of conception, N.S. had a mutation in a gene, known as an SCN2A gene. Exhibit 13. This mutation was not detected until about sixteen months after he was born.

On September 12, 2013, when he was about six months old, before the genetic mutation was found, N.S. underwent a physical therapy evaluation "due to concerns regarding his gross motor skills and development." Exhibit 39 at 13. N.S. was found to have delays in gross motor development, and was "functioning at -2.00 standard deviations below the mean than that of his peers." Id. at 15-16. Dr. Selina Cali recommended that he should have an occupational therapy evaluation. Id. at 16.

The following day, N.S. was evaluated by occupational therapist Melissa Gianquinot using the Developmental Assessment of Young Children-Second Edition (DAYC-2), informal observation/clinical opinion, and parent interview. Exhibit 39 at 7. N.S. scored above average in the expressive language domain; average in the communication and adaptive behavior domains; below average in the cognitive, fine motor, and social-emotional domains; and poor in the physical development domain. Id. at 9-10. Ms. Skorupska emphasizes that this evaluation did not mention seizures or epilepsy. Pet'r's Br. at 10.

During N.S.'s six-month well-child examination, his pediatrician found that he was generally normal. However, he had not yet achieved the milestones of looking for a dropped item or feeding himself. Exhibit 24 at 19-21 (Sep. 25, 2013).

N.S. was seen by another occupational therapist, Elizabeth Esposito, on September 27, 2013. This therapist stated that N.S. "has not yet met any of his developmental milestones." Examples of the abilities typical for a six-month-old include: rolling over, sitting up, reaching and grasping toys and tracking toys consistently to and past midline. N.S. "made no eye contact and at times did not seem aware of others in his environment." Exhibit 39 at 20.

N.S. again saw his pediatrician on October 19, 2013. He did not make eye contact, he did not follow a toy, he was not sitting, and he was not rolling over. Exhibit 24 at 16. The doctor, Joanna B. Lis, assessed him as having hypotonia and an unspecified delay in development. Id. at 17.

In early November 2013, N.S. had two episodes of nonstop crying. See Exhibit 7 at 20-22 (emergency room records from November 1, 2013); Exhibit 24 at 132 (pediatrician's follow up on November 6, 2013).

A pediatric neurologist, Steven Schwartzberg, evaluated N.S. on November 13, 2013. Exhibit 2 at 4-5. N.S. had not yet rolled over. Dr. Schwartzberg determined that N.S. had diminished tone in all four extremities. He assessed N.S. as having "diffuse hypotonia and developmental delay predominantly affecting motor skills." Id. at 5. Dr. Schwartzberg recommended that N.S. increase his therapies. Dr. Schwartzberg also ordered an EEG and a brain MRI.

2. Expert Commentaries on N.S.'s Development before Vaccination

The Secretary's expert, Dr. Raymond, highlighted problems with N.S.'s September 13, 2013 evaluation, commenting:

> The major issue with this evaluation is that it is unclear what was scored from observation and what was credited by parental report. This becomes even more appreciable in the discrepancy between what the evaluations found in terms of gross motor and fine motor where one can only be scored for actions performed. On the DAYC-2, NS was only -1.13 SD below the mean, but when actually

> assessed during the Peabody, he was -2.40 SD below the mean and profoundly delayed in that assessment.

Exhibit EE at 1. Dr. Raymond further opined:

> This is to some extent immaterial because when seen by pediatric neurologist Dr. Schwartzberg on November 13, 2013, at 8 months and prior to any immunization, [N.S.] was clearly severely delayed in all domains.

Id. Dr. Raymond opined that N.S. "was severely developmental[ly] delayed prior to the immunization." Exhibit A at 6. He explained:

> It is well-established that NS was hypotonic and not meeting developmental milestones, so it is apparent that the genetic disorder that he had was already affecting his brain and had been doing so since birth. This is concordant with what has been seen in other children affected by early infantile epileptic encephalopathies due to mutations in SCN2A.

Id.

Dr. Ghacibeh acknowledged that it is "evident and unquestionable" that N.S. "was already exhibiting symptoms related to his genetic mutation" before vaccination. Exhibit 50 at 2. However, he characterized N.S. as "showing signs of mild developmental delay affecting primarily his motor function, most likely due to his genetic mutation." It was not until after vaccination, in Dr. Ghacibeh's analysis, that N.S. "developed infantile spasms," and "had a severe developmental regression which left him with profound intellectual disability and global developmental delay." Id. Dr. Ghacibeh summarized:

> To reiterate, there is no doubt that [N.S.]'s underlying genetic mutation was the cause of his early delay. However, as we see from the referenced literature, mutations on the SCN2A gene can result in a spectrum of disorder, ranging from the mild and benign to the severe. While it is impossible to know for sure what would have been [N.S.]'s ultimate phenotype as related to his mutation, the course of his development up to the day he had received the vaccine was taking a fairly favorable

> trajectory, which suggests that he would have most probably had mild delay. However, after receiving the HiB vaccine, he developed refractory epilepsy and severe developmental regression.

Id. at 3.

Dr. Raymond, in contrast, opined that N.S. was "not mildly delayed in one domain, but rather globally delayed prior to seizure onset." Exhibit C at 1. Dr. Raymond noted that, when N.S. was eight months old, he was "at best meeting two-month milestones" across various domains. Id. In summary, Dr. Raymond stated that N.S. was "a child with a severe, generalized epilepsy with developmental delays," and that his genetic mutation "was the sole cause" or his condition and "was not caused or exacerbated by the vaccination that he received." Id. at 3.

Dr. Ghacibeh did "not agree" with Dr. Raymond's conclusion that N.S. had severe delay prior to vaccination. Exhibit 59 at 2. Maintaining that N.S. "likely suffered mild motor delay at that time," Dr. Ghacibeh highlighted that neither Dr. Schwartzberg's examination nor impression indicated that N.S. had severe delay. Id. Dr. Ghacibeh maintained this position in his next report, stating that, pre-vaccination, N.S. "had exhibited mild delay in motor development but normal cognitive development." Exhibit 212 at 1. He reiterated that Dr. Schwartzberg's "initial evaluation clearly reports that [N.S.]'s cognitive development seemed on track, and he only exhibited mild motor delay which was to be addressed [with] physical therapy." Dr. Ghacibeh again opined that N.S. "likely suffered mild motor delay" and then "had a severe regression after the onset of his epilepsy." Id.

Dr. Raymond maintained his position and "continue[d] to strongly disagree with Dr. Ghacibeh's assessment." Exhibit K at 3. He raised the point that Dr. Schwartzberg recommended that N.S. increase his therapies from once a week to three times per week; gave the family instructions for hearing, feeding, and special instructional evaluations; and planned for numerous tests and consultations. Id. Dr. Raymond stated: "These recommendations including the significant increase in the frequency of therapy are commensurate with the severity of this child's delays and not those of a 'mild motor delay.'" Id. at 4.

When Ms. Skorupska retained Dr. Huq, he took a position similar to that of Dr. Ghacibeh. Dr. Huq noted "concerns of mild developmental delay prior to the Hib vaccination." Exhibit 68 at 4. He later described N.S. as "a healthy child with

some developmental concerns mainly in the motor domain, but he was not severely delayed." Id. at 27.

Just as he disagreed with Dr. Ghacibeh, Dr. Raymond disagreed with Dr. Huq's characterization of N.S. as "not severely delayed." Dr. Raymond reiterated his previous point that, at eight months of age, N.S. was "at best" meeting two-month milestones, which he described as "a significant delay." Exhibit T at 12. He also again noted the treatments and recommendations N.S. received from Dr. Schwartzberg. Id.

### B. Vaccination and Next Two Weeks

N.S. returned to his pediatrician on November 16, 2013. He was assessed with a "developmental coordination disorder." Exhibit 24 at 4. As part of this appointment, N.S. received the allegedly harmful Hib vaccine. Exhibit 1 at 4-5.[2]

On November 22, 2013 (six days after vaccination), N.S. was taken to the emergency department at Staten Island University Hospital. The history, which was given by N.S.'s mother and father, states that N.S. had been vomiting after every meal and having intermittent episodes of choking for the past three days. Exhibit 29 at 30. N.S. was also noted to be "afebrile." Id. at 138.[3] The attending note further stated that N.S. had "been receiving extensive outpatient evaluation for chronic vomiting and lethargy," and had been "diagnosed with reflux and multiple formula changes due to formula intolerance." Id. at 31. The doctor in the emergency department consulted N.S.'s pediatrician, who reported that N.S. "has been not developing appropriately and was feeding poorly." Id. at 36. A document associated with his admission states that N.S. has a two-day history of

[2] "Severe adverse events following administration of Hib vaccine are uncommon, making it one of the safest vaccines currently available. In a study of >4000 infants, there were no differences in the type and frequency of severe adverse events occurring among those receiving Hib conjugate vaccine and those receiving a placebo." Exhibit F (World Health Organization, Information Sheet: Observed Rate of Vaccine Reactions: Haemophilus Influenzae Type B (Hib) Vaccine (2012)) at 1.

[3] Ms. Skorupska asserts that N.S. started suffering from "infantile spasm type seizures accompanied by fever." Pet'r's Br. at 2. The Secretary disputed this assertion. Resp't's Br. at 33. Ms. Skorupska did not counter the Secretary's arguments. See Pet'r's Reply. In the hospital, Ms. Skorupska denied fevers. Exhibit 29 at 149 (Nov. 22, 2013).

spasms. Id. at 94-95. Pursuant to this history, N.S. started having spasms on November 20, 2013 (or four days after vaccination).

In the emergency department, a doctor ordered, among other tests, a complete blood count. Although the number of lymphocytes were reported as higher than expected (Exhibit 29 at 173), a treating doctor said the CBC was normal. Id. at 168.

As N.S. was stable and appeared well-hydrated, the doctors determined that it would be appropriate to discharge N.S. and to have him follow up for an outpatient GI workup. Exhibit 29 at 36. However, at the request of his mother, N.S. was admitted to the hospital. Id. An initial inpatient history reported that N.S. had "global delay." Id. at 95. The doctor also checked the boxes on the form to indicate that N.S. had deficits in "Gross Motor," "Fine Motor," and "Speech." Id. at 97. N.S. underwent a video EEG. See id. at 150 (report from neurology / epilepsy attending), 167 (ordering form). He was diagnosed with spasms and epileptic encephalopathy. Exhibit 38 at 5. A doctor ordered treatment with adrenocorticotropic hormone (ACTH). Exhibit 29 at 153. A brain MRI was normal. Exhibit 29 at 168-69, Exhibit 7 at 47 (Nov. 26, 2013). N.S. was discharged on November 27, 2013. Exhibit 29 at 47. Instructions included following up with genetics. Id. at 280.

### C. Treatment and Genetic Testing

Following the diagnosis of infantile spasms, N.S. saw a variety of doctors, who recorded his status and prescribed a variety of medications. Medications included ACTH, topiramate (Topamax), and vigabatrin (Sabril). Because these records are generally not relevant to determine what caused the infantile spasms, these records are not detailed in this decision. For details, see Resp't's Br. at 6-7. (Ms. Skorupska's brief does not cite any medical records from this time.)

In short, in December 2013 and January 2014, N.S.'s doctors struggled to get control of his seizures. See Exhibit 5 at 5 (December 12, 2013 visit during which a neurologist added topiramate); Exhibit 15 at 1-2 (December 18, 2013 visit during which a different neurologist (Dr. Wolf) added Sabril).

After these medications failed to control the spasms, Dr. Wolf recommended IVIG. Exhibit 30 at 836 (Apr. 18, 2024). In May 2014, Dr. Wolf oversaw a course of IVIG, which was administered in Beth Israel Medical Center. Exhibit 38 at 3-5; Exhibit 30 at 800-01 (duplicate). Initially, Dr. Wolf stated the clusters of spasms had "no improvement." Exhibit 38 at 5. In a follow up appointment, Dr. Wolf

reported that after the IVIG, there was a "great result of no spasms for 1.5 week only then they returned at 1 cluster per day. But then more recently." Exhibit 15 at 26.

The results of the genetic testing were returned on July 11, 2014. Exhibit 13. There was a mutation in the SCN2A gene. "The SCN1A and SCN2A genes encode α subunits of the neuronal voltage-gated sodium channel." Exhibit 193 (Dr. Huq's second report) at 6. "Sodium channels are extremely important in neuronal transmission and interneuronal communication and their role spans the spectrum of early neuronal development as well as ongoing brain function in adults. Disorders in sodium channels, referred to as channelopathies, produce a spectrum of clinical disorders, ranging from asymptomatic carries to severe epilepsy and developmental delays." Exhibit 50 (Dr. Ghacibeh's first report) at 2.

In short, N.S.'s SCN2A contained a variant that Gene Dx classified as a "disease-causing mutation." Exhibit 13 at 1. More specifically, in position 3631, the amino acid was changed from guanine to adenine. This change in amino acid meant in position 1211 there was glutamic acid, rather than lysine. Exhibit A (Dr. Raymond's report) at 4.

After the genetic test was returned, N.S. was seen by a clinical geneticist, Ethylin Wang Jabs, on July 23, 2014. Exhibit 23 at 25. Dr. Jabs documented that the infantile epilepsy panel "was significant for a heterozygous deleterious variation in the SCN2A gene. . . . This has been reported in other patient (Ogiwara et al., 2009) with infantile spasms that progressed to generalized epilepsy." Id. Dr. Jabs's impression was that N.S. has "infantile spasms due to a known pathogenic variant in the *SCN2A* gene." Id. at 25. To support the "hypothesis" that this mutation is a "deleterious change," Dr. Jabs stated that the gene is "well-conserved evolutionarily and is predicted to be deleterious from programs that analyze protein function after mutations. It was also tested in vitro in the same paper that described the patient with this mutation and was found to have increased activity and reduced latent state." Id.

The view of Dr. Jabs---that the genetic mutation was pathogenic---was at least repeated by one other doctor. In the context of a visit to an emergency department for lethargy in January 2015, the doctor wrote that N.S. is a "22 mo boy with PMHx [past medical history] for significant SCN2A mutation and resulting seizure disorder and developmental delay." Exhibit 30 at 389 (emphasis added).

As discussed extensively below, the parties differ about the role this variant played in N.S.'s life and plays in this litigation. "Petitioner's position is that the variant SCN2A gene is not enough to cause [N.S.'s] current mental state. While he may have been at an increased risk to develop some deficits due to the genetic mutation, the vaccination (not simply the genetic mutation) is what caused his current mental state." Pet'r's Br. at 4. In contrast, the Secretary argues: "It is beyond question that N.S.'s mutation explains his entire course." Resp't's Br. at 16.

## II. Procedural History

Represented by Attorney Robert Krakow, Ms. Skorupska initiated this case by filing a petition November 15, 2016. Over the next year, Ms. Skorupska submitted various medical records. She filed an affidavit on September 18, 2017. Exhibit 40.

The Secretary reviewed this material and determined that Ms. Skorupska was not entitled to compensation. Resp't's Rep., filed pursuant to Vaccine Rule 4 on Dec. 4, 2017.

Ms. Skorupska was instructed to file a report from an expert. Order, issued Dec. 13, 2017 (setting a deadline of March 12, 2018). For a variety of reasons, an expert report was not filed. Ms. Skorupska was ordered to show cause why the case should continue. Order, issued Apr. 30, 2019. It appeared that Ms. Skorupska would wrap up her case. See Pet'r's Status Rep., filed May 24, 2019.

However, Ms. Skorupska elected to maintain her case. Mr. Krakow was allowed to withdraw his representation via an order issued August 7, 2019, and a December 17, 2019 decision awarded Ms. Skorupska compensation for Mr. Krakow's work.

Representing herself, Ms. Skorupska submitted a report from Arthur E. Brawer, a rheumatologist who has sometimes supported claims that a vaccine injured a person. Dr. Brawer's report is Exhibit 42.

In response to Dr. Brawer, the Secretary submitted a response from Gerald Raymond, who is a medical geneticist and neurologist. Dr. Raymond generally opined that "The pathogenic mutation in [N.S.'s] SCN2A gene is the sole cause of his epileptic encephalopathy and was not caused or exacerbated by the immunization that he received." Exhibit A at 7.

Ms. Skorupska, still appearing pro se, submitted a responsive report from Dr. Brawer on August 5, 2020. Exhibit 46.

It appeared that this second report from Dr. Brawer completed the disclosure of opinions from experts. Thus, the parties were directed to advocate through briefs. Order, issued Aug. 24, 2020.

Attorney Phyllis Widman became counsel of record on September 28, 2020. In some ways, Ms. Widman's participation re-started the case.

A status conference was held on December 3, 2020. The Secretary asserted that Dr. Brawer is not qualified to discuss the causes of a neurologic condition. In this context, Ms. Widman requested an opportunity to seek a report from a neurologist. See Order, issued Dec. 3, 2020. This December 3, 2020 order suspended the schedule for submitting briefs.

After receiving enlargements of time, Ms. Skorupska submitted a three-page report from Georges Ghacibeh. Exhibit 50. Dr. Ghacibeh is a neurologist. He opined: "While it is impossible to know for sure what would have been [N.S.'s] ultimate phenotype as related to his mutation, the course of his development up to the day he had received the vaccine was taking a fairly favorable trajectory, which suggests that he would have most probably had mild delay. However, after receiving the HiB vaccine, he developed refractory epilepsy and severe developmental regression." Exhibit 50 at 3.

The Secretary again responded with a report from Dr. Raymond. Exhibit C. Another round of reports followed. See Exhibit 59 (Dr. Ghacibeh's second report) and Exhibit K (Dr. Raymond's third report). In this third report, Dr. Raymond stated that N.S.'s variant has been reported in children with epilepsy seven times.

Ms. Skorupska stated that additional reports were not needed. Pet'r's Status Rep., filed Dec. 16, 2022. Thus, the parties were informed that an order for briefs would be forthcoming. Order, issued Dec. 19, 2022.

Ms. Widman requested a status conference, which was held on January 12, 2023. She now realized that she should strengthen Ms. Skorupska's case by adding a report from a geneticist. The Secretary ultimately deferred to the special master's discretion, but maintained that inescapable science shows that the mutation is pathogenic. Thus, incurring additional costs would not be justified. However, Ms. Skorupska was given an opportunity to present the report of a geneticist. Order, issued Jan. 16, 2023.

Ms. Skorupska submitted a report from Mahbubul Huq on March 20, 2023. Dr. Huq is a board-certified clinical geneticist and pediatric neurologist. His approximately 30-page report cites approximately 90 articles. Exhibit 68.

Dr. Huq's report kicked off another series of reports. See Exhibit T (Dr. Raymond's fourth report); Exhibit 193 (Dr. Huq's second report); Exhibit EE (Dr. Raymond's fifth report); and Exhibit 212 (Dr. Ghacibeh's third report). The Secretary then advised that additional reports were not necessary. Resp't's Status Rep., filed Feb. 2, 2024.

A status conference was held on February 27, 2024. The parties were directed to file briefs as explained in the August 24, 2020 order.

For a variety of reasons, Ms. Skorupska did not submit a brief quickly. Eventually, Ms. Skorupska argued that she was entitled to compensation. Pet'r's Br., filed Sep. 30, 2024. Shortly before the brief was filed, Ms. Skorupska revised her petition. Am. Pet., filed Sep. 16, 2024. The amended petition alleges that the Hib vaccine caused N.S. to suffer a variety of conditions, including infantile spasms. The amended petition alternatively alleges that the Hib vaccine significantly aggravated a pre-existing condition.

Ms. Skorupska's September 30, 2024 brief was deficient in that it failed to address one of the elements of compensation (timing). Thus, she was directed to present a supplement. Order, issued Oct. 7, 2024. She did so. Pet'r's Supp'l Br., filed Oct. 15, 2024.

The Secretary argued that Ms. Skorupska was not entitled to compensation and maintained the case could be resolved without a hearing. Resp't's Br., filed Dec. 2, 2024.

Ms. Skorupska attempted to answer the Secretary's arguments in a relatively short reply, which was filed on February 4, 2025. Upon review of this material one question came up about a citation to evidence in the reply brief. Ms. Skorupska provided the material in a status report, filed Sep. 17, 2025.[4] The case is ready for adjudication.

[4] The September 17, 2025 status report remains slightly confusing. Although it cites to Exhibit 148, the medical records are actually contained within Exhibit 227.

The case can be resolved without a hearing. Special masters possess discretion to decide whether an evidentiary hearing will be held. 42 U.S.C. § 300aa-12(d)(3)(B)(v) (promulgated as Vaccine Rule 8(c) & (d)), which was cited by the Federal Circuit in Kreizenbeck v. Sec'y of Health & Hum. Servs., 945 F.3d 1362, 1365 (Fed. Cir. 2018). Here, a hearing is not required because Ms. Skorupska has had a full and fair opportunity to present her case.

## III. Standards for Adjudication

A petitioner is required to establish her case by a preponderance of the evidence. 42 U.S.C. § 300aa–13(1)(a). The preponderance of the evidence standard requires a "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 v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010) (citations omitted). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991).

Distinguishing between "preponderant evidence" and "medical certainty" is important because a special master should not impose an evidentiary burden that is too high. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379-80 (Fed. Cir. 2009) (reversing special master's decision that petitioners were not entitled to compensation); see also Lampe v. Sec'y of Health & Hum. Servs., 219 F.3d 1357 (Fed. Cir. 2000); Hodges v. Sec'y of Health & Hum. Servs., 9 F.3d 958, 961 (Fed. Cir. 1993) (disagreeing with dissenting judge's contention that the special master confused preponderance of the evidence with medical certainty).

## IV. Diagnosis

In Broekelschen v. Sec'y of Health and Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010), the Federal Circuit recognized that in some circumstances, the special master may "first determine which injury was best supported by the evidence in the record before applying the Althen test." Here, based upon Dr. Raymond's third report, the Secretary asserts that N.S. suffers from "developmental and epileptic encephalopathy 11 (DEE11)." Resp't's Br. at 15, citing Exhibit K at 5.

Dr. Huq enjoyed an opportunity to address Dr. Raymond's opinion regarding diagnosis. Under the heading "NS's Diagnosis and Genetic Test Results," Dr. Huq stated, "NS developed epileptic spasms." Exhibit 68 at 4. Later, Dr. Huq wrote, "NS developed infantile spasms." Id. at 27. However, Dr. Huq did

not respond to the proposal that the appropriate diagnosis is "developmental and epileptic encephalopathy 11." Likewise, although Ms. Skorupska enjoyed an opportunity to address the Secretary's argument regarding DEE11, she did not. See Pet'r's Reply.

Nevertheless, for the ease of analysis, this decision will proceed upon the assumption that an appropriate diagnosis for N.S. is infantile spasms. This assumption is made because, in part, it is not readily apparent that any difference in nomenclature would change the analysis. See Exhibit T (Dr. Raymond's report in response to Dr. Huq's first report) at 12 and 15 (apparently accepting the diagnosis of infantile spasms).

## V. Part One: Loving Analysis

Ms. Skorupska's September 16, 2024 Amended Petition alleges both an initial onset claim and a significant aggravation claim. In such a situation, the test is whether the vaccinee manifested problems before the vaccination. Paluck v. Sec'y of Health & Hum. Servs., 104 Fed. Cl. 457, 468 (2012), aff after remand on non-relevant grounds, 786 F.3d 1373 (Fed. Cir. 2015); see also Lampe v. Sec'y of Health & Hum. Servs., 219 F.3d 1357, 1364-65 (Fed. Cir. 2000) (ruling that the special master did not err in reasoning that because the child-vaccinee's seizure disorder started after the second dose of the diphtheria-tetanus-pertussis vaccine, the petitioners could proceed only on a theory that the third dose significantly aggravated the seizure disorder); Childs v. Sec'y of Health & Hum. Servs., 33 Fed. Cl. 556, 559-60 (1995) (ruling that when the vaccinee did not have a history of a problem before the vaccination, petitioners could not proceed on a theory of significant aggravation). Here, ample evidence shows that N.S. was experiencing at least some neurologic problems / developmental delay before the vaccination. Exhibit 2 at 4-5 (Dr. Schwartzberg's Nov. 13, 2013 evaluation). Although Ms. Skorupska's experts (Dr. Ghacibeh and Dr. Huq) assert N.S. was only mildly delayed, even from their perspective he *was* delayed pre-vaccination. Thus, it is appropriate to categorize Ms. Skorupska's case as presenting a significant aggravation claim.

As confirmed in W.C. v. Sec'y of Health & Hum. Servs., 704 F.3d 1352, 1357 (Fed. Cir. 2013), the elements of an off-Table significant aggravation case were stated in Loving. There, the Court blended the test from Althen v. Sec'y of Health & Hum. Servs., 418 F.3d 1274, 1279 (Fed. Cir. 2005), which defines off-Table causation cases, with a test from Whitecotton v. Sec'y of Health & Hum.

Servs., 81 F.3d 1099, 1107 (Fed. Cir. 1996), which concerns on-Table significant aggravation cases. The resulting test has six components. These are:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also pertinent), (3) whether the person's current condition constitutes a "significant aggravation" of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144.

In addressing a significant aggravation claim, special masters may focus upon the last three elements in the Loving test, which correspond to the three prongs of the well-established Althen test. Vinesar v. Sec'y of Health & Hum. Servs., No. 18-440V, 2023 WL 5427935, at *28 (Fed. Cl. Spec. Mstr. July 28, 2023), mot. for rev. denied, 170 Fed. Cl. 681 (2024), aff'd in non-precedential op., No. 2024-1787, 2025 WL 2945665 (Fed. Cir. Oct. 17, 2025); Hennessey v. Sec'y of Health & Hum. Servs., No. 01-190V, 2009 WL 1709053, at *42 (Fed. Cl. Spec. Mstr. May 29, 2009), mot. for rev. denied, 91 Fed. Cl. 126 (2010).

Ms. Skorupska states that "Dr. Huq's theory is that the vaccine caused inflammation and epileptic encephalopathy. The inflammation caused altered connectivity, which in turn, caused epilepsy." Pet'r's Br. at 57; accord Pet'r's Reply at 4. Ms. Skorupska's assertion that Dr. Huq's theory is based upon inflammation is accurate. See Exhibit 68 at 73 (asserting that inflammation causes infantile spasms). The mechanism of inflammation is through cytokines. Pet'r's Br. at 6-7, citing Exhibit 68 at 11-12. The Secretary, too, discussed inflammation, challenging the theory. Thus, whether N.S. experienced inflammation is critical to petitioner's claim, as even if she were to prevail on the fourth Loving / first Althen prong, she would have to establish that the theory applies to N.S.'s case. To address this issue, this Decision evaluates the Loving / Althen prongs out of order, beginning with the fifth Loving prong / second Althen prong.

### A. Loving Prong 5 / Althen Prong 2

In evaluating the second Althen prong, which corresponds to the fifth Loving prong, special masters may consider whether the vaccinee responds in a way predicted by the expert's theory. Hibbard v. Sec'y of Health & Hum. Servs., 698 F.3d 1355, 1364 (Fed. Cir. 2012); La Londe v. Sec'y of Health & Hum. Servs., 110 Fed. Cl. 184, 205 (2013), aff'd, 746 F.3d 1334 (Fed. Cir. 2014). Special masters may also consider the views of treating doctors. Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1326 (Fed. Cir. 2006). These two aspects are considered below.[5]

#### 1. Response Predicted by the Theory

As stated above, Ms. Skorupska's theory is based upon inflammation. She asserts that various blood tests "can be associated with inflammation." Pet'r's Br. at 6.[6] Later, Ms. Skorupska similarly argues that the evidence of inflammation buttresses Dr. Huq's theory:

> Dr. Huq supports this theory of significant aggravation based on the evidence of inflammation in N.S.'s body post-vaccination. "One reason to think that [N.S.'s] vaccination caused significant aggravation of effects of the E122K variant is the evidence of interaction between vaccine induced inflammation and SCN2A gene." Exhibit 193 at 5.

Pet'r's Br. at 57.

The Secretary contends that the evidence does not support a finding that N.S. experienced harmful inflammation. See Resp't's Br. at 31. After quoting the passage from page 57 of Ms. Skorupska's brief, the Secretary asks: "Respectfully, what evidence?" Resp't's Br. at 31. Citing Dr. Raymond, the Secretary argues that there is "no indication of inflammatory markers or MRI features in this case to

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[5] Arguably, another consideration in any analysis of Althen prong two / Loving prong five could include an examination of the presence (or absence) of alternative causes. However, the alternative cause analysis is deferred to section VI., below.

[6] For the underlying blood tests, Ms. Skorupska cited Exhibit 148 at 172-73. However, informal communications revealed that the correct citation is Exhibit 227 at 550-51 (results from Nov. 22, 2013).

suggest an inflammatory event or autoimmune injury, whether vaccine-related or not." Id.

Ms. Skorupska answers that her primary brief "provides extensive evidence that inflammation in N.S.'s body became evident following the Hib vaccine administration." Pet'r's Reply at 5.[7] She brings forward an argument based upon N.S.'s positive response to IVIG. Id. at 5-6.

Ms. Skorupska has not persuasively established that N.S. suffered deleterious inflammation within a relevant time of the vaccination. To start with the last point first, IVIG is a complicated issue. Although Dr. Ghacibeh maintained that IVIG stopped the seizures (see Exhibit 59 at 2), Dr. Raymond raised several challenges, including the fact that N.S. was receiving other therapies at the same time. Exhibit K at 3. Moreover, the improvement after IVIG did not last very long. See Exhibit 15 at 26. Thus, Ms. Skorupska is oversimplifying the question.

While Dr. Ghacibeh's opinion regarding IVIG is not persuasive, Ms. Skorupska does have some minimal expert support for an argument based upon IVIG. The same cannot be said for her reliance on blood tests. In Dr. Raymond's second report, he stated that there was no evidence of inflammation. Exhibit C at 3. Ms. Skorupska's experts did not refute this point. For example, Dr. Ghacibeh reasoned the absence of inflammatory markers does not mean that N.S. did not experience inflammation. Exhibit 59 at 2. The problem with such reasoning, as the Secretary points out (Resp't's Br. at 31), is that ultimately Ms. Skorupska bears a burden of proving her case.

With respect to the question of inflammation, the material that Ms. Skorupska provided in her September 17, 2025 status report was reviewed. This material does not support a finding that N.S. had information. Ms. Skorupska attached four pages:

[7] Given that Ms. Skorupska recognizes in the same paragraph of her reply that inflammatory markers were absent, her characterization of her evidence regarding inflammation as "extensive" appears to be an attorney's rhetorical flourish.

| Evidence Cited to Establish Inflammation | | | |
|---|---|---|---|
| Pg in SR | Pet'r's Cite in Ex. 148 | Actual Cite in Ex. 227 | Comment |
| 1 | 109 | 464 | Intake form. Chief complaint includes "involuntary muscle movements." Inflammation is not mentioned. |
| 2 | 172 | 550 | Blood tests from Nov. 26, 2013. Abnormally high values include bun to creatinine ratio, ammonia, and white blood cells. Ms. Skorupska has not cited any evidence in which a medical professional interpreted these results as consistent with inflammation. |
| 3 | 173 | 551 | Blood tests from Nov. 22, 2013. Abnormally high values include mean platelet value (slightly high), percentage of lymphocytes, number of lymphocytes, and number of lymphocytes. Ms. Skorupska has not cited any evidence in which a medical professional interpreted these results as consistent with inflammation. |
| 4 | 298 | 443 | Part of medical record from N.S.'s visit to the emergency room on Nov. 1, 2013. Medical professionals did not diagnose N.S. as suffering from inflammation. |

Moreover, after N.S.'s blood tests from November 2013 were returned, N.S.'s treating doctors did not diagnose him with or treat him for inflammation. Similarly, neither Dr. Ghacibeh nor Dr. Huq pointed to the results of blood tests as evidence supporting an assertion that N.S. suffered inflammation. The omission of this type of opinion combined with Dr. Raymond's opinion that there is no evidence that N.S. suffered inflammation is a persuasive reason for finding that N.S. did not experience inflammation.

2. Treating Doctors

The parties were directed to identify doctors who commented upon whether the Hib vaccine could have contributed to N.S.'s medical condition. Order for Briefs, issued Aug. 24, 2020, at 6-7. They were expected to cite relevant evidence by exhibit number and page number.

The parties say relatively little about treating doctors. The relevant portion of Ms. Skorupska's brief does not discuss any statements of treating doctors. See Pet'r's Br. at 59-62 (discussing Dr. Huq's, Dr. Ghacibeh's, and Dr. Brawer's opinions about the logical sequence of cause and effect).

The Secretary asserts that the treating doctors do not assist Ms. Skorupska. The Secretary argues: "no treating physician attributed N.S.'s condition in general, or spasms and later seizures more specifically, to vaccination. Indeed, the treating physician statements heavily weigh against causation in this case, because they did, on aggregate, attribute N.S.'s condition – without reservation – to his SCN2A mutation." Resp't's Br. at 32.[8] Strong support for the Secretary's opinion comes from the July 23, 20214 report from the clinical geneticist, Dr. Jabs. She wrote that she counseled N.S.'s parents that the genetic mutation "may be causative," depending upon the outcome of genetic testing on N.S.'s parents. Exhibit 23 at 26. She also characterized N.S.'s genetic mutation as "deleterious." Id. at 25.

Overall, Ms. Skorupska has not presented a logical sequence of cause and effect connecting the Hib vaccination to the change in N.S.'s health in November 2013 (regardless of whether this change is categorized as a new injury for Althen or the significant aggravation of a pre-existing problem for Loving). The Federal Circuit has clarified that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 549 (Fed. Cir. 1994). However, "notation of absence is not requirement of presence." Howard v. United States, No. 16-1592V, 2023 WL 4117370, at *7 (Fed. Cl. May 18, 2023) (denying motion for review and commenting that, "Because Petitioner's theory relied on the pathogenetic role of certain antibodies, Petitioner cannot then fault the Decision for noting the absence of evidence

[8] The Secretary's argument would have been stronger if the Secretary supported his assertion by citing exhibit numbers and page numbers for the medical records in which treating doctors linked N.S.'s genetic mutation to his neurologic problems. See Resp't's Br. at 32.

preponderantly indicating their presence"), aff'd, No. 2023-1816, 2024 WL 2873301 (Fed. Cir. June 7, 2024). Here, Ms. Skorupska's theory relied on the presence of cytokine-driven inflammation. Without a showing that this inflammation did occur, she cannot establish a logical sequence of cause and effect under the fifth Loving prong / second Althen prong. Ms. Skorupska's failure to meet her burden of proof on this element means that she is not entitled to compensation.

### B. Loving Prong 6 / Althen Prong 3

The timing prong actually contains two parts. A petitioner must show the "timeframe for which it is medically acceptable to infer causation" and that the onset of the disease occurred in this period. Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 542-43 (2011), recons. denied after remand on other grounds, 105 Fed. Cl. 353 (2012), aff'd without op., 503 F. App'x 952 (Fed. Cir. 2013).

Ms. Skorupska's position regarding timing is not clear. Part of the lack of clarity may derive from an inconsistency in Dr. Huq's first report. Dr. Huq wrote: "As the initial brain injury occurred due to activation of the innate immunity by [the] Hib vaccine, the timing of epileptic spasms 5 days after the vaccination is appropriate." Exhibit 68 at 7-8. He repeats this sentence on page 28. But, in this context, Dr. Huq stated, "NS developed infantile spasms seven days after Hib vaccination." Id. at 28.

The relevant portion of Ms. Skorupska's original brief did not engage with timing. See Pet'r's Br. at 63-64. She did not identify a period for which an inference of aggravation is appropriate, and she did not state when N.S.'s pre-existing condition was first aggravated. She was, accordingly, directed to clarify her position. Order, issued Oct. 7, 2024.

Ms. Skorupska presented more fulsome arguments. As to the interval for which an inference of aggravation is appropriate, Ms. Skorupska stated that the appropriate range of time would be "within the first 72 hours – six days." Pet'r's Supp'l Br., filed Oct. 15, 2024, at 1. Although Ms. Skorupska cited two medical articles, they do not propose six days. The first is by Tro-Baumann, which found that the majority of children with Dravet's syndrome experienced febrile seizures

within 72 hours of a vaccination against diphtheria-tetanus-pertussis. Exhibit 224.[9] The second is by Verbeek, which found febrile seizures within 24 hours of a vaccination in children with Dravet's syndrome. Exhibit 225.[10]

Next, to fit N.S.'s initial aggravation within these periods, Ms. Skorupska relies upon her September 18, 2017 affidavit. There, she averred that during the night after the vaccination, N.S. developed a "mild fever." Exhibit 40 ¶ 5. She added that in the emergency room, she realized that N.S. "had been having spasms for at least two days, maybe more." Id. at 8. At least one medical record from this time corroborates Ms. Skorupska's account, at least in respect to the two days of trouble. See Exhibit 29 at 94-95 (report dated November 22, 2013 and stating Ms. Skorupska "reports a 2 day history of new onset spasms").

The Secretary challenges Ms. Skorupska's arguments regarding timing in at least two respects. First, the Secretary disagrees with Ms. Skorupska's reliance upon the Tro-Baumann and Verbeek articles because those articles were written about children experiencing febrile seizures in the context of Dravet's syndrome. In the Secretary's view, because N.S. did not have a febrile seizure, "data concerning febrile seizures are completely irrelevant to establish an appropriate temporal relationship between the Hib vaccine in this case and the onset of infantile spasms." Resp't's Br. at 34. Second, the Secretary points out another mismatch: "Even if febrile seizure data were relevant here, N.S.'s spasms were first observed four days after vaccination, outside the time frame of within three days cited in Tro-Baumann, or within a day cited in Verbeek." Id.[11]

Ms. Skorupska largely fails to answer these arguments. She maintains that she "provided evidence of a clear temporal relationship between the administration of the Hib vaccine and the onset of N.S.'s seizures." Pet'r's Reply at 5. However, she did not address the Secretary's arguments about (the lack of relevance for)

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[9] Blanca Tro-Baumann et al., A retrospective study of the relation between vaccination and occurrence of seizures in Dravet syndrome, 52 EPILEPSIA 175 (2011). Filed as Exhibit 224.

[10] Neinke E. Verbeek et al., Prevalence of SCN1A-related dravet syndrome among children reported with seizures following vaccination: a population-based ten-year cohort study, 8 PLOS ONE 1 (2013). Filed as Exhibit 225.

[11] A third potential argument is that N.S. was having infantile spasms before his mother observed them, possibly before the vaccination. See Resp't's Br. at 33 n.14. However, preponderant evidence does not establish that N.S. was experiencing infantile spasms before the vaccination.

febrile seizures or the development of infantile spasms after the period predicted by the Tro-Baumann article.

Under these circumstances, crediting Ms. Skorupska's proof regarding timing is difficult. If it is assumed that Tro-Baumann and Verbeek, two articles about febrile seizures, provide information about the time that afebrile seizures develop after vaccination, then preponderant evidence favors a finding that the seizures would manifest within about 72 hours of the vaccination. But, the evidence does not support a conclusion that N.S. displayed symptoms of a seizure within the first 72 hours post-vaccination. In an affidavit written in 2017, Ms. Skorupska averred that N.S. "had been having spasms for at least two days, maybe more" as of November 22, 2013, placing the onset at four days after vaccination, "maybe" sooner. Exhibit 40 at 2. Ms. Skorupska also states that N.S. had a fever the night of vaccination, and, "in retrospect, she felt as though N.S. had been having seizures possibly for the 'entire week,' meaning from the first day, or two, of the vaccination." Pet'r's Supp'l Br. at 3.

Ms. Skorupska's account does not overcome the medical records created when N.S. went to the hospital in November 2013. These medical records, which were generated contemporaneously with N.S.'s illness, note only a two-day history of spasms, i.e., four-days post-vaccination. This is not to indicate that Ms. Skorupska is presenting a false narrative; special masters have often refrained from crediting accounts presented years later because of a faulty memory, not dishonesty. See Hodge v. Sec'y of Health & Hum. Servs., No. 09-453V, 2023 WL 4186513, at *71 (Fed. Cl. Spec. Mstr. May 24, 2023), mot. for rev. denied, 168 Fed. Cl. 117 (2023); Barnett v. Sec'y of Health & Hum. Servs., No. 19-1578V, 2021 WL 6211590, at *6 (Fed. Cl. Spec. Mstr. Dec. 10, 2021); Duda v. Sec'y of Health & Hum. Servs., No. 19-31V, 2021 WL 4735857, at *8 (Fed. Cl. Spec. Mstr. Aug. 10, 2021); Mueller v. Sec'y of Health & Hum. Servs., No. 06-775V, 2011 WL 1467938, at *9 (Fed. Cl. Spec. Mstr. Mar. 16, 2011); Velchek v. Sec'y of Health & Hum. Servs., No. 02-1479, 2005 WL 2847451, at *17 (Fed. Cl. Spec. Mstr. Oct. 28, 2005). Here, Ms. Skorupska's affidavit—written nearly four years after N.S.'s vaccination and, additionally, phrased in uncertain terms—is too far-removed and too speculative to be credited over the medical records.

Ms. Skorupska argued that the two events (vaccination and onset of infantile spasms) could not simply be viewed as a "coincidence." Pet'r's Br. at 64. However, in several cases, special masters have found that a sequence of events in which a child-vaccinee's febrile seizure occurred within one day of a vaccination

did not mean that the vaccine caused the child's epilepsy. Vinesar, 2023 WL 5427935 at *32.

In sum, Ms. Skorupska has not presented any persuasive evidence to justify a timeframe of up to six days. After 72 hours is credited as an acceptable timeframe, Ms. Skorupska has not demonstrated that N.S. developed symptoms within this time. Accordingly, Ms. Skorupska has failed to meet her burden regarding timing. Regardless, if Ms. Skorupska were found to have prevailed on the timing element, she would still not be entitled to compensation due to deficiencies in other parts of her case. Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144 (Fed. Cir. 1992) ("Temporal association is not sufficient, however, to establish causation in fact.").

### C. Loving Prong 4 / Althen Prong 1

The first Althen prong, which corresponds to the fourth Loving prong, requires a petitioner to present a reliable and persuasive medical theory. Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359 (Fed. Cir. 2019) (citing Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548 (Fed. Cir. 1994)). Ms. Skorupska presents the theory that the Hib vaccine caused inflammation, which "caused altered connectivity, which in turn, caused epilepsy." Pet'r's Br. at 57. Ms. Skorupska further states that N.S.'s genetic mutation made him more susceptible to the inflammatory response, but that his "genetic mutation alone is not a sound explanation for [his] infantile spasms," as his "genetic variant would typically cause something different than infantile spasms" such as delays or autism. Id. at 57-58. Ms. Skorupska summarizes, "The theory is that [N.S.]'s susceptible genetic mutation made him more likely to have the particular reaction to the vaccine," and that she "has set forth his medical theory causally connecting his injury of infantile spasms and the sequelae to the Hib vaccination that he received in 2013." However, while Ms. Skorupska may theorize that the vaccine caused N.S.'s condition, she does not actually describe in her briefs the underlying mechanism to explain *how* the vaccine could have done so. Instead, she simply states that she is relying on all of her experts' reports. Pet'r's Br. at 58.

Citing various cases, the Secretary asserts that a theory based upon cytokines is too general to be persuasive. The Secretary argues that "there is an utter lack of evidence in this case that the Hib vaccine can cause proinflammatory cytokines that ultimately, and deleteriously, affect voltage-gated sodium channels encoded by SCN2A." Resp't's Br. at 24. Additionally, the Secretary notes that vaccines are meant to stimulate the immune system, and so invocation of an innate immune

response is not sufficient to explain causation, as it would mean that all vaccines and pathogens could cause N.S.'s condition. Id. at 26. Finally, the Secretary argues that Ms. Skorupska and Dr. Huq's assertion of molecular mimicry is "unwarranted," as no treating doctor "even suspected that N.S. had an autoimmune condition." Id. at 27. The Secretary further notes that generalized theories of molecular mimicry are frequently invoked and rejected in the program. Id. at 27-28.

A finding that a petitioner has not met one element of the Althen prongs justifies a denial of compensation. Ms. Skorupska has not carried her burden under the other Loving / Althen prongs. As discussed more thoroughly in the previous sections, the evidence does not preponderate in a finding that N.S. experienced cytokine-driven inflammation, nor did he exhibit inflammatory symptoms within the relevant timeframe. Without a showing that N.S. experienced cytokine-driven inflammation, Ms. Skorupska cannot prevail with a theory relying on such inflammation, regardless of whether she has met her burden under this prong. Additionally, neither party discussed Ms. Skorupska's theory in great detail in their briefs. See Pet'r's Br. at 57-58; Resp't's Br. at 24-28; Pet'r's Reply at 3.[12] Thus, a fulsome analysis of the theory is not necessary.

## VI. Part Two: Alternative Cause

The foregoing analysis is a basis for determining that Ms. Skorupska failed to meet her burden of proving with preponderant evidence that the Hib vaccine significantly aggravated N.S.'s neurologic problems. See Osenbach v. Sec'y of Health & Hum. Servs., No. 16-419V, 2023 WL 5714809, at *29-32 (Fed. Cl. Spec. Mstr. Aug. 8, 2023) (finding Dr. Huq's theory of inflammation unpersuasive), mot. for rev. denied, unpublished op., aff'd, No. 2024-1663, 2025 WL 2387944 (Fed. Cir. Aug. 18, 2025). To a degree, the analysis has been incomplete because it has

[12] Although Ms. Skorupska cites three articles about molecular mimicry (see Pet'r's Br. at 41-42), she does not present any detailed argument. Rather, the parties devoted more discussion to inflammation. Likewise, this Decision focuses on cytokine-driven inflammation. Importantly, Ms. Skorupska has not adequately established that N.S. suffered from an autoimmune condition. See Exhibit C at 3; Resp't's Br. at 27, 31. Thus, even if it were assumed that molecular mimicry was a reliable theory to explain how the Hib vaccine can cause infantile spasms, Ms. Skorupska would remain not entitled to compensation because N.S.'s medical history does not align with the theory. See Hibbard v. Sec'y of Health & Hum. Servs., 698 F.3d 1355, 1364 (Fed. Cir. 2012). For these reasons, this Decision does not elaborately evaluate molecular mimicry.

largely not addressed the "elephant in the room," which is N.S.'s genetic variation. See Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1380 (Fed. Cir. 2012).

If, for the sake of argument, it were assumed that Ms. Skorupska had met her burden of proof, then the Secretary may show N.S.'s "condition . . . is due to factors unrelated to the administration of the vaccine." 42 U.S.C. § 300aa–13(1)(B). "If a petitioner successfully satisfies the Loving inquiry, the burden shifts to the government to prove by a preponderance of the evidence that a 'factor unrelated' to the vaccine caused the petitioner's injuries." Sharpe v. Sec'y of Health & Hum. Servs., 964 F.3d 1072, 1080 (Fed. Cir. 2020). As stated earlier, the parties differ widely about the significance of the genetic variation. Ms. Skorupska maintains that the genetic mutation "primed N.S. to have the adverse reaction to the Hib vaccine." Pet'r's Br. at 3. Her "position is that the variant SCN2A gene is not enough to cause Petitioner's current mental state. While he may have been at an increased risk to develop some deficits due to the genetic mutation, the vaccination (not simply the genetic mutation) is what caused his current mental state." Id. at 4. In contrast, the Secretary maintains: "It is beyond question that N.S.'s mutation amply explains his entire course." Resp't's Br. at 16.

### A. Method of Analysis / Precedent Cases

Special masters may consider the method of analysis and outcome of similar cases, although those outcomes are not binding. Oliver v. Sec'y of Health & Hum. Servs., 900 F.3d 1357, 1363-64 (Fed. Cir. 2018); Nunez v. Sec'y of Health & Hum. Servs., 144 Fed. Cl. 540, 548 (2019), aff'd on non-related grounds, 825 F. App'x 816 (Fed. Cir. 2020).

The topic of genetic variants and their potential to cause harm has been discussed in multiple cases that reached the Federal Circuit. As recounted extensively, the first four opinions from the Federal Circuit ruled that special masters were not arbitrary in finding that a genetic mutation in an SCN1A gene was the sole basis for a child's condition. Vinesar, 2023 WL 5427935, at *1-20 (citing cases). But, then, the tide may have shifted. The Federal Circuit vacated an opinion in which the special master found a genetic mutation explained the child-vaccinee's worsening. Sharpe, 964 F.3d 1072; see also Vinesar, 2023 WL 5427935, at *14-18. Then, the Federal Circuit went a step further and reversed the (undersigned) special master's finding of genetic causation. Sanchez v. Sec'y of

Health & Hum. Servs., 34 F.4th 1350 (Fed. Cir. 2022); see also Vinesar, 2023 WL 5427935, at *18-20.[13]

The Secretary argues that Ms. Skorupska's case "is analytically no different from the Dravet syndrome cases in which respondent has uniformly prevailed through Federal Circuit review." Resp't's Br. at 16. The Secretary also attempts to distinguish Sharpe. Id. at 17-19. However, the Secretary omits any discussion of Sanchez.

The omission of Sanchez from the Secretary's brief can be excused because Ms. Skorupska cites no Federal Circuit cases about vaccines and genetics. See Pet'r's Br. The omission of Federal Circuit genetic cases, especially Sanchez, from Ms. Skorupska's brief is less easily justified.

The lack of argument based upon Sanchez from Ms. Skorupska is regrettable. As stated in Vinesar, 2023 WL 5427935, at *45-47, the Federal Circuit's opinion in Sanchez has introduced questions about how special masters (at least the undersigned special master) should approach cases with genetic issues. For example, the Federal Circuit's opinion in Sanchez could be interpreted as requiring evidence from the Secretary about a genetic variant that is stronger than the evidence previously credited in cases such as Hammitt v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 719 (2011), aff'd sub nom. Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373 (Fed. Cir. 2012). Future precedential opinions from the Federal Circuit may provide additional guidance. See Strickland v. United States, 423 F.3d 1335, 1338 & n. 3 (Fed. Cir. 2005) (stating that if a trial court thinks Federal Circuit opinions are inconsistent with Supreme Court precedent, the trial court may note the conflict and urge en banc consideration).

### B. Evidence

Several pieces of overlapping evidence support a finding that the E1211K variant is the sole cause of N.S.'s medical condition.

[13] In two recent non-precedential opinions, the Federal Circuit affirmed judgments denying compensation in cases in which the child-vaccinee had a genetic mutation without discussing genetics. See Vinesar v. Sec'y of Health & Hum. Servs., No. 2024-1787, 2025 WL 2945665 (Fed. Cir. Oct. 17, 2025); Osenbach v. Sec'y of Health & Hum. Servs., No. 2024-1663, 2025 WL 2387944 (Fed. Cir. Aug. 18, 2025).

An important and foundational article is by Ogiwara and colleagues.[14] These researchers discovered an *SCN2A*-E1211K mutation in a person who suffered from infantile spasms. He was born with mild asphyxia. Ogiwara at 1051. His seizures began at age 11 months. Ogiwara at 1047. The researchers compared the genetic mutation (c3613G>A) to a cohort of approximately 300 individuals. This genetic mutation was found "only in affected individuals." Id. at 1048. The researchers then conducted "whole-cell patch-clamp recordings" and other tests. ("Patch clamps" allow researchers to measure the flow of current through ion channels. Dorland's Illus. Med. Dict. at 362 (33rd ed.).) The researchers determined that E1211K is "evolutionarily conserved among vertebrae and invertebrae VGSC [voltage gated sodium channel] α subunits." Id. at 1049. The researchers also tested how the E1211K variant affected a cell's electrophysiologic property. They found "E1211K . . . markedly altered the voltage-dependence of $Na_v1.2$ indicating high pathologic potential[] of E1211K." Id. The researchers concluded: "our present study and others' provide solid genetic evidence implicating *SCN2A* in the etiology of human epilepsies." Id. at 1052.

In 2014, when analyzing the results of N.S.'s genetic testing, Dr. Jabs cited the Ogiwara article. She noted that the Ogiwara researchers tested the mutation and they found the mutation changed the functioning of the protein. This testing supported Dr. Jabs's opinion that N.S.'s genetic mutation was a "deleterious change." Exhibit 23 at 25.

Dr. Raymond cited the 2009 Ogiwara article in his first report. Exhibit A at 5. In his view, Ogiwara and colleagues "clearly demonstrated that this mutation seriously impairs the normal gating actions of the channel." Id. at 6.

Dr. Huq's response to Ogiwara was underwhelming. Dr. Huq stated that: "Only one other individual with SCN2A E1211K variant has been reported but that individual also had birth asphyxia which likely contributed to or modified the pathogenesis of his epilepsy." Exhibit 68 at 4; accord Id. at 7, 10, 28.[15] Although

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[14] I. Ogiwara et al., De novo mutations of voltage-gated sodium channel alpha II gene SCN2A in intractable epilepsies, 73 NEUROLOGY 1046 (2009). Filed as Exhibit A-2.

[15] As discussed below, by the time Dr. Huq wrote his first report, Dr. Raymond had identified other cases in which individuals had the same genetic mutation. See Exhibit C at 1-2. Thus, the evidentiary basis for Dr. Huq's assertion that "only one other individual" is not readily apparent.

he highlighted the potential importance of asphyxia, Dr. Huq did not engage with other meaningful parts of Ogiwara. Dr. Huq did not contest Ogiwara's assertion that E1211K is evolutionarily conserved. Dr. Huq also did not contest the electrophysiologic consequences to an alteration in E1211K.

The electrophysiologic testing carries great weight because this testing shows that the genetic variant interferes with how the sodium channel is supposed to function. See Exhibit C at 2 (Dr. Raymond's second report: "E1211K produced severely affected channels with electrophysiologic properties very different than the normal functioning voltage gated sodium channel"). In other words, the gene directed the creation of a protein that does not act as it should. Oligawa's finding regarding the electrophysiologic dysfunction appears to be confirmed by other researchers. Exhibit E (Lauxmann).[16] Due to this variation in how the sodium channel was created in N.S., N.S.'s sodium channel had a deficiency. It is difficult to see how the vaccination affected N.S.'s anatomy.[17]

Ogiwara also lays a foundation for an answer to another potential challenge to the significance of N.S.'s genetic variant. Dr. Huq acknowledges that the "E1211K variant has not been detected in healthy individuals." Exhibit 193 at 2. Although Dr. Huq does not cite a basis for this assertion, Ogiwara did not find the

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[16] Stephan Lauxmann et al., Relationship of electrophysiological dysfunction and clinical severity in SCN2A-related epilepsie, 12 HUM. MUTAT. 1942 (2018). Filed as Exhibit E.

[17] Although the electrophysiologic properties are important to the undersigned (see Whitecotton v. Sec'y of Health & Hum. Servs., 81 F.3d 1099, 1108 (Fed. Cir. 1996) (stating that special masters have discretion in how they weigh evidence)), conceivably, the undersigned is overvaluing this testing. In Sanchez, in one of the critical articles about the significance of the genetic mutation in that case (the Parfait article), "the researchers used fibroblasts to see how the gene performed in tissue cultures. . . . Their experiment 'confirm[ed] the deleterious effect of this mutation.'" Sanchez, 2020 WL 5641872, at *59. Based upon this work, the petitioners' geneticist (Dr. Niyazov) conceded that the genetic mutation was "an 'altered function' mutation." Id. at *60. However, upon an appeal, the Federal Circuit stated that "There is no evidence . . . that [the child-vaccinee's] mutations would have resulted in the same progression and severity of his [disease], absent the vaccine." Sanchez, 34 F.3d at 1356. The Federal Circuit did not discuss the fibroblast testing.

This discussion about whether the Federal Circuit's precedential opinion in Sanchez bars the consideration of the electrophysiologic testing is academic in the sense that Ms. Skorupska has made no argument about Sanchez. Ms. Skorupska also did not discuss the Ogiwara's electrophysiologic testing. See Pet'r's Reply.

mutation when they looked for it among approximately 300 healthy individuals. Ogiwara at 1048. Dr. Huq's response is that "population databases contain DNA sequence information of only a tiny percentage of healthy population." Exhibit 193 at 2. Dr. Raymond persuasively refutes a concern about population databases. Exhibit EE at 2 (relying, in part, on the gnomAD database). Special masters have rejected concerns that population databases are unlikely to detect healthy individuals with the potentially harmful genetic mutation. See Harris v. Sec'y of Health & Hum. Servs., No. 07-60V, 2011 WL 2446321, at *15 (Fed. Cl. Spec. Mstr. May 27, 2011), mot. for rev. granted, 102 Fed. Cl. 282 (2011), reinstated sub nom., Snyder v. Sec'y of Health & Hum. Servs., 553 Fed. App'x 994 (Fed. Cir. 2014); Stone v. Sec'y of Health & Hum. Servs., No. 04-1041V, 2010 WL 1848220, at *25 (Fed. Cl. Spec. Mstr. Apr. 15, 2010), mot. for rev. denied after remand, 99 Fed. Cl. 187 (2011), aff'd, 676 F.3d 1373 (Fed. Cir. 2012).

The lack of unaffected individuals contrasts with the number of examples in the literature of children with the same mutation and who developed neurologic problems similar to N.S.'s problems. Dr. Raymond cited Ogiwara in his first report. Exhibit A at 5. In his second report, Dr. Raymond cited two more articles (Wolff and Wong).[18] Exhibit C at 1-2. In his third report, Dr. Raymond emphasized that these people had the "exact same variant." Exhibit K at 5. Then, after Dr. Huq mistakenly asserted that there was only one case in the literature, Dr. Raymond found more cases and presented them in his fourth report. Exhibit T at 14. Finally, in the fifth report, Dr. Raymond brought forward two more examples. Exhibit EE at 4.

The Secretary aptly summarizes this evidence:

> (Case No. 1) Ex. A, Tab 1: Marcus Wolff et al., Genetic and phenotypic heterogeneity suggest therapeutic implications in SCN2A-related disorders, 140 BRAIN 1316-36 (2017). The subject with an E1211K mutation is listed as subject number 49 in Table 3. Id. at 11. The subject had West syndrome (WS) (see page 6 for abbreviation, WS) with seizure outcome noted to be

[18] Markus Wolff et al., Genetic and phenotypic heterogeneity suggest therapeutic implications in SCN2A-related disorders, 140 BRAIN 1316 (2017). Filed as Exhibit 55.

Virginia C. N. Wong et al., SCN2A mutation in a Chinese boy with infantile spasm - response to Modified Atkins Diet, 37 BRAIN DEV. 729 (2015). Filed as Exhibit D.

intractable. Id. (last followed at 4 years of age). Cognition at both onset and follow-up was "severe intellectual disability" (SD) (see page 12 for abbreviation, SD). Id.

(Case No. 2) Ex. A, Tab 2: I. Ogiwara et al., De novo mutations of voltage-gated sodium channel [alpha II] gene SCN2A in intractable epilepsies, 73 NEUROLOGY 1046-53 (2009). This article supports Dr. Raymond's statement that "the glutamine at position 1211 is highly evolutionarily conserved among vertebrate and invertebrate voltage gated sodium channels and [the authors] went on to demonstrate that significantly altered the functional properties of the channel." Ex. A at 5 (citing Ex. A, Tab 2, at 4-5); see also Ex. T at 13 (Dr. Raymond). With regard to the E1211K subject discussed in the article, the authors state: "One de novo mutation, E1211K, was identified in a patient with sporadic infantile spasms that progressed into severe symptomatic generalized epilepsy." Ex. A, Tab 2, at 6.

(Case No. 3) Ex. D: Virginia C.N. Wong et al., SCN2A mutation in a Chinese boy with infantile spasm – response to Modified Atkins Diet, 37 BRAIN & DEV. 729-32 (2015). This is a case report of the same E1211K mutation. The subject "had developmental delay since birth. He developed flexor spasm of his upper limbs occurring in clusters since 15 months. … He had developmental regression and autistic features later during subsequent follow up. Electroencephalography (EEG) showed modified hypsarrhythmia." Id. at 2 (noting "severe intellectual disability" at age 6). Characterizing the nature of the mutation, the authors state, "this mutation affected highly conserved amino acid … and the mutation was predicted to be deleterious to protein function and pathogenic by PolyPhen-2, SIFT and Align-GVGD analysis. This mutation has been reported previously in a patient with sporadic IS, marked

developmental delay and severe intellectual disability." Id. (citing Ogiwara (Ex. A, Tab 2)).

(Case No. 4) Ex. Q: Hane Lee et al., Clinical Exome Sequencing for Genetic Identification of Rare Mendelian Disorders, 312(18) JAMA 1880-87 (2014) (including relevant Supplementary Online Content). On page 27, the authors report a case of "p.Glu1211Lys" like in this case. The clinical indications are listed as "developmental delay, epilepsy, seizures, spasms, autism, [m]icrocephaly, wide palpebral fissures, hypertonia, mitochondrial [sic], complex IV, cytochrome c oxidase deficiency, Rett syndrome, Lennox Gastaut." Id.

(Cases No. 5-7) Ex. R: Qi Zeng et al., SCN2A-Related Epilepsy: The Phenotypic Spectrum, Treatment and Prognosis, 15(809951) FRONTIERS MOLECULAR NEUROSCIENCE (2022). This is a report of three cases overall. On page 5, the authors report 2 cases of E1211K, both with commensurately severe outcomes as the current case, one subject noted to be unable to sit alone or speak (last follow-up at age 6 years, 11 months), the other with delay before seizure onset, walking at 4 years, 8 months, and unable to speak (last follow-up at age 5). On page 7, the third subject was also delayed before seizure onset and could not walk or speak.

(Cases No. 8-9) Ex. V: Géza Berecki et al., Functional correlates of clinical phenotype and severity in recurrent SCN2A variants, 5(1) COMMUNICATIONS BIOLOGY 1-13 (2022) (Article No. 515) (doi: 10.1038/s42003-022-03454-1) (including Supplementary Data 1: Phenotypic data of 179 individuals with 38 recurrent SCN2A variants, and 2 individuals with unique variants (Ex. V at 14)). The authors reported two previously-unreported cases of E1211K mutation both with moderate delays prior to seizure development, like in the current case, and both with severe delay "at last review," like in the current case. Id. at 14 (also listing the cases cited in Ogiwara

(Ex. A, Tab 2), Wong (Ex. D), and Wolff (Ex. A, Tab 1)).

(Case No. 10) Ex. HH: Balamurugan Nagarajan et al., Landscape of genetic infantile epileptic spasms syndrome—A multicenter cohort of 124 children from India, 00 EPILEPSIA OPEN 1-22 (2023) (doi:10.1002/epi4.12811). In Table 1, a subject is listed as having the c.3631G>A variation, like in the current case. Id. at 5. All the subjects had "infantile-onset (2 months-2 years) epileptic spasms and classical or modified hypsarrhythmia on EEG with or without developmental delay or regression." Id. at 3 (defining Infantile Epileptic Spasms Syndrome for the purpose of the report). The subject with variation c.3631G>A is coded as "Serial no. 16." Id. at 5. In the outcomes table (Table 4) on page 13, that patient had generalized tonic (GT) seizures at 6 months and epileptic spasms (ES) at 7 months. At 14 months, she developed stereotypies, and her phenotypic characteristics included central hypotonia (C HYP) and clinodactyly (CLDY). By 81 months of age, she was non-ambulatory (NAMB) and autistic (AU). Id. at 13 (see page 19 for explanation of abbreviations). The authors note that "the longterm neurodevelopmental outcome was poor in most children with SCN2A (5/6 non-ambulatory, 4/7 autistic, one progressed to LGS [Lennox-Gastaut syndrome])." Id. at 10.

(Case No. 11) Ex. II (Eye, Eye): Sylvia Vidal et al., The utility of Next Generation Sequencing for molecular diagnostics in Rett syndrome, 7:12288 SCIENTIFIC REPORTS 1-11 (2017) (doi:10.1038/s41598-017-11620-3). The patient with the same mutation as N.S. is highlighted on page 4. That study evaluated Rett syndrome-like patients, "an early-onset neurodevelopmental disorder that almost exclusively affects girls and is totally disabling." Id. at 1 (emphasis added) (noting characteristics to be "a period of apparently normal development (up to the age of 6–18

> months), followed by a regression characterized by loss of speech and purposeful hand use and motor apraxia that may be associated with epilepsy and dysautonomic features"). Contextually, the patient with the same missense mutation as N.S. had a severe disorder. See also Id. at 5 ("we detected 2 SNVs [single nucleotide variants] … in SCN2A genes, which are associated with mental retardation, stereotypic movements, epilepsy, and/or cerebral malformations").

Resp't's Br. at 13-15.

This consistency of outcomes contributes to finding that the E1211K variant causes neurologic problems. The consistency of outcomes for this variant further weakens an argument that variations among SCN2A genes produces a variety of outcomes. See Exhibit 50 at 2, Exhibit 68 at 8, Exhibit 212 at 2; see also Pet'r's Br. at 57. Dr. Raymond agrees that some SCN2A genetic variants lead to different results. See Exhibit T at 4. But, those other genetic variants are not N.S.'s variant. From the evidence presented, N.S.'s variant, the E1211K variant, appears to lead to neurologic problems always.

For all these reasons, a preponderance of the evidence supports the finding that N.S.'s genetic variant caused and was the sole cause of his neurologic problem. Dr. Raymond's opinion regarding the sole cause is persuasive. See Exhibit A at 7 ("The pathogenic mutation in [N.S.'s] SCN2A gene is the sole cause of his epileptic encephalopathy"); Exhibit C at 3 (N.S.'s "pathogenic mutation in SCN2A . . . is the sole cause of his early infantile epileptic encephalopathy"); Exhibit EE at 6 (same).

The finding that the E12111K variant impaired N.S.'s neurologic development should not be controversial. Even Dr. Huq stated the "missense variant in SCN2A gene E1211K . . . is considered to be pathogenic." Exhibit 68 at 6.[19] Dr. Huq's opinion that this "pathogenic variant" merely meant that he was

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[19] To reinforce the conclusion that the E1211K variant is pathologic, Dr. Raymond's final report cites an article with guidelines in how to interpret variants. Exhibit EE at 2-3, citing Exhibit BB (Sue Richards et al., "Standards and Guidelines for the Interpretation of Sequence Variants: A Joint Consensus Recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology," 17(5) Genet Med. 405 (2015)). However, these Guidelines are relatively complicated and difficult to understand. Thus, the

"predisposed to having seizures" is not in line with the multiple researchers who have identified the mutation as causative. See Terran v. Sec'y of Health & Hum. Servs., 195 F.3d 1302, 1316 (Fed. Cir. 1999) (authorizing special masters to consider, among other factors, whether an opinion "enjoys general acceptance within a relevant scientific community").

For these reasons, the evidence preponderates in favor of finding that N.S.'s genetic variant was the sole cause of his neurologic problems, including the infantile spasms and developmental delay. Thus, even if it were found that Ms. Skorupska met her burden of presenting a persuasive case for each of the Loving prongs, she would remain not entitled to compensation because the Secretary would have met his burden to present a factor unrelated to the vaccination caused N.S.'s condition.

## VII. Conclusion

Ms. Skorupska has consistently maintained that the Hib vaccine harmed her son. She has persevered during this litigation, even representing herself for a time. Her dedication and love for her son is admirable. However, these traits cannot be a basis for awarding compensation. Congress requires that special masters evaluate claims based upon the evidence.

Here, Ms. Skorupska's evidence is lacking. The primary flaw is that she did not establish that N.S. developed inflammation as her experts' theory predicted. In addition, it is more likely than not that N.S.'s variant in the E1211K gene was the sole cause of his neurologic problems. Thus, Ms. Skorupska is not entitled to compensation.

The Clerk's Office is instructed to enter judgment in accord with this decision unless a motion for review is filed. Information about filing a motion for review, including the deadline, can be found in the Vaccine Rules, which are available on the website for the Court of Federal Claims.

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Guidelines do not affect the outcome of Ms. Skorupska's case. In the future, Dr. Raymond may wish to proceed through the Guidelines step-by-step.

**IT IS SO ORDERED.**

s/Christian J. Moran
Christian J. Moran
Special Master