In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS
No. 02-235V
Filed: August 18, 2015

KELLIE MILLER and RON MILLER, as mother and father of A.H.M., a minor,	*	
Petitioners,	*	Autism; Mitochondrial Disease, Mitochondrial Disorder, or Mitochondrial Dysfunction; Entitlement; Lack of a Factual Predicate in Support of a Claim; Insufficient Proof of Causation; Expert Qualifications and Bias
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Michael L. Cave, Esq., Cave Law Firm, Baton Rouge, LA, for petitioners.
Linda S. Renzi, Esq., U.S. Department of Justice, Washington, DC, for respondent.

DECISION[1]

Vowell, Chief Special Master:

On March 26, 2002, Kellie and Ron Miller ["Ms. Miller," "Mr. Miller," or "petitioners"] filed a petition on behalf of their minor daughter, A.H.M., seeking compensation under the National Childhood Vaccine Injury Act[2] ["Vaccine Act"]. This petition raised the same claims made in thousands of other petitions filed between 1997 and 2009—that various vaccines or a preservative contained in some vaccines

[1] The E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002), requires that this decision be publicly available. In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] The National Vaccine Injury Compensation Program ["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.A. §§ 300aa-10 *et seq*. (2012). All citations to the Vaccine Act in this decision will be to 42 U.S.C. § 300aa.

caused autism spectrum disorders ["ASD"].[3]

Over the last four years, Mr. and Ms. Miller have modified their claim repeatedly, raising new theories and re-litigating old ones, but have failed to produce reliable evidence that any vaccine or combination of vaccines that A.H.M. received actually caused her condition. Accordingly, I hold that petitioners are not entitled to compensation and the petition is dismissed.

I. Procedural History.

A. The Omnibus Autism Program ["OAP"].

A.H.M.'s case, filed more than 13 years ago, was filed prior to the creation of the OAP. Beginning in 1997, but peaking numerically in 2002-03, thousands of petitions were filed alleging either that the measles, mumps, and rubella ["MMR"] vaccine or thimerosal, an ethyl mercury preservative used in multi-dose vials of vaccine, caused ASD. The sheer volume of petitions filed—more than 1,300 new petitions in 2002 alone—led to the creation of the OAP.[4] Omnibus programs had been used previously to make causation determinations in groups of cases alleging that a particular vaccine caused a specific injury, but the OAP was, by far, the largest such grouping of similar cases.[5]

Following a series of meetings with an informal advisory committee comprised of petitioners' counsel representing many of the Program claimants plus legal and medical representatives of the Secretary of Health and Human Services, the Office of Special Masters ["OSM"] adopted a plan that would allow a period of discovery, followed by the selection and litigation of test cases on the theories of causation presented. Autism General Order #1 at 2-3. The conclusions reached on general causation in the test cases would be used to resolve the remaining individual claims. *Id.* at 3. Petitioners

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[3] I use the abbreviation "ASD" to refer to the broad category of autism spectrum disorders. The Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 5th ed. 2013) ["DSM-V"] uses this term to cover all autistic disorders, replacing the earlier version which used the umbrella term "pervasive developmental disorder" ["PDD"]. *See* Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 4th ed. text revision 2000) ["DSM-IV-TR"]. The symptoms recognized by the medical community at large as those of ASD have not changed, but the criteria for diagnosis have been refined, and the distinctions drawn in the DSM-IV-TR among the diagnoses of autistic disorder, pervasive development disorder–not otherwise specified ["PDD-NOS"], and Asperger's disorder have been eliminated.

[4] *See* Autism General Order #1, issued July 3, 2002 (found at 2002 WL 31696785, 2002 U.S. Claims LEXIS 365, or http://www.uscfc.uscourts.gov/sites/default/files/autism/Autism+General+Order1.pdf) (last visited on May 11, 2015).

[5] *See Hennessey v. Sec'y, HHS,* No. 01-190V, 2009 WL 1709053 (Fed. Cl. Spec. Mstr. May 29, 2009), *aff'd,* 91 Fed. Cl. 126 (2010) (discussing the different types of omnibus proceedings conducted in the Vaccine Program).

were allowed to "opt in" or "opt out" of the proceedings and future claimants could automatically "opt in" by filing the short form petition included as Attachment B to Autism General Order #1. Autism General Order #1 at 6-8.

Attorneys representing petitioners created the Petitioners' Steering Committee ["PSC"] to coordinate the OAP litigative effort. The PSC acknowledged that there was insufficient evidence at the time the OAP was created to prove vaccine causation, but averred that such evidence could be found through discovery and ongoing scientific investigations. Petitioners sought and received an extended period of time to conduct discovery and prepare to litigate test cases.

Nearly five years after the OAP was created, litigation in the test cases began. The PSC presented two different theories on the causation of ASD in two sets of test cases. The first alleged that thimerosal-containing vaccines and the MMR vaccine, in combination, could cause ASD (Theory 1). The second alleged that thimerosal-containing vaccines could cause ASD (Theory 2).[6]

Decisions in each of the three Theory 1 test cases, which were tried in 2007, rejected petitioners' causation theories. *Cedillo v. Sec'y, HHS,* No. 98-916V, 2009 WL 331968 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 89 Fed. Cl. 158 (2009), *aff'd*, 617 F.3d 1328 (Fed. Cir. 2010); *Hazlehurst v. Sec'y, HHS,* No. 03-654V, 2009 WL 332306 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 88 Fed. Cl. 473 (2009), *aff'd*, 604 F.3d 1343 (Fed. Cir. 2010); *Snyder v. Sec'y, HHS,* No. 01-162V, 2009 WL 332044 (Fed. Cl. Spec. Mstr. Feb. 12, 2009), *aff'd*, 88 Fed. Cl. 706 (2009).[7]

Decisions in the three Theory 2 test cases, which were tried in 2008, also rejected the causation theory presented. Petitioners did not seek review of the special masters' decisions. *Dwyer,* 2010 WL 892250; *King v. Sec'y, HHS,* No. 03-584V, 2010 WL 892296 (Fed. Cl. Spec. Mstr. Mar. 12, 2010); *Mead*, 2010 WL 892248.

An impressive body of medical and scientific evidence was adduced in the OAP test cases, with the three special masters who heard this evidence finding that the issue of vaccine causation was "*not a close case*". *See, e.g.*, *King*, 2010 WL 892296, at *90 (emphasis in original); *Snyder*, 2009 WL 332044, at *198. Each of the three special

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[6] *Dwyer v. Sec'y, HHS,* No. 03-1202V, 2010 WL 892250, at *2 (Fed. Cl. Spec. Mstr. Mar. 12, 2010). In keeping with the intent that the OAP test case evidence be available to aid in resolving the remaining OAP cases, the evidence (including expert reports, transcripts of testimony, trial presentation materials (trial exhibits), and lists of the medical journals and other documents filed by the parties) is posted on the Court of Federal Claims website (http://www.uscfc.uscourts.gov/docket-omnibus-autism-proceeding) (last visited on May 11, 2015). The parties in the test case hearings all filed explicit written consent to make these materials publicly available. *See, e.g.*, *Mead v. Sec'y, HHS,* No. 03-215V, 2010 WL 892248 (Fed. Cl. Spec. Mstr. Mar. 12, 2010), at *1 n.1 (referencing petitioners' and respondent's written consent in that case).

[7] Petitioners did not appeal the Court of Federal Claims decision in *Snyder* to the Federal Circuit.

masters independently determined that the medical theories advanced were not reliable and that the test case petitioners had failed to produce preponderant evidence of causation. The OAP test case litigation concluded in 2010, with the last Federal Circuit decision affirming the dismissal of the final Theory 1 test case.[8]

With appellate review completed, orders were issued on a rolling basis to the remaining 4,800 petitioners, requiring them to inform the court whether they intended to dismiss their case or proceed on a different theory or with different evidence. Petitioners were cautioned that the special masters did not intend to re-litigate the test case theories, and that a reasonable basis to proceed would require either new evidence on the old theories or new theories of causation.[9]

B. A.H.M.'s Petitions.

The original petition filed in this case claimed that A.H.M. had ASD and raised both of the theories of causation presented in the OAP.[10] This petition claimed that A.H.M. "suffered mercury poisoning, autism spectrum disorder, and pervasive developmental disorder" caused by the MMR and diphtheria, tetanus, and pertussis ["DTP"] vaccines she received on September 15, 1999.[11] Unlike most OAP cases, some medical records were filed with the original petition. After evaluating them, respondent filed a Vaccine Rule 4 report on June 26, 2002, recommending that the claim be dismissed.

Thereafter, at petitioners' request, their case was included in the newly-created OAP. Order, issued Aug. 8, 2002. The case was reassigned to Special Master George Hastings and, pursuant to the transfer order, stayed until the general causation issues raised in the OAP could be decided.[12] Special Master Hastings presided over all OAP

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[8] *Cedillo v. Sec'y, HHS*, 617 F.3d 1328 (Fed. Cir. 2010).

[9] *See* Order, issued Apr. 29, 2011, at 2.

[10] Two petitions were filed in 2002 involving A.H.M.'s claimed vaccine injury. The first, No. 02-235V, was filed on March 26, 2002 by attorney Terry Bennett. A second petition, No. 02-2051V, was filed by petitioners' current attorney, Michael Cave, on December 30, 2002. When respondent brought the duplicate filing to the court's attention (*see* Respondent's Motion, filed Feb. 26, 2003, at 1), the special master then presiding ordered petitioners' two attorneys to confer and decide which petition should go forward. The later-filed petition was resolved by a motion for a voluntary dismissal. *See* Petitioners' Motion, filed Jan. 15, 2004, at 1; Order Concluding Proceedings, issued in No. 02-2051V on Feb. 6, 2004. Michael Cave became attorney of record in this case on February 10, 2004.

[11] Original Petition at 3. The petition erroneously indicated A.H.M. received a DTP vaccination but the medical records show she received a diphtheria, tetanus and acellular pertussis ["DTaP"] vaccination. *Compare id.* at 2 (original petition) *with* Petitioners' Exhibit ["Pet. Ex."] 5, p. 1 (vaccination record). Hereinafter, I refer to this vaccination by its correct formulation—DTaP.

[12] The reassignment order indicated that counsel for petitioners had requested, formally or informally, that the case be included in the OAP, with the understanding that further proceedings would be stayed. Order, issued Aug. 8, 2002, at 1. Petitioners were also informed that they could proceed independently of

cases until early in 2007 when about 5,200 OAP cases were divided among the three special masters selected to hear the test cases.[13] This case was reassigned to me on March 22, 2007.

While the OAP test cases were being litigated, petitioners were ordered to file medical records to position their case for resolution after the test cases were resolved. Petitioners ignored the order issued on January 15, 2008, but complied with a similar order issued March 31, 2009.[14]

In April 2011, after the final appellate decision in the test cases was issued, petitioners were ordered to inform the court whether they intended to proceed with their claim, either on a new theory of causation or with new evidence on one or both of the rejected causation theories. Order, issued Apr. 29, 2011. On July 18, 2011, petitioners filed the amended petition which now constitutes the operative petition for their vaccine injury claim.[15] This petition expressly claimed that the varicella vaccination received on June 4, 1999 was a causal vaccination, in addition to the vaccinations received on September 15, 1999.

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the OAP at any time by so informing the court. *Id*. at 2

[13] *See* Notice Regarding Reassignments, issued in the Autism Master File by then-Chief Special Master Golkiewicz on Jan. 11, 2007. The notice can be found at http://www.uscfc.uscourts.gov/sites/default/files/autism/1%2011%2007.pdf (last visited on May 11, 2015).

[14] Exhibits 1-10 accompanied the original petition. On April 29, 2009, petitioners refiled Pet. Exs. 1-8, splitting what had previously been Pet. Ex. 1 into two exhibits (Pet. Exs. 1 and 2) and omitting what had been filed as Pet. Exs. 9-10 (documents which detailed incurred medical expenses). This resulted in changing most of the exhibit numbers by one digit. For example, the affidavit of a treating physician filed in lieu of an expert report changed from Pet. Ex. 7 to Pet. Ex. 8, based on the refiled exhibits. An additional problem complicating references to the medical records is that the pagination used in 2009 for some of the exhibits differed from the pagination used in 2002. For example, in the version of Pet. Ex. 4 filed in 2002, petitioners did not assign a page number to the certification sheet, but in the version filed in 2009, they did so. This generated some confusion because previously filed documents, such as the Rule 4 report summarizing the evidence, had used the original exhibit numbers. I therefore ordered all future filings to reference the exhibits using the 2002 exhibit numbers Order, issued December 7, 2011. I also ordered petitioners to refile as separate exhibits any pages filed in 2009 which had not been filed in 2002. *Id*. Petitioners failed to do so. During the hearing, I used the 2002 page numbers, as did petitioners. *See* Petitioners' Exhibit List, filed Apr. 22, 2013 at ECF 58. Throughout this decision, I do so as well.

To avoid confusion between medical records and other filings, medical records are cited throughout this decision using a page number format, *e.g*., "Pet. Ex. 5, p. 1." Other exhibits and filings, including motions, expert reports, briefs, and journal articles, are cited using an "at" format, *e.g.*, "Petition at 3."

[15] Hereinafter any references to "petition" are to the amended petition filed on July 18, 2011. At several places, this petition uses the name of a child other than A.H.M. to refer to the vaccinee, but the information discussed and allegations made in the petition appear to be those related to A.H.M. Included with this petition was an unsigned and undated affidavit labeled as Ex. 1. Because this affidavit simply repeated the matters asserted in the petition, I consider the unexecuted affidavit as a part of the petition.

The amended petition and theories raised in subsequent filings recharacterized A.H.M.'s injury as an encephalopathy rather than ASD, caused by vaccines which either triggered or aggravated a mitochondrial disorder. Nevertheless, petitioners have continued to rely on either or both of the causal mechanisms identified in the OAP.

As the filed medical records did not establish the diagnosis of a mitochondrial dysfunction claimed in the amended petition, and the affidavit of Dr. Amy Holmes, a treating physician,[16] filed with the original petition in 2002 as Pet. Ex. 7, was inadequate to establish vaccine causation, I ordered petitioners to file updated records by September 26, 2011 and an expert report by October 26, 2011.[17] Order, issued July 28, 2011. After several extensions of time and one order to show cause for missed deadlines, apparently due to Mr. Cave's inability to locate his clients (*see* Motion for an Extension of Time, filed Nov. 23, 2011, at 2), petitioners filed the report of Dr. Stephanie Cave on December 1, 2011.[18] Due to significant problems with Dr. Cave's report, I ordered petitioners to file a supplemental report curing the defects listed or the report of another expert.[19] Order, issued Dec. 6, 2011, at 4. My concerns about how the familial relationship between Dr. Cave and Mr. Cave (Dr. Cave is Mr. Cave's mother) could affect petitioners' case were communicated to Mr. Cave orally and in writing in this same order. *Id*. at 2, n.3. Petitioners filed a supplemental report from Dr. Cave, her curriculum vitae ["CV"], and some references on January 23, 2012.[20]

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[16] Doctor Amy Holmes was in practice for about three years with Dr. Stephanie Cave, petitioner's expert witness in this case. This period of practice overlapped with the period in which Dr. Holmes treated A.H.M. Tr. at 24.

[17] This was the last "paper" filing in this case, as I ordered the case converted to electronic filing once Mr. Cave's office had received the certification necessary to make electronic filings. *See* Order, issued Aug. 23, 2011.

[18] Petitioners failed to assign an exhibit number to this report at the time of filing. It was subsequently designated Pet. Ex. 11. *See* Order, issued Dec. 7, 2011.

[19] I provided petitioners with a non-exhaustive list of the significant problems with Dr. Cave's expert report: (1) the inclusion of an allegation which, if correct, would render the petition untimely filed; (2) the failure to include her CV or any of the referenced materials, to identify tests she was relying upon for her diagnosis of mitochondrial dysfunction, to identify the vaccines and "toxic components" to which she referred, and to identify the medical records that supported her assertions; (3) the identification of medical records which did not reflect what she claimed they said; (4) her reliance on theories of causation considered and reject in the OAP Theory 2 test cases without providing new evidence; and (5) the failure of the report to address the *Althen* factors. Order, issued Dec. 6, 2011, at 2-3. This was neither Dr. Cave's first expert report in the Vaccine Program nor the first report that required supplementation to meet basic requirements. *See Blake v. Sec'y, HHS*, No. 03-31V, 2014 WL 2769979, at *3 (Fed. Cl. Spec. Mstr. May 21, 2014).

[20] This filing included Dr. Cave's supplemental expert report (labeled as Pet. Ex. 11-a), her CV, and some of the supporting medical literature referenced in the report. Unfortunately, the supporting documents included only a few complete medical journal articles, with other references consisting of selected pages or the abstracts of the complete reference. A few days later, petitioner filed these same documents on CD, this time labeling Dr. Cave's CV as Pet. Ex. 12. The references were labeled as Pet. Ex. 11-a, Tabs 1-23. Petitioners filed the complete version of some of the references on May 3, 2013 (*see* Pet. Exs. 22-

A few weeks later, Mr. Cave stated he had not produced updated medical records establishing that A.H.M. had the claimed diagnosis of a mitochondrial dysfunction because he still had not located his clients.[21] *See* Status Report, filed Feb. 13, 2012. In early April 2012, Mr. Cave reported that he had located his clients. *See* Order, issued Apr. 4, 2012, at 1. Three months later, petitioners filed more medical records and a statement of completion.[22] None of the records filed indicated that A.H.M. had the diagnosis of a mitochondrial dysfunction on which Dr. Cave's opinion rested.[23]

On March 30, 2012, respondent filed a second Vaccine Rule 4 report addressing the amended claim. Respondent again recommended against compensation. Respondent's 2012 Rule 4 Report at 10. On August 23, 2012, respondent filed the expert report and CV of Dr. Max Wiznitzer. *See* Respondent's Exhibits ["Res. Ex."] A-B. The accompanying medical literature was filed as Res. Ex. A, Tabs 1-6.

A Vaccine Rule 5 status conference was held on October 12, 2012. Possible hearing dates were among the matters discussed.[24] In January 2013, I set the case for a May 10, 2013 entitlement hearing. Pre-Hearing Order, issued Jan. 29, 2013, at 1. That order required each of the parties to file a list of witnesses they intended to call, and an indication whether the witnesses would be testifying in person, by telephone, or by video teleconference. I also informed the parties that: "*Failure of a party to list a witness shall result in the exclusion of that witness's testimony at the hearing absent a showing of a compelling reason for the failure.*" *Id.* at 2 (emphasis in original). The parties filed their witness lists on April 22, 2013, and pre-hearing submissions, including additional medical literature, on May 3 and 14, 2013. These filings indicated Dr. Cave would be testifying for petitioners and Dr. Wiznitzer for respondent.

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34), but never filed complete copies of all of them.

[21] It appears that Mr. Cave was unable to find his clients during the entire nine months between filing the amended petition (which contained the unexecuted affidavit) in July 2011 and April 2012.

[22] Petitioners labeled these additional records as Ex. 2, pp. 114-47, in spite of the fact that two records were already designated as Pet. Ex. 2. The "official" Pet. Ex. 2, filed with the original petition in March 2002, contained Ms. Miller's labor and delivery records from Norton Hospital, part of the Alliant Health System. The records filed in July 2012 and labeled as Pet. Ex. 2 showed testing for A.H.M. from 2000-2002 at the Kosair Children's Hospital, also part of the Alliant Health System. Unfortunately, the pagination in the 2012 Ex. 2 was not consecutive to the earlier version. I have thus designated this 2012 filing as Pet. Ex. 2a, pp. 1-34. *See* Order, issued Aug. 4, 2015. This order serves to clarify the exhibit numbers for various documents filed. Citations in this decision are based on the exhibit numbers set forth in this order.

[23] Although Dr. Cave diagnosed A.H.M. with mitochondrial dysfunction, she had not seen A.H.M. in about 10 years at the time she did so. One of the many reasons for ordering petitioners to file a new expert report or a supplemental one from Dr. Cave was that Dr. Cave did not explain how she reached this diagnosis. *See* Order, issued Dec. 6, 2011, at 2.

[24] Although a minute entry reflecting this status conference was entered, no order documenting the matters discussed was filed.

At the beginning of the hearing, I noted for the record that petitioners were not present. At that point, Mr. Cave stated that they were not present because he thought the hearing was limited to expert testimony alone. Transcript ["Tr."] at 4. He added that if petitioners were to testify, he would have expected me to travel to petitioners' location because travel would have been burdensome for the parents.[25] Tr. at 5-6. He acknowledged that I had agreed to the proposed hearing date (which was not one of the dates during which I had indicated that I was available) only if the hearing could be conducted in Washington, DC.[26] He averred that petitioners had wanted to testify at the hearing and that he could arrange to take Ms. Miller's testimony telephonically. He indicated that he and Dr. Cave had talked with Ms. Miller the evening prior to the hearing. Tr. at 6-7. Later Mr. Cave acknowledged that he had not been in contact with A.H.M.'s parents for some months before the hearing. Tr. at 9. When I asked him how he knew the parents wanted to testify or whether he had consulted with his clients about any desire to testify, he did not answer my question. *Id.*

Although I directed Mr. Cave to talk again with Ms. Miller and determine if she had anything to add to her affidavit (Tr. at 12-13), he did not request to have Mr. or Ms. Miller testify, telephonically or otherwise. Mr. Cave did not raise the issue of petitioners' testimony in petitioners' post-hearing memorandum. *See* Petitioners' Post-Hearing Memorandum, filed Dec. 16, 2013 ["Pet. Post-Hearing Memo"].

The only hearing witnesses were Drs. Cave and Wiznitzer. Based on discussions during the hearing involving diagnostic criteria for mitochondrial disorders, I filed a complete copy of the medical journal article discussed, N. Wolf & J. Smeitink, *Mitochondrial disorders--A proposal for consensus diagnostic criteria in infants and*

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[25] Mr. Cave did not explain why travel would have been "burdensome." Later in the hearing, Dr. Cave commented that A.H.M.'s parents were separated. Tr. at 62. Doctor Cave asserted that, per conversations with A.H.M.'s mother, she was doing well, participating in school and extracurricular activities such as cheerleading. Tr. at 50-51. Thus, in spite of A.H.M.'s ASD diagnosis, there did not appear to be any impediment to petitioners' travel.

[26] Based on Mr. Cave's startling claim that he misunderstood the nature of the hearing, I asked the law clerk present at the Rule 5 status conference on September 27, 2012 to read his notes from the status conference into the record. Those notes reflected a discussion concerning the locations of the parties and witnesses and where the hearing should be held. The notes reflected that Mr. Cave and his mother, Dr. Cave, worked in Louisiana; petitioners lived in Florida; respondent's expert was in Ohio; and respondent's counsel and I were in Washington, DC. The notes contained my statement that: "Petitioners do not have to be present [at the hearing] but they are welcome to testify. They are also welcome to observe via videoconferencing or listen in on the telephone." Tr. at 10. Respondent's counsel agreed that the notes comported with her recollection of the matters discussed at the status conference. Tr. at 7-8, 11. The notes also reflected that I had provided the parties with several weeks in March and April 2013 that I had available for the hearing, but apparently these weeks were unacceptable to the parties, as the prehearing order setting what was characterized as an "entitlement hearing" was subsequently scheduled for May 10, 2013. It is my recollection that Mr. Cave did not oppose the hearing location, and in fact commented that he had a brother residing in the Washington, DC area, and that he and Dr. Cave would welcome the opportunity to visit with him.

children, NEUROL. 59 (9): 1402-05 (2002), Court Ex. I [hereinafter "Wolf & Smeitink, Court Ex. I"], after the hearing.[27] I also provided the parties the opportunity to file any comments, supplemental reports from their respective experts, or arguments concerning Court Exhibit I. The parties filed their simultaneous post-hearing submissions on December 16, 2013, including supplemental reports from their experts and additional medical literature.[28] *See* Pet. Exs. 35-37; Res. Exs. D and D, Tab 1.

II. The Changing Nature of Petitioners' Vaccine Injury Claim.

In their amended petition, petitioners claimed that A.H.M. suffers from mitochondrial dysfunction and a "progressive decrease in brain function with encephalopathy." Petition at 1. Petitioners identified the varicella vaccination received on June 4, 1999, and the MMR, oral polio ["OPV"], "DTP/Hib"[29] vaccinations received on September 15, 1999, as causal of her "encephalopathic condition attendant symptoms." Petition, ¶ 1.r. The petition also alleged that A.H.M. "suffered mitochondrial dysfunction and that her continuing problems are a sequella [sic] of the mitochondrial dysfunction." Petition, ¶ 3, at 5. Presumably, this paragraph incorporates the claim that A.H.M.'s mitochondrial dysfunction was caused or aggravated by the vaccinations identified as causal, but the petition did not state that explicitly. Precisely how petitioners squared their claim in the petition that A.H.M. suffered a progressive decrease in brain function with their claim that A.H.M. improved on treatment and now has fewer deficits was never elucidated.

Petitioners and their expert have modified their claim on several occasions since filing the amended petition. In her initial expert report, Dr. Cave appeared to claim that different vaccines were causal. She asserted that A.H.M.'s mitochondrial dysfunction first manifested after vaccinations administered in November 1998 when A.H.M. was six months old,[30] followed by language problems after a varicella vaccination (for which no

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[27] The article's supplemental materials (which define the parameters of the clinical and laboratory diagnostic criteria) can be found on the www.neurology.org website by clicking on the link for the Nov. 12, 2002 issue (Volume 59, Issue 9). Petitioners filed the abstract of the Wolf & Smeitink article on January 6, 2012 as Pet. Ex. 11-a, Tab 9. Respondent filed a portion of the supplemental material (the general criteria) on May 14, 2013 as Res. Ex. C and on December 16, 2013 as Res. Ex. D, Tab 1. Res. Ex. D, Tab 1 is a clearer copy of the Wolf general criteria filed as Res. Ex. C on May 14, 2013, and thus any citations will be to the copy filed as Res. Ex. D, Tab 1.

[28] This deadline was established after two unopposed requests for a delay in the post-hearing briefing schedule were granted. *See* Motions, issued Sept. 6, 2013 and Dec. 2, 2013.

[29] This petition repeated the error in the original petition regarding the type of diphtheria, pertussis, and tetanus vaccine A.H.M. received.

[30] *See* Pet. Ex. 11 at 1 (the first expert report from Dr. Cave). Reliance on symptoms occurring after the November 1998 vaccinations would render the claim untimely. § 16(a) (Vaccine Act's statute of limitations). In spite of my cautions about making this assertion (*see* Order, issued Dec 6, 2011, at 2), Mr. Cave appeared to be asserting during his cross examination of Dr. Wiznitzer that the hepatitis B vaccine A.H.M. received at birth played a causal role (*see* Tr. at 205-06).

date was specified), and that A.H.M. "lost touch with her surroundings and became encephalopathic" after vaccinations administered on September 15, 1999. Pet. Ex. 11 at 1.

Less than one month prior to hearing, petitioners argued for the first time that they also had proven a "Table" injury[31] claim of encephalopathy. *See* Petitioners' Pre-Hearing Memorandum, filed Apr. 22, 2013 ["Pet. Pre-Hearing Memo"], at 4-9. Additionally, they separated their off-Table claim into two claims (one for encephalopathy and one for mitochondrial dysfunction), treating each as an independent claim. *Id.* at 9, 11. They alleged that A.H.M.'s mitochondrial dysfunction was either caused or aggravated by the vaccines she received. *Id.* at 11.

In later filings, petitioners focused on a comprehensive off-Table injury claim involving mitochondrial dysfunction and encephalopathy. *See* Pet. Post-Hearing Memo at 5-8. They did not address the Table injury again but did "adopt and incorporate" the arguments set forth in their pre-hearing brief. Post-Hearing Memo at 1.

Petitioners' hearing presentation and post-hearing arguments were not well-developed or presented. In spite of my cautions during several status conferences and in orders that I did not intend to permit re-litigation of the rejected OAP test case theories, absent new evidence, Dr. Cave presented testimony about mercury, oxidative stress, and brain inflammation that had been heard and rejected in the OAP test cases, without producing any new evidence.[32] The presentation of their new mitochondrial

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[31] A "Table" injury is an injury listed on the Vaccine Injury Table, 42 C.F.R. § 100.3, corresponding to the vaccine received within the time frame specified. If a Table injury is established, vaccine causation is presumed. § 11(c)(1)(C)(i). In contrast, proof of causation is required for "off-Table" injury claims. § 11(c)(1)(C)(ii). An "off-Table" injury is any injury not listed on the Vaccine Injury Table, 42 C.F.R. § 100.3 or an injury listed on the Table but occurring outside the time frame specified.

[32] In the Theory 1 test case litigation, one of petitioners' experts, Dr. Marcel Kinsbourne, opined that the measles virus "caused an inflammatory process in the brain leading to encephalopathy." *Snyder*, 2009 WL 332044, at *87. He offered a related theory in both the Theory 1 and Theory 2 test case litigation, claiming that the measles virus in the MMR vaccine (Theory 1) or the mercury in thimerosal-containing vaccines ["TCVs"] (Theory 2) caused neuroinflammation in the brains of autistic children which produced excess levels of an excitatory neurotransmitter, glutamate, resulting in a state of over-arousal manifested by the autistic behaviors exhibited by these children. *See Snyder*, 2009 WL 332044, at *87-88; *Dwyer*, 2010 WL 892250, at *27. To support the portion of this theory involving brain inflammation, Dr. Kinsbourne relied on the Vargas study (the abstract of which was filed in this case as Pet. Ex. 11-a, Tab 14) to demonstrate that an inflammatory process involving glial activation was found on autopsy in the brains of autistic children. *Snyder*, 2009 WL 332044, at *87 and n.274. Doctor Kinsbourne claimed that activated microglia released proinflammatory cytokines which caused astrocytes to release excess glutamate. *Id.* at *88. Doctor Wiznitzer, respondent's expert in this case, also testified in the test cases. He disagreed with Dr. Kinsbourne's interpretation of the conclusions of the Vargas study, pointing out that the authors indicated that the inflammation seen could have been the effect of the brain pathology found, not the cause of it. *Id.* at *88.

The test case evidence showed that the brain damage caused by the measles virus was "an entirely different type of damage than is found in autism." *Id.*, at *93. Moreover, Dr. Kinsbourne's "excess

disorder theory was highly speculative, poorly explained by Dr. Cave, and did not fit the facts of petitioners' case. She also contended that monosodium glutamate ["MSG"], a component of the varicella vaccine, was "proinflammatory and highly destructive to cells," citing to Pet. Ex. 28,[33] an article that reiterated the excitotoxicity theory presented and rejected in the OAP test cases. Pet. Ex. 11-a.

III. Expert Qualifications, Bias, and Credibility Challenges.

A. Petitioners' Experts.

1. Dr. Amy Holmes.

Technically, petitioners presented reports on causation from two physicians: one from Dr. Amy Holmes, filed with the original petition, and the more recent reports from Dr. Stephanie Cave. Although Dr. Holmes also offered an opinion regarding the cause of A.H.M.'s condition (*see* Affidavit, Pet. Ex. 7), she is better categorized as a treating physician in this case. As I informed petitioners in 2011, Dr. Holmes' affidavit was not sufficient to establish causation. *See* Order, issued July 28, 2011, at 1.

Doctor Holmes did not provide a supplemental report or testify at hearing. She did, however, treat A.H.M. from May 2000 until she retired in 2002. *See* Pet. Ex. 8; Tr. at 24. Thus, I viewed her statements regarding causation contained in the medical records and affidavit as the opinion of one of A.H.M.'s treating physicians and analyzed and weighed her opinion accordingly. *See* Pet. Exs. 7, 8 (affidavit and medical records).[34]

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glutamate theory lacked any evidentiary basis, as there was no evidence of excess glutamate in autistic children. *Id.* at *93.

During the Theory 2 test case litigation, petitioners offered an additional theory presented by Dr. Richard Deth, that the mercury in TCVs affected biochemical processes causing metabolic problems which produced oxidative stress. *Dwyer*, 2010 WL 892250, at *27. He opined that this oxidative stress affected "neuronal function in the areas of attention and cognition." *Id.* I concluded that respondent's experts on oxidative stress were far more qualified to opine on vaccinations and oxidative stress than Dr. Deth. *Id*. at *13-15. I ultimately concluded that the theories advanced involving mercury and oxidative stress to be "illogical, contrary to the weight of the evidence, and, ultimately, unpersuasive." *Id.* at *165.

I informed petitioners that, based on the test case decisions, they needed to provide "different evidence or theories not presented in the test cases" in order to proceed. Order, issued Apr. 29, 2011, at 2. Throughout this proceeding, petitioners and their expert continued to rely on theories offered and fully litigated in the OAP test case litigation. Doctor Cave was even less qualified to opine on oxidative stress caused by vaccines than Dr. Deth, and ultimately proffered little that was new or different from the matters presented in the OAP test cases, other than her mitochondrial dysfunction theory.

[33] R. Blaylock & A. Strunecka, *Immune-Glutamatergic Dysfunction as a Central Mechanism of the Autism Spectrum Disorders*, CURRENT MEDICINAL CHEMISTRY, 16(1): 1-14 (2009). *See* n.32, above for a discussion of the excess glutamate theory.

[34] It appears that, shortly after Dr. Holmes wrote her affidavit on causation, she may have filed a vaccine

One of the studies relied upon by the petitioners in the OAP test cases which compared mercury content of hair samples from autistic and non-autistic children was authored by Dr. Holmes.[35] Doctor Cave testified that A.H.M. was one of the ASD children in this study. Tr. at 266-68. Although Dr. Holmes' study was not filed in this case, Dr. Cave testified about it on rebuttal. Tr. at 266.

2. Dr. Stephanie Cave.

Prior to attending medical school, Dr. Cave earned a Master's degree in clinical chemistry and taught clinical biochemistry for five years at Louisiana State University ["LSU"] in Baton Rouge. Tr. at 14-15; Pet. Ex. 12 at 1. She attended the LSU School of Medicine from 1979-1983 (*id.*) and completed her residency at the Earl K. Long Memorial Hospital in Baton Rouge from 1983-86 (Tr. at 15; Pet. Ex. 12 at 1). Since earning her medical degree, she has practiced family medicine and is currently in private practice as a family physician in Baton Rouge. Tr. at 16-17; Pet. Ex. 12 at 1. She is board certified in family medicine.

Her only training in several of the disciplines in which she offered expert opinions is from medical school classes and blocks of training during her family practice residency. Tr. at 15-16. Mr. Cave creatively referred to these blocks of training as "subspecialties," a term more appropriately applied to training in addition to, rather than as part of, residency training. Doctor Cave offered opinions grounded in several medical specialties in this case, including neurology, genetics, toxicology, mitochondrial disease, and immunology, all of which were clearly beyond her area of expertise.

The majority of Dr. Cave's patients are children who come to her for treatment after being diagnosed elsewhere with autism. Tr. at 17, 107. She estimated she has

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injury claim involving an autism spectrum disorder on behalf of a minor child. Mr. Cave represented Mr. Charles Weinstein and Amy Holmes in the case. *See Hebert v. Sec'y, HHS*, 66 Fed. Cl. 43 (2005) (listing the case of Charles Weinstein and Amy Holmes v. Sec'y, HHS, No. 02-2059V, as one of the trailing cases in a combined motion for review of the special master's decisions dismissing as untimely a number of claims filed by Mr. Cave). Judge Baskir denied the motions for review. *Hebert*, 66 Fed. Cl. at 43, 49.

[35] The study was based on hair samples from baby haircuts of autistic patients in Dr. Holmes' practice, and control samples obtained from non-autistic children. However, the validity of any conclusions Dr. Holmes drew was undercut by the levels of mercury purportedly found, "with the control children having mercury levels almost 15 times higher than the mean U.S. level." *Dwyer,* 2010 WL 892250, at *102. More importantly, five studies attempted to duplicate Dr. Holmes' results and were unable to do so. *Id.* Scientific evidence is evidence "grounded in the methods and procedures of science." *See Daubert v. Merrell Dow Pharmaceuticals*, 509 U.S. 579, 590 (1993). When determining scientific validity, one of the factors to consider is whether a technique or theory has been or can be tested. *Id*. at 593. The fact that Dr. Holmes' findings could not be replicated severely undercuts their reliability. Although Dr. Holmes' study was not filed in this case, Dr. Cave appeared to rely on it. *See* Tr. at 266. In the Theory 2 test cases, the special masters similarly found the study to be of little value. *See, e.g.*, King, 2010 WL 892296, at *48-49.

treated more than 10,000 children with autism since 1996. Tr. at 17; *see also* Pet. Ex. 12 at 1.

Doctor Cave's treatment of children with ASD involves, in her words, "normalizing" their "cellular chemistry." Tr. at 22. She still prescribes chelation to remove heavy metals in spite of the lack of any controlled study showing it can reduce ASD symptoms or cure autism. Tr. at 23, 74. During her testimony, she acknowledged that chelation agents do not cross the blood brain barrier, and thus cannot remove mercury from the brain. Tr. at 74. *See also Dwyer*, 2010 WL 892250, at *104.

Doctor Cave testified before a Congressional subcommittee hearing on the issue of mercury in vaccines. She also presented testimony to the Louisiana legislature regarding thimerosal and the MMR vaccine and their connection to autism. Tr. at 18; Pet. Ex. 12 at 6. She has spoken at numerous conferences regarding a relationship between autism and vaccines. She authored a book on vaccines published in 2001, which was revised in 2010, and authored several articles published between 2000 and 2008, but none appeared to involve original research. Tr. at 19-21; Pet. Ex. 12 at 6. In her book, Dr. Cave recommended a revised schedule for childhood vaccinations, with no vaccines administered until a child is four months old and administering the usual childhood vaccinations over a more extended period. Tr. at 58-62. She did not identify any research upon which her recommendations for the alternative vaccination schedule were based.[36]

B. Respondent's Expert: Dr. Max Wiznitizer.

Doctor Wiznitzer earned his medical degree in 1977 from Northwestern University. Tr. at 116; Res. Ex. B at 1. He participated in a three year residency at the Cincinnati Children's Hospital and a one year child development fellowship at the Cincinnati Center for Developmental Disorders. *Id.* He then trained in child neurology at the University of Pennsylvania and completed a two year National Institutes of Health fellowship in disorders of cognitive or higher cortical function in children at the Albert Einstein College of Medicine. Tr. at 116; Res. Ex. B at 1-2. He is board certified in pediatrics, neurology (with special qualification in child neurology), and neurodevelopmental disabilities. Tr. at 116-17; Res. Ex. B at 5.

At the time of the hearing, Dr. Wiznitzer was employed as a pediatric neurologist at the Rainbow Babies and Children's Hospital in Cleveland, Ohio. Tr. at 115-16; Res. Ex. B at 1. He was also an associate professor at Case Western Reserve University,

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[36] Tr. at 58-60. She testified that her book (titled "*What Your Doctor May Not Tell You About Vaccinations*") "gives a schedule for vaccines that [she] think[s] is safer than the schedule that they're using." Tr. at 59. She credited her use of this revised vaccination schedule in her own practice with preventing any of her patients from developing autism. Tr. at 59. She also testified about an unidentified study that found delaying vaccinations reduced asthma by 42%. Tr. at 60.

School of Medicine. Tr. at 116; Res. Ex. B at 2. His clinical practice is not exclusively devoted to autism treatment, but he sees many patients with ASD, and he routinely diagnoses autism spectrum disorders. Tr. at 117-18. Working with mitochondrial disease specialists, he also diagnoses mitochondrial disease. Tr. at 118.

Doctor Wiznitzer has authored or co-authored approximately 60 peer reviewed articles, many of which deal with ASD. He also peer reviews papers and serves on the editorial boards for various medical journals. Tr. at 117; Res. Ex. B at 6. Doctor Wiznitzer testified for the respondent in the Theory 1 OAP test cases and was described in the test case decisions as experienced, credible, and persuasive.[37]

Doctor Wiznitzer testified that he reviews medical records for the Department of Vaccine Injury Compensation (DVIC) in Vaccine Act cases and, in some of those reviews, he has found that the child experienced a Table encephalopathy. Tr. at 209-10. He was a member of a panel that developed criteria for definitions for conditions such as encephalitis, multiple sclerosis, and acute disseminated encephalomyelitis for the Vaccine Adverse Events Reporting System. Tr. at 207.

C. Issues Concerning Expert Qualifications.

Several issues concerning the backgrounds and possible biases of the witnesses that testified at the hearing emerged before and during the hearing.

1. Financial Issues.

Both Drs. Cave and Wiznitzer have previously provided opinions and testified in other vaccine injury cases. When petitioner's counsel, Mr. Cave, questioned Dr. Wiznitzer regarding the percentage of his income that he earns from testifying,[38] I

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[37] *Hazelhurst*, 2009 WL 332306, at *12 (finding Dr. Wiznitzer to be "a credible and persuasive witness"); *Cedillo*, 2009 WL 331968, at *84 (noting Dr. Wiznitzer's extensive experience in treating children with autism); *Snyder*, 2009 WL 332044, at *11 (noting that Dr. Wiznitzer and other respondent's experts "had far more experience in treating children with ASD and much more extensive research experience in and publications concerning ASD").

[38] Mr. Cave sought information about Dr. Wiznitzer in addition to the information contained in his CV. He argued that he would be entitled to such information under Fed. R. Civ. P. 26, and argued that this rule applied in Court of Federal Claims cases. That federal rule does not directly apply to cases filed in the Court of Federal Claims. And, although Court of Federal Claims Rule 26 is nearly identical to Fed. R. Civ. P. 26, discovery in the Vaccine Program is more circumscribed. Had Mr. Cave asked for this information prior to the hearing, and explained why it was relevant and necessary to a fair hearing, he might have made a better case for obtaining it. *See* § 12(d)(2)(E) (indicating special masters are not bound by the usual rules of discovery); Vaccine Rule 7 (indicating there is no right to discovery but allowing a party to move for formal discovery, orally in a status conference or by motion, if the party believes informal discovery has not been sufficient).

judicially noted that Dr. Wiznitzer often testified for respondent in vaccine cases and observed that he had testified before me approximately five times in the previous year.[39]

While I have considered Dr. Wiznitzer's compensation for his frequent appearances as an expert witness for respondent and the fact that he has authored opinions in additional cases in which his testimony was not required, virtually all witnesses who testify as experts are compensated. Both experts in this case have that common financial interest, regardless of the outcome of the case. In view of their extensive involvement in patient care, neither of these witnesses could be classified as a "professional witness."

However, Dr. Cave's practice appears to depend in large measure on her alternative medical approach to the treatment of ASD. Thus, she has a financial interest in establishing the bona fides of her treatment methods, in that mainstream physicians do not claim to prevent or cure autism. Doctor Cave does. Establishing, via a court decision, that vaccines can cause ASD or ASD-like symptoms could be expected to increase her book sales, as well as have a similar impact on her private medical practice. Thus her financial interest in the outcome of this case is likely greater than Dr. Wiznitzer's.

2. Qualifications to Opine.

By any measure, except perhaps the sheer number of ASD patients seen, Dr. Wiznitzer's qualifications to opine on the causes of ASD and whether A.H.M. has ASD, or a mitochondrial dysfunction masquerading as ASD, far exceed those of Dr. Cave. Doctor Wiznitzer is more highly credentialed in relevant areas such as pediatrics and neurology than Dr. Cave. Then-Special Master (now Judge) Sweeney took note of the difference in qualifications of these same two experts in a prior case. *See Nilson v. Sec'y, HHS,* No. 98-797V, 2005 WL 6122524, at *9, 13-14, 20 (Fed. Cl. Spec. Mstr. Aug. 31, 2005), ***aff'd,*** 69 Fed. Cl. 678 (2006). Furthermore, Dr. Wiznitzer has conducted research on ASD and co-morbid conditions, has peer-reviewed publications on ASD and related topics, and has been awarded research grants in relevant fields; Dr. Cave has not.

Despite her lack of training, research, or board certification in relevant disciplines, Dr. Cave opined on a range of medical conditions, but offered little support for her opinions. In contrast to Dr. Cave's sweeping statements about causation (*see, e.g.*, Tr. at 70-71) and opinions in areas in which she has neither the training nor the experience to opine credibly, Dr. Wiznitzer candidly acknowledged the limits of his expertise, often noting when he did not feel qualified to opine on a medical issue. The wide latitude

[39] Given Dr. Wiznitzer's expertise in autism and my assignment as one of the three special masters who were hearing autism cases at the time of this hearing, his frequent appearances before me would be expected.

afforded to an expert "is premised on an assumption that the expert's opinion will have a reliable basis in the knowledge and experience of his discipline." *Daubert*, 509 U.S. at 592.[40] This assumption does not apply when an expert is opining outside of her discipline.

3. Bias.

A more significant problem with Dr. Cave's testimony was apparent from the time her first expert report was filed: she was retained to offer expert testimony in a case in which her son was counsel of record. I informed Mr. Cave of my concerns that this relationship might impact on Dr. Cave's credibility, but he elected to proceed with Dr. Cave as his expert witness despite them. *See* Order, issued Dec. 6, 2011, at 2, n.3.

Familial relationships alone may raise the issue of bias.[41] Strictly speaking, the familial relationship may say more about the judgment of petitioners' counsel in calling his mother as an expert witness than about Dr. Cave herself. But, this appears to be their regular practice, rather than an isolated instance: my experience in other cases in which Mr. Cave represents petitioners and Dr. Cave has treated or offered opinions on causation suggests frequent cross-referral of cases between the legal and medical

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[40] The Federal Circuit has approved the use of the *Daubert* factors by special masters in evaluating the reliability of and the weight assigned to expert opinions in Vaccine Act cases. *See Moberly v. Sec'y, HHS*, 592 F.3d 1315, 1324 (Fed. Cir. 2010); *Andreu v. Sec'y, HHS,* 569 F.3d 1367, 1379 (Fed. Cir. 2009); *Terran v. Sec'y, HHS*, 195 F.3d 1302, 1316 (Fed. Cir. 1999).

[41] Under common law, "a witness may be impeached by showing that he or she is biased, has an interest in the outcome of the litigation, is prejudiced in some relevant way, or has a motive to testify in a particular way." *Behlar v. Hanlon*, 199 F.R.D. 553, 556-57 (D. Md. 2001) (citing *United States v. Abel*, 469 U.S. 45, 49-52 (1984)). Family relationships, business relationships, and financial interest in the outcome of a case have been named as examples of relationships or circumstances that permit a finding of bias or prejudice. *Behler*, 199 F.R.D. at 557 (quoting 1 Michael Graham, HANDBOOK OF FEDERAL EVIDENCE, § 607.7 (4th ed. 1996)). The family relationship may be between the witness and a party to the litigation or may be between the witness and an individual who is involved or has an interest in the litigation. *See, e.g.*, *In re Pennsylvania Footwear Corp. v. Midatlantic Bank, N.A.*, 204 B.R. 165 (Bankr. E.D.Pa. 1997) (finding the expert's property appraisal suspect since it was originally prepared for the appraiser's son who was considering purchasing the property). In Vaccine Act cases, counsel may be paid for their services by the Program, regardless of the outcome of the case, as a non-prevailing petitioner may have attorney's fees and costs paid, provided the case was brought in good faith (a very low standard) and upon a reasonable basis. § 15(e)(1); *see Sebelius v. Cloer*, 133 S.Ct. 1886, 1896-97 (2013) (holding even an untimely filed petition "may qualify for an award of attorney's fees if it is filed in good faith and there is a reasonable basis for its claim"). An expert opinion supporting causation is one factor used by special masters in determining if there is a reasonable basis for the claim. *See Heath v. Sec'y, HHS*, No. 08-86V, 2011 WL 4433646, at *12 (Fed. Cl. Spec. Mstr. Aug. 25, 2011) (holding an expert report provided support for a finding of reasonable basis until it was determined the report was based on erroneous facts). Thus, obtaining an opinion on causation from Dr. Cave could make it easier for Mr. Cave to be compensated for bringing a questionable claim for vaccine compensation. Such compensation could include Dr. Cave's fees as well.

practices.[42] *See, e.g., Mooney v. Sec'y, HHS*, No. 05-266V, 2013 WL 3874444, at *3, 4 n.14 (Fed. Cl. Spec. Mstr. July 3, 2013).

Doctor Cave's poor qualifications to opine as an expert and her familial relationship to petitioners' counsel do not preclude her appearance as a witness under the more flexible evidentiary standards in effect in Vaccine Act cases[43] but both bear on the weight accorded to her testimony. However, the most troubling aspects of Dr. Cave's opinion and testimony were their content. I discuss Dr. Cave's opinions in more detail after setting forth A.H.M.'s relevant medical history, in order to place these concerns in context.

IV. A.H.M.'s Medical and Developmental History.

A. Early Growth and Development.

A.H.M. was born in mid-May 1998 after an unremarkable pregnancy.[44] Pet. Ex. 1, pp. 1-2. Her Apgar scores were 8 and 9.[45] Pet. Ex. 1, p. 2. She received a hepatitis B vaccination the day she was born. Pet. Ex. 5, p. 1. Until she was eight months old, she saw her pediatrician only for well-child visits and was assessed as having normal growth and development. Pet. Ex. 4, pp. 9-10. She received routine childhood vaccinations with no recorded reactions.[46]

Numerous videos show A.H.M. as an alert and healthy infant who occasionally smiled at the person holding or filming her. *See* Titles 1-6.[47] In a video recorded around Christmas 1998, someone was taking A.H.M.'s photograph. A.H.M. looked in

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[42] Doctor Cave has been the only medical expert in the five autism cases Mr. Cave has continued to litigate since the decisions in the OAP test cases. *See, e.g.*, *Blake*, 2014 WL 2769979, at *3 n.11. In addition to the *Blake* and *Mooney* cases, Mr. Cave was or is counsel of record in No. 02-2056V, 03-775V, and 08-108V. At least one report by Dr. Cave was filed in each of these cases.

[43] *See* § 12(d)(2). Vaccine Act proceedings were intended to be, and generally are, less adversarial and more informal, expeditious, and flexible than proceedings in civil litigation conducted outside the Program.

[44] Because Ms. Miller had miscarried three times and lost an anencephalic child at 26 weeks, her pregnancy with A.H.M. was considered "high risk." Pet. Ex. 6, p. 1.

[45] An Apgar score is a numerical assessment of a newborn's condition (with lower numbers indicating problems), usually taken at one minute and five minutes after birth. The score is derived from the infant's heart rate, respiration, muscle tone, reflex irritability, and color with from zero to two points awarded in each of the five categories. DORLAND'S ILLUSTRATED MEDICAL DICTIONARY ["DORLAND'S"] at 1682 (32d ed. 2012).

[46] Pet. Ex. 5, p. 1. A.H.M. received a second hepatitis B vaccination at her one month well-child visit; DTaP, haemophilus influenza type b ["Hib"], and inactivated polio ["IPV"] at her two and four month well-child visits; and DTaP, Hib, and hepatitis B at her six month well-child visit. *Id.*

[47] Because the videotape submitted consisted of short clips usually only a minute or two in length, I will not reference the elapsed time, using only the assigned title number as the identifying reference.

the direction of the camera when her name was called. *See* Title 8. Doctor Wiznitzer testified she was "looking at the individual talking to her" when he discussed this video.[48] Tr. at 132.

At her nine month well-child visit on February 17, 1999, A.H.M. was assessed as having normal growth and development. She was pulling up to stand and saying "mama" and "dada." Pet. Ex. 4, p. 8. She received no vaccinations at this visit.

Shortly before her first birthday, A.H.M. was taken to the emergency room ["ER"] with vomiting, diarrhea, and a fever of 103.4° Fahrenheit.[49] Pet. Ex. 16, p. 2. She was diagnosed with febrile illness and gastroenteritis. *Id.*, p. 3. This ER visit occurred a week prior to the first vaccination alleged as causal by petitioners. It appears to be the only time petitioners sought emergency medical treatment for A.H.M. during her early childhood.

A.H.M. visited her pediatrician a week later on May 19, 1999 for her one year well-child visit. Recorded milestones reflected that she was walking well and saying "mama" and "dada" with specificity. Pet. Ex. 4, p. 8. She was described as alert and active and assessed as a well-child with normal growth and development. *Id.*

Several undated videos appear to have been filmed at some point during the first half of 1999.[50] The first clip showed A.H.M. interacting with her mother. *See* Title 9. Doctor Wiznitzer described her as "showing some interaction" and babbling, but A.H.M. used no discernable words. Tr. at 132. In the next video, A.H.M. was playing with a phone. As Dr. Wiznitzer testified, A.H.M. sometimes responded with a slight smile but exhibited little vocalization. *See* Title 10; Tr. at 132-33. In the third video segment, she smiled slightly while swinging, and looked at the person filming, but broke off eye contact as the video concluded. *See* Title 11.

B. A.H.M.'s Condition Following the Allegedly Causal Vaccinations.

A.H.M. received the varicella vaccination recommended at her one year well-child visit on June 4, 1999, when she returned to the pediatric practice with her older brother. Pet. Exs. 4, p. 8; 5, p. 1. This vaccination is the first one petitioners alleged as causal.

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[48] Although Dr. Cave indicated she reviewed the videotape submitted by petitioners, she did not make any observations regarding the video evidence. Tr. at 109. Doctor Wiznitzer testified about the significance of various segments as they were played at the hearing. Tr. at 130-140.

[49] Hereinafter, all temperatures will be reported in degrees Fahrenheit.

[50] These videos (Titles 9-11) appear to be in chronological order. Title 8, the video prior to this group, was dated December 24, 1998, and the video (Title 12) following this group was dated July 18, 1999. I thus conclude that Titles 9-11 were most likely recorded at some point between these two dates.

On July 2, 1999, almost a month after the varicella vaccination, A.H.M. was seen for a rash on her legs, hips, and face that had begun about two weeks earlier. She also had a low grade fever, cough, and runny nose. Pet. Ex. 4, p. 7. She was described as alert and active with no fever and was diagnosed with an upper respiratory infection ["URI"] and contact dermatitis. *Id.* Although petitioners pointed to this rash as evidence that A.H.M. suffered a Table encephalopathy (Pet. Pre-Hearing Memo at 6-7),[51] A.H.M.'s pediatrician did not indicate the rash or the fever was connected to A.H.M.'s vaccination.

In videos dated between July 18, 1999 and August 1, 1999, A.H.M. showed more pronounced abnormal behaviors: a lack of vocalization and eye contact, a failure to respond to others, and a fascination with objects. *See* Titles 11-16. Doctor Wiznitzer gave detailed testimony regarding A.H.M.'s behavior, describing it as "distinctly different" than the behaviors he had seen in previously recorded clips. Tr. at 133. He characterized the behavior as typical autistic behavior, but not evidence of an encephalopathy. Tr. at 135-37, 139-40. In the July 18, 1999 video, A.H.M. dropped the electrical cord she was holding and reached for the camera. *See* Title 12. Contrasting her behavior with that seen in Title 8 (recorded around Christmas 1998), Dr. Wiznitzer pointed out that A.H.M. paid more attention to the camera than the person filming. Tr. at 133. He described her behavior as being "distinctly different" from the behavior shown on Title 8. Tr. at 133. In the July 18, 1999 video, A.H.M. was babbling, but no discernable words could be identified. *See* Title 12.

In the July 19, 1999 video, A.H.M. pushed a plastic buggy on the deck while her father watched, her mother filmed, and her brother and three dogs played with a ball. *See* Title 13. She looked at the ball when her brother bounced it and at a toy lawn mower as he pushed it, but showed little interest in her brother or the dogs. *See id.* Doctor Wiznitzer commented on her lack of attention to those around her and noted that she had few vocalizations during the video. Tr. at 133-34.

The next three videos were filmed inside.[52] *See* Titles 14-16. Commenting on Title 14, Dr. Wiznitzer testified that he heard sounds, but concluded that they did not originate from A.H.M. Tr. at 134. Referring to Title 15, he described A.H.M. as holding onto her father, but not looking in his face. *Id.* He pointed to A.H.M.'s continued fascination with the camera in Title 16, which was dated August 1, 1999. *Id.* He described A.H.M. as exhibiting a lack of responsive smiling, facial expression, and attention to others, as well as being unresponsive to her name. Tr. at 134-35.

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[51] A rash is not one of the symptoms used to determine whether the vaccinee experienced a Table encephalopathy. *See* 42 C.F.R. § 100.3(b)(2).

[52] Title 15 is undated, but Titles 14 and 16 are dated July 22, and August 1, 1999 respectively.

When A.H.M. returned to her pediatrician on August 10, 1999, for a cough and fever, Ms. Miller reported that A.H.M. had experienced colds off and on over the prior six weeks, a loose and productive cough for the last few days, fever, increased fussiness when lying down, and a decreased appetite. Pet. Ex. 4, p. 7. Her temperature at the visit was normal, and she was diagnosed with a URI and ear infection. *Id.* She failed to show for a follow-up appointment approximately a week later, suggesting that she had improved on treatment. *Id.*

In early September 1999, A.H.M. visited her pediatrician again for nighttime coughing. *Id.*, p. 6. Her temperature was 100.5°, but she was alert and active. She was diagnosed as suffering from croup. *Id.*

At her next well-child visit on September 15, 1999, A.H.M. still had a cough and runny nose, but her cough was much better. Pet. Ex. 4, p. 6. At this visit, her parents reported a concern about her speech ("doesn't say much"). *Id.* Her pediatrician assessed her as having mild developmental and speech delay. *Id.* A.H.M., then 16 months old, could say four to five words, but she did not seem to understand or follow directions or otherwise indicate her wants and needs. She was not pointing to body parts. However, she walked and ran well, used utensils, and was loving and appropriately affectionate. *Id.* A.H.M. received the allegedly causal DTaP, Hib, OPV, and MMR vaccinations at this visit. Pet. Ex. 5, p. 1.

In medical histories provided almost ten years later, Ms. Miller claimed that A.H.M. suffered a sudden reaction to her September 15, 1999 vaccinations. She described this immediate reaction (occurring within eight hours of the vaccination (*see* Pet. Ex. 18, p. 8) as including a fever of 105° (*see* Pet. Ex. 20, p. 2). However, there is no evidence to support these claims in the contemporaneously-created medical records or in other histories provided during the almost ten year period following the vaccinations.

A.H.M. did not return to her pediatrician after the September 15, 1999 vaccinations until October 5, 1999, when she was seen for a croupy cough and a runny nose. The cough was described as the same one she had during the prior month. Pet. Ex. 4, p. 5. Her temperature was normal, and she was playful, active, and in no apparent distress. *Id.* At a visit on October 21, she was experiencing a low grade fever, not sleeping, and "still coughing all the time." *Id.* She had a slight fever (her temperature was 100° at the visit), and she was again described as playful and active. Her pediatrician assessed her cough as having an allergic component and increased her Zyrtec dosage. *Id.* By her next visit on October 29, she was "[d]oing alot (sic) better" but still had a loose cough during the day. *Id.*, p. 4. Her illness (described as allergic rhinitis) was "much improved." *Id.* No concerns about any reactions to the September vaccinations were recorded at any of these visits. *See id.*, pp. 4-5.

During her testimony, Dr. Cave pointed to these three visits in October 1999 as proof that A.H.M.'s health went "downhill" after her September 15, 1999 vaccinations. Tr. at 30.

C. Referral, Diagnosis, Early Intervention Evaluations, and Therapy.

At A.H.M.'s 18 month well-child visit on November 12, 1999, the "History of Present Illness" section (abbreviated on the form as "HPI") reflected that her parents were still concerned about her speech. A headnote on the form before the HPI section reflected "Speech + Hearing Evaluation – Not talking yet" as the reason for the visit.[53] Pet. Ex. 4, p. 4. A notation in the body of the form reflected a concern that she was "not making progress," presumably referring to A.H.M.'s speech. *Id.* (quotes original). The Assessment/Plan section of the form indicated that the treating physician had concluded that A.H.M. had mild developmental/speech delay for which she would be referred to "1st Steps" for an evaluation. *Id.*

A.H.M. received her First Steps evaluation in her home on December 10, 1999. Pet. Ex. 19, p. 1. Her mother was present and provided the case history. Her father later provided some additional information via telephone. According to the report, her parents' view of the problem was a "possible speech delay." *Id.* Ms. Miller reported that A.H.M. had achieved motor milestones at the appropriate ages. *Id.*, pp. 1-2. The parents' reports of her language differed slightly from the contemporaneous records; petitioners reported that the use of "dada" had begun at 12 months of age, while the contemporaneous record of her one year well-child visit reflected that she was using both "mama" and "dada" with specificity (Pet. Ex. 4, p. 8) and an even earlier record reflected that she said both words at nine months of age (*id*.). Her vocabulary at the time of the evaluation was reported as three to four words. Pet. Ex. 19, p. 2.

The Battelle Developmental Inventory was administered during the home visit that day. This test assesses personal-social, adaptive, motor, communication, and cognitive skills. A.H.M. scored more than two standard deviations below the norm in every area of development assessed. Pet. Ex. 19, p. 2 (chart). A.H.M., then 18 months old, was, on average, functioning at the level of a nine month old infant. Her lowest score was in communication, where she was observed to function at an age-equivalent of six months. Her strongest performance was in motor skills, with an age-equivalent score of 13 months. *Id.* She qualified for participation in the First Steps early intervention program. *Id.*, pp. 5-6. Additional testing and evaluation were recommended. *Id.*, p. 6.

Specific observations during the evaluation included a failure to respond to familiar names, such as "Where's mama?"; inconsistent response to her own name; engaging in parallel play without initiating contact with her brother; a focus on television or videos; failing to attempt to stack blocks or place rings on a post; a lack of response to simple verbal commands; humming instead of babbling or using jargon; a lack of

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[53] This notation that A.H.M. was not talking yet generated considerable discussion during the hearing. Tr. at 27-29, 90-98, 112-13.

gestures (other than lifting her arms to be held); a lack of pointing to desired objects; repetitive play; and failing to reach around a barrier to obtain a toy. Pet. Ex. 19, pp. 2-5.

After the First Steps evaluation, Ms. Miller met with A.H.M.'s pediatrician the same day. She reported that a possible diagnosis of "PDD" was mentioned, and that additional testing would be performed. Pet. Ex. 4, p. 3.

In two undated videos filmed about the same time as the First Steps evaluation (Christmastime 1999), A.H.M. was playing with toys. Titles 17-18. These were the last of the videos that Dr. Wiznitzer discussed at the hearing. *See* Tr. at 136. He described A.H.M. as awake and alert and noted that she played with and manipulated objects in her environment. However, he commented that she ignored the people around her and their attempts to get her attention. Tr. at 135-36. He characterized A.H.M.'s behavior as demonstrating "selective awareness." Tr. at 136.

An in-home speech and language evaluation took place approximately a month later on January 20, 2000. Pet. Ex. 19, p. 7. A.H.M. followed simple directions when they were accompanied by gestures but, without the gestures, she could not do so. She did not understand verbs in context. *Id*., p. 8. A.H.M. communicated nonverbally by pushing, pulling, and pointing. *Id.* She used jargon and said "no" during the evaluation. Based on her parents' assertion that she could use the words "mama," "daddy," and "why," they were also included in scoring the extent of her vocabulary. *Id.*, pp. 7-8. A.H.M. scored higher on the Preschool Language Scale testing administered at this evaluation than she had on the First Steps testing. At 20 months of age, A.H.M.'s auditory comprehension was at a 12 month level, her expressive communication was scored at a 15 month level, and her total language skills were assessed at the 14 month level. *Id.* She was assessed as having mild delay in both receptive and expressive language. Speech therapy was recommended. *Id.*, p. 9.

The language evaluation was followed about a week later by a developmental intervention assessment. Pet. Ex. 19, pp. 10-14. Her fine and gross motor skills appeared to be at age level. Per parental report, A.H.M. could perform cognitive tasks such as identifying some body parts and the neighbor's dog, but she was unable to identify objects in a book. *Id.*, p. 11. A.H.M. showed good eye contact but little interaction with the evaluator. *Id.*, p. 12. She "jabbered a lot," repeating the same vocal patterns over and over, but said few understandable words. *Id.*, pp. 12-13. Ms. Miller's main concerns were centered on A.H.M.'s language development; she reported that A.H.M. did not use many words or communicate her wants and needs very effectively. *Id.*, p. 13. With regard to sensory processing problems, the evaluator reported no "red flags" but added that she "did notice [A.H.M.] wave her fingers, some paper, and a few other things in front of her face." *Id.*, p. 13. A.H.M. "usually had something in each hand and she frequently rubbed her thumbs on the object." *Id.*, p. 12. Developmental intervention was recommended "based on parental concerns, her language delay, and her lack of interaction" with the examiner. Focus on functional play skills was recommended. *Id.*

On February 2, 2000, A.H.M. was evaluated by an interdisciplinary team at the University of Louisville's Child Evaluation Center ["CEC"].[54] Pet Ex. 6, pp. 1-14. After a comprehensive battery of tests, she was diagnosed with "Central Nervous System Dysfunction manifested as muscle hypotonia, deficient motor skills, and sensory processing concerns." She showed "features of Pervasive Developmental Disorder-Not Otherwise Specified" and "global developmental delay."[55] *Id.*, p. 9.

During the evaluation at the CEC, A.H.M. was hard to engage, and showed reduced eye contact and scant shared enjoyment. Pet. Ex. 6, p. 2. She used jargon, but no identifiable words.[56] *Id.*, pp. 1-2. She "showed inconsistent and poor response to her name," seldom sought comfort from her parents, "and engaged in some repetitive play." *Id.*, p. 3. Due to what were described as A.H.M.'s "significant social and communication skill difficulties," the examiners were unable to formally assess her cognitive skills. *Id.* A variety of therapies were recommended and additional diagnostic testing was ordered. *Id.*, pp. 10-14.

Laboratory testing, including genetic testing, metabolic disease screening tests (including tests for storage disorders and urine organic and amino acid screening), and blood testing for lead and thyroid function, was performed as part of the CEC evaluation. Pet. Ex. 6, pp. 15-35. The only abnormality detected on testing was a mild

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[54] The team included a pediatrician, a developmental pediatrician, a psychologist, two speech and language pathologists, and an occupational therapist. Pet. Ex. 6, p. 14.

[55] A.H.M. received a psychological evaluation that included the Baley Scales of Infant Development and the Developmental Profile II; a speech and language evaluation that included the Rossetti Infant-Toddler Language Scale, the Communicative Function Assessment Checklist, and the Profile of Communication and Symbolic Behaviors; and an occupational therapy evaluation that included administration of the Peabody Developmental Motor Scales, as well as physical and neurological examinations. Pet. Ex. 6, pp. 1-9. The report reflected that PDD-NOS is:

> a diagnostic label given to the developmental disability that is characterized by a variable combination of difficulties in three areas: significant impairments in language and communication, significant impairments in social skills, and the presence of restricted or stereotyped interests or activities. There are no specific tests for PDD-NOS and the diagnosis is made on a clinical basis. Pervasive Developmental Disorders are currently thought of as neurobiological disorders. In a small percent of cases, an associated medical condition can be identified. Atypical brain development in the early weeks of pregnancy and genetic factors seem to play a role in the development of this condition. . . With appropriate interventional strategies, developmental progress can be expected; however children with Pervasive Developmental Disorders typically have lifelong weaknesses in early identified areas of deficiency.

Id., pp. 9-10.

[56] This lack of words was noted on the neurological examination, but in another part of the evaluation, she was observed to vocalize four syllables, one two-syllable combination, babble, and sing along with a familiar song. She was reported to say "mama" and "dada" meaningfully, name one object frequently, imitate new words, and "use true words with jargon-like utterances." Pet. Ex. 6, p. 5. By history, she began to babble at 15-16 months of age, and "now occasionally inserts words." *Id*., pp. 1-2.

elevation of glycine.[57] *Id.*, p. 21. The results of the remaining tests were all normal.[58] These urine organic acid test results were discussed at some length during the hearing.[59]

During the February 2, 2000 CEC evaluation, A.H.M. had nasal congestion and fluid in her left ear. Pet. Ex. 6, p. 2. She visited her pediatrician the next day for treatment and was diagnosed with an ear infection. Pet. Ex. 4, p. 3. At this visit, A.H.M. was reported to have 12-15 persistent words, to repeat words, and to have "lots of jargoning." *Id.* The report of 12-15 persistent words was likely an overestimate, as no identifiable words were heard at the CEC evaluation the prior day and the assessment of her language expression at the CEC reflected that she was not saying fifteen meaningful words, used gestures rather than talking, did not imitate words overheard in conversation, could not ask simple questions, and could not name five to seven familiar objects on request. Pet. Ex. 6, p. 5.

She visited her pediatrician in March and again in April for vomiting and diarrhea. Pet. Ex. 6, p. 2. Fevers of 99.9° and 100° were recorded in conjunction with her ear infection in February and gastrointestinal illness in March respectively, but no fever was recorded in April. At the March visit, her pediatrician discussed the milk and wheat free diet "prescribed by [the] CEC," commonly referred to as a gluten-free, casein-free ["GF/CF"] diet.[60] Pet. Ex. 4, p. 2.

The next videos appear to have been filmed after these evaluations, as they show A.H.M. interacting with a therapist. *See* Titles 19-20. A.H.M. pointed to the card requested by the therapist and mimicked the therapist's actions in making a toy squeak. Title 19. In the second video, the therapist called A.H.M.'s name several times and rolled a ball to get her attention. A.H.M. babbled expressively but uttered no discernable words. Title 20.

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[57] A repeat test performed at the same laboratory in April 2000 showed glycine levels within normal limits. Pet. Ex. 16, p. 34.

[58] These test results all appear in the CEC records, Pet. Ex. 6. Some of the testing was performed at Hardin Memorial Hospital ["Hardin"] and the remainder at the University of Chicago. Duplicates of the tests performed at Hardin were also filed in Pet. Ex. 16, the records from that hospital.

[59] Urine organic acids were normal on a sample taken on February 22, 2000. Pet. Exs. 6, p. 20; 16, p. 24. The report was simply a summary sheet stating that the screen "shows no specific abnormalities," and did not contain a list of the various organic acids tested and the levels of acids found. A subsequent urine organic acid screen was performed on April 18, 2000, also at the University of Chicago laboratory, and was likewise reported only as showing "no specific abnormalities." Pet. Ex. 16, p. 35.

[60] The GF/CF diet involves the elimination of gluten-containing foods (primarily wheat products) and casein, a substance found in milk products. Many children with autism are placed on this diet, with reported improvements in behavior, although controlled studies of its efficacy are lacking. *Snyder*, 2009 WL 332044, at *163. Because this diet was not included in the long list of recommendations in the CEC report (Pet. Ex. 6, pp. 10-14), it is not clear if the examiners mentioned it or if petitioners learned about it elsewhere.

D. Treatment by Dr. Holmes.

On May 23, 2000, A.H.M. saw Dr. Holmes in Baton Rouge, LA, for the first time. In the notes from the office visit, Dr. Holmes observed that A.H.M.'s eye contact was "not good," her interaction was "poor," and her behavior was "zoned out." Pet. Ex. 8, p. 8. In the HPI section of the form, Dr. Holmes wrote "no talking," but also indicated that A.H.M. had "some expressive language, decent receptive." *Id.* Her parents also reported that she had "chronic diarrhea" but when they removed fruit juice from her diet, it stopped. Doctor Holmes' impression was that A.H.M. had "probable ASD" and "irritable bowel." She wanted to rule out "neurotoxicity." *Id.* She instructed A.H.M.'s parents to continue the GF/CF diet and administer Epsom salt baths. *Id.*, p. 7. She also ordered a number of tests and diagnosed A.H.M. with neurotoxicity, nutritional deficiency, mineral deficiency, and irritable bowel. *Id.*

A.H.M.'s parents signed a consent form that acknowledged that some of the treatments and tests offered by Dr. Holmes were not used by the majority of doctors in the community and were not considered part of the standard of care. Pet. Ex. 8, pp. 22-23. The tests ordered by Dr. Holmes included "urine organix profile," plasma amino acids, hair analysis for "toxic" and "essential" metals, and stool analysis. *Id.*, pp. 16, 18, 20-21. After receiving A.H.M.'s test results, Dr. Holmes determined there was "presumptive evidence of heavy metal toxicity" due in part to what she characterized as increased amounts of heavy metals in A.H.M.'s hair. *Id.,* p. 10. However, the level of mercury in A.H.M.'s hair (.21 mcg/g) was within the laboratory's reference range (*id.*, pp. 20). In spite of this normal level, Dr. Holmes informed petitioners about "Autism + Mercury" and recommended they begin "mercury chelation."[61]

A.H.M. also was treated and monitored by Dr. Jeffrey Wampler[62] during Dr. Holmes' treatment protocols. She directed A.H.M.'s treatment through email communications with Ms. Miller and written instructions for, and at least one telephone call with, Dr. Wampler. Pet. Exs. 8, pp. 25-108; 14, pp. 136-46. Doctor Wampler primarily supervised testing, including A.H.M.'s urine mercury levels but he also offered an opinion regarding causation to petitioners' original attorney.[63] He credited the

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[61] Pet. Ex. 8, p. 9. Aside from Dr. Cave's opinion, no new evidence showing chelation to be effective in treating ASD was filed in this case. Evidence from the OAP Theory 2 test cases indicated that chelation had not been shown to be a beneficial and effective treatment for autism. *See King*, 2010 WL 892296, at *49; *Dwyer*, 2010 WL 892250, at *104-05; *Mead*, 2010 WL 892248, at *36 n.74. Even Dr. Cave acknowledged that chelation could not remove mercury from the brain (Tr. at 74), where it would have to be in order to contribute to a neurological problem like ASD.

[62] *See* Pet. Ex. 8, pp. 107-08 (email dated May 31, 2000), 24 (letter to Dr. Wampler from Dr. Holmes). Doctor Wampler's practice in Louisville, Kentucky was much closer to A.H.M.'s home.

[63] Citing unspecified test results showing high mercury levels, Dr. Wampler concluded A.H.M. had mercury intoxication, adding that it was difficult to pinpoint the sources of mercury exposure. Pet. Ex. 14, pp. 7-8. He indicated that the MMR vaccine also may have contributed to A.H.M.'s autism, citing the work of Dr. Andrew Wakefield. Pet. Ex. 14, p. 7. Doctor Wampler wrote that "Dr. Wakefield has, I think, has

mercury chelation, dietary restrictions implemented by her parents, and the more traditional therapies A.H.M. was receiving with improving her behavior. Pet. Ex. 14, p. 6.

Initially, Dr. Wampler was hesitant to begin chelation. In email to Dr. Holmes, Ms. Miller indicated Dr. Wampler would "not be doing anything" until testing confirmed A.H.M. was "lead or mercury toxic," at which time "chelation may be attempted." Pet. Ex. 14, p. 101. After a July 7, 2000 call with Dr. Holmes, Dr. Wampler agreed that A.H.M. could begin chelation. *Id.*, p. 99.

The urinary metals testing was performed by Doctor's Data laboratory, a testing facility that performed similar tests on the children in the OAP test cases. *King,* 2010 WL 892296, at *80, 82. As I noted in *Dwyer*, 2010 WL 892250, at *182 n.687, concerns about this laboratory's testing methods have been raised, particularly with regard to comparisons of metal levels found during chelation to reference ranges established in non-chelated individuals. The results reported for A.H.M. in Dr. Wampler's and Dr. Holmes' records were difficult to interpret. No baseline testing for urinary mercury (or other metals) was performed.[64] The only mercury testing performed on A.H.M. prior to chelation was the hair analysis. A cross-walk of the baseline levels in hair and urine did not appear in the records filed, and, to the best of my recollection from the voluminous evidence filed in the OAP test cases, none has been established.

Without a baseline, it is difficult to assess the significance of the first "post provocation" (after chelation) test result, which showed a level of 11.8 micrograms (expressed as "µg/g creatinine" on the test report) per grams of creatinine on July 26, 2000.[65] Pet. Ex. 14, p. 122. While quite high compared to the reference range listed, there was no reference range for chelated individuals. It is thus impossible to determine if this result was high as compared to levels detected in other individuals undergoing chelation. On August 25, 2000, after more chelation, A.H.M.'s mercury level was below

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proven to my satisfaction that in certain individuals there is a link between chronic measles infection or autoimmune process initiated by the measles, mumps, rubella reaction that is causing regressive autistic behavior." *Id*. This letter was authored in March 2001, long before the Theory 1 test case evidence showed that Dr. Wakefield's research findings could not be duplicated and that the journal article he published linking MMR and autistic regression was withdrawn from publication because it contained fraudulent data. *See Snyder*, 2009 WL 332044, at *78-83. Doctor Wakefield's work was severely criticized by each of the special masters in the OAP Theory 1 test case hearings (*Cedillo,* 2009 WL 331968, at *110-11; *Hazelhurst,* 2009 WL 332306, at *86-92; *Snyder*, 2009 WL 332044, at *78-83) with one witness calling Dr. Wakefield's work "scientific fraud." *See Hazelhurst*, 2009 WL 332306, at *87, *aff'd,* 604 F.3d 1343, 1347-48 (Fed. Cir. 2010). Another witness testified that he asked that his name be removed from the paper before it was published because the test results reported were different from those he found on tests he performed. *Snyder*, 2009 WL 332044, at *79, n.245.

[64] In this context, a baseline would be a test performed prior to beginning chelation.

[65] The laboratory reference range was between 1-3 µg/g (micrograms per gram of creatinine) and the report form reflected that this sample was obtained "Post provocative challenge."

the detection limit of the test. *Id*., p. 111. Inexplicably, the mercury level was much higher in October 2000 (7.6 µg/g (*id*., p. 105)). A later result of 7.7 µg/g was referenced in a January 25, 2001 email from Ms. Miller to Dr. Holmes (*see* Pet. Ex. 8, p. 55), but I could not find a test report in the records reflecting this result. Ms. Miller inquired about why the level would be rising. *Id*. Doctor Holmes did not explain why this result would make sense.[66] Pet. Ex. 8, p.88.

Other testing in the summer of 2000 found normal immunoglobulin levels and one slightly high test involving liver function, but the results were otherwise not remarkable. Pet. Ex. 2a, pp. 25-26, 30-33. The only test of significance for the issues in this case was a serum lactic acid test performed on July 31, 2000. Although it was ordered by Dr. Wampler *(id*., p 143), Dr. Holmes requested it because she wanted to rule out a possible mitochondrial disorder, based on a high urine lactate level on an earlier test (*see* Pet. Ex. 8, p. 10). A high blood or cerebral spinal fluid ["CSF"] lactate level may indicate a mitochondrial disorder, but may also be elevated due to a struggling child or the use of a tourniquet to obtain the sample. A.H.M.'s lactate level was low. Pet. Ex. 2a, p. 146; *see also* Wolf & Smeitink, Court Ex. I, at 1404 (high serum lactate is a metabolic abnormality that may be indicative of mitochondrial disease).

In email messages to Dr. Holmes, Ms. Miller reported improvements but also complained of "excessive 'stimmy'" behavior in August 2000 (Pet. Ex. 8, p. 70) and some regression in behavior, to include an increase in temper tantrums in May 2001 (*id.*, p. 38). In April 2001, Ms. Miller asked Dr. Holmes about a treatment she had found on the internet, FGF2 therapy.[67] Doctor Holmes responded that the treatment "seems to work very well for those children who start it before age 6" and recommended Dr. Luis Aguilar in Guadalajara, Mexico[68] as the only source she would trust to provide this treatment to A.H.M. Pet. Ex. 8, pp. 40-41.

A.H.M. visited Dr. Aguilar during the summer of 2001. *See* Pet. Ex. 8, p. 31. From the email correspondence between Dr. Holmes and Ms. Miller, it appears Dr. Aguilar commented that A.H.M.'s brain was "not the brain of an autistic child." Pet. Ex. 8, p. 32. He agreed that the cause of A.H.M.'s condition could have been mercury, but also considered the possibility of a lack of oxygen during birth. *Id.* In her email response, Dr. Holmes wondered "what the EEG[69] would have shown 14 months ago?,"

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[66] Although Dr. Holmes usually was very responsive to Ms. Miller's questions, she ducked this question, responding only "Kellie, 7.7? Wow!! That's how much was dumped–that's great." *Id*., pp. 55-56.

[67] *See* Pet. Ex. 8, pp. 40-41. FGF2 stands for fibroblast growth factor 2. *See* http://www.ncbi.nlm.nih.gov/pmc/articles/PMC138918 (last visited on April 22, 2015).

[68] All the email discussions regarding Dr. Aguilar, including some regarding traveling to Mexico for treatment, indicated that he was located in Mexico.

[69] EEG stands for electroencephalogram. "The EEG is a graphic recording of the electrical activity of the brain" and is typically performed "to identify and evaluate patients with seizures." MOSBY'S MANUAL OF DIAGNOSTIC AND LABORATORY TESTS (4th ed. 2010) ["Mosby's Labs"] at 573.

adding "[g]uess we will never know." Pet. Ex. 8, p. 31. This exchange suggests that Dr. Aguilar performed an EEG on A.H.M. As autism is not a structural disease (*see* Tr. at 175 (testimony of Dr. Wiznitzer)), it cannot be diagnosed (or ruled out) by EEG. In reviewing hundreds of ASD claims, I have yet to see any reference to an EEG being diagnostic of or ruling out an ASD diagnosis, although EEGs are frequently performed to determine if a seizure disorder is also present as a co-morbid condition.

After the summer 2001 visit to Dr. Aguilar, Ms. Miller emailed Dr. Holmes indicating she was overnighting a report from Dr. Aguilar and forwarding a letter she wrote to DrAmysKids,[70] but only the first sentence of the letter was included in the pages filed. No report, test results, or medical records from Dr. Aguilar were filed.

The records reflect that Dr. Holmes and Ms. Miller sought assistance from several airlines in October and November 2001 for second trip to see Dr. Aguilar but there is no evidence A.H.M. saw him again. Pet. Ex. 8, pp. 110-13.

A.H.M. saw Dr. Holmes only twice, first on May 23, 2000, and again in November 6, 2001, when A.H.M. returned to Baton Rouge to receive a secretin injection.[71] That visit was the last contact with Dr. Holmes, based on the records filed. Pet. Ex. 8, p. 25. Doctor Wampler's treatment of A.H.M. appears to have continued for several more months. His medical records indicate A.H.M. received an autism assessment by a home health nurse in late 2001 and that A.H.M. saw him for a rash in May 2002. *See* Pet. Ex. 14, pp. 148-49. The results of the autism assessment were not filed.

The last two videos were not dated. Based on A.H.M.'s appearance and the piles of leaves, they could have been recorded in the fall of 2000 or 2001. *See* Titles 21-22. In the first video (Title 21), A.H.M. was sitting in the leaves, feeling their texture. In the last video (Title 22), she was running around and playing apart from the other children and adults. She occasionally looked up as the other children and adults made noise, and glanced at an older woman who called her name, but she ignored a Frisbee that the woman threw to her. *Id.*

E. Later Treatment Records.

A.H.M.'s later treatment records are quite sparse. She apparently became a patient of Dr. Jeff Bradstreet in 2003, upon referral from Dr. Holmes, who had retired.[72]

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[70] It appears that "DrAmysKids" was a forum for parents of the children Dr. Holmes treated.

[71] *See* Pet. Ex. 8, pp. 136-39. Doctor Wampler was not willing to administer this treatment. *See id.*, p. 79.

[72] Doctor Bradstreet, a Florida physician, testified on behalf of his patient in one of the OAP test cases. *Snyder*, 2009 WL 332044, at *21-22. In that case, his records included office notes as well as laboratory tests. *Id*. The letter to petitioners' counsel forwarding Dr. Bradstreet's records in this case specifically references "Medical Lab Records," and does not mention treatment records. Pet. Ex. 13, p. 1.

See Pet. Exs. 13, p. 2; 20, p. 2. It is difficult to determine what treatment Dr. Bradstreet provided, or if he even saw A.H.M. in person, as his medical records are simply the results of tests performed at many different laboratories from May 2003 to January 2012.[73] *See* Pet. Ex. 13.

For routine care, A.H.M. was seen at Sharonville Pediatrics from late 2002 through early 2005. A well-child visit in late 2002 and several visits for illnesses (bronchitis, sinusitis, an ear infection, pharyngitis, and URI) throughout 2004 and 2005 are reflected in these records. Pet. Ex. 15, pp. 1-5.

A.H.M. saw a new pediatrician, Dr. Jonathan Mumma, on May 14, 2009 for a checkup prior to moving to Florida in June 2009. *See* Pet. Ex. 18. That fall, A.H.M. was referred for an autism spectrum evaluation by her new middle school in Florida. *See* Pet. Ex. 20. The report from the evaluation listed A.H.M.'s diagnoses as "Asperger's Syndrome" and attention deficit hyperactivity disorder. The background section indicated that A.H.M. was receiving special education services from her school in Ohio. *Id.*, p. 1. This record is incomplete, stopping mid-sentence and the conclusions reached by the evaluators are unknown. *Id.*, p. 6. A.H.M. was reported to exhibit several symptoms typical of autism, such as self-stimulating behavior, difficulty with a change in routine, and sensitivity to sound. *Id.*, p. 5.

These two more recent records contain the first mention of a sudden reaction to A.H.M.'s September 15, 1999 vaccinations. When providing a medical history to Dr. Mumma, Ms. Miller claimed that A.H.M. received her 15 and 17 month vaccinations at one time and suffered a reaction eight hours later. Pet. Ex. 18, p. 8. During A.H.M.'s ASD evaluation for the new school district in Florida, Ms. Miller claimed the reaction was immediate and included a fever of 105°. She stated that A.H.M. "began saying single words with communicative intent at 14 months of age and then at 17 months [A.H.M.] stopped talking all together and did not speak again until about the age of 3." Pet. Ex. 20, p. 2.

F. Expert Testimony Regarding A.H.M.'s Symptoms.

Both physicians testified about A.H.M.'s symptoms and the conclusions they drew from the medical records and other evidence.

Doctor Cave claimed that A.H.M. exhibited symptoms of encephalopathy following her June 4, 1999, vaccination and that these symptoms became more severe after her September 15, 1999 vaccinations. Tr. at 26-27; *see also* Pet. Post-Hearing Memo at 2. Claiming that the "worst injury" was caused by A.H.M.'s September 15,

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[73] These records include the 2008 test result reporting a mild elevation in whole blood lactate which is discussed, *infra*, in Section V.C. The records also contain normal plasma lactate levels reported in 2010 and 2011. *See* Pet. Ex. 13, pp. 53, 61.

1999 vaccinations (Tr. at 33), Dr. Cave testified that A.H.M. regressed after the June 4, 1999 varicella vaccination, had more illnesses after the September 15, 1999 vaccinations, and was no longer talking by November 1999 (Tr. at 27).

She described A.H.M. as having words and a lot of playful behavior before the June vaccination, and then compared A.H.M.'s condition before that vaccination to her appearance at the initial visit with Dr. Holmes, which occurred almost a year later. Tr. at 29. Doctor Cave mentioned the specific symptoms of poor eye contact, "zoned out" behavior, chronic diarrhea, and lack of speech noted by Dr. Holmes as evidence of encephalopathy and regression. Tr. at 25, 29-30. She also relied upon Ms. Miller's claim of a sudden reaction to the September 15, 1999 vaccinations as evidence that A.H.M. experienced a vaccine-related encephalopathy. *See* Tr. at 88-89.

Doctor Cave was not precise in her use of the terms "regression" and "encephalopathy." At times, she appeared to be using the term "encephalopathy" in the context of the severe, acute encephalopathy described in the Vaccine Injury Table. *See, e.g.*, Tr. at 25; *see* 42 C.F.R. 100.3(b)(2) (definition of a Table encephalopathy found in the Qualifications and Aids to Interpretation ["QAI"]). At other times, she and Mr. Cave used the term more generally, but she never really defined it. Tr. at 32-33, 63, 76.

Doctor Wiznitzer opined that A.H.M. did not suffer an encephalopathic reaction or a developmental regression in response to her vaccinations. Tr. at 120-22; Res. Ex. A at 7. Distinguishing between regression and stagnation,[74] he testified A.H.M. showed the delay in development (which he termed "stagnation") often seen in children with autism spectrum disorders, not a regression. Tr. at 121-30. He testified that he did not find "any good documentation in the contemporaneous medical records" (including Dr. Holmes' intake form from May 23, 2000) showing A.H.M. suffered a "clear loss of acquired skills." Tr. at 121.

When testifying about the absence of an encephalopathic condition in A.H.M., Dr. Wiznitzer clarified that he was referring to an "acute encephalopathy" rather than the more general use of the term. "Encephalopathy" simply means "there's something wrong with the brain." Tr. at 138. Doctor Wiznitzer explained that although both "autism" and "encephalopathy" are terms reflecting a problem with the brain, they are not interchangeable. Tr. at 230-31. According to Dr. Wiznitzer, there is no diagnostic entity of "encephalopathy with autistic features." Autism is a separate diagnostic entity with specific diagnostic criteria, and all the required criteria must exist before the diagnosis can be assigned. Tr. at 231. Doctor Cave made the point that the majority of the children with autism would be considered to have some degree of encephalopathy

[74] Doctor Wiznitzer defined regression as "a loss of skills that have been acquired and clearly are present and functional," providing the example of a child who was walking well and then no longer walking. Tr. at 121. He defined stagnation as a failure to move on from simpler to more complex skills. Tr. at 122.

(Tr. at 270), but this would be in the broad, general use of the term, rather than in the sense of the child having an acute encephalopathy. And, according to Dr. Wiznitzer, the correct diagnosis for children who meet the ASD diagnostic criteria would, nevertheless, be autism, not encephalopathy. Tr. at 230-31.

He described three main developmental patterns seen in children with ASD. Tr. at 127-29. He opined that A.H.M.'s pattern of development fit nicely in the most common group. These children appear to be developing normally until approximately six to twelve months of age when they start showing "deviations" from the normal social, language, and play behaviors. Tr. at 128-29. He described this phenomenon as a failure "to turn on certain centers in the brain," much like the failure to flip a switch at the appropriate time. The resulting difference becomes more apparent over time, and in part of this group of children, development stagnates. Tr. at 129-30. Although some children with ASD regress or lose skills that they once demonstrated, he opined that A.H.M. showed no evidence of regression. Tr. at 136-37.

Doctor Wiznitzer also observed that there was no sign A.H.M. suffered the impairment of consciousness alleged by petitioners and Dr. Cave. Tr. at 138-39. Rather, he maintained A.H.M.'s lack of eye contact and response to others was selective behavior indicative of autism and not evidence of impairment in consciousness or a lack of responsiveness to her environment. Tr. at 139. Summarizing the video evidence submitted, he testified that the videos showed a change in A.H.M.'s social behavior and language beginning in the summer of 1999, but not the regression or loss of skills or the impairment of consciousness that Dr. Cave asserted had occurred. Tr. at 137.

V. Factual Findings.

In summary, after reviewing the entire record and weighing the experts' testimony, I conclude that the symptoms and behaviors that Dr. Cave relied upon for her conclusion that A.H.M. regressed did not occur as she described them. Although A.H.M.'s impairments, including loss of some eye contact, stagnation in language and social development, and lack of response to her name, could be termed an encephalopathy in the broad use of the term, A.H.M. never experienced an acute encephalopathy, much less one that meets the strict Table definition.[75] The behaviors A.H.M. displayed beginning in the summer of 1999 and which became more overt and obvious throughout the following year are the behaviors that are diagnostic of ASD. That diagnosis fully accounts for her behavioral symptoms. *See* Res. Ex. A, Tab 1[76] at

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[75] *See* 42 C.F.R. 100.3(b)(2) (definition of a Table encephalopathy found in the Qualifications and Aids to Interpretation ["QAI"]).

[76] C. Johnson, et al., *Identification and Evaluation of Children with Autism Spectrum Disorders*, PEDIATR. 120(5): 1183-1215 (2007) [hereinafter "Johnson, Res. Ex. A, Tab 1"].

9-11; *White v. Sec'y, HHS,* No. 04-337V, 2011 WL 6176064, at *4-9 (Fed. Cl. Spec. Mstr. Nov. 22, 2011).

With regard to the mitochondrial disorder diagnosis made by Dr. Cave based on medical records alone, I summarize the relevant testimony and report and the conflicting testimony of Dr. Wiznitzer. I discuss the diagnostic criteria Dr. Cave used, and make factual findings regarding whether A.H.M. had the symptoms relied upon by Dr. Cave, either at all or to the degree required by the diagnostic criteria. I ultimately conclude that there is insufficient evidence that A.H.M. actually has either a mitochondrial disorder or mitochondrial dysfunction.

A. Law Pertaining to Evidentiary Conflicts.

Conflicts between the events described in contemporaneous records and those described in documents prepared or testimony given several years later are common in Vaccine Act cases. Two general legal principles guide the resolution of conflicts between contemporaneous records and later-adduced evidence. The first is that the absence of a reference to specific symptoms in a medical record does not conclusively establish the absence of symptoms during that time frame. *See, e.g.*, *Murphy v. Sec'y, HHS*, 23 Cl. Ct. 726, 733 (1991), *aff'd*, 968 F.2d 1226 (Fed. Cir. 1992) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance" (citation omitted)).

The second principle addresses the degree of reliance commonly accorded to contemporaneous records. Special masters frequently accord more weight to contemporaneously-recorded medical symptoms than those recounted in later medical histories, affidavits, or trial testimony. "It has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight." *Murphy*, 23 Cl. Ct. at 733 (citation omitted); *see also Cucuras v. Sec'y, HHS*, 993 F.2d 1525, 1528 (Fed. Cir. 1993) (medical records are generally trustworthy evidence). Memories are generally better the closer in time to the occurrence reported and when the motivation for accurate explication of symptoms is more immediate. *Reusser v. Sec'y, HHS*, 28 Fed. Cl. 516, 523 (1993). Inconsistencies between testimony and contemporaneous records may be overcome by "clear, cogent, and consistent testimony" explaining the discrepancies. *Stevens v. Sec'y, HHS*, No. 90-221V, 1990 WL 608693, at *3 (Fed. Cl. Spec. Mstr. Dec. 21, 1990).

B. Factual Findings Regarding Symptoms and Onset.

1. Sudden Vaccine Reaction.

I find that A.H.M. did not have a sudden or dramatic reaction to either the June 1999 varicella vaccination or the September 1999 combination of vaccinations. She did not have a fever caused by these vaccinations.

Doctor Cave testified that petitioners had an unrecorded meeting with A.H.M.'s

pediatrician shortly after the September 1999 vaccinations. She relayed that petitioners were told by the pediatrician that the reaction was not vaccine-related. *See* Tr. at 88. However, Dr. Cave offered no explanation for the lack of documentation for this meeting or for petitioners' failure to mention it to any other physician (including Dr. Holmes). She "thought" that the report of this meeting was in the CEC records (Tr. at 89), but the CEC records (Pet. Ex. 6) do not reflect any mention of the September 1999 vaccinations, much less a reaction to them. A.H.M. had multiple pediatric visits for illness in the two months after the September 1999 vaccinations, but none of the records from these visits attribute the illnesses, which were apparently allergic in nature, to the September vaccinations.

Ms. Miller's later reports of A.H.M.'s vaccinations and reactions thereto contain numerous assertions that are contradicted by, or inexplicably missing from, the contemporaneous medical records, including the extent of A.H.M.'s vocabulary, the abrupt loss of all language, and the presence of a high fever. While I do not question Ms. Miller's veracity, her recollection of events occurring many years earlier is clearly flawed. Thus, Dr. Cave's reliance on these reports, made about 10 years after the events in question, is misplaced.

One of A.H.M.'s treating physicians, Dr. Wampler, specifically questioned Ms. Miller about any reaction to the September 1999 MMR vaccination. He reported that "[A.H.M.'s] mother said that there was really no high fever or other systemic reactions at the time the MMR vaccine was taken" but only a regression in speech shortly after vaccination. Pet. Ex. 14, p. 6. The fact that this report by a treating physician, who thought the MMR vaccine could cause autism, was made in a letter to petitioners' former attorney of record (*see* Pet. Ex. 14, pp. 4-8), is particularly telling, as Dr. Wampler was clearly looking for evidence of a vaccine reaction or regression to explain A.H.M.'s ASD.

2. Increased Illnesses.

I find that A.H.M.'s health in the two months after the September 15, 1999 vaccinations was not significantly different from her health in the months prior to those vaccinations.

Doctor Cave pointed to the three "sick visits" in October 1999, as evidence that A.H.M. had "many sick visits," that her health "generally went downhill," and that she was chronically ill with respiratory and other problems after receiving the September 15, 1999 vaccinations. Tr. at 27, 30. She testified that this demonstrated an "insult to the immune system at the very least." Tr. at 30. Precisely what point Dr. Cave was making with regard to A.H.M.'s encephalopathy, regression, or any mitochondrial disorder by these claims of increased illnesses was never articulated.

The evidence does not support Dr. Cave's assertions about increased ill health after the September vaccinations. A.H.M. had been suffering from coughs, colds, and low grade fevers throughout the summer and fall. *See* Pet. Ex. 4, pp. 5-7. Her

pediatrician attributed these illnesses to an ear infection, URI, and allergies. *See id.* After her Zyrtec dosage was increased on October 21, 1999, she returned to the doctor only once more (on October 29) and was described as "much improved." *Id.*, p. 5. A.H.M. did not visit her pediatrician for illness again until February 2, 2000 when she was diagnosed with another ear infection. Pet. Ex. 4, p. 3.

3. Language Skills.

With regard to A.H.M.'s receptive and expressive language skills, I make the following factual findings:

a. A.H.M. was saying "mama & dada" at nine months of age. She was using these words in reference to her parents at twelve months of age.

b. She gained words over the summer after receiving the varicella vaccination in June 1999. She had four to five words at the time of her September 15, 1999 vaccinations. In November 1999, she had the same number of words.

c. As early as September 1999, petitioners were concerned about A.H.M.'s speech.

d. There is no reliable evidence of a regression in speech, defined as a loss of words previously used, in the five months after her September 15, 1999 vaccinations.

e. Although some records contain notations such as "not talking yet," the same records, taken as a whole, reflect some speech, just not the level of speech expected in a child of A.H.M.'s age at the time the record was created.

f. As reflected by her pediatric records, A.H.M. had a mild developmental and speech delay in both September and November 1999.

g. During the period from November 1999 through January 2000, A.H.M. had a vocabulary of three to five words at any given time, including "mama," "dada," "why," "no," and "Dusty."[77] Essentially, her speech development was stagnant from September 1999 through the visit to Dr. Holmes in May 2000. Although she did not talk at that visit, she still had some expressive language.

h. It is unlikely that A.H.M. had 12-15 words in February 2000.[78]

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[77] *See, e.g.*, Pet. Exs. 19, p. 2 (A.H.M. was observed to imitate her mother and repeat why); 6, p. 7 (Dusty refers to the neighbor's dog).

[78] Although Ms. Miller reported to A.H.M.'s pediatrician at a February 2000 visit that she spoke 12-15 words (*see* Pet. Ex. 4, p. 3), other records indicated that A.H.M.'s vocabulary was far more limited. On

Doctor Cave's testimony about A.H.M.'s language demonstrated a lack of familiarity with and a selective reading of the information contained in A.H.M.'s records. She initially testified that A.H.M. had acquired 12 to 15 words prior to the alleged causal vaccinations and thereafter stopped talking. She pointed to the medical records from November 1999[79] and May 2000 doctor visits as evidence that A.H.M. was no longer talking. Tr. at 93, 96-97. After I informed her that the entry indicating A.H.M. had acquired 12 to 15 words[80] was created in February 2000 (between the two visits she cited, and five months *after* the last vaccinations alleged as causal) (Tr. at 93; *see also* Tr. at 95 (respondent's counsel also clarifying the date that entry was created)), she altered her testimony, claiming A.H.M. suffered a regression following the September 1999 vaccinations, improvement in February 2000 and a second regression before the May 2000 visit with Dr. Holmes. Tr. at 112-13.

Doctor Cave ignored other entries in the two records she referenced which indicated the description of "not talking" did not equate to lost speech or no words. Doctor Cave claimed an entry in the November 12, 1999 records that A.H.M. was "[n]ot talking yet" (Pet. Ex. 4, p. 4) was proof that A.H.M. had lost speech since the September 1999 vaccinations. This assertion not only ignores the qualifier "yet," which, read literally, would indicate that she had not been talking previously. It also ignores the remainder of the office visit notes, which reflect that the concern about A.H.M.'s speech was ongoing and that she was "not making progress." *Id.*

Doctor Cave also pointed to Dr. Holmes' intake interview from May 2000 (Pet. Ex. 8, p. 8) as proof that A.H.M. had lost words after the September 1999 vaccinations. Tr. at 96. Even assuming her assertions to be correct, this note, written in May 2000, can hardly be said to follow closely the allegedly-causal September 1999 vaccinations. Doctor Cave's assertions were not correct. Although the note indicated that, by history, A.H.M. had "no talking," this entry was immediately followed by "some expressive language." Pet. Ex. 8, p. 8. Read together, these entries are consistent with the reports from the fall of 1999, both to the pediatrician and to the Early Steps evaluators. A.H.M. had some vocabulary, but was not talking the way her parents expected. Doctor Holmes' entries are likewise consistent with the CEC reports.

February 2, 2000, one day before the visit to the pediatrician where this report was made, A.H.M. had a speech and language evaluation which reflected the use of only a few words. *See* Pet. Ex. 6, p. 5.

[79] Both Dr. and Mr. Cave used an incorrect date, calling the visit on November 12, 1999 (*see* Pet. Ex. 4, p. 4) the "November 22, 1999" visit (Tr. at 27). There was no visit on November 22, 1999, in the records filed.

[80] As noted earlier, I have little confidence that the report of 12-15 words made by Ms. Miller to A.H.M.'s pediatrician in February 2000 was completely accurate, given the speech and language evaluation performed the prior day did not reflect much language usage. However, this report to the pediatrician does demonstrate that, regardless of the extent of her vocabulary, A.H.M. was still using some words. The speech and language evaluation reflected the use of some words as well. *See* Pet. Ex. 6, p. 5.

Nowhere in the contemporaneous records is there a report that A.H.M. stopped talking or lost words. As Doctor Wiznitzer testified (Tr. at 121-22, 124), none of A.H.M.'s treating physicians noted a regression in speech or any other skills.

The video evidence reflects that A.H.M. did not often talk or use discernable words, but the videos recorded both before and after the allegedly causal vaccinations are remarkably similar regarding the extent of A.H.M.'s expressive communication. *See* Tr. at 137 (Dr. Wiznitzer's testimony).

4. Other Developmental Symptoms and Onset.

With regard to other symptoms and their onset, I make the following factual findings:

a. There is no evidence of any other type of developmental regression after the September 1999 vaccinations. Although A.H.M. began displaying more behaviors consistent with ASD, symptoms of ASD had begun to manifest in the summer of 1999. The additional ASD-type behaviors, including changed play and eye contact, did not include the loss of skills previously displayed.

b. Doctor Cave's assertions about a developmental regression were based on a selective reading of the medical records. Her contention that A.H.M. "went from words to no words, from eye contact to no eye contact, from playful behavior to zoned out behavior" (Tr. at 90; *see also* Tr. at 69-70) were hyperbole. What happened to A.H.M. was not a loss of skills; it was the progression of atypical behavior patterns commonly seen in ASD. No treating physician, not even Dr. Holmes, diagnosed a regression of skills.

c. A.H.M. exhibited symptoms of social deficits common in children with ASD, such as poor eye contact, failure to respond to her name, ignoring others, and a fascination with specific objects, as early as July 1999. Even in the five months after the September 1999 vaccinations, eye contact was not non-existent; it was selective and less extensive than the eye contact seen in a typically developing child.

d. A.H.M. was not unresponsive to her environment; she was selectively responsive. She paid more attention to objects than people. However, she was capable of interaction and remained affectionate with her parents.

e. No treating physician who evaluated A.H.M. in the winter and spring of 1999-2000 diagnosed her as encephalopathic. Although her poor eye contact and lack of social interaction with people could be characterized, in the broad use of the term, as an encephalopathy, A.H.M. never experienced an acute encephalopathy as defined by the Vaccine Injury Table.

f. These behavioral symptoms manifested over the summer of 1999, at a time period when it is expected that deficits in social behaviors (behavioral symptoms) typically become apparent. *See* Johnson, Res. Ex. A, Tab, 1 at 8-9. As Dr. Wiznitzer testified, the deviations in development generally begin around twelve months of age. Tr. at 129-30, 136-37.

C. Mitochondrial Disorder.[81]

1. Evidence Concerning Mitochondrial Disorders.

No treating physician or specialist ever diagnosed A.H.M. with a mitochondrial disorder, mitochondrial disease, or mitochondrial dysfunction. Prior to Dr. Cave's initial expert report, the only mention of a mitochondrial disorder anywhere in the evidence filed was a comment by Dr. Holmes that an elevated urinary lactate might be indicative of a mitochondrial disorder, and a suggestion that a serum lactate level be obtained. See Pet. Ex. 8, p. 10. She repeated both this comment and the suggestion that a serum lactate level be obtained in a June 19, 2000 letter to Dr. Wampler. *Id*., p. 24 Serum lactate testing performed in July 2000 in response to this suggestion was essentially normal. Pet Ex. 14, p. 121 (reporting lactic acid as low in a blood sample drawn on July 31, 2000). Nevertheless, ostensibly applying the Morava criteria discussed below, Dr. Cave diagnosed A.H.M. with a mitochondrial disorder. Tr. at 44; *see also* Pet. Ex. 11-a at 7-8 (citing the scoring checklist found at Pet. Ex. 11-a, Tab 11); Pet. Ex. 35 at 1-2.

2. The Morava Diagnostic Criteria.

The Morava criteria were formulated as a simplified "bedside version" of the Nijmegen mitochondrial disease criteria used in diagnosing children.[82] *See* Pet. Ex. 26

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[81] Doctor Cave drew a distinction between the terms "mitochondrial disorder" and "mitochondrial dysfunction." Equating mitochondrial disease and disorder, she claims "mitochondrial dysfunction can be thought of as a less severe form of mitochondrial disease." Pet. Ex. 11-a at 6 (citing the abstract of an article which was later filed as Pet. Ex. 24 (D. Rossignol & R. Frye, *Mitochondrial Dysfunction and Autism Spectrum Disorders: a Simplified Approach*, AUTISM SCIENCE DIGEST: THE JOURNAL OF AUTISMONE, Issue 02: 21-27 (undated) [hereinafter "Rossignol & Frye, Pet. Ex. 24"]. In spite of drawing this distinction, she and Mr. Cave often used "dysfunction" and "disorder" interchangeably. For example, although Dr. Cave acknowledged the Morava criteria are used to diagnose mitochondrial *disorders*, she and Mr. Cave often substituted the term dysfunction for disorder when discussing the points which should be awarded. *See, e.g.,* Tr. at 43. When asked to clarify her theory of causation, Dr. Cave responded that the "metal toxicity from the vaccines mainly" caused a mitochondrial *disorder* in A.H.M., manifested in behavior and regression. Tr. at 76. Because I am making factual findings in this section regarding the bases for the points awarded by Dr. Cave under the Morava criteria, I use the term mitochondrial disorder. I will discuss mitochondrial dysfunction in Section V.D.

[82] The Nijmegen criteria (also referred to as the "Wolf" criteria or as the "consensus mitochondrial disease criteria ["MDC"]) are found in Wolf & Smeitink, Court Ex. I. *See also* Morava, Pet. Ex. 26, which identifies the MDC as the source of the scoring system used. Reference 5 in the Morava study is the Wolf &

(E. Morava, et al., *Mitochondrial Disease Criteria: Diagnostic applications in children*, NEUROLOGY 67: 1823-26 (2006)) [hereinafter "Morava, Pet. Ex. 26"]. The Morava study was a follow-on to the original Nijmegen study to determine the specificity of diagnoses of mitochondrial disorder made using the Nijmegen criteria. *Id*. at 1823. In this context, "specificity" refers to the ability to differentiate those with a mitochondrial disorder from those with a nonmitochondrial multisystem disorder.[83]

The Morava diagnostic criteria are divided into three sections, with scoring limitations for each section, as well as in the subsections that form Section I, the clinical criteria. Section II covers metabolic and imaging studies, and Section III, the findings on muscle biopsy (morphology). No more than 4 points may be awarded in each section. As A.H.M. did not have a muscle biopsy, only Sections I and II of the Morava criteria are discussed.

Section I covers the three basic clinical presentations in mitochondrial disorders: muscular (Section I.A); central nervous system ["CNS"] (Section I.B); and multisystem disease (Section I.C). The total point score allowed in Section I is a maximum of 4 points, with no more than 2 points each allowed in Sections I.A and I.B, and a maximum of 3 points in Section I.C. The maximum score from Section II is 4 points, with each test or syndrome-like presentation limited to 1 or 2 points. *See* Morava, Pet. Ex. 26, Table, at 1824 [hereinafter "Morava Table"]. A score of 8-12 points results in a diagnosis of a definite mitochondrial disorder, 5-7 points a probable mitochondrial disorder, 2-4 points a possible mitochondrial disorder, and 1 point identifies someone unlikely to have a mitochondrial disorder. *Id*.

The Morava Table simply lists symptoms, such as "exercise intolerance," "seizures," or "GI tract" (in Section I) and lists tests such as "elevated lactate" or "ethylmalonic aciduria" (in Section II) without any explanation of the parameters of the criteria. However, the references in the Morava article to the MDC (Nijmegen) criteria, with reference 5 citing to the Wolf & Smeitink article (Court Ex. I), reflect an adoption of the parameters found in that article and the www.neurology.org website's supplemental

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Smeitink article filed as Court Ex. I, which is identified as the source for the scoring system used in the Morava study, and was a product of the Nijmegen Center for Mitochondrial Disorders, Nijmegen, the Netherlands. I note that the senior researcher listed on the Morava study, Pet. Ex. 26, is Dr. Smeitink, the co-author of Court Ex. I. *Id*. at 1823. I will refer to the criteria proposed in Wolf & Smeitink, Court. Ex. I, as the Nijmegen criteria, rather than the Wolf criteria or MDC.

[83] The researchers determined the specificity of the Nijmegen criteria by comparing the scores computed using the Nijmegen criteria in two groups of patients: (1) a group of patients with a genetic basis for their mitochondrial disease and (2) another group of patients who were suspected to have a mitochondrial disease, but were diagnosed based on genetic analysis with a nonmitochondrial multisystem organ disorder. Morava, Pet. Ex. 26, at abstract. Part of the purpose of the study was to determine if the Nijmegen criteria score could be used to inform the decision whether to conduct a muscle biopsy, an invasive procedure often performed under general anesthesia, with specific risks for patients with some types of mitochondrial disorders. *Id*. at 1825.

materials, a portion of which were filed as Pet. Ex. 25. During the hearing, Dr. Wiznitzer testified that the more detailed Wolf (Nijmegen) criteria were the source of the Morava criteria. Tr. at 148-49; *see* Res. Ex. D, Tab 1.

3. Doctor Cave's Application of the Morava Criteria to A.H.M.

a. Scoring Problems.

Doctor Cave's lack of understanding of the Morava criteria is best illustrated by her initial expert report and score sheet. Using the list of symptoms and tests appearing on the Morava Table, she scored A.H.M. as having 8 points, to arrive at a "definite" mitochondrial disorder diagnosis.[84] Her award of 5 points on Section I exceeded the point total of 4 permitted on Section I. Her score on Section II was 3 points, but her expert report and the score sheet filed along with it differed as to which test results correlated with which points.[85]

At the hearing, Dr. Cave testified that she awarded 5 points in Section I, again exceeding the maximum points allowed in that section. In Section II, she awarded 2 points (this time reflecting the correct amount) for an elevated lactate level and 1 point for an elevated ethylmalonic acid level. Tr. at 39. She did not award any points for the elevated lactate/pyruvate ratio that she had scored in her expert report. For the first time, she awarded 2 points for elevated urinary tricarboxylic acid ["TCA"][86] for a total of 5 points on this section. Tr. at 39-42. Thus, in her testimony, Dr. Cave awarded more points than the maximum allowed for both Sections I and II. The maximum score, based on the use of Sections I and II, was 8, not the 10 points she awarded.

Her difficulty in following the scoring criteria was not limited to math errors or remaining within the score system parameters. Each time she opined on the application of the Morava Table to A.H.M., she arrived at a different total or used different symptoms.

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[84] In her second expert report, Dr. Cave awarded 1 point each for exercise intolerance, muscle weakness, developmental delay, loss of skills, and gastrointestinal tract involvement, for a total of 5 points in Section I. Pet. Ex. 11-a at 7. This point total matched the total on the Morava criteria score sheet filed as Pet. Ex. 11-a, Tab 11.

[85] In scoring Section II, Dr. Cave's expert report reflected that she awarded 1 point each for elevated levels of lactate, the lactate/pyruvate ratio, and levels of ethylmalonic acid. Pet. Ex. 11-a at 7-8. When she completed the score sheet, she checked boxes for only elevated lactate (which is worth 2 points, not the 1 point Dr. Cave scored on the expert report) and 1 point for lactate/pyruvate ratio. Pet. Ex. 11-a, Tab 11 at 2.

[86] The term "tricarbon" (referring to urinary tricarbon acid ["TCA"]) appears in the Morava Table, Pet. Ex. 26, at 1824, but Dr. Cave used the term tricarboxylic acid. See Pet. Ex. 35 at 1. The tricarboxylic acid cycle is also referred to as the Krebs or citric acid cycle. *See* DORLAND'S at 454.

b. Symptoms.

What Dr. Cave determined to be symptoms matching the lists appearing in Section I of the Morava Table reflected a fundamental misunderstanding of the nature of the symptoms involved in mitochondrial disorders and how such disorders are diagnosed. She also ignored or misunderstood the requirements for awarding points for metabolic and imaging studies in Section II.

In her expert report and at the hearing, Dr. Cave awarded points for exercise intolerance, muscle weakness, developmental delay, regression (loss of skills), and gastrointestinal disease as clinical symptoms scored in Section I of the Morava Table. Pet. Ex. 11-a, Tab 11; Tr. at 35-44 (cites from transcript). After the hearing, Dr. Cave apparently eliminated the points she awarded for regression,[87] exercise intolerance,[88] and gastrointestinal disease,[89] reducing the point total she awarded on Section I to 2 points: 1 point for developmental delay and 1 point for muscle weakness. Pet. Ex. 35 (supplemental report commenting on Court Ex. I) at 1. Petitioners' post-hearing brief adopted Dr. Cave's rescoring for Section I. Pet. Post-Hearing Br. at 3. Effectively, the reduced score on Pet. Ex. 35 was an acknowledgment that Dr. Cave had misapplied the clinical criteria in her second expert report and testimony.

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[87] As noted in the factual findings above, A.H.M. did not experience a developmental regression, and no one, other than Dr. Cave, ever opined that she did. Although Dr. Cave apparently conceded in her supplemental report that A.H.M. did not experience a developmental regression by not awarding any points for this clinical symptom, petitioners continued to argue that A.H.M. experienced developmental regression following her June and September 1999 vaccinations in their post-hearing filings. Pet. Post-Hearing Br. at 6.

[88] The rescoring was necessary, as the evidence at the hearing established that A.H.M. did not have exercise intolerance. Doctor Cave testified she awarded a point for exercise intolerance based on an entry in the report from the February 2000 CEC evaluation indicating A.H.M. had "difficulty coordinating balance reactions during movement interaction." Tr. at 36 (referencing Pet. Ex. 6, p. 8). Referencing the more detailed description in the Nijmegen criteria which described exercise intolerance as "defined by abnormal premature fatigue, weakness, muscle aches or cramps after normal play or activities as daily living" (Tr. at 152 (citing Res. Ex. D, Tab 1 at 1)), Dr. Wiznitzer maintained the CEC entry referred to a difficulty with fine and gross motor skills and was not indicative of exercise intolerance. *See* Tr. at 152.

[89] As Dr. Wiznitzer testified, the category of gastrointestinal tract symptoms in mitochondrial disease includes only specific symptoms (the list of qualifying symptoms was filed, post-hearing, as Res. Ex. D, Tab 1), and A.H.M. did not have those symptoms. Tr. at 159-60. Doctor Cave testified that she awarded 1 point for gastrointestinal involvement due to "chronic diarrhea" and a pathogenic organism for which A.H.M. received treatment. Tr. at 37. She relied on test results from Great Smokies Diagnostic Laboratories which showed enterobacter cloacae present in A.H.M.'s stool. Tr. at 38; *see* Pet. Ex. 8, p. 186 (for test results). The medical records do not reflect a chronic diarrhea diagnosis. At best, Dr. Holmes diagnosed irritable bowel syndrome, based on the parents' reports that A.H.M. had suffered from diarrhea, which stopped when juice was removed from her diet. Pet. Ex. 8, p. 8. As A.H.M.'s diarrhea was explained by diet and the presence of bacteria, it did not meet the diagnostic criteria for "GI tract" as listed on the Morava Table, and explained by the Nijmegen supplemental materials, Res. Ex. D, Tab 1, at 2; Tr. at 181-83.

c. Metabolic Studies.

In her post-hearing report, Dr. Cave reduced the score on Section II to 3 points, 1 point for elevated ethylmalonic acid and 2 points for elevated tricarboxylic acid. *Id.*; Pet. Ex. 35 at 1. She eliminated the points she had awarded previously for elevated lactate (or lactic acid)[90] and an elevated lactate/pyruvate ratio.[91]

d. Evaluating the Post-Hearing Scoring.

Doctor Cave's post-hearing rescoring, necessitated by evidence demonstrating that A.H.M. did not have the symptoms or test results required by the Morava diagnostic criteria, reduced A.H.M.'s point total to 5, a "probable" mitochondrial disorder under the Morava scoring system. However, respondent's expert, Dr. Wiznitzer, had previously challenged the factual basis for, or Dr. Cave's interpretation of, most of the remaining symptoms or test results for these 5 points. I resolve the factual issues largely in favor of Dr. Wiznitzer's testimony, although I note that Dr. Wiznitzer's scoring was not without some of the same problems that Dr. Cave demonstrated.[92]

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[90] During the hearing, Dr. Cave testified that she awarded points for an elevated blood lactate level based on testing ordered by Dr. Bradstreet in November 2008. Tr. at 41, 84; see Pet. Ex. 14, p. 50 (test results). Admitting the lactate level was only "slightly over the top limit" (2.1 mmol/L with a normal range being 0.4-2.0), she maintained the designated two points should be awarded. Tr. at 42, 84. Additional serum lactate testing ordered by Dr. Bradstreet in 2010 and 2011 (approximately one and two years after the slightly elevated result) showed normal plasma lactate levels. The January 5, 2010 plasma result was 13.2 mg/dL with a normal range of 4.5-19.8. Pet. Ex. 14, p. 53. A January 3, 2011 plasma result was 9.7 mg/L using the same normal range. *Id.*, p. 61. Doctor Wiznitzer noted that the Wolf/Nijmegen criteria (and thus the Morava criteria) require a high lactate level on three separate occasions before points can be awarded. Tr. at 160; *see also* Res. Ex. D, Tab 1 at 3. He testified that the slightly elevated lactate level reported in 2008 was likely due to the tourniquet used during the blood draw and the results obtained by Dr. Holmes in 2003 were from A.H.M.'s urine, not blood. Tr. at 147-48, 160; *see* Pet. Exs. 8, p. 16; 14, p. 50. Blood lactate testing subsequent to the urine lactate testing was normal or below. These July 31, 2000 test results were filed in Dr. Wampler's records at Pet. Ex. 14. p. 121.

[91] Although Dr. Cave awarded one point for an elevated lactate/pyruvate ratio in her second expert report (Pet. Ex. 11-a at 7-8), she abandoned this argument during the hearing, stating that A.H.M. had "10 points without even considering lactate pyruvate ratios." Tr. at 41-42. Petitioners maintained this stance after the hearing as well. Pet. Post-Hearing Br. at 2-3. They could hardly do otherwise, as there were no tests that measured plasma lactate/pyruvate ratios in A.H.M.'s records.

[92] Like Dr. Cave, Dr. Wiznitzer had some difficulty with the Morava criteria's scoring. He initially indicated a score of 4 would mean a mitochondrial disorder was "unlikely" (Tr. at 160), but corrected that statement himself only minutes later, acknowledging that a score of 4 would be in the "possible" mitochondrial disorder range (Tr. at 162). He made one other mistake, in that he awarded 1 point instead of 2 points in his expert report for testing showing elevated urinary TCAs. Res. A. at 8 (expert report); Morava Table, Pet. Ex. 26 (indicating that this test scored 2 points). He corrected that error in his testimony. Tr. at 160. Nevertheless, he demonstrated a far greater understanding of the Morava criteria and mitochondrial disorder diagnosis requirements than did Dr. Cave.

Doctor Wiznitzer testified that A.H.M. did not suffer from a mitochondrial disorder. Tr. at 140. Often referencing the more detailed explanation for these categories provided by the Wolf (Nijmegen) criteria, he challenged every point awarded by Dr. Cave except the 1 point for developmental delay (Tr. at 155-56). His scoring would place A.H.M. in the category of an unlikely mitochondrial disorder (Pet. Ex. 11-a, Tab 11 at 2).

Although Dr. Cave lowered A.H.M.'s point total to 5 after the hearing, she maintained this score was sufficient to support her opinion on causation as it placed A.H.M. in the range of probable mitochondrial disorder. Pet. Ex. 35.

4. Conclusions Regarding Application of the Morova Criteria for Diagnosis.

I find that proper scoring of the Morava criteria in this case results in a diagnosis of, at best, a possible mitochondrial disorder, and the more reliable score would place her in the "unlikely" category. A.H.M.'s developmental delay was the only symptom that met the clinical Morava/Nijmegen criteria, accounting for 1 point.[93] At best, the score for the metabolic criteria would add 3 points, but given the erratic results from MetaMetrix Clinical Laboratory ["MetaMetrix"], coupled with the contradictory reports from the University of Chicago laboratory finding A.H.M.'s urinary organic acids showed no specific abnormalities (Pet. Exs. 6, pp. 20, 35), no points should be awarded in this category.

a. Clinical Criteria-Muscle Weakness.

I find that A.H.M. did not have muscle weakness meeting the requirements under the Morava criteria. Except in the case of a young infant, this criterion requires reduced muscle power.

Post-hearing, Dr. Cave continued to maintain that A.H.M. displayed muscle weakness (Pet. Ex. 35 at 1), based on a reference to hypotonia in A.H.M.'s pediatric records (Pet. Ex. 6, p. 9). As Dr. Wiznitzer explained, low muscle tone (hypotonia) is not the same as muscle weakness. Tr. at 155, 180. She acknowledged that she had reviewed the Nijmegen (Wolf) article filed as Court Ex. I (Tr. at 36), but had apparently never looked at the supplemental materials referenced in the article. Those materials were filed post-hearing by respondent. Res. Ex. D, Tab 1. These supplemental materials fully supported the testimony of Dr. Wiznitzer that hypotonia alone in a toddler is not sufficient to meet the diagnostic criteria of "muscle weakness."[94]

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[93] Both Dr. Cave and Dr. Wiznitzer agreed that A.H.M. suffered developmental delay. Tr. at 36; Pet. Ex. 35 at 1 (Doctor Cave); Tr. at 156 (Doctor Wiznitzer). That symptom accounted for 1 point under Section I of the Morava Table.

[94] Muscle weakness is defined as:

> reduced muscle power (as evident by formal testing revealing weakness, if possible, or

Differentiating between muscle tone and muscle strength, Dr. Wiznitzer described muscle tone as the idle state of a car engine, which "tells you nothing about how fast the car can run" (Tr. at 153), and muscle strength as the car's horsepower (Tr. at 154). He pointed to the portion of the CEC evaluation indicating A.H.M. had diminished muscle tone but adequate muscle strength. Tr. at 153; *see* Pet. Ex. 6, p. 2. He added that, according to the Wolf criteria, muscle tone or hypotonia could be used to fulfill the criteria of muscle weakness as claimed by Dr. Cave but only until the child is six months old. Tr. at 154-55; *see* Res. Ex. D, Tab 1 at 1. He then quoted the examples provided in the Nijmegen criteria for a child less than six months of age, explaining that "head lag on traction" involved a baby whose head lags or falls back when you pull her up from the arms; "poor head control" involved a baby whose head falls in different directions when placed in a seated position; "slip through" involves a baby who, when picked up, falls through the adult's arms; and "frog-like posture" as an infant whose legs fall into a frog-like position when placed supinely. Tr. at 154-55. A.H.M. displayed none of these classic indications of muscle weakness.

Thus, Dr. Wiznitzer concluded that no points should be awarded for muscle weakness. I agree.

b. Metabolic Studies.

In her post-hearing report, Dr. Cave awarded points for elevated levels of TCA (2 points) and ethylmalonic acid (1 point) based on the results of testing ordered by Dr. Holmes in May 2000. Tr. at 41-42.

These elevated levels were detected in urine testing ordered by Dr. Holmes in May 2000 and conducted by MetaMetrix.[95] Pet. Ex. 8, p. 16. The ethylmalonic acid level appears on the test as "ethylmalonate," under the heading of "Fatty Acid Metabolism," and was reported as 6.6 µg/mg creatinine. The TCA results appear under the heading of "Energy Production (Citric Acid Cycle). All of the TCA results were well above the reference ranges on this MetaMetrix test. *Id.*

> signs as Gower's sign or absent or bad head control or delayed motor milestones[,] the latter only if mental development is normal or much advanced in comparison to motor development) or muscular hypotonia (in the newborn period and up to a developmental age of 6 months with head lag in traction test, poor head control, slipping through-sign, frog-like posture when awake and with limply hanging head and limbs in ventral suspension)

Res. Ex. D, Tab 1, at 1. A.H.M. did not meet these criteria, as her hypotonia was not evident in the newborn to six months of age period, and she did not show a lack of strength or delayed motor milestones.

[95] During direct examination, respondent's counsel briefly inquired about why MetaMetrix's results differed from the results on the same tests performed just months earlier, but neither party introduced any further evidence regarding the reliability of this laboratory's testing generally. Tr. at 140-45.

Doctor Wiznitzer considered the MetaMetrix test results to be suspect because similar testing by the University of Chicago had produced normal results in February and April 2000, several months earlier and, thus, closer in time to the allegedly causal vaccines. Tr. at 140-42; *see* Pet. Ex. 16, p. 24 (February 2000 test results); *id.*, p. 35 (April 2000 test results).[96] He testified, referring to the MetaMetrix results, that he rarely saw tests with such across-the-board abnormal results. Tr. at 142-44. He thought that that "the MetaMetrix result was affected by a low creatinine level in the urine and that the result is not a true measure of the organic acid content in the urine." Tr. at 144; *see* Pet. Ex. 8, p. 16 (test results showing a urinary creatinine level of 21 mg/dl).[97]

Mr. Cave cross-examined Dr. Wiznitzer about his reliance on the University of Chicago testing, noting the results were simply a summary, rather than a report identifying the specific organic acids tested and the results for each, unlike the MetaMetrix results.[98] Nevertheless, Dr. Wiznitzer asserted that "all labs that do urine organic acids will include the TCAs." Tr. at 185.

MetaMetrix performed urine organic acid testing in November 2000 and March 2001, during the time period when A.H.M. was undergoing chelation. Pet. Exs. 8, pp. 174, 179 (test results), 9, 25; 27, 35 (references to chelation during these time frames); 14, pp. 99, 105, 111, 122 (reference to chelation and laboratory tests from July through late October 2000 reflecting that urine tests were performed "post provocation," a reference to chelation). The November test for ethylmalonate showed a level of 49.4, more than 10 times the reference range and more than seven times the May 2000 result. The TCA testing results were mixed. Several organic acids were within the reference ranges, three remained high, and one was well below the reference range reported. *Id*., p. 179.

The March 2001 test reported an ethylmalonate level of 5.1, above the reference range, but much closer to the May 2000 results rather than the November 2000 results. The TCA testing results were also mixed in March 2001, but they were very different with regard to specific acids. Pet. Ex. 8, p. 174. The chart below reflects the extremely wide variation in results reported in the three tests performed by MetaMetrix:

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[96] The results for each urinary organic acid were not reported individually. The report simply stated that: "The urine organic acids screen shows no specific abnormalities." Pet. Ex. 16, pp. 24, 35.

[97] Two later tests performed by MetaMetrix laboratory in November 2000 and March 2001 had creatine levels of 16 and 38, respectively. Pet. Ex. 8, pp. 179, 174. There were a smaller number of high values (less than 50%) in the March test results which had the higher creatine level as compared to the November 2000 results which (like the May 2000 results) showed across the board elevated levels.

[98] Both reports from February and April 2000 testing by the University of Chicago indicate only a "urine organic acids screen" showing no abnormalities. Pet. Exs. 6, p. 20; 16, p. 35.

Urine Organic Acid	May 2000		Nov 2000		Mar 2001		Reference Range
Ethylmalonate	6.6	H	49.4	H	5.1	H	≤ 4
Citrate	2339	H	795.0		2607.0	H	500-2300
Cis-Aconitate	676	H	208.0		124.0		5-250
Isocitrate	1766	H	1801.0	H	563.0		50-800
a-Ketoglutarate	71.7	H	<0.4	L	12.8		3-25
Succinate	63.8	H	147.0	H	213.9	H	5-35
Fumarate	1.32	H	0.49		2.85	H	0.2-1.2
Malate	7.5	H	14.10	H	5.5		≤6
Hydroxymethylglutarate	3.54	H	7.94	H	1.45	H	0.2-1

c. Conclusion.

A.H.M.'s clinical presentation justifies the award of only 1 point. The point total for the metabolic and imaging studies is a closer call, but without a rational explanation for the highly variable results, I find it difficult to attach much weight to MetaMetrix's findings of high ethylmalonic acid and TCAs. That uncertainty, particularly in view of the University of Chicago laboratory tests reporting essentially normal results,[99] leads me to conclude that no points should be awarded.

Even if I thought points should be awarded, A.H.M.'s total under the Morava criteria would increase from only 1 to 4 points for the MetaMetrix test results. This amount still is not sufficient to raise her score to the range for a "probable" mitochondrial disorder, taking her from "unlikely" to "possible."

D. Mitochondrial Dysfunction.

In her post-hearing report, Dr. Cave reiterated a point she made during her testimony: that mitochondrial dysfunction may exist in individuals who cannot be diagnosed with mitochondrial disease. Pet. Ex. 35 at 1-3. She described this dysfunction as lacking the genetic basis present in mitochondrial disorders, and contended that medical studies demonstrate that those with ASD diagnoses have more mitochondrial dysfunction than typically developing children. Her post-hearing report

[99] In this regard, I note that, although the University of Chicago did not report results individually for each type of organic acid, when a result on other tests was abnormal, a specific note was made by the laboratory director. *See* Pet. Ex. 16, p. 23 (reporting a mild elevation of glycine on a urine amino acids screen in February 2000 and indicating that it might be significant in diagnosing a disorder of glycine metabolism) and p. 34 (urine amino acids screen report from April 2000 specifically reporting that glycine was within normal limits). While I cannot conclude from the summary reports of urine organic acids screening that all of A.H.M.'s urine organic acids were within normal limits or reference ranges, I am confident that a pattern of abnormal results would have been reported. Given their use in diagnosing mitochondrial disorders, elevations of ethylmalonic acid and TCAs would have been significant findings, and, if they existed, the laboratory report would have noted any elevated levels.

implied that mitochondrial dysfunction is a milder form of a mitochondrial disorder. *See id*. at 2. She did not explain how there can be a mild form of a genetic disorder that lacks any genetic basis.

Doctor Cave and petitioners, however, provide no evidence that A.H.M. suffers mitochondrial dysfunction other than a score under the Morava criteria insufficient to establish she suffers from a mitochondrial disorder. No treating doctor ever diagnosed A.H.M. with mitochondrial disorder, disease, or dysfunction. Doctor Holmes was the only treating doctor to mention the possibility of a mitochondrial disorder and, given the serum lactate testing results, dropped the issue.

There is simply no evidence that A.H.M. showed any sign or symptom that her mitochondria were dysfunctional. In her post-hearing report, Dr. Cave did not point to any test results (other than the MetaMetrix urine organic acids results discussed above) that suggested that A.H.M.'s mitochondria had diminished function. In citing to medical journal articles that she claimed demonstrated that children with ASD had markers of mitochondrial dysfunction (Pet. Ex. 35 at 1, referring to an unspecified literature review by Rossignol and Frye[100]), she did not point to any tests other than those discussed above (the TCA tests) that showed any impairment in mitochondrial function. She claimed that treatment by Dr. Holmes improved the citric acid chemistry (Pet. Ex. 35 at 2), but the March 2001 testing as compared to the May 2000 testing, as reflected on the table above, belies that claim. Inexplicably, Dr. Cave referred to a "MTRR"

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[100] Doctor Cave did not specify to which of the several review articles or abstracts authored by D. Rossignol and R. Frye filed as petitioners' exhibits in this case she referred in her post-hearing report. *See* Pet. Ex. 11-a, Tab 7 (R. Frye & D. Rossignol, *Mitochondrial Dysfunction Can Connect the Diverse Medical Symptoms Associated With Autism Spectrum Disorders*, PEDIATRIC RESEARCH, 69(5), Part 2: 1-16 (2011); Pet. Ex. 11-a, Tab 8 (D. Rossignol & R. Frye, *A review of research trends in physiological abnormalities in autism spectrum disorders: immune dysregulation, inflammation, oxidative stress, mitochondrial dysfunction and environmental toxicant exposures*, MOL. PSYCHIAT., published online (doi:10.1038/mp.2011.165 (2011)); Rossignol & Frye, Pet. Ex. 24; and Pet. Ex. 36, R. Frye & D. Rossignol, *Mitochondrial physiology and autism spectrum disorder*, OA Autism (2013) ["hereinafter, Frye and Rossignol, Pet. Ex. 36"]. A portion of the article filed as Pet. Ex. 24 was also filed as Pet. Ex. 11-a, Tab. 6. The review article filed as Pet. Ex. 11-a, Tab 8 was not helpful as it simply counted journal articles that implicated an association between a physiological abnormality and ASD and finding that many more journal articles discussing mitochondrial dysfunction had been filed more recently, without discussing the substance of the findings. Petitioners' Ex. 11-a, Tab 7 lists some features of mitochondrial dysfunction seen in children with ASD, but other than developmental delay and the autism diagnosis itself, there is no evidence that A.H.M. had the symptoms or the laboratory abnormalities discussed (excessive fatigability, epilepsy, neuropathy, endocrine disorders, gastrointestinal abnormalities, abnormalities in pyruvate and acyl-carnitines, etc.). Doctor Cave was likely referring to Rossignol & Frye, Pet. Ex. 24, as the source of her claim that mitochondrial dysfunction could be considered as a less severe form of mitochondrial disease (Pet. Ex. 35 at 1-2) as the authors make this claim on the first page of the article. However, although the authors asserted that studies had documented secondary mitochondrial dysfunction present in many children with ASD, the studies discussed test evidence of dysfunction such as the levels of glutathione, proinflammatory cytokines, and proprionic acid. *Id*. at 24, 26. A.H.M. did not have any of these tests.

polymorphism as restricting "her ability to detox through the glutathione pathway in the liver." Pet. Ex. 35 at 3. There was no evidence I could find in this record reflecting this polymorphism test result and Dr. Cave did not cite to any record showing it.

I conclude that there is insufficient evidence in this record to establish that A.H.M. suffered mitochondrial disease, disorder, or dysfunction.

VII. Evaluating Petitioners' Causation Claims.[101]

There are two ways to establish entitlement to compensation under the Vaccine Act's no-fault system. First, petitioners may demonstrate that A.H.M. suffered a vaccine-specific injury listed on the Vaccine Injury Table within the requisite time period set forth in the Table (a "Table injury"). To prove a Table injury, petitioners must show that the first symptom or manifestation of the onset…of any such illness, disability, injury, or condition…occurred within the time period after vaccine administration set forth in the Vaccine Injury Table." *Shalala v. Whitecotton,* 514 U.S. 268, 270 (1995) (quoting 42 U.S.C. §11(c)(1)(C)(i)). In such cases, causation is presumed. *See* 42 C.F.R. § 100.3.

Second, petitioners may demonstrate by preponderant and reliable evidence that an injury was caused in fact or significantly aggravated by a vaccine listed on the Table ("actual causation" or "causation-in-fact" or "significant aggravation"). *See* § 11(c)(1)(C); *see also Moberly v. Sec'y, HHS,* 592 F.3d 1315, 1321 (Fed. Cir. 2010) (causation in fact claims); *W.C. v. Sec'y, HHS,* 704 F.3d 1352, 1357 (Fed. Cir. 2013); *Loving v. Sec'y, HHS,* 86 Fed. Cl. 135, 144 (2009); *Hennessey v. Sec'y, HHS,* No. 01-190V, 2009 WL 1709053 at *40 (Fed. Cl. Spec. Mstr. May 29, 2009), *aff'd,* 91 Fed. Cl. 126 (2010) (significant aggravation claims).

To prove actual causation, petitioners must demonstrate by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Althen v. Sec'y, HHS*, 418 F.3d 1274, 1278 (Fed. Cir. 2005); *see also Grant v. Sec'y, HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992); *Hines v. Sec'y, HHS*, 940 F.2d 1518, 1525 (Fed. Cir. 1991); *de Bazan v. Sec'y, HHS*, 539 F.3d 1347, 1351-52 (Fed. Cir. 2008); *Caves v. Sec'y, HHS,* 100 Fed. Cl. 119, 132 (2011), *aff'd per curiam*, 463 Fed. Appx. 932, 2012 WL 858402 (Fed. Cir. 2012) (holding that each *Althen* factor must

[101] The only claim not discussed in this decision is an off-Table claim (based on an unpublished paper authored by Theresa Deisher, Ph.D.) involving human DNA in vaccines which petitioners bought to my attention "[o]ut of an abundance of caution." *See* Pet. Pre-Hearing Memo. at 14. Petitioners acknowledged the claim "had not been specifically raised via expert report or amended petition." *Id.* Moreover, they submitted no evidence regarding this claim and did not mention it in subsequent filings. There is no evidence in the record to support this claim.

be established by preponderant evidence). The applicable level of proof is the "traditional tort standard of 'preponderant evidence.'" *Moberly*, 592 F.3d at 1322 (citing *de Bazan*, 539 F.3d at 1351; *Pafford v. Sec'y, HHS*, 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Capizzano v. Sec'y, HHS*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *Althen,* 418 F.3d at 1278).

Petitioners' assertions regarding the factual predicate for their claims are so far removed from the facts of this case that I could deny petitioners' claims on that basis alone. However, I elect to discuss their poorly articulated Table claim and their causation in fact case. Under the facts of this case, it was unreasonable to maintain that A.H.M. experienced a Table encephalopathy.

A. The Table Injury Claim.

To establish a Table encephalopathy, petitioners must demonstrate A.H.M. suffered an "encephalopathy" as defined by the QAI section to the Vaccine Injury Table within five to fifteen days of her MMR vaccination or within seventy two hours of her DTaP vaccination.[102]

1. The Table Definitions.

According to the QAI, a vaccinee is considered to have suffered a Table encephalopathy if the vaccinee manifests an injury encompassed in the definition of an acute encephalopathy within the appropriate time period, and if a chronic encephalopathy is present for more than six months after the immunization. 42 C.F.R. § 100.3(b)(2).

An acute encephalopathy is "one that is sufficiently severe so as to require hospitalization (whether or not hospitalization occurred)." 42 C.F.R. § 100.3(b)(2)(i). For a child younger than 18 months of age,[103] presenting without an associated seizure event, an acute encephalopathy is indicated "by a significantly decreased level of consciousness . . . lasting for at least 24 hours." 42 C.F.R. § 100.3(b)(2)(i)(A). A significantly decreased level of consciousness is indicated by the presence of one of three clinical signs for a period of at least 24 hours: "(1) Decreased or absent response to environment (responds, if at all, only to loud voice or painful stimuli); (2) Decreased or absent eye contact (does not fix gaze upon family members or other individuals); or (3) Inconsistent or absent responses to external stimuli (does not recognize familiar people or things)." 42 C.F.R. § 100.3(b)(2)(i)(D). Sleepiness, irritability (fussiness), high-

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[102] 42 C.F.R. § 100.3(a). Although petitioners included only the portion of the Vaccine Table which deals with an MMR Table encephalopathy (*see* Pet. Pre-Hearing Memo. at 5), I will consider whether A.H.M. suffered a Table encephalopathy following the administration of either the MMR or DTaP vaccines.

[103] A.H.M. was not 18 months old until November 14, 1999, placing her case in the "younger than 18 months" period for purposes of evaluating whether she experienced an acute encephalopathy.

pitched and unusual screaming, persistent inconsolable crying, and bulging fontanelle are insufficient, standing alone or in combination, to demonstrate an acute encephalopathy. 42 C.F.R. § 100.3(b)(2)(E).

A chronic encephalopathy is defined in the QAI as "a change in mental or neurologic status, first manifested during the applicable time period [that] persists for a period of at least 6 months from the date of vaccination." 42 C.F.R. § 100.3(b)(2)(ii). If a person returns to a typical neurologic state after suffering an acute encephalopathy, he or she is not presumed to have suffered residual neurologic damage and "any subsequent chronic encephalopathy shall not be presumed to be a sequela of the acute encephalopathy." *Id.*

"The symptoms associated with an acute encephalopathy are neither subtle nor insidious." *Waddell v. Sec'y, HHS*, 2012 WL 4829291, at *6 (Fed. Cl. Spec. Mstr. Sept. 19, 2012). As noted in *Waddell*, "[t]he hospitalization requirement underscores how serious the symptom presentation must be after vaccination to merit classification as a Table encephalopathy." *Id*. at *7 (citing to Revision of the Vaccine Injury Table, 60 Fed. Reg. 7,685, 7,687 (Feb. 20, 1997) (preamble to final rule) ("[W]e did not intend that hospitalization be viewed as an absolute requirement to establish an acute encephalopathy, but rather as an indicator of the severity of the acute event.").

In contrast, "encephalopathy"[104] as commonly used in the medical community encompasses a much broader class of injuries than the more stringent definition of acute encephalopathy found in the QAI. As explained in *Waddell*, "[t]he scope of the medical term 'encephalopathy' is more expansive than the narrower, statutory definition set forth in the Table." *Id*. at *12 (referencing *Hazelhurst*, 2009 WL 332306, at *26-29). The QAI definition of acute encephalopathy simply does not encompass every type of brain dysfunction to which the broader meaning of "encephalopathy" applies.

2. Evaluating Petitioners' Table Encephalopathy Claim.

There is nothing in the record to indicate A.H.M. suffered any reaction to the DTaP and MMR vaccines she received on September 15, 1999, much less an encephalopathy as defined in the QAI section to the Vaccine Injury Table. Certainly, she did not suffer an acute encephalopathy within the time periods required by the Vaccine Injury Table. *See* 42 C.F.R. § 100.3(a)II.B and III.B.

In their pre-hearing memorandum, petitioners list as proof of A.H.M.'s encephalopathy the rash A.H.M. experienced in July 1999, the speech delay noted just prior to vaccination on September 15, 1999, and the concerns noted during A.H.M.'s February 23, 2000 evaluation. *See* Pet. Memo at 5-7. Not only do these symptoms and

[104] Encephalopathy is defined very broadly as "any degenerative disease of the brain." Dorland's at 614

concerns not qualify as proof of encephalopathy, but the record does not show that any of these conditions began or were significantly aggravated in the time periods set forth for the MMR and DTaP vaccines. In fact, both the rash and speech delay occurred before A.H.M. even received these vaccinations. Although A.H.M. developed a rash within a few weeks of the varicella vaccination she received in June, since there is no Table injury associated with that vaccine, any reaction is irrelevant to the Table injury claim.

Even if petitioners could establish the symptoms and behavior they describe occurred or were significantly aggravated during the appropriate time frame, they fail to constitute an encephalopathy "sufficiently severe as to require hospitalization." 42 C.F.R. § 100.3(b)(2)(i). Petitioners argue that A.H.M.'s autistic symptoms are sufficient to establish that she suffered the decreased level of consciousness that is required to establish an acute encephalopathy in a child younger than 18 months. *See* 42 C.F.R. § 100.3(b)(2)(i)(A). There is a difference, however, in the various symptoms and behaviors autistic children often exhibit and the clinical signs listed in the QAI. *See* 42 C.F.R. § 100.3(b)(2)(i)(D).

For example, A.H.M.'s inconsistent response to her name (*see, e.g.*, Pet. Ex. 19, p. 3) is not equivalent to the decreased or absent response to the environment enumerated in the QAI. *See* 42 C.F.R. § 100.3(b)(2)(i)(D)(1). To meet the lack of response described in the QAI, a child must not respond, or respond only to a **loud** voice or **painful** stimuli. *See id.* (emphasis added). This lack of response is more profound than the inconsistent response shown by many autistic children like A.H.M.

Likewise, A.H.M.'s lack of eye contact is not equivalent to the decreased or absent eye contact listed in the QAI. *See* 42 C.F.R. § 100.3(b)(2)(i)(D)(2). To meet the QAI description, a child must fail to gaze on family members as well as other individuals. *See id.* At times, A.H.M. showed good eye contact such as that noted at her January 26, 2000 evaluation. *See* Pet. Ex. 19, p. 12. At other times, she showed a lack of eye contact with others but good eye contact with family members. *See* Pet. Ex. 20, p. 3.

Finally, although A.H.M. sometimes exhibited a lack of response, there is nothing to indicate she failed to recognize familiar people and things. *See* 42 C.F.R. § 100.3(b)(2)(i)(D)(3). A.H.M.'s autistic symptoms alone do not qualify as any of the clinical signs of the decreased level of consciousness required by the QAI, which clearly requires symptoms which are more intense and consistent than those A.H.M.displayed and which manifested during the very specific time periods provided.

Doctor Wiznitzer eloquently explained this difference during the hearing. He testified that the lack of eye contact needed to satisfy one of the clinical signs of an acute encephalopathy stems from a lack of visual function because the brain is not awake and alert. Tr. at 211-12. The signals coming into the eye are "not processed in any manner because there's an impairment in consciousness." Tr. at 213. The information comes into the brain but is not processed. *Id.* In contrast, the autistic child

is receiving and processing information but is being selective in how she responds. Tr. at 213-14. This child is not making eye contact due to an impairment in social behavior, not because she is failing to process the visual information. Tr. at 214. An autistic child has a deficit in socialization and social communication. Tr. at 217. She will respond to her favorite TV show but not to her name. This behavior is different than that of a child experiencing acute encephalopathy.

A.H.M.'s condition after receiving the MMR and DTaP vaccinations does not meet the requirements established by the Vaccine Injury Table for encephalopathy. Because petitioners failed to demonstrate that A.H.M. suffered a Table injury, they can prevail only on an actual causation claim.

B. Causation in Fact Claim.

1. Law Pertaining to Off-Table Claims.

The Federal Circuit has set forth three factors petitioners must establish to prove causation in off-Table cases. *See Althen*, 418 F.3d 1274, 1278 (Fed. Cir. 2005). *Althen* requires petitioners to provide: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* All three prongs of the *Althen* test must be satisfied by preponderant evidence. *De Bazan v. Sec'y, HHS*, 539 F.3d 1347, 1351-52 (Fed. Cir. 2008); *Caves v. Sec'y, HHS*, 100 Fed. Cl. 119, 132 (2011), *aff'd per curiam*, 463 Fed. Appx. 932, 2012 WL 858402 (Fed. Cir. 2012) (finding that "[w]hen a petitioner seeks to demonstrate causation in fact by meeting the three *Althen* requirements, each of those requirements must be proven by a preponderance of the evidence"). Petitioners may satisfy this evidentiary burden by relying either on "medical records *or* medical opinion." *Althen,* 418 F.3d at 1279 (emphasis in original).

A petitioner is not required to establish identification and proof of specific biological mechanisms, as "the purpose of the Vaccine Act's preponderance standard is to allow the finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body." *Althen*, 418 F.3d at 1280. The petitioner does not have to show that the vaccination was the sole cause, or even the predominant cause, of the injury or condition; showing that the vaccination was a "substantial factor"[105] and

[105] The Restatement (Third) of Torts has eliminated "substantial factor" in the factual cause analysis. § 26 cmt. j (2010). Because the Federal Circuit has held that the causation analysis in Restatement (Second) of Torts applies to off-Table Vaccine Act cases (*see Walther v. Sec'y, HHS*, 485 F.3d 1146, 1151 (Fed. Cir. 2007); *Shyface v. Sec'y, HHS,* 165 F.3d 1344, 1352 (Fed. Cir. 1999), this change does not affect the determination of legal cause in Vaccine Act cases: whether the vaccination is a "substantial factor" is still a consideration in determining whether it is the legal cause of an injury. *See Stone v. Sec'y, HHS,* 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[T]he causation standard in off-Table Vaccine Act cases is to be applied consistently with the principles set forth in the Second Restatement of Torts.").

a "but for" cause of the injury are sufficient for recovery. *Shyface,* 165 F.3d at 1352; *see also Pafford,* 451 F.3d at 1355 (petitioner must establish that a vaccination was a substantial factor and that harm would not have occurred in the absence of vaccination).

Although a petitioner cannot be required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect,"[106] when a party files medical literature, a special master may weigh and evaluate that medical literature. When the filed literature fails to support the medical theory alleged, it can be an important factor in determining whether petitioner has met her burden to show vaccine causation.

Causation is determined on a case by case basis, with "no hard and fast *per se* scientific or medical rules." *Knudsen v. Sec'y*, *HHS,* 35 F.3d 543, 548 (Fed. Cir. 1994). Close calls regarding causation must be resolved in favor of the petitioner. *Althen*, 418 F.3d at 1280; *but see Knudsen*, 35 F.3d at 550 (when evidence is in equipoise, the party with the burden of proof fails to meet that burden).

Althen requires that a petitioner in an off-Table causation case present a reliable medical theory by which a vaccine can cause the injury in question. *Althen*, 418 F.3d at 1278. This first prong of *Althen's* three-part causation test has also been characterized as the equivalent of the "Can it cause?" inquiry used in toxic tort litigation. *See Pafford v. Sec'y, HHS*, No. 01-165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *aff'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). The medical theory must be a reputable one, although it need only be "legally probable, not medically or scientifically certain." *Knudsen,* 35 F.3d at 548-49. Theories of causation must be reliable as well. *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 590 (1993); *Moberly,* 592 F.3d at 1324.

Althen's second prong requires a logical sequence of cause and effect between the vaccine and the injury. It has been characterized as addressing the "Did it cause?" or specific causation query. *See Pafford*, 2004 WL 1717359, at *4. Circumstantial evidence and medical opinions may be sufficient to satisfy this requirement. *Capizzano*, 440 F.3d at 1325-26. Opinions of treating physicians may also provide the logical connection. *See Andreu v. Sec'y, HHS*, 569 F.3d 1367, 1376 (Fed. Cir. 2009); *see also Moberly,* 592 F.3d at 1323; *Capizzano*, 440 F.3d at 1326.

The third *Althen* factor requires a proximate temporal relationship between the allegedly causal vaccine and the injury suffered. The requirement of temporal connection necessitates a showing that the injury occurred in a medically or scientifically reasonable period after the vaccination, not too soon (*see de Bazan*, 539

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[106] *Capizzano v. Sec'y, HHS,* 440 F.3d 1317, 1325 (Fed. Cir. 2006).

F.3d at 1352) and not too late (*see Pafford*, 451 F.3d at 1358). Merely showing a temporal connection between a vaccination and an injury is insufficient, standing alone, to establish causation. *Grant*, 956 F.2d at 1148. A temporal relationship, even when coupled with the absence of any other identified cause for the injury, is not enough to demonstrate probable cause under the Vaccine Act's preponderance standard. *Moberly*, 592 F.3d at 1323 (citing *Althen*, 418 F.3d at 1278).

2. Applying *Althen*.

a. Lack of a Reliable Medical Theory.

To satisfy the first prong of the *Althen* test, petitioners must provide "a medical theory causally connecting the vaccination and the injury." *Althen*, 418 F.3d at 1278 (quoting *Grant v. Sec'y, HHS*, 956 F.2d 1144, 1148 (Fed. Cir. 1992)). Although petitioners are not required to provide an exact mechanism for their theory, the medical theory must be a reliable one. *Knudsen*, 35 F.3d at 548 ("This 'logical sequence of cause and effect' must be supported by a sound and reliable medical or scientific explanation."). Petitioners must prove the existence of this medical theory by a preponderance of the evidence. *Broekelschen v. Sec'y, HHS*, 618 F.3d 1339, 1350 (Fed. Cir. 2010). In other words, petitioners must show that it is more likely than not that the received vaccine **can** cause the alleged injury. *Pafford*, 451 F.3d 1352, 1355-56 (Fed. Cir. 2006) (emphasis added).

In her expert reports, Dr. Cave asserts the vaccines A.H.M. received caused A.H.M. to suffer mitochondrial dysfunction resulting in autistic symptoms (which Dr. Cave called an encephalopathy) (*see* Pet. Ex. 11-a at 8) or caused a regression and encephalopathy which was more pronounced in A.H.M. because she suffers from mitochondrial dysfunction (*see* Pet. Ex. 11 at 1). Doctor Cave switched with abandon between these variations of her theory. When asked on cross examination to clarify her theory of causation, Dr. Cave answered, "I think it's the metal toxicity from the vaccine mainly that caused the mitochondrial disorder, and it manifested in behavior and regression." Tr. at 76. She claimed, however, that her theory would be sufficient even without the presence of mitochondrial dysfunction. Tr. at 86.

Doctor Cave provided few details about and little support for her theory. The medical literature offered by Dr. Cave either did not indicate what she claimed or did not provide support for her theories. She referred to oxidative stress on several occasions but failed to explain how the vaccines A.H.M. received could have caused oxidative stress sufficient to result in the damage she describes.[107] Identifying the "toxins" in the

[107] When asked if she had any expertise regarding oxidative stress, Dr. Cave replied only that she has treated patients who were ""oxidatively stressed" and taught seminars on the subject. Tr. at 100. She also maintained that vaccines cause more oxidative stress than infections, claiming she reached this conclusion based on "what [she has] seen with patients." Tr. at 103. She listed specific biomarkers for oxidative stress but offered no evidence to establish A.H.M. suffered from it. Tr. at 103-04.

vaccines A.H.M. received, Dr. Cave listed monosodium glutamate and heavy metals such as aluminum and mercury but sometimes included other components as well as the live virus itself. *See, e.g.*, Pet. Ex. 11-a at 10-11. She offered no information regarding the quantities required for toxicity, the quantities present in the vaccines, or any reliable evidence that these amounts could cause mitochondrial dysfunction, impair mitochondrial dysfunction or cause ASD to develop.

Doctor Cave also labeled A.H.M.'s condition immunocytotoxicity. *See id.* at 8. She stated that "[t]he mitochondrial dysfunction combined with the glial cell excitotoxicity from a toxin like MSG (Varicella vaccine) as well as the metals, affecting developing neurons-altering their function and development." Pet. Ex. 35 at 3. Except for the added element of mitochondrial dysfunction and the expansion of toxins to include MSG and metals other than mercury, this theory was discussed during the OAP Theory 2 test case litigation. *See, e.g., Dwyer*, 2010 WL 892250, at *27. Many of articles and studies that Dr. Cave claimed as support for her theories were discussed in the OAP litigation.

Doctor Cave testified that she based her definition of oxidative stress on the work of Jill James, Ph.D. and Richard Deth, Ph.D. Tr. at 101. Doctor Deth's opinions and testimony were thoroughly analyzed in the theory 2 OAP test cases and determined to be lacking.[108] As I indicated in *Dwyer*, "[i]n the course of the hearing, nearly every premise of [Dr. Deth's] causation theory, other than that of the ubiquity of mercury exposure in children (with or without autism), was seriously undermined, where not completely demolished." 2010 WL 892250, at *110. Although Dr. Deth claimed two studies from Dr. James provided the "the strongest evidence in support of his hypothesis" (*Dwyer*, 2010 WL 892250, at *109), I found Dr. James's work did not support, and often contradicted, Dr. Deth's assertions (*see id.*, at *133-42).

Doctor Cave also identified the work of Drs. Burbacher, Vargas, and Hornig as supportive of her theories. *See* Tr. at 52-55, 262-63. For example, she cited Dr. Vargas's study as "reporting the neuroactivation and neuroinflammation in the brains of patients with autism."[109] As discussed in *Dwyer*, the study did show "extensive microglial and astroglial activation" in the presence of a pathology which most likely occurred prenatally. *Dwyer*, 2010 WL 892250, at *49-50. There is no connection to or

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[108] I found his opinions and testimony farfetched and weak, lacking coherence and logic. *Dwyer*, 2010 WL 892250, at *108-09, 115. For example, while opining that levels of mercury lower than that contained in vaccines could induce oxidative stress, Dr. Deth "claimed the ability to detect the effects of mercury on cells at levels 100–1000 times lower than levels used by other researchers." *Dwyer*, 2010 WL 892250, at *110. One of my colleagues described Dr. Deth's approach to be "simply unscientific, and wholly invalid," his testimony to be unpersuasive, and the errors in his presentation to be "too numerous to detail." *King*, 2010 WL 892296, at *63.

[109] Pet. Ex. 11-a at 10. The article regarding the Vargas study was filed as Pet. Ex. 29 (D. Vargas et al., *Neuroglial Activation and Neuroinflammation in the Brain of Patients with Autism*, ANN. NEUROL. 57(1) 67-81 (2005)).

even a mention of, vaccines in the Vargas study. *See* Pet. Ex. 29. Likewise, I am familiar with the Hornig[110] and Burbacher[111] studies and neither provides the support Dr. Cave claimed.[112]

The new studies referenced by Dr. Cave share similar flaws and shortcomings. Discussing the articles involving MSG, Dr. Wiznitzer testified that the dosages administered were 10,000 and 54,000 times higher than what A.H.M. received via vaccination.[113] At that dose, there was structural injury to the test animals' brains but nothing to show they exhibited behavioral symptoms related to autism and, thus, no connection between MSG and autism. Tr. at 174-75. Doctor Wiznitzer similarly criticized Dr. Cave's claims regarding aluminum, stating that "[a]luminum is the third most common element on this planet" and has never been linked to autism by the "mainstream medical community." Tr. at 175. He testified that the article referenced by Dr. Cave "is basically a speculative paper that according to many vaccine safety experts . . . has many flaws in its logic and makes inappropriate assumptions."[114]

Although Dr. Cave acknowledged that the *Poling* claim was compensated as a Table injury for which causation is presumed, she cited the Poling case report, Pet. Ex. 34, as supporting her theory of causation.[115] Pet. Exs. 11 at 1; 11-a at 14. She also

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[110] M. Hornig, et al., *Neurotoxic effects of postnatal thimerosal are mouse strain dependent*, MOL. PSYCHIATRY 1-13 (2004). Petitioners did not file this article. I note that in the Theory 2 test cases, evidence was adduced that Hornig's results could not be duplicated. *Dwye*r, 2010 WL 892250 at *106.

[111] Pet. Ex. 11-a, Tab 17 (T. Burbacher et al., Comparison of Blood and Brain Mercury Levels in Infant Monkeys Exposed to Methylmercury or Vaccines Containing Thimerosal, ENVIRON HEALTH PERSPECT 113:1015-20 (2005).

[112] As discussed in *Dwyer*, "the [Burbacher] study did not mirror human infant dosing." 2010 WL 892250, at *90. "The amounts of mercury received differed, and the time frame between vaccinations was shorter." *Id.*

[113] Tr. at 174. Petitioners filed only the abstracts of these articles. *See* Pet. Ex. 11-a, Tab 4; 11-a, Tab 5. Respondent later filed the complete articles. *See* Res. Ex. A.2 (J. Olney & L. Sharpe, *Brain Lesions in an Infant Rhesus Monkey Treated with Monosodium Glutamate*, SCIENCE, NEW SERIES 166(3903): 386-88 (1969)); Res. Ex. A.4 (J. Olney, *Brain Lesions, Obesity, and Other Disturbances in Mice Treated with Monosodium Glutamate*, SCIENCE, NEW SERIES 164(3880): 719-21 (1969)).

[114] Tr. at 176; *see* Pet. Ex. 30 (L. Tomljenovic, C.A. Shaw, *Aluminum Vaccine Adjuvants: Are they Safe?*, CURRENT MEDICINAL CHEMISTRY 18(17): 2630-37 (2011).

[115] In the recent Federal Circuit decision in a mitochondrial disorder case*, Paluck v.Sec'y, HHS*, No. 2014–5080, 2015 WL 2403354, at *8-9 (Fed. Cir. May 20, 2015), the panel relied to some degree on the Poling case study in reversing the special master's decision denying compensation. The panel also found that the special master and respondent had conceded the plausibility of the petitioner's causation theory in *Paluck*. The Poling case settlement itself cannot be viewed as a concession that vaccines can trigger or aggravate mitochondrial disorders. Because the Vaccine Injury Table represents a blend of science and policy (*Shyface v. Sec'y, HHS*, 165 F.3d 1344, 1352-53. (Fed. Cir. 1999) (quoting legislative history acknowledging that the Table may provide compensation to some whose injuries are not vaccine-caused); *Shifflett v. Sec'y, HHS*, 30 Fed. Cl. 341, 345 (1994) (observing that Congress designed the Vaccine Injury Table to be "overinclusive"); a settlement of a Table injury case cannot be viewed as a

relied upon a series of articles written in the wake of this decision, one of which she authored.[116] In the article she authored, Dr. Cave erroneously implied that causation was found in the *Poling* case.[117]

The Poling child was diagnosed with a mitochondrial disorder after muscle biopsy and experienced a regression within the Table injury time frame; neither of these factors is present in A.H.M.'s case. I note that the Frye & Rossignol article filed post-hearing as Pet. Ex. 36 discussed the controversy raised by the Poling case (*id*. at 2) and expressed doubt that the proposed causal mechanism (metabolic decompensation in a child with an underlying mitochondrial disorder as the result of fever) would apply in cases of mitochondrial dysfunction rather than mitochondrial disease. *Id*. There was no evidence that A.H.M. actually experienced a fever after either her June or September vaccinations, other than a report first made about a decade later by Ms. Miller.

Although petitioners are not required to provide proof of a biological mechanism for their theories, petitioners must demonstrate that the theory or theories proposed are reliable. *See Moberly*, 592 F.3d at 1324. They have failed to do so. Doctor Cave's background, training, and experience are inadequate to validate the theories she cobbled together from the scientific and medical literature she cited. Her reports,

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concession of Prong 1 of *Althen* in every case involving the same vaccine and injury. Rather, the Table makes actual causation irrelevant when an injury meets all the Table criteria. *Quinn v. Sec'y, HHS*, No. 90-0884V, 1992 WL 183197, at *6-7 (Fed. Cl. Spec. Mstr. July 15, 1992) (when the Table injury criteria for encephalopathy are met, the fact that the vaccine in question has never been shown to cause the injury resulting in the encephalopathy is irrelevant). And, even if a case meets the Table injury criteria, respondent may still defend on the basis of a factor unrelated, but only if the "factor unrelated" is not an idiopathic or unknown cause. § 13(a)(2)(A); *Snyder v. Sec'y, HHS*, 553 Fed.Appx. 994, 999-1000 (Fed. Cir. 2014) (presumption of causation in a Table encephalopathy case rebutted by a factor unrelated—evidence of genetic disorder known to cause seizure disorders). Moreover, a concession by respondent in one case does not constitute a concession in another, as science and medicine are not immutable, and evidence filed in one case may not be filed in another case. For example, a concession that the measles vaccine can cause an encephalopathy occurring within five to 15 days of a vaccination is not a concession that it can do so at times shorter or longer. *Shyface,* 165 F.3d at 1351 (quoting the legislative history of the Vaccine Act asserting that a similarity to conditions or time periods in the Table would not be sufficient to demonstrate vaccine causation). Based on the record in this case, I cannot conclude that petitioners have demonstrated the plausibility of their theory, much less established a theory by preponderant evidence.

[116] *See* Pet. Exs. 22, 24, 34, 36. The Poling case report, Pet. Ex. 34, referenced by Dr. Cave in her post-hearing report, was authored by the father of the child described in the report, a fact he failed to disclose when the article was published.

[117] Doctor Cave described the *Poling* vaccine decision as a landmark case in which "Poling's family was awarded funds for ongoing medical care of an autistic child who was **found** to have mitochondrial dysfunction exacerbated by vaccines that left her with autistic behavior and seizures." Pet. Ex. 22 at 1 (emphasis added) (S. Cave, *The History of Vaccinations in the Light of the Autism Epidemic*, ALTERNATIVE THERAPIES 14(6): 54-57 (2008)). *Poling ex rel. Poling v. Sec'y, HHS*, No 02-1466V, 2011 WL 678559 (Fed. Cl. Spec. Mstr. Jan. 28, 2011) (award of fees and costs reflecting that the case was compensated as a Table injury claim).

testimony, and other evidence do not constitute preponderant evidence that vaccines can cause autism or mitochondrial dysfunction, or mitochondrial dysfunction masquerading as ASD.

b. Lack of a Logical Sequence of Cause and Effect.

To satisfy the second prong of the *Althen* test, petitioners must establish a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278. In other words, petitioners must show that the received vaccine actually **did** cause the alleged injury. *Pafford*, 451 F.3d at 1356 (emphasis added). Testimony from a treating physician may assist petitioner in meeting her burden of proof under the second *Althen* prong. *Capizzano*, 440 F.3d at 1326.

In this case, no treating doctor, other than Dr. Holmes, opined that A.H.M.'s injuries were caused by the vaccines she received, and Dr. Holmes' opinion was based on her belief that the thimerosal found in vaccines caused A.H.M.'s injuries, the theory considered and rejected in the OAP test cases.

This case is not a close call. There is nothing in the record, other than the assertions of petitioners and their expert, to establish A.H.M. suffered from encephalopathy, regression, or mitochondrial dysfunction, elements which are crucial to establishing causation. Without a fever and evidence of a mitochondrial decompensation, they cannot establish facts that parallel even their expansive reading of the Poling case as one involving actual causation rather than a Table encephalopathy. The basic requirements of the theories advocated by Dr. Cave are not present in this case. Thus, even if I accepted Dr. Cave's theories as reliable, petitioners have not shown A.H.M.'s vaccinations caused her injuries. Petitioners have failed to establish a logical cause and effect in this case.

c. Lack of a Temporal Relationship.

Finally, when proving that a vaccine was the cause of an injury, petitioner must also show "a proximate temporal relationship between vaccination and injury." *Althen*, 418 F.3d at 1278. Petitioner must prove "that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *De Bazan v. Sec'y, HHS*, 539 F.3d at 1352. Failure to provide a proximate temporal relationship will result in a denial of compensation. *Id.* at 1353.

The medical records and videotape in this case show a child exhibiting the traditional symptoms of autism. The correlation in timing relied upon by petitioners and Dr. Cave is not supported by the record in this case, certainly not regarding A.H.M.'s September 15, 1999 vaccinations. Although the onset of A.H.M.'s autistic behaviors coincide somewhat with the first vaccination alleged as causal (the varicella vaccination), the onset and developmental pattern A.H.M. exhibited is common for children with ASD. Moreover, the only "toxin" petitioners allege is contained in the

varicella vaccination is MSG and the evidence petitioners submitted regarding MSG and ASD was extremely weak. There is no evidence that A.H.M.'s symptoms or behaviors appreciably worsened after the September vaccinations.

VIII. Conclusion.

Petitioners claimed both a Table and non-Table injury but have failed to prove any of their claims. After considering the record as a whole, I find that petitioners have failed to establish entitlement to compensation. The petition is dismissed. The clerk shall enter judgment accordingly.

IT IS SO ORDERED.

s/Denise K. Vowell
Denise K. Vowell
Chief Special Master