OFFICE OF SPECIAL MASTERS

No. 90-3327V

(Filed: May 5, 1998)

* *		
MICHELLE CONNOR, Custodial Parent of CHARLES E. CONNOR, JR., a Minor,	*	
Petitioner,	*	**TO BE PUBLISHED**
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	
* *		

Curtis R. Webb, Twin Falls, ID, for petitioner.

Michael P. Milmoe, Washington, DC, for respondent.

DECISION AND ORDER

MILLMAN, Special Master

On October 1, 1990, Michelle Connor, on behalf of her son, Charles E. Connor (hereinafter "Charles"), filed a petition for compensation under the National Childhood Vaccine Injury Act of 1986[(1)] (hereinafter the "Vaccine Act" or the "Act"). Petitioner has satisfied the requirements for a prima facie case pursuant to 42 U.S.C. § 300aa-11(c) by showing that: (1) she has not previously collected an award or settlement of a civil action for damages arising from the vaccine injury, (2) the MMR vaccination was administered to Charles in the United States and, (3) she has incurred $1,000.00 in unreimbursable medical expenses prior to filing the petition.

Petitioner alleges that Charles suffered an on-Table residual seizure disorder (hereinafter "RSD") as a result of his MMR vaccination. 42 U.S.C. §§300aa-11(c)(1)(C)(I); 14(a)(I)(D). Respondent defends by arguing that a known factor unrelated, an undefined metabolic disorder, caused Charles' condition.

The court held a hearing in this case on December 10 and 30, 1997. Testifying for petitioner were Michelle Connor and Dr. Thomas Schweller. Testifying for respondent were Dr. Samuel J. Horwitz and

Dr. David B. Bettis.

FACTS

Charles was born on November 30, 1984. Med. recs. at Ex. 1, p. 1. He received an MMR vaccination on July 18, 1986, when he was seventeen and one-half months old. Med. recs. at Ex. 7, p. 267.

On July 24, 1986, Charles fell and was taken to Southeast Baptist Hospital Emergency Room (ER) due to a mouth injury and a seizure. Med. recs. at Ex. 7, p. 265.[(2)] The medical records from this visit reflect the following. During the prior week, Charles had fallen, experiencing a Jacksonian-like seizure ten minutes after the fall. Med. recs. at Ex. 7, p. 267. The medical records further reflect that, after the July 24, 1986 incident, Charles experienced a seizure during which he was awake and cried intermittently. Id. However, the histories given by Mr. and Mrs. Connor differ as to Charles' behavior that day. Id. While Mrs. Connor stated that Charles cried a little, got up, and walked around without distress after he fell, Mr. Connor stated that Charles sat down and leaned against the wall, while his arms stiffened, and his lips turned blue. Id. His neurological examination was negative and his CT scan was normal. Med. recs. at Ex. 7, pp. 267-68.

On August 18, 1986, Charles was again taken to Southeast Baptist Hospital ER due to a seizure which involved all extremities[(3)] and right facial jerking. Med. recs. at Ex. 7, p. 272. A history given by Mr. Connor reflects that Charles' extremities became very rigid for about three minutes. Id. During the past three and one-half weeks, Charles had a second type of seizure during which he had right facial twitching, right arm twitching, and staring to the right. Id. This seizure lasted for approximately four to five minutes. Id. He was put on Phenobarbital. Med. recs. at Ex. 7, p. 272. On examination, Charles was alert and smiling and his neurological examination was nonfocal. Med. recs. at Ex. 7, pp. 270, 272.

On August 19, 1986, Charles was taken to Bexar County Hospital. Med. recs. at Ex. 7, p. 302. The history from this visit reflects the following. In the beginning of July 1986, Charles took Amoxicillin for an ear infection. Id. Thereafter, his balance was off, he fell down, and he experienced staring episodes. Id. He acted as if he were drunk. Id. He received his last immunization on July 18, 1986, which was two weeks after his ear infection. Med. recs. at Ex. 7, p. 302. On July 24, 1986, he had a seizure approximately twenty minutes after hitting his head. Id. During the seizure, which lasted three to five minutes, he was stiff and his eyes rolled to the back of his head. Id. He was sleepy after the seizure ended. Id. On July 28, 1986, he had another seizure, which presented with a fixed stare and lasted for approximately two minutes. Med. recs. at Ex. 7, p. 302. On August 18, 1986, he had a third seizure during which he was rigid, trembled on the right side, looked to the right and had cyanosis about his lips. Id.

Charles had an EEG on September 18, 1987 which was normal. Med. recs. at Ex. 7, p. 281. On February 19, 1988, he saw Dr. Richard W. Wilson, a neurologist. Med. recs. at Ex. 9, p. 66. Mrs. Connor could not think of any specific episode or incident which preceded his first seizure. Id. There is a history of seizures on his father's side. Id. Dr. Wilson opined that neurological disease caused Charles' seizures, bilateral corticospinal tract dysfunction, and delayed development of speech and fine coordination. Med. recs. at Ex. 9, p. 65. He further noted that Charles has a lifelong, nonprogressive, static problem in the general category of cerebral palsy. Med. recs. at Ex. 9, p. 65.

On May 3, 1988, Charles saw Dr. David Bettis, a pediatric neurologist, who stated that Charles' progressive gait abnormality raised the possibility of a degenerative central nervous system disease, such as metachromatic leukodystrophy. Med. recs. Ex. 10, p. 182.

From May 3 to May 14, 1988, Charles was in St. Luke's Regional Medical Center. Med. recs. at Ex. 10,

p. 175. The medical record from this visit reflects that his paternal first cousin has temporal lobe epilepsy. Id. The records further note that Charles has a chronic history of seizure disorder and developmental delay. Id. He has a progressive neurological disease with changing mental status and decreased motor movement. Id.

On June 26, 1988, Charles saw Dr. A. Wakelee Bledsoe, a pediatrician. Med. recs. at Ex. 9, p. 63. At the time of this visit, he had an elevated CPK (268,000), an elevated LDH and SGOT as well as a lack of hyperbilirubinemia. Id. He had a severe episode of myoglobinuria. and painful, decreased movements which indicated a muscle disease. Id.

In a September 26, 1988 medical record, Dr. Robert J. Burnett noted that Charles was hospitalized in April 1988 for lethargy and abnormal CPK elevation. Med. recs. at Ex. 9, p. 61. This was due to a strange muscular disorder which appeared to be an unusual form of muscular dystrophy. Id. Two maternal uncles had muscular dystrophy deaths, suggesting a familial disorder. Med. recs. at Ex. 9, p. 62. The record further notes that Charles' urine carnitine was high, which supports a diagnosis of carnitine palmitoyl transferase[(4)] deficiency. Id. However, Charles did extremely well on carnitine and thyroxine, having daily developmental improvement. Id. In addition, his seizure activity had stopped, and his EEG was normal. Id. Dr. Burnett further noted that "[i]t is tempting to speculate that a metabolic disorder rather than anticonvulsant treatment underlies Charles' deterioration of development. With cessation of seizure activity and normal EEG, one questions the presence of neurologic disease." Med. recs. at Ex. 9, p. 62. However, Dr. Burnett also stated that he has hyperactive reflexes and Achilles contractures. Id.

On January 15, 1993, Charles again saw Dr. Bledsoe, who stated that he has a rare metabolic disorder resulting in seizures and near comatose episodes. Med. recs. at Ex. 11, p. 27. This disorder has been termed a fatty acid defect. Med. recs. at Ex. 11, p. 43.

Charles returned to Dr. Bledsoe on May 18, 1993. Med. recs. at Ex. 11, p. 26. During this visit, Dr. Bledsoe noted that Charles appeared to have a carnitine deficiency since birth. Id. Without Carnitor, his life would be in jeopardy from acute encephalopathy and muscle weakness. Id.

TESTIMONY

Michelle Connor testified first for petitioner. She has a deceased first cousin with muscular dystrophy and her ex-husband has a nephew with temporal lobe epilepsy. Tr. at 26, 28. She stated that Charles was generally healthy prior to receiving his MMR vaccine on July 18, 1986. Tr. at 8. He was developing normally and had just started walking. Tr. at 8-9. He had taken his first step at tens months of age and began speaking at one year of age. Id. At four to five months of age, he sat up without assistance. Tr. at 9.

Although Charles was cranky after receiving his MMR, he was not sick or feverish. Tr. at 11. Mrs. Connor testified that, on July 24, 1986, Charles fell off a table. Tr. at 12. After the fall, he started choking and turned blue. Id. His eyes rolled back, his arms and legs stiffened, and his right side shook. Tr. at 12-13. The Connors took Charles to the emergency room where he had a CT scan which was normal. Tr. at 16.

Mrs. Connor did not remember Charles having a second seizure on July 28, 1986. Id. She did, however, remember his third seizure on August 18, 1986. Id. During this seizure, Charles was afebrile. Id. He turned blue, his eyes rolled back, and he jerked more on his right side. Tr. at 16. She took him to the hospital and tests were run. Tr. at 17. Although he was already on Phenobarbital, it was again prescribed. Id.

In May 1988, Charles was going into a coma unbeknownst to Mrs. Connor. Tr. at 27. He went to sleep and she could not wake him. Id. He was in a coma for one week. Tr. at 28. He had a similar episode in July 1996. Tr. at 28-29.

Mrs. Connor testified that Charles changed after his July 24, 1986 seizure.[(5)] Tr. at 18. After the seizures began, he lost ground. Tr. at 19-20. He drooled, did not talk, and lost energy.[(6)] Id. Within a month of his first seizure, he had problems walking. Tr. at 24. He never resumed talking as he did prior to the vaccination. Id. Physically, he did not grow normally. Tr. at 21-22. He looks and acts as if he were six or seven years of age, rather than thirteen years of age. Tr. at 22. He gets along with preschoolers better than with those his own chronological age. Tr. at 22-23. He was not toilet trained until five years of age. Tr. at 23.

Currently, Charles is in special education school and he receives occupational therapy one to two times a week. Tr. at 21, 23. He talks but he cannot speak in sentences. Tr. at 30-31. Strangers cannot understand him. Tr. at 31. Thus, he receives speech therapy two times per week. Id. Charles' balance poor and he is constantly on his toes. Tr. at 44. As a result, he cannot run well. Tr. at 32. He had surgery to correct the tightening of his heel cords three years ago, but it did not work and more surgery is required. Id.

Charles still seizes today. Tr. at 19. He responds well to Tegretol; however, it controlled his seizures better during the early part of his treatment. Id. His current seizures involve crying, glazed eyes, bilateral shaking, and jerking for a few seconds. Tr. at 29-30. They seem to be getting longer lately. Tr. at 30. He knows when they are coming. Tr. at 29-30. He generally has three to four seizures per month. Id.

Charles was put on Carnitor, which helps break down fatty acids. Tr. at 34. His dosage has increased since it was initially prescribed. Id. When his dosage of carnitine was temporarily reduced, he seemed more sluggish and his speech more slurred. Tr. at 42, 44. The doctors have never told Mrs. Connor which metabolic disorder Charles has. Tr. at 52. He was hypothyroid five to six years ago and was put on medication which resolved the condition. Tr. at 56-57. A couple of years ago, he was hypothyroid again and put on Synthroid. Tr. at 57. Currently, his thyroid condition is normal. Id.

EXPERTS

Dr. Thomas Schweller testified next for petitioner. He is a neurologist, board-certified in pediatrics and neurology with special competency in child neurology and electromyography. Tr. at 63. While fifty percent of his time involves medical-legal matters, such as workmen's compensation and personal injury cases, forty percent of his time is devoted to social security assessments. Id. The remaining ten percent of his time involves treatment of seizure disorders in care facilities. Id.

Dr. Schweller opined that Charles' seizure disorder was caused by the MMR.[(7)] Tr. at 78. Dr. Schweller opined that Charles had no symptoms of either a neurological disorder or seizures prior to receiving the MMR vaccine. Tr. at 65. Rather, within six days after receiving MMR, Charles suffered his first seizure on July 24, 1986. Tr. at 65, 66. He had his second seizure on July 28, 1986, and his third seizure on August 18, 1986. Tr. at 65. All of these seizures were afebrile, presenting with symptoms such as clonic shaking of the upper extremities, eye rolling, stiffness, and staring episodes. Tr. at 65-66. Dr. Schweller further stated that Charles' seizure disorder resulted in additional symptoms such as regression of speech, mental retardation, and developmental delay. Tr. at 67. He stated that the seizure disorder, developmental delay and mental retardation are features of neurological difficulties. Id. Thus, based solely on a temporal association, Dr. Schweller opined that Charles' speech and psychomotor problems and mental retardation were sequelae of his seizures. Tr. at 67-68. However, later in his testimony, Dr. Schweller stated that although seizures manifest what is occurring in the brain, they, themselves, do not

injure the brain.[8] Tr. at 126-27. As a rule, a one- to three-minute seizure itself will not cause developmental problems. Tr. at 127. Thus, Dr. Schweller concluded that seizures are not the direct cause of Charles' mental retardation or speech delay. Id. Rather, the seizures are a reflection of what was occurring in his brain. Tr. at 127-28. Dr. Schweller further stated that there is no evidence that Charles' brain disorder commenced prior to receiving the MMR because he was attaining milestones. Tr. at 128. The onset of his brain injury coincides with the MMR.[9] Id.

Dr. Schweller did not think that Charles' fall on July 24, 1986 caused his first seizure because he did not have any altered consciousness. Tr. at 90-91. In addition, the seizures persisted although the fall was insignificant. Id. Even if Charles had fallen twice prior to July 24, 1986, Dr. Schweller's testimony would not differ because children fall all the time. Tr. at 91-92. Moreover, a loss of balance would be expected if Charles has an inborn error of metabolism because it affects muscles or the brain system. Tr. at 93.

Charles was born with a metabolic disorder, i.e., an inborn error of metabolism.[10] Tr. at 129. His May 1988 hospitalization reflects that he has a metabolic disturbance of the muscles called rhabdomyolysis. Tr. at 69. Rhabdomyolysis is a nonspecific condition where muscle proteins break down and enter the urine, impairing kidney function. Id. This disorder can be life-threatening. Id.

Metabolic disturbances can be caused by a variety of conditions including carnitine palmitoyl transferase deficiency. Tr. at 71. Carnitine palmitoyl transferase deficiency occurs when the muscles are unable to metabolize fatty products. Id. Dr. Schweller stated that it is a common, rare disorder which involves symptoms similar to those exhibited by Charles while hospitalized.[11] Id. He further explained that this condition involves a cellular defect in the mitochondria of the muscles. Id. These mitochondrial disorders are commonly caused by an inborn error of metabolism due to a genetic defect in some structural part of the body which changes the way that certain cells function. Tr. at 72. A wide spectrum of diseases cause inborn errors of metabolism. Tr. at 73.

Dr. Schweller believes that the source of Charles' metabolic disorder appears to be in his muscles. Tr. at 75. This is why doctors continually want to biopsy Charles' muscles and why he is on carnitine.[12] Id. Furthermore, Charles' tight heel cords reflect a muscle disease rather than a central nervous system problem. Id. Although he does not have an upper motor neuron component, he does have signs of spasticity. Tr. at 75-76.

Although the majority of metabolic disorder involving muscles do not also involve a brain abnormality, it is possible for an inborn error of metabolism to involve both brain chemistry and muscles. Tr. at 85-87. MELAS (mitochondrial encephalopathy, lactic acidosis, and stroke) and carnitine deficiency are examples of this.[13] Id. Carnitine deficiency involves both muscle weakness and some mental confusion which is almost encephalopathic. Tr. at 87. Dr. Schweller is uncertain whether or not Charles has a carnitine deficiency. Tr. at 87-88. Although he does not know exactly why Charles takes carnitine, he stated that it may help to stabilize the metabolism of his muscles. Tr. at 130.

Dr. Schweller does not believe that Charles' metabolic disorder is related to his seizures for two reasons. Tr. at 77. First, the exact inborn error of metabolism is unknown. Tr. at 77-78. Second, his metabolic disorder has manifested itself with muscular problems rather than brain problems. Id. Metabolic disorders of the central nervous system result in progressive deterioration and death within a relatively short period of time. Tr. at 97. Charles, however, does not have signs of progressive mental deterioration. Tr. at 76. In fact, his mental condition has stabilized. Tr. at 76. Individuals who have metabolic disorders which affect their central nervous system are in a much more serious condition than

Charles. Tr. at 97. His static condition clearly argues against his having a metabolic disease of the central nervous system.[(14)] Id.

Dr. Schweller further testified that in order to know the future course of a metabolic condition, it is necessary to know the particular metabolic deficiency from which an individual suffers. Tr. at 82. For instance, it is necessary to know whether the condition is either systemic, i.e., involving multiple organs, or predominantly located in one organ.[(15)] Id. A great number of inborn errors of metabolism have seizures as a component, particularly a lot of white matter diseases, e.g., ceroid lipofuscinosis, leukodystrophies, myoclonic epilepsy, and amino acidurias. Id. It is very common for inborn errors of metabolism to have seizure disorders as a component if they involve brain chemistry. Tr. at 84-85. However, if they involve the liver, kidney, and muscles, they are much less likely to have seizure disorders as a component. Tr. at 85.

While Dr. Schweller believed that Charles had post-MMR encephalopathy because he had seizures, he did not believe that Charles had encephalitis because he did not have alteration of consciousness. Tr. at 95. Dr. Schweller did not believe that Charles had a metabolic encephalopathy, although he may have had it at the time of his rhabdomyolysis. Tr. at 109. Metabolic encephalopathy is an altered consciousness due to a toxic substance in the blood stream. Tr. at 108.

Dr. Samuel J. Horwitz testified for respondent. He is a professor of pediatrics and neurology, and is board-certified in pediatrics and neurology with special competency in child neurology. Tr. at 142-43. He has treated about twelve children similar to Charles. Tr. at 143. Although he had a large clinical practice until June 1997, he now teaches exclusively. Id. He practiced for thirty years, seeing thousands of children. Tr. at 144. Several hundred of his patients had metabolic disorders. Id.

In Dr. Horwitz's opinion, Charles has an underlying metabolic disorder which caused his seizure on July 24, 1986. Tr. at 152-53. Mitochondria are vital to maintaining the metabolism of all tissues. Tr. at 154. The brain has a resistance to seizures; however, metabolic disorders affect chemistry, which, in turn, lowers the seizure threshold. Tr. at 155. Thus, seizures generally accompany these types of disorders. Id. Charles has epilepsy and doctors know the cause of epilepsy in only one-quarter of the cases. Tr. at 155-56. It is speculative to presume that either Charles' fall or the vaccine caused his seizure.[(16)] Tr. at 153.

Dr. Horwitz testified that a metabolic disorder is a chemical disorder of the body. Tr. at 144. The consequences of such disorders range from acute disorders to chronic disability to death. Id. In Dr. Horwitz's opinion, Charles suffers from a metabolic condition of a mitochondrial type. Tr. at 188. Seizures are considered to be a common manifestation of this disorder and Charles' condition, in its entirety, was caused by this disorder.[(17)] Tr. at 188, 205. Dr. Horwitz further noted that Charles' metabolic disorder has permanent consequences. Tr. at 145. He will probably have acute flare-ups with residual worsening, even devastation. Id.

Metabolic disorders can involve mutiple organs, may have acute exacerbations, and affect the muscular and nervous systems as well as other organs (thyroid, kidney, liver, and eye). Tr. at 164-65. Charles' disorder is not limited to his muscles. Tr. at 145. Rather, his metabolic condition involves multiple organs, including the brain, thyroid, and kidney. Id. The involvement of his brain is evidenced by: (1) episodes of ataxia and incoordination which were not related to muscle weakness or anticonvulsants and, (2) stroke-like episode with hemiparesis and hemianopsia.[(18)] Tr. at 146. His kidney involvement is evidenced by an episode of elevated protein and blood in his urine which was not associated with either rhabdomyolysis or myoglobinuria. Tr. at 145-47.

Dr. Horwitz opined that Charles' metabolic condition can be further classified as a mitochondrial disorder which affects the muscles in the artery in the brain. Tr. at 151, 188. Carnitine is very involved in mitochondrial metabolism. Tr. at 179. It is not unusual for a mitochondrial disorder to affect the central nervous system as more than half do. Tr. at 205. Charles' laboratory results are predictive of a central nervous system disease. Tr. at 197. He has had abnormal EEGs, abnormal thyroid tests, abnormal carnitine, rhabdomyolysis and myoglobinuria which are all related to central nervous system disease.(19) Id. In addition, Charles' mental retardation was more apparent in the second and third year of life, which is consistent with mitochondrial disorder. Tr. at 217. In Dr. Horwitz's opinion, all of Charles' symptoms, including his seizures, are due to his disorder.(20) Tr. at 188, 205. There are no reports that vaccination exacerbates this disorder. Tr. at 172.

Dr. Horwitz noted that mitochondrial disorders can be classified according to a spectrum of severity. Tr. at 180. Dr. Horwitz considers Charles to have been born with a moderately severe mitochondrial disorder because he is very impaired. Tr. at 180. While the severity of the disorder itself does not change, its symptomatology will progress over time because of the growth and change in the patient's metabolism. Tr. at 181. It is difficult to predict the severity of a disorder based on the number of organs afflicted because one never knows which organ could be targeted next. Tr. at 211. For instance, Charles had an episode where his heart was enlarged. Id. Although his heart was normal, it could be affected in the future. Id.

Charles' condition is not static. Tr. at 201. Rather, he has a progressive metabolic disorder. Tr. at 177. He was normal or near normal prior to July 24, 1986; however, he is abnormal now. Id. His condition progressed by acute flare-ups, increased tone, and increased reflexes, indicating that the nervous tissue in Charles' brain and spinal cord are involved. Tr. at 177-78. His heel cords are constantly tightening because the process is on-going. Id.

Dr. Horwitz found Charles' family history of muscle disease to be significant. Tr. at 183-84. However, even if Charles and his relatives had the same disorder, there is a huge spectrum of severity depending on the extent of the genetic material involved. Tr. at 184. With regard to Charles' thyroid condition, Dr. Horwitz noted that it dissipated as did his many other symptoms. Tr. at 184-85. Something insulted Charles' thyroid, causing it to malfunction; however, once the insult was treated, his thyroid condition disappeared. Tr. at 185.

Charles had neither a post-MMR encephalopathy nor overt manifestations of encephalopathy prior to July 1986. Tr. at 185, 187. However, Dr. Horwitz believes that Charles did subsequently have an acute encephalopathy when he was extremely ill with rhabdomyolysis and kidney dysfunction. Tr. at 186. Because his loss of consciousness was not attributable to either of these illnesses, Dr. Horwitz concluded that he had an acute encephalopathy. Id. Currently, however, he has a chronic encephalopathy. Tr. at 187. Although it is impossible to determine when his chronic encephalopathy began, Dr. Horwitz stated that he exhibited symptoms of it prior to his episode of rhabdomyolysis. Id.

Dr. David Bettis testified next for respondent. He has intermittently been Charles' treating pediatric neurologist since 1988.(21) Tr. at 245. He has consulted with Charles' pediatrician, Dr. Bledsoe, as well as Dr. Neil Buist. Id. Although Charles suffers from numerous abnormalities for which an exact cause has not been diagnosed, he opined that Charles has an undefined metabolic disorder.(22) Tr. at 246. This disorder manifested itself by significant motor and speech delay, seizures, mental retardation, increased tone and spasticity. Id. Although he is unable to arrive at a unified diagnosis for these problems, he is suspicious that a metabolic disorder is the more likely cause. Tr. at 246-47. Furthermore, Dr. Bettis believes that Charles could have a mitochondrial disease because such conditions can involve symptoms such as seizure disorder, developmental delay, and stroke.(23) Tr. at 248-49.

Dr. Bettis believes that Charles was born with this disorder. Tr. at 248. Charles did not experience any events either during delivery or within his first year of life to which his problems can be attributed. Tr. at 247-48. At one year of age, Charles was a toe walker, which is abnormal. Tr. at 247. This abnormality continued in the form of increased tone and reflexes in his legs and positive clonus. Id. He had seizure onset at eighteen months. Id. Dr. Bettis has not seen a clear cut worsening trend in Charles' disorder. Tr. at 248. However, Charles has continued to make some progress despite significant delay. Id. Thus, Dr. Bettis would classify Charles' disorder as being static rather clearly progressive or degenerative. Id.

Charles' metabolic disorder has affected his brain, resulting in a seizure disorder, borderline microcephaly, and developmental delay. Tr. at 249. Dr. Bettis believes that seizures are part of Charles' overall constellation of symptoms. Tr. at 250. All of his present symptoms are a consequence of his brain abnormality. Id.

Dr. Bettis is highly skeptical that MMR caused Charles' problems. Tr. at 250-51. First, he has never seen medical literature linking Charles' symptoms to a vaccine injury. Tr. at 251. Charles' symptomatology is not the type of clinical presentation related to a vaccine injury. Id. Secondly, Charles exhibited neurological symptoms, i.e., toe walking, prior to his vaccination. Tr. at 252. Therefore, his seizure onset was a coincidence to the MMR. Id. Thirdly, none of Charles' other doctors considered the vaccine to be the cause of his condition.[(24)] Tr. at 254-55. He noted that Dr. Buist discussed the possibility of a metabolic disorder; however, Dr. Buist made no mention of a vaccine-related cause. Tr. at 255. Thus, Dr. Bettis concluded that Charles has an undefined metabolic disorder which caused all of his symptoms. Id.

DISCUSSION

The Vaccine Act affords petitioner two theories of recovery, thereby allowing causation to be proven by showing that either: (1) a Table-injury occurred or, (2) the vaccine was the cause-in-fact of the injury. With reference to the former theory, Section 14(a) of the Act contains a Vaccine Injury Table. If the injuries in this Table occur within the statutorily defined time period, petitioner has proven that a "Table-injury" has occurred, therefore, creating a rebuttable presumption of causation.[(25)] To rebut this presumption, respondent must provide affirmative evidence demonstrating that a known factor unrelated was the cause-in-fact of the petitioner's condition.[(26)] Based on this statutory framework, the court now turns to petitioner's specific claims.

1. On-Table RSD

Petitioner herein alleges that Charles suffered an on-Table RSD. RSD is deemed a Table-injury under the Act and is defined in 42 U.S.C. § 300aa-14(b)(2):

A petitioner may be considered to have suffered

a residual seizure disorder if the petitioner did

not suffer a seizure or convulsion unaccompanied

by fever or accompanied by a fever of less than

102 degrees Fahrenheit before the first seizure

or convulsion after the administration of the

vaccine involved and if--

(A) in the case of a measles, mumps, or rubella

vaccine or any combination of such vaccines, the

first seizure or convulsion occurred within 15

days after administration of the vaccine and 2 or

more seizures or convulsions occurred within 1

year after the administration of the vaccine

which were unaccompanied by fever or accompanied

by a fever of less than 102 degrees Fahrenheit....

Petitioner herein has met the aforementioned criteria. There is no question that he had a seizure within fifteen days of his MMR vaccination, which was then followed by two afebrile seizures within one year of vaccination. The medical records clearly reflect that Charles suffered afebrile seizures on July 24, 1986, July 28, 1986, and August 18, 1986. Thus, petitioner has prevailed in presenting a prima facie case of on-Table RSD.

2. Sequelae

Once petitioner has proven that a Table injury occurred, petitioner must still prove that his current condition is a sequela of the vaccine injury. Petitioner has the burden of proving sequela by a causation-in-fact standard. Song v. Secretary, HHS, 31 Fed. Cl. 61 (Fed. Cir. 1994); Hossack v. Secretary, HHS, 32 Fed. Cl. 769, 774 (Fed. Cl. 1995). In order to meet this burden "a preponderance of the evidence must show that some logical, direct causal link exists between the presumed Table injury and the alleged sequela." Hossack, supra, at 776.

Petitioner herein alleges that the developmental and speech delay, mental retardation and seizures from which he suffers are sequelae of his Table injury. Although the court heard expert testimony on this issue, the undersigned is unable to find that petitioner has met his burden of proving all these sequelae. Dr. Schweller, petitioner's expert, originally testified that Charles' speech delay and mental retardation were caused by his seizures based on temporal association.(27) However, he later stated that Charles' seizure disorder did not cause his mental retardation or speech delay.(28) Dr. Schweller further testified that seizures do not lead to injury in the brain except for anoxia.(29) While Dr. Schweller initially linked Charles' condition to his seizures based on temporal association, his later testimony traced it to metabolic events in his brain. Although the seizures reflected what was occurring in his brain, he said they did not cause mental retardation and developmental delay.

Even if Dr. Schweller had not changed his mind and instead ascribed Charles' mental retardation and other delays to his inborn error of metabolism, petitioner would have still failed to meet her burden because temporal association is insufficient to prove causation. Grant v. Secretary, HHS, 956 F.2d 1144

(Fed. Cir. 1992). Where the Table injury is unrelated to the child's ultimate damage, petitioner may not recover damages. Jordan v. Secretary, HHS, No. 91-113V, 1998 WL 106131 (Fed. Cl. Spec. Mstr. Feb. 23, 1998). Since petitioner's expert has not linked causally any of Charles' current condition to his RSD except for the seizures themselves, petitioner may recover solely for the seizures from which he still suffers.

3. Factor unrelated

After petitioner has satisfied her burden on seizures as sequelae, the burden shifts to respondent to prove that Charles' seizure disorder was caused by a known factor unrelated. Among the listed known factors unrelated in the statute are metabolic disturbances. 42 U.S.C. § 300aa-13(a)(2)(B).

Although each witness testified that Charles has a metabolic disturbance, the court must determine whether or not respondent has proven that Charles' undefined metabolic disorder is the cause in fact of his seizures.(30)

To determine whether respondent has met this burden, the court turned to the medical literature respondent submitted as well as the testimony. Respondent's Exhibit A discusses mitochondrial disorders, stating that they can affect any tissue in the body.(31) R. Ex. A, p. 1201. The text further states that "the characteristic changes described in muscle are not commonly seen in the central nervous system (CNS) and neuropathologic changes in mitochondrial encephalomyopathies tend to be nonspecific." R. Ex. A, p. 1203.

There are patterns in which mitochondrial disorders affect the CNS. Id. The first pattern is comprised of microcephaly and ventricular dilatation, which are sometimes associated with agenesis of the corpus callosum, as well as other maladies seen in infants with severe congenital lactic acidosis. Id. The court is unaware that Charles has microcephaly, agenesis of the corpus callosum, or lactic acidosis.

A second pattern consists of bilateral symmetrical subcortical lesions in the basal ganglia, and other brain structures. Id. There is no evidence that Charles has any structural defects except enlarged ventricles. Although his MRI reflected delayed myelination, this is not the same as lesions.(32)

A third pattern consists of multifocal encephalomalacia, characterizing strokelike lesions of the MELAS (mitochondrial encephalomyopathy, lactic acidosis, and strokelike episodes) syndrome. R. Ex. A, p. 1203. Charles had one strokelike episode but does not have lactic acidosis. Finally, a fourth pattern is spongy encephalopathy which Charles does not have. Id.

A table within the text lists the symptomatology of CNS tissue which is afflicted with mitochondrial diseases. R. Ex. A, p. 1214 (Table 52-4). Under only three diseases, MERRF,(33) MELAS,(34) and MILS,(35) are seizures a symptom. Id. MERRF also includes ataxia, myoclonus, weakness, short stature, sensorineural hearing loss, lactic acidosis, ragged-red fibers on muscle biopsy, and maternal inheritance. Id. MELAS includes ataxia, psychomotor regression, hemiparesis/hemianopsia, cortical blindness, migrainelike headaches, dystonia, weakness, short stature, sensorineural hearing loss, ragged-red fibers on muscle biopsy, and maternal inheritance. Id. Finally, MILS further includes psychomotor retardation, dystonia, weakness, and maternal inheritance. R. Ex. A, p. 1214 (Table 52-4).

While Charles clearly exhibits some of these features, many are absent from his clinical picture. On muscle biopsy, there was no notation of ragged-red fibers. P. Ex. 29A. Moreover, none of the witnesses can define his metabolic disease.

The above table also describes three other mitochondrial diseases which do not include seizures. However, these diseases include other symptoms that Charles exhibits, e.g., weakness, psychomotor regression. R. Ex. A, p. 1214 (Table 52-4). A second table includes a flowchart for evaluating patients with suspected mitochondrial encephalomyopathy. R. Ex. A, p. 1213 (Table 52-3). With regard to clinical features, the table reads "[c]ast a wide net...," noting that any combination of symptoms or symptom in isolation is suspicious of mitochondrial disease.[(36)] Id. In addition, Dr. Bettis testified that there are several hundreds of mitochondrial disorders.[(37)]

The text further states that mitochondrial DNA (mtDNA) "is ubiquitous and all tissues in the body depend, to a greater or small degree, on oxidative metabolism, mtDNA mutations can affect every organ, and mtDNA-related disorders are typically multisystemic." R. Ex. A, p. 1213. It continues by noting that "[m]ost patients with mtDNA mutations have various 'nonsyndromic' combinations of the symptoms and signs listed in the table [52-4]." Id. at 1214.

Respondent's Exhibit B describes three major categories of mitochondrial diseases: defects of fatty acid oxidation, defects of pyruvate metabolism, and defects of the respiratory chain.[(38)] R. Ex. B, pp. 1342-45. Defects of fatty acid oxidation are associated with symptoms of altered consciousness. Id. Defects of pyruvate metabolism are not associated with neurologic problems. Id. at 1344. Finally, defects of the respiratory chain are associated with brain and muscle dysfunction. Id.

Clearly, from the testimony of Drs. Horwitz and Bettis and the medical literature respondent submitted, the range of mitochondrial diseases is immense. Drs. Horwitz and Bettis opined that all of Charles' symptoms are due to an undefined mitochondrial defect. Moreover, although Dr. Horwitz did not know what metabolic disorder Charles has, Dr. Horwitz concluded that all of Charles' symptoms must come from this undefined disorder.

While such a conclusion may be tenable medically, it is invalid legally. Although respondent does not have the burden of showing which metabolic disease Charles has because the Vaccine Act permits a metabolic disturbance itself to be sufficient to constitute a known factor unrelated, respondent does have the burden of showing what the known factor unrelated causes. Only by making this connection can respondent defeat the statutory presumption that Charles' MMR vaccination caused his seizure disorder.

While respondent has succeeded in proving that Charles has a metabolic disturbance, it has failed to prove that it caused his seizures. Arguing that the disease must have caused the seizures because Charles has seizures is insufficient proof. Such logic is mere tautology. The medical testimony reflects that there are hundreds of metabolic disorders having varying symptoms, some of which Charles has and others of which he does not have. Respondent cannot work backward from the symptom to an unknown, preexisting condition to prove causation, particularly where petitioner has the statutory presumption of causation from the vaccination.[(39)] Respondent has failed to prove that a known factor unrelated, rather than MMR, was the cause in fact of his seizure disorder.[(40)]

Accordingly, Charles may recover damages for his seizure disorder, which the court assumes will consist of the cost of anticonvulsants and visits to a neurologist. The parties may work out any other items of compensation directly relating to Charles' seizures. However, Charles may not recover for his other damages, e.g., mental retardation, developmental delay, because petitioner has failed to prove that these are sequelae of his Table injury. Petitioner's own expert attributed these other signs and symptoms to Charles' inborn error of metabolism and testified that his seizures did not cause him any harm, identical to the testimony of respondent's expert.

CONCLUSION

Petitioner is entitled to a program award. The court hopes that the parties will be able to settle the damages portion of this case and will schedule a status conference in aid of determining damages or encouraging settlement. Considering the limited nature of the damages in this case, the court assumes neither party will need a life care plan.

IT IS SO ORDERED.

DATE: _____ _____

Laura D. Millman

Special Master

1. The National Vaccine Injury Compensation Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, 42 U.S.C.A. §300aa-1 et seq. (West 1991), as amended by Title II of the Health Information, Health Promotion, and Vaccine Injury Compensation Amendments of November 26, 1991 (105 Stat. 1102). For convenience, further references will be to the relevant subsection of 42 U.S.C.A. § 300aa.

2. For the purposes of clarity, please note that Exhibits 1-7 can be found in Appendix I while Exhibits 8-10 can be found in Appendix II.

3. Although the seizure involved all extremities, it was stronger on the right side. Med. recs. at Ex. 7, p. 272.

4. In the medical records, this is referred to as "palmitoryltransferare" deficiency.

5. Since Charles could neither walk nor crawl after taking Phenobarbital, the doctors thought initially that he was overdosed; however, it was later discovered that he was allergic to Phenobarbital and Dilantin. Tr. at 18-19.

6. His speech changed within a couple of weeks after the onset of the seizures. Tr. at 19-20.

7. In addition, Dr. Schweller believes that the MMR caused the delayed myelination which appeared on Charles' MRI. Tr. at 78.

8. This is, however, with the exception of anoxia. Tr. at 127.

9. Dr. Schweller could not draw any conclusions as to what was actually occurring in the brain. Tr. at 128. However, when asked whether the MMR was the cause of the underlying brain disorder, he explained causation in terms of being a "logical sequence of events." Id.

10. Dr. Schweller has not treated any patients who are exactly like Charles. Id. He does treat children with metabolic disorders, including rhabdomyolysis. Tr. at 107.

11. However, Dr. Schweller later testified that he is unsure whether Charles has a mitochondrial disorder. Tr. at 88. While Charles has a problem affecting his white matter, he does not have a primary white matter disease. Tr. at 89. Someone with a typical white matter error of metabolism would be in a wheelchair. Id.

12. Carnitine helps stabilize muscles. Tr. at 75.

13. Dr. Horwitz, whose testimony is discussed infra, stated that Charles has a MELAS-like syndrome but not MELAS because he has not been diagnosed with lactic acidosis, although it may be undetected. Tr. at 198-99.

14. Although Dr. Schweller does not believe that Charles has a progressive disorder because he is not losing ability and making some progress, Charles has not caught up with his peers. Tr. at 131, 134.

15. Dr. Schweller believes that Charles has solely an abnormality in his muscles in disagreement with respondent's expert, Dr. Horwitz that Charles has a multi-organ malfunction. Tr. at 105-06. Dr. Schweller has not seen any involvement of Charles' brain. Tr. at 106.

16. Dr. Horwitz found no medical significance in Charles' two falls prior to his MMR. Tr. at 152

17. In support of his opinion, Dr. Horwitz relied on respondent's exhibits. See Berg, B.O., Principles of Child Neurology, ch. 52, "The Mitochondrial Disorders," by S. DiMauro, et al., 1201-32 (R. Ex. A); Swaiman, K.F., Pediatric Neurology - Principles and Practice, 2d ed., ch. 69, "Mitochondrial Diseases," by D.C. DeVivo and S. DiMauro, 1335-56 (R. Ex. B).

Dr. Horwitz stated that these texts discuss mitochondrial disorders, reflecting that the central nervous system is the first tissue they affect. Tr. at 188-89. In addition, seizures are the first symptom of these disorders. Tr. at 188-89. In fact, seizures are a major component of one mitochondrial disorder called MERRF. Tr. at 189. These texts are discussed at length infra.

18. Hemianopsia is a condition in which an individual loses vision on one side, indicating that the opposite side of the brain is affected. Tr. at 148. Dr. Horwitz opined that this condition was unrelated to Charles' seizures. Tr. at 149. Rather, there was loss of blood supply to one side of his brain which generally occurs in mitochondrial disorders. Id.

19. While the EEG and carnitine deficiency reflect a central nervous disease, they do not reflect a mitochondrial problem. Tr. at 197-98.

20. Dr. Horwitz noted Charles' May 4, 1988 MRI showed enlarged ventricles and abnormal myelination, i.e., abnormal white matter, which, in turn, reflect that something is wrong with the structure of the brain. Tr. at 157. He attributes this abnormality to the metabolic disorder because the white matter is produced by cells which contain mitochondria and, therefore, have metabolic processes. Tr. at 158. A change in the white matter is then seen because there is either a chemical disturbance of those cells or

because the myelin is always active. Tr. at 158-59. Charles' seizures cannot be *per se* attributed to the abnormal myelination, however, because seizures are caused by the gray matter in the brain. Tr. at 159.

21. The first time Charles visited Dr. Bettis was in May 1988 when had an elevated liver enzyme (SGOT). Tr. at 257. Dr. Bettis stated that this condition is symptomatic of some metabolic disorders; however, he also had Dilantin-induced liver dysfunction at that time, which qualifies as another possible cause. Id.

22. Dr. Bettis testified that metabolic disorders make up a broad category in medicine. Tr. at 271-72. They can be exacerbated by medication, illness with a high fever, too much exercise, certain kinds of foods, exposure to heat or cold, and high or low electrolytes. Id. In many circumstances, the exacerbating circumstances are temporary. Id.

23. Dr. Bettis based this belief on the fact that Charles had one-sided weakness in childhood.

24. Although not argued by petitioner, Dr. Bettis also noted that there was no significant aggravation because there was no history of a drastic change in Charles' condition after the onset of his seizures. Tr. at 252-53.

25. The court need not discuss the causation-in-fact standard as petitioner in this case is proceeding strictly under a Table-injury theory.

26. 42 U.S.C § 13(a)(1)(B).

27. Tr. at 67-68.

28. Dr. Schweller stated that " . . . I am not in this case saying that the seizures are causing, are the direct cause of the mental retardation or speech delay. They are more a reflection of what's happening in the brain." Tr. at 127.

29. Dr. Schweller testified that " . . . [A]s a rule, a one- to three-minute seizure [such as Charles had] of itself is not causing problems with development." Tr. at 127.

30. Because the undersigned found that Charles' seizures were the only condition caused by his RSD, respondent need prove causation only with respect to this symptom.

31. Berg, B.O., Principles of Child Neurology, ch. 52, "The Mitochondrial Disorders," by S. DiMauro, et al.,1201-32.

32. However, the text further mentions that the brain can microscopically show demyelination in mitochondrial disorders. R. Ex. A, p. 1203.

33. MERRF consists of myoclonic epilepsy and ragged-red fibers. Harrison's Principles of Internal Medicine, p. 2116 (12th ed. 1991). It presents between the first and fifth decades with generalized seizures, myoclonus, dementia, hearing loss, and ataxia. Id.

34. MELAS is slowly progressive and is characterized by mitochondrial myopathy, encephalopathy, lactic acidosis, strokelike episodes including alternating hemiparesis, hemianopsia or cortical blindness, and focal or generalized seizures. Id.

35. MILS is Leigh's syndrome, which is an autosomal recessive subacute necrotizing encephalomyelopathy. Id. at 437.

36. The text is referring to the symptoms discussed in Table 52-4.

37. Tr. at 263.

38. Swaiman, K.F., Pediatric Neurology - Principles and Practice, 2d ed., ch. 69, "Mitochondrial Diseases," by D.C. DeVivo and S. DiMauro, 1335-56.

39. This case is similar to Spence v. Secretary, HHS, No. 95-57V (April 13, 1998), where petitioner had trisomy 5p which is a rare genetic disorder. In that case, petitioner prevailed because respondent could not prove what symptoms this very rare condition would have produced.

40. Whitecotton v. Secretary, HHS, 81 F.3d 1099 (Fed. Cir. 1996).