In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 13-253V

Filed: October 26, 2017

WILLIAM RODRIGUEZ *and* BRENDA RODRIGUEZ *as the Parents and Natural Guardians of* C.R., *a Minor,*	*	PUBLISHED
Petitioners,	*	Diphtheria-Tetanus-acellular Pertussis ("DTaP"); Measles Mumps Rubella ("MMR"), Polio and Varicella Vaccinations; Onset of Juvenile Dermatomyositis ("JDM"); Plausible Medical Theory; Entitlement to Compensation
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Mark T. Sadaka, Esq., Mark T. Sadaka, LLC, Englewood, NJ, for petitioners.
Linda S. Renzi, Esq., U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

Roth, Special Master:

On April 9, 2013, William and Brenda Rodriguez ("petitioners") filed a petition on behalf of their minor child, C.R., pursuant to the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10 *et seq.*[2] ("Vaccine Act" or "the Program"). Petitioners allege that C.R. developed juvenile dermatomyositis ("JDM") as a result of the Diphtheria-Tetanus-acellular Pertussis ("DTaP"), Measles-Mumps-Rubella ("MMR"), Polio, and Varicella vaccinations he received on August 30, 2011. *See* Petition, ECF No. 1.

[1] This decision will be posted on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). As provided in 42 U.S.C § 300aa-12(d)(4)(B), however, the parties may object to the decision's inclusion of certain kinds of confidential information. To do so, each party may, within 14 days, request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, this decision will be available to the public in its present form. *Id.*

[2] National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (1986). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2012).

Upon review of the evidence submitted in this case, the undersigned finds that petitioners are entitled to compensation under the Vaccine Act. Petitioners have carried their burden of showing by preponderant evidence that C.R.'s JDM was triggered by the vaccinations he received on August 30, 2011, and respondent has failed to rebut that showing with sufficient evidence of an alternative cause. The case shall accordingly proceed to damages.

I. Medical and Procedural Background

A. C.R.'s Medical History

The following medical history is taken from C.R.'s relevant medical records as well as the testimony of petitioners Brenda and William Rodriguez, C.R.'s biological parents. Both petitioners testified at the hearing, and although each was sequestered while the other testified, their testimony was generally consistent with each other and with the contemporaneous medical records.

1. C.R.'s Health Prior to the Vaccines

C.R. was born on August 27, 2007. He is the oldest of three children and lives on a small farm in the rural, mountainous area of Northern Georgia, where petitioners raise chickens, ducks, and goats for personal use. C.R. was a generally healthy child with no chronic health conditions. According to petitioners, C.R. liked to play with his toys, hike with his dad, and play outside with his brother. Pet. Ex. 2 at 17-19; Pet. Ex. 4 at 43; Tr. 185, 269.

On August 29, 2008, after receiving Hepatitis A, Varivax and MMR vaccines, C.R was diagnosed with an upper respiratory infection and viral exanthema. Pet. Ex. 2 at 18. At a well-child visit on August 26, 2010, C.R. was noted to be a three year old who played well with others with no concerns. Labs from the prior year were noted to be normal. Pet. Ex. 1 at 19-22, 40.

On August 15, 2011, C.R. began preschool at Rabun County Head Start. Pet. Ex. 81 at 1; Tr. 187. C.R. loved going to school—after his first week, his teacher reported that he was energetic and would not nap during nap time. Pet. Ex. 81 at 2; Tr. 209. On August 30, 2011, three days after his fourth birthday, C.R received the vaccinations at issue in this case—DTaP, MMR, polio and varicella—at Clayton Medical Associates ("Clayton").[3] Pet. Ex. 3 at 1-2; Pet. Ex. 81 at 2.

2. C.R.'s Health Following the Vaccines

Upon arriving home after receiving the vaccinations, C.R. took a nap and awoke complaining that his head hurt. He had a slight fever, vomited, was given Tylenol, and sent back to bed. Pet. Ex. 81 at 2; Tr. 275. According to petitioners, C.R. vomited several times during the night and had a headache and high fever that did not break for about 48 hours. Pet. Ex. 81 at 2; Tr. 193, 276. Mrs. Rodriguez recalled changing his bed several times that night because of his

[3] C.R. received his vaccinations at Toccoa Clinic Medical Associates, an affiliate of Clayton, but he was treated following the vaccinations at the Clayton location. *See* Pet. Ex. 1 at 1; Pet. Ex. 3 at 1. For ease of reference, both clinics will be referred to as "Clayton."

being sick. Tr. 194. C.R. slept in the next morning when he was usually up with the sun. Tr. 195.

According to petitioners, within the first week after his vaccinations, C.R. developed a rash on his knuckles, which started as a small patch. Pet. Ex. 81 at 2; Tr. 269. It was a scaly, purple rash, inflamed on the proximal knuckles. Tr. 196-97. He also had a patch on his chest, which petitioners thought was heat rash, so they did not bring him to the doctor immediately. Tr. 197, 269. His face was also a pinkish-red color. Over the next week, the rash spread to C.R.'s elbows and knees. Pet. Ex. 81 at 2; Tr. 199, 269. Around this time, petitioners received a note from Rabun County Head Start advising that many of the children had been diagnosed with hand-foot-and-mouth disease ("HFMD").[4] Petitioners were therefore not initially concerned when they saw the rash on C.R.; they assumed it was HFMD. Pet. Ex. 81 at 2; Tr. 198.

C.R. continued not feeling well and, on September 12, 2011, he was seen at Clayton for vomiting, sore throat, fever, and headache. Pet. Ex. 1 at 11; Pet. Ex. 81 at 2. His rash was spreading more to the distal knuckles, elbows, and knees. Tr. 199-200. His sore throat and vomiting had started the night before. Tr. 201-02; 278. He also had a rash on his chest that "popped up" with the "flu like symptoms." According to Mrs. Rodriguez, the rash on C.R.'s chest was completely different than the one on his joints. Tr. 206-07. The medical records also noted that C.R. had enlarged tonsils and lymphadenopathy, as well as a dry "patch of erythema." Pet. Ex. 1 at 11-12. A strep test was negative, but labs were ordered, including a CBC and Lyme Titer. C.R. was diagnosed with fever, acute pharyngitis, vomiting, and rash. *Id*. at 12, 37. Labs were negative for Lyme and typhus fever, and there was no isolated strep, but his CBC showed elevated levels for his white blood cell count,[5] sedimentation rate,[6] gran count,[7] lymphocytes,[8] and C-reactive protein.[9] *Id*. at 35-39. C.R. was prescribed Augmentin for 10 days and Zofran for vomiting. *Id*. at 11.

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[4] Hand-foot-and-mouth disease is a typically mild exanthematous eruption caused by coxsackievirus A16 and enterovirus 71. It is most often seen in preschool children and is characterized by a sore throat, blisters on the tongue, and a rash on the palms of the hands and soles of the feet. *See Dorland's Illustrated Medical Dictionary* 535 (Saunders eds., 32nd ed. 2012) ("*Dorland's*").

[5] A high white blood cell count can indicate infection, inflammation, trauma, tissue damage, medication, allergy, acute or chronic leukemia, or bone disease. *Dorland's* 1028.

[6] A sedimentation rate test measures the rate at which red blood cells (erythrocytes) settle in a test tube over one hour. The more red cells that fall to the bottom, the higher the sedimentation rate. *See Dorland's* 1594. When inflammation is present in the body, certain proteins cause red blood cells to stick together and fall more quickly than normal. These proteins are produced by the liver and the immune system under many different abnormal conditions, such as infection, cancer, or autoimmune disease. *Dorland's* 936.

[7] High gran blood count can indicate a variety of things from cancer, kidney failure, and infection. *Dorland's* 425.

[8] Lymphocytes are essentially a type of white blood cell that protects the body against disease and infection. *See Dorland's* 1084. Whenever the overall health of the body is penetrated by an invading organism, the lymphocytes are called on to attack the invader. An autoimmune disorder will cause the lymphocytes to misidentify some of their own tissue as being foreign substances and attack them. *See id.* at 181.

[9] C-reactive protein measures general levels of inflammation in the body. High levels of C-reactive protein are caused by infections and many long-term diseases. But a C-reactive protein test cannot show the cause or location of the inflammation; other tests are needed to do so. *Dorland's* 1891.

Mrs. Rodriguez noted that until this point in time, C.R. had never been sick or had rashes, vomiting, or a fever. Tr. 207; *see generally* Pet. Ex. 2. She also testified that the rash on C.R.'s knuckles, elbows, and knees was dry, cracked hardened skin that was raised and inflamed. Tr. 279. At this visit, petitioners were told that C.R. probably had HFMD, which was going around the community. Tr. 208. According to Mrs. Rodriguez, she did not, as a mother, believe that C.R. had HFMD. Tr. 229-31. Both petitioners testified that C.R.'s rash persisted and did not look like the other children that had HFMD. The rash was not on C.R.'s palms and bottoms of his feet like the other children; it started on his knuckles and extended down. Tr. 224, 262, 281. But they had no reason to question the medical providers. Pet. Ex. 81 at 2; Tr. 224, 262.

By the middle of September, C.R. had become sleepy throughout the day and had less energy. Pet. Ex. 81 at 2; Tr. 209, 278. While at school, he had started napping but still wanted to sleep when he got home. He continued to complain of headaches and body aches. Pet. Ex. 81 at 2. The school had informed Mrs. Rodriguez that they noticed that he was tired, a little off-balance, and emotional. Tr. 218. He had bonded with his teacher, and she told Mrs. Rodriguez that he was not acting like himself; he was whiny and not his usual energetic self. Tr. 231-32. Mrs. Rodriguez noted that C.R. continued to have the rash throughout September, while the rash on all the other children at school had cleared up. Pet. Ex. 81 at 2. Although it was fall, C.R. complained that the sun was too hot and that his rash hurt. Even when he was in the car, with tinted windows and the air conditioning on, he complained that the sun hurt his rash. *Id.*; Tr. 219. He seemed a little more sensitive too. Tr. 210. Petitioners also noted that, although the family ordinarily did a lot of hiking on the trails around their home, they had to stop hiking in September because C.R. just could not do it anymore. Tr. 216-17, 270-71. According to Mr. Rodriguez, about a month after the vaccinations, C.R. started to complain when he took him hiking. He would say he was tired and that his legs hurt, and Mr. Rodriguez would have to carry him home. Tr. 270-71.

Mrs. Rodriguez was shown photographs of C.R. taken at various times from September through December of 2011, which she described as showing a sick child, with redness on his cheeks, rash on his chest and knuckles, and puffy eyes. Tr. 210-11, 213, 217, 238-39, 241-45; Pet. Exs. 74, 75. His hands were shiny and puffy. Tr. 219.

On October 4, 2011, Mrs. Rodriguez brought C.R to Clayton with a high fever and persistent rash on his hands and face. Pet. Ex. 1 at 9. The rash on his hands was the same (it remained the same since its onset), but the night before he had developed a rash on his face. Tr. 221. His knuckles were pinkish, dry, and inflamed. Tr. 234. Dawn Morgan, a certified pediatric nurse practitioner, noted that C.R. had red, dry "patches over his knuckles and abdomen" that he was scratching because it was itching.[10] Pet. Ex. 1 at 10. Ms. Morgan said that it was HFMD, but Mrs. Rodriguez responded that all of the other children's rashes had cleared up. Pet. Ex. 81

[10] According to Mrs. Rodriguez, Ms. Morgan examined C.R. at each visit. Tr. 187, 200-04.

at 2. C.R. was diagnosed with Fifth disease[11] and atopic dermatitis.[12] Claritin was recommended to relieve the itching, and Ms. Morgan indicated that the Fifth disease would resolve itself. C.R. was also referred to a dermatologist, who saw him that same day. Pet. Ex. 1 at 9-10; Pet. Ex. 81 at 2.

The dermatologist, Dr. Russell Burke, noted that C.R. was red and had a rash on his trunk, face and legs for 5 weeks, as well as scratches and sores on his legs. He also noted that C.R. had a fever a few weeks ago and that he had HFMD. According to Mrs. Rodriguez, Dr. Burke held up C.R's hands, looked at his knuckles, lifted his shirt, looked at his torso, and said he had eczema. Tr. 228; Pet. Ex. 81 at 2. She also noted that C.R. had never been diagnosed with eczema before. Tr. 208. Dr. Burke's report states that C.R. had "[s]lapped cheek erythema," eczema on his chest, back, abdomen, and right and left extremities. And like Ms. Morgan, Dr. Burke gave a diagnosis of atopic dermatitis and erythema infectiosum (i.e., Fifth disease). He prescribed a topical steroid cream. Pet. Ex. 1 at 24. Petitioners indicated that they had no reason to question Dr. Burke's assessment. They further noted that Dr. Burke was the only dermatologist in town and that everyone used him. Tr. 262-63.

Mrs. Rodriguez was asked at the hearing if the Claritin or antibiotics noted in C.R.'s medical chart helped with his rash. She said that it only took away the rash on his chest, but the knuckles and the knees never changed. Tr. 235.

Following the appointment with Dr. Burke, petitioners researched eczema and HFMD rashes on the internet and in books they had at home. They noted that C.R's rash did not look like those rashes; his was pinkish-purple in color and was located on his joints and not his torso. C.R.'s rash looked exactly as it did in September and was not going away. Pet. Ex. 81 at 3. According to Mr. Rodriguez, C.R. never had any kind of rash that looked like that before; their research showed that C.R.'s rash looked more like a rheumatic issue. Tr. 277, 286-88. Mr. Rodriguez stated that it took about a month after the vaccinations for him to realize that this was not going away and that it was not HFMD. Tr. 288.

Mrs. Rodriguez stated that, by October of 2011, C.R. had started walking more slowly while dragging his feet, struggling to walk normally. He stopped running around, and he became clumsy. He could no longer give his usual big "Koda bear hugs." He also became noticeably weaker—he could not get up from the floor, button his pants, or dress himself to get ready for school, and he struggled to turn on and hold his electric toothbrush. Pet. Ex. 81 at 3.

On October 11, 2011, Mrs. Rodriguez brought C.R. back to the pediatrician. He had a worsening cough, continued runny nose, a red eye, and enlarged red tonsils. Pet. Ex. 1 at 7-8. Mrs. Rodriguez stated that he still had the rash on his hands and elbow. He also had bug bites on

[11] Fifth disease, also called erythema infectiosum, is a moderately contagious, often benign epidemic disease seen mainly in children and caused by the B19 virus. The primary characteristic is a rash of abrupt onset that begins as redness of the cheeks, which appear to have been slapped; later is a maculopapular rash on the trunk and limbs; when this fades, there may be central clearing that leaves a lacelike pattern. *See Dorland's* 533, 643.

[12] Atopic dermatitis is a common, chronic type of dermatitis, thought to be hereditary, sometimes associated with other allergic reactions such as allergic rhinitis, hay fever, or asthma. Severe pruritus leads to scratching, rubbing, and typical signs of eczema. *Dorland's* 494.

his legs. She believes Ms. Morgan told her at that visit that C.R. must have come in contact with HFMD again. Tr. 239-41. Ms. Morgan diagnosed him with sinusitis and bronchitis, and she prescribed Cefzil and Rondec. Pet. Ex. 1 at 7-8. The cough went away. Tr. 241.

Near the end of October, according to Mrs. Rodriguez, C.R. complained that his body hurt and that he felt really weak. C.R. dressed as a Ninja for Halloween, and the family went to the local drive-in movie theater for a trick-or-treating event. People would decorate their cars, and the children would walk down the rows of cars trick or treating. C.R. walked down one row of cars and asked to go home; he was too tired to stay. Pet. Ex. 81 at 3; Tr. 237-38. By December of 2011, C.R. was missing school due to fatigue and pain. He struggled with hand movements, and his handwriting became sloppy. Pet. Ex. 81 at 3; Tr. 243. His teacher sent home a note that he was taking long naps. Tr. 246-47. Mrs. Rodriguez recalled that on Christmas morning, C.R. was holding the rail trying to come down the stairs. He was complaining and begged them to slow down. He was sluggish and could not get up off the floor. He struggled to play. Tr. 245-46.

On January 9, 2012, C.R. returned to Clayton with continued rash on his hands, face and back. He was noted to have a low grade fever for the past month, difficulty turning on his toothbrush, and increased fatigue. Pet. Ex. 1 at 4-5.[13] Ms. Morgan asked C.R. to squeeze her hand, and he could not do it. Pet. Ex. 81 at 3. Ms. Morgan also documented noticeable rash over C.R.'s hands and a red, raised, irregular-shaped rash over his face and forehead. He continued to have dry red rash over his knuckles and abdomen. Pet. Ex. 1 at 4-5, 10; Tr. 249 He was diagnosed with fever, rash, and muscle weakness, and prescribed Orapred (a steroid) and Zyrtec. Pet. Ex. 1 at 6. According to Mrs. Rodriguez, blood work was ordered, and this was when they actually believed that it was *not* HFMD given the other symptoms. Pet. Ex. 81 at 3; Tr. 250-51. C.R. still had the pinkish rash on his face, but it had extended over his nose and become raised. Tr. 251-52. Mrs. Rodriguez testified that this rash was not the same as the rash on his knuckles and knees; that rash was rougher and smaller on his joints. Tr. 253. The rash on his hands extended from the knuckle to his wrist. Tr. 254-55.

On January 16, 2012, C.R. returned to Clayton. He had a positive ANA, a standard symptom of systemic autoimmune disorders.[14] He was negative for Lyme disease. Pet. Ex. 1 at 27-32. The assessment on that day was rash/dermatitis, and muscular weakness of the hands. *Id.* at 1-3. C.R. was referred to a rheumatologist. Pet. Ex. 81 at 4.

[13] The record for this visit notes that C.R. had been seen by a dermatologist who diagnosed viral rash. Pet. Ex. 1 at 4. When Mrs. Rodriguez was asked about this notation, she stated that she had not been told he had a viral rash; she was told that he had eczema. She was also asked about a reference to a flea infestation, which she testified occurred during the summer of 2011, before C.R. received his vaccinations. Tr. 248-49; *see* Pet. Ex. 1 at 13 (noting C.R. to have "dry, insect bites" on his date of vaccination).

[14] An antinuclear antibody ("ANA") test measures the amount and pattern of antibodies in the blood that work against the body. Normally, the body's immune system attacks and destroys foreign substances, but in autoimmune diseases, the immune system attacks and destroys the body's normal tissues. It does so by producing antibodies that attach to the body's own cells as though they were foreign substances, often causing them to be damaged or destroyed. *See Dorland's* 101.

On January 23, 2012, C.R. was examined by Dr. Kelly Rouster-Stevens, a rheumatologist at Emory Children's Center in Atlanta. Mr. Rodriguez advised Dr. Rouster-Stevens that C.R. had never been sick until his vaccinations, and that he had been constantly sick since receiving his vaccinations. Tr. 276. Dr. Rouster-Stevens summarized C.R.'s medical history as a four year old who was previously healthy but developed "rash, fatigue and worsening weakness since 09/2011," which was "around the time [C.R.] had received several immunizations." Pet. Ex. 4 at 41. C.R. "developed cough rhinorrhea, headache and low grade fevers" the day after he received his vaccinations and "has 'constantly been sick'" ever since. *Id*. She also noted that C.R.'s rash had been worsening, his activity level had declined, and he complained that the sun felt hot on his skin. She noted that he had a five-day course of prednisone without improvement; "he continues with marked rash and weakness." *Id*. Dr. Rouster-Stevens noted C.R.'s family medical history was also noted: his parents (petitioners) were healthy 25- and 29-year-old individuals, his paternal grandparents had diabetes, a paternal uncle had psoriasis,[15] and a paternal great-aunt had multiple sclerosis. *Id*. at 42.

Upon examination, Dr. Rouster-Stevens noted dilated blood vessels; mild violaceous discoloration;[16] Gottron's papules[17] covering his knuckles, elbows, and knees; and dilated nail bed capillaries. She also noted C.R.'s decreased strength and fatigue, his inability to do a sit-up, and his positive ANA. Dr. Rouster-Stevens concluded that C.R.'s "history and physical exam findings are most concerning for juvenile dermatomyositis," and that "[a]lthough lupus is in the differential, it is much less likely in a child of his age and the Gottron's papules noted on his exam are more suggestive of JDM." *Id*. at 43. Dr. Rouster-Stevens ordered more testing and prescribed 15 milligrams of prednisone daily. *Id*. at 43-44.

At a follow-up examination with Dr. Rouster-Stevens on February 29, 2012, C.R.'s rash had improved with the steroids but he was not back to baseline. C.R had stopped going to school at this point due to his compromised immune system, constant fatigue, and inability to keep up with the other children. His hamstrings were tight, and he was slightly Cushingoid.[18] Dr. Rouster-Stevens confirmed that C.R. had JDM and discussed Methotrexate. Since petitioners felt that Methotrexate had too many side effects, they opted to continue treating with the same dosage of daily prednisone. *Id*. at 36-40; Pet. Ex. 81 at 4.

MRIs performed of C.R.'s pelvis and thighs on March 28, 2012, revealed diffuse muscle signal heterogeneity, consistent with JDM. Pet. Ex. 76 at 36.

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[15] Mr. Rodriguez advised Dr. Rouster-Stevens that his brother had become paralyzed after receiving vaccinations and had to relearn how to walk. According to Mr. Rodriguez, his brother has continued to suffer from eczema, psoriasis, and immune issues, and the doctors never had a diagnosis for what his brother suffered after his vaccinations. Tr. 272-74.

[16] Violaceous is used to describe discoloration of the skin; usually as having a violet color. *Dorland's* 2057.

[17] Gottron's papules is a sign in dermatology, consisting of discolored lichenoid flat-topped papules over the knuckles. *Dorland's* 1373.

[18] Cushingoid resembles the features, symptoms, and signs associated with Cushing syndrome—a complex of symptoms caused by hyperadrenocorticism due either to a neoplasm of the adrenal cortex or adenohypophysis, or to excessive intake of glucocorticoids. Symptoms may include adiposity of the face, neck, and trunk, as well as muscular wasting and weakness. *Dorland's* 1827.

C.R. remained on prednisone through the summer and returned to Dr. Rouster-Stevens on August 6, 2012. He continued to have generalized weakness, facial rash which flared from heat, and redness around his eyes. The record indicated that he had improved overall. He still waddled but was less stiff. Upon examination he was noted to have diffuse erythematous rash on his eyelids, cheeks, elbows, and knees with low muscle tone, and significantly decreased muscle strength. He was unable to lift his head off the bed, and refused to sit on the floor. The diagnosis on that date was JDM, with weakness despite six months of prednisone. Intravenous immunoglobulin ("IVIG") was suggested. Pet. Ex. 4 at 31-35.

C.R. was presented for physical therapy evaluation on September 27, 2012. He was noted to have impaired joint mobility, motor function, muscle performance and range of motion associated with connective tissue dysfunction. Physical therapy three times a week was ordered. Pet. Ex. 5 at 2-3.

At his follow-up with Dr. Rouster-Stevens on October 26, 2012, C.R. was noted to be making gradual progress but still had erythema covering his entire body and decreased strength. IVIG was discussed along with reducing the steroid dosage. Pet. Ex. 4 at 27-30. On December 6, 2012, C.R. was reassessed by the physical therapist, who noted that he appeared to be stronger. He could walk further, and he was hiking and running better. *Id*. at 22-24.

On January 11, 2013, C.R. returned to Dr. Rouster-Stevens. He had a marked rash, a high-pitched voice, dilated vessels on his hard palate, and diminished strength. Although petitioners were hesitant, they agreed to proceed with IVIG treatments. *Id*. at 14-15, 17. IVIG treatments were started in February of 2013 and continued through July. Pet. Ex. 72 at 23. Following his treatment, C.R. was noted to be off medications with minimal skin findings. Pet. Ex. 73 at 19. By the summer of 2013, C.R. was stronger and some of his energy returned, although he continued to be thin and did not gain weight easily. Pet. Ex. 81 at 5.

At his five year check up on August 8, 2013, C.R. was noted to have bumps on his scalp. It was also noted that he was taking a multivitamin, fish oil, Vitamin D, curcumin, turmeric and folic acid. He had undergone large doses of intravenous and oral steroids to reduce his autoimmune process, which had been discontinued two months prior. Pet. Ex. 75 at 7-15.

On September 29, 2014, C.R. was examined by Dr. Rouster-Stevens. He was then seven years old with JDM in remission. He had been off therapy for over a year and there was no rash or weakness. Mrs. Rodriguez was concerned that C.R. was not gaining weight as he should. She also felt he was not as strong as he should be. Upon examination there was no sign of weakness. There was no warmth or swelling of the joints; he had normal range of motion in all four extremities. His labs were unremarkable. Pet. Ex. 79 at 54.

C.R. had a relapse in August 2015. Pet. Ex. 83 at 2-5; Pet. Ex. 84 at 334-35. According to Mr. Rodriguez, the rash appeared on his knuckles, and it was the same rash he developed on his knuckles shortly after his vaccinations. Tr. 285. Petitioners testified that the rash did not get as bad this time since they knew what it was and what to do. Tr. 257-58. He was started on IVIG twice weekly in September 2015, which continued through February 18, 2016. Following

six months of IVIG, he was continued on prednisone until July 2016, when he was tapered off. *See generally* Pet. Ex. 84. At C.R.'s July 22, 2016, visit, it was noted that he had Vitamin D deficiency and was on a supplement. He had been noted to have decreased TSH with normal free T4 in the past and was being monitored. *Id.* at 19. The rash never totally went away. Tr. 283.

Petitioners testified that Dr. Rouster-Stevens has discussed the future with them—explaining the potential for recurrences and advising that, due to C.R.'s immune issues, he needs to be careful around other people, stay out of the sun, and keep his stress levels low. Tr. 256-57, 283-85. According to Mrs. Rodriguez, C.R. has some emotional problems now; he does not think very highly of himself. He gets extra help in school because he is behind. He has trouble remembering things. He's very thin and cannot gain weight even though he eats well. Tr. 258-59. According to Mr. Rodriguez, he cannot keep up with the other children. He cannot play sports, although he wants to, because he is clumsy. His muscles are weak. He is able to hike now up to two or three miles. Tr. 281-83.

Mrs. Rodriguez also testified that, following C.R.'s diagnosis of JDM, she told Ms. Morgan what the diagnosis was, and Ms. Morgan asked her to spell it for her so she could look it up, stating that she had never heard of it before. Tr. 259.

B. Experts

Petitioners' expert is Dr. Eric M. Gershwin. *See* Pet. Ex. 7 (expert report). Dr. Gershwin graduated from Stanford University and is board certified in internal medicine and allergy and clinical immunology. He completed his residency in internal medicine at Tufts-New England Medical Center. He is currently a Professor of Medicine in the Division of Rheumatology and Allergy and Clinical Immunology at the University of California, Davis. Pet. Ex. 8.

Respondent's expert is Dr. Carlos D. Rose. *See* Resp. Ex. A (expert report). Dr. Rose graduated from University of Buenos Aires School of Medicine and is board certified in pediatric rheumatology. He completed a residency in internal medicine followed by a two year fellowship in rheumatology. He is currently in private practice in pediatric rheumatology. Resp. Ex. B.

C. Procedural History

Petitioners filed their petition on April 9, 2013, and filed medical records and a statement of completion the following week. ECF Nos. 6-7. This case was originally assigned to Chief Special Master Dorsey.[19] Respondent filed a Rule 4 Report on July 3, 2013, stating that, based "[on] the existing record, petitioners have failed to provide preponderant evidence in support of their claim," and that respondent therefore "recommends that compensation be denied and the case be dismissed." Resp. Rule 4 Report, ECF No. 10.

On February 20, 2014, petitioners filed an expert report from Dr. Gershwin. ECF No. 19. Respondent filed an expert report from Dr. Rose on June 3, 2014. ECF No. 23. Petitioners filed

[19] This case was reassigned to me on October 23, 2015. *See* Notice of Reassignment, ECF No. 63.

additional medical records, a statement of completion, and a supplemental report from Dr. Gershwin on October 17, 2014. ECF Nos. 38-40. Respondent filed a supplemental report from Dr. Rose on November 6, 2014. ECF No. 41.

An entitlement hearing was held on May 23-24, 2016. After the hearing, the parties were encouraged to discuss settlement. A status conference was held on December 2, 2016, during which the parties indicated that settlement of this matter did not appear feasible. The parties were then ordered to file post-hearing briefs. Order, issued Dec. 2, 2016, ECF No. 102. Petitioners' post-hearing brief was filed on March 10, 2017, and respondent's post-hearing brief was filed on April 10, 2017. ECF Nos. 107-8. This matter is now ripe for decision.

II. Legal Framework

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. § 11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface v. Sec'y of Health & Human Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999).[20] Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing § 13(a)(1)(B)).

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. § 11(c)(2). Medical records created contemporaneously with the events they describe are presumed to be accurate and "complete" such that they present all relevant information on a patient's health problems. *Cucuras v. Sec'y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). In making contemporaneous reports, "accuracy has an extra premium" given that the "proper treatment hang[s] in the balance." *Id.* Contemporaneous medical records that are clear, consistent, and complete warrant substantial weight "as trustworthy evidence." *Id*. Indeed, "where later testimony conflicts with earlier contemporaneous documents, courts generally give the contemporaneous documentation more weight." *Campbell ex rel. Campbell v. Sec'y of Health & Human Servs.*, 69 Fed. Cl. 775, 779 (2006); *see United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1948). But petitioners can support their claim with oral testimony if it is credible and consistent with the medical records. *See, e.g., Stevenson ex rel. Stevenson v. Sec'y of Health*

[20] The Vaccine Act also requires petitioners to show by preponderant evidence that the "residual effects or complications" of the alleged vaccine-related injury lasted for more than six months. § 11(c)(1)(D)(i). It is undisputed that this six-month requirement is satisfied in this case.

& Human Servs., No. 90-2127V, 1994 WL 808592, at *7 (Fed. Cl. Spec. Mstr. June 27, 1994) (crediting the testimony of a fact witness whose "memory was sound" and "recollections were consistent with the other factual evidence"). In short, "the record as a whole" must be considered. § 13(a).

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993), requires that courts determine the reliability of an expert opinion before it may be considered as evidence. "In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establishes a standard of evidentiary reliability." *Id*. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly ex rel. Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010). The *Daubert* factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Human Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Human Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). And nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder ex rel. Snyder v. Sec'y of Health & Human Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

Finally, although this decision discusses some but not all of the literature in detail, the undersigned reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Human Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

III. Analysis

Because petitioners do not allege an injury listed on the Vaccine Injury Table, their claim is classified as "off-Table." As noted above, for petitioners to prevail on an "off-Table" claim, they must show by preponderant evidence that C.R.'s injury resulted from by the vaccinations at

issue. *Capizzano*, 440 F.3d at 1320. Doing so shifts the burden to respondent to show that the injury was caused by factors unrelated to the vaccinations. *Deribeaux*, 717 F.3d at 1367.

A. Petitioners' Burden to Show Causation

To prove causation, petitioners must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Human Servs.*, 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that petitioners show by preponderant evidence that a vaccination C.R. received caused his injury "by providing: (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.* at 1278. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Human Servs.*, 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast *per se* scientific or medical rules." *Knudsen v. Sec'y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

1. Reputable Medical Theory

The first *Althen* prong requires petitioners to provide a "reputable medical theory" demonstrating that the vaccines received *can* cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). To satisfy this prong, petitioners' "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu ex rel. Andreu v. Sec'y of Health & Human Servs.*, 569 F.3d 1367, 1379 (Fed. Cir. 2009) (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id.* at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde v. Sec'y of Health & Human Servs.*, 746 F.3d 1334, 1341 (Fed. Cir. 2014). In this case, petitioners have offered a reputable medical theory of causation that the vaccinations at issue can trigger JDM.

JDM is an autoimmune disease that requires both a genetic predisposition and environmental insult. Resp. Ex. A at 6. It is part of a group of diseases characterized by inflammation of muscles and skin, which leads to necrosis and dysfunction of the muscles. Tr. 20; Pet. Ex. 7 at 5. The disease can first manifest as entirely skin-based and later lead to muscle weakness. The incidence of JDM is rare—approximately 1.2 to 17 cases per million. The average length of time from onset of symptoms to diagnosis is approximately four to five-and-one-half months. Tr. 20-21.

According to both experts, the etiology of JDM is somewhat elusive; it involves some interplay among genetic, hormonal, environmental, and immunologic factors. Pet. Ex. 7 at 6;

Resp. Ex. A at 11. Data does suggest, however, that innate and adaptive immunity play a fundamental role in the onset and pathology of the disease. *Id.* at 7. Thus, a brief overview of the immune system and its relationship to JDM is instructive for assessing the theory of causation.

a. The Immune System and JDM

In general, a body's response to foreign invaders, antigens or pathogens, is controlled by the immune system. The immune system can be divided into two branches: the innate immune system and the adaptive immune system.

The innate immune system provides the first bodily defense to an immune challenge, and it can be activated within hours of the attack. Components of the innate immune system include macrophages,[21] cytokines,[22] and natural killer cells.[23] Tr. 15-16; Pet. Ex. 7 at 1. Certain innate immune cells—plasmacytoid dendritic cells—produce various cytokines, especially Type I interferons, to control inflammation and regulate or mediate an immune attack. Tr. 16, 46-47; Pet. Ex. 7 at 1. Type I interferons are critical for inflammation, and are controlled by over 400 genes which are produced in order to help protect against unknown and varied infections. Tr. 47. Dr. Gershwin cited *Griffin*[24] (Pet. Ex. 80), a study that speaks extensively about Type I interferons and the role of the innate immune system in childhood myositis. Tr. 60; Pet. Ex. 80 at 488.

The adaptive immune system, unlike the innate immune system, can take several days or even months to develop a protective response, but it does so in a more advanced way: by recognizing specific antigens. The adaptive immune system contains both B cells and T cells. *See supra* note 8. B cells make the antibodies that fight against the foreign substances.[25] T cells can be divided into two groups: cytotoxic T cells and helper T cells. Cytotoxic T cells kill cells infected with viruses. Helper T cells (of which there are many types) provide "help" to the various cells: Th1 cells help cytotoxic T cells kill infected cells; Th2 cells help B cells make antibodies; and Th17 cells help cells at the mucosal level respond to infections by generating interleukin-17, a type of cytokine.[26]

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[21] Macrophages are cells that kill ingested microorganisms; they also digest and present antigens to T and B cells. *Dorland's* 103.

[22] Cytokines are proteins released by cells upon contact with an antigen as a means of generating an immune response. *Dorland's* 466.

[23] Natural killer cells are a type of white blood cell that contains enzymes that can kill tumor cells or cells infected with a virus. They do this by secreting cytokines that stimulate and guide the response of other agents of innate immunity and the cells of the adaptive immune system. *Dorland's* 319.

[24] Thomas A. Griffin & Ann M. Reed, *Pathogenesis of Myositis in Children*, 19 Current Opinion in Rheumatology 487 (2007).

[25] Antibodies are immunoglobulin molecules that have a specific amino acid sequence by virtue of which it interacts only with the antigen that induced its synthesis in cells of the lymphoid series (especially plasma cells), or antigen closely related to it. Immunoglobulins are glycoproteins that function as antibodies. *Dorland's* 100, 919.

[26] *See supra* note 22.

Dr. Gershwin explained that when a vaccine is administered, there can be an overproduction, or dysregulation, of certain cytokines, which happens immediately. Tr. 51, 57. A particularly pro-inflammatory interferon signature could likely cause several changes within cells, including in the endoplasmic reticulum.[27] Tr. 52. Dr. Gershwin explained that the interferon signature and endoplasm reticulum are like two cogs in a wheel; they should turn together. But in the genetically at-risk person, the wheel involving immunity gets stuck and keeps emitting Type I interferons. Tr. 53-54. Dr. Gershwin cited *Nagaraju*[28] (Pet. Ex. 34), a study showing that an upregulation was associated with inflammation and the pathways involved in the endoplasmic reticulum, explaining the unfolding of proteins that become activated in the muscle tissue of patients with myositis. The study described this process as a "self-sustaining loop." Dr. Gershwin clarified that once it starts, it is perpetuated by the adaptive immune system because cytotoxic T cells will continue to be cytotoxic, which will lead to unrelenting inflammation. Tr. 56-58; Pet. Ex. 34 at 10. Thus, JDM is a response by both the innate and adaptive immune systems. Tr. 54-55.

According to Dr. Gershwin, analysis of publicly-available data shows that 107 of the 226 most upregulated genes in JDM muscle tissue are interferon-inducible. Pet. Ex. 7 at 7. The most extensive study—*Greenberg*[29]—compared 40 adults with other forms of Idiopathic Inflammatory Myopathies ("IIM"). Of the 14 most upregulated genes in dermatomyositis tissue, 12 were Type I interferon-inducible. "This contrasts with only three of the 21 most upregulated genes in other IIMs vs. normal being Type I interferon-inducible." *Id*. Dr. Gershwin noted that though many Type I interferon-inducible genes were upregulated to some degree in idiopathic inflammatory myopathies, none was elevated to the extent that was found in dermatomyositis tissue. The study also demonstrated Type I interferon action by detecting expression of a Type I interferon-inducible antiviral protein in affected dermatomyositis tissue. That protein was expressed in myofibers, capillaries and other blood vessels in dermatomyositis tissues as well as affected dermatomyositis skin, "which suggests a common pathogenic mechanism involving Type I interferon-mediation of both muscle and skin inflammation in dermatomyositis." *Id*. at 7; *see* Pet. Ex. 80 at 2; *see also* Resp. Ex. F at 3.

Dr. Gershwin pointed out that upon stimulation, plasmacytoid dendritic cells differentiate into mature cells that induce cytokines and activate other bystander cells that increase inflammation. This is best seen under viral exposure. However, there are no known viruses that produce JDM, but in a genetically susceptible host, something leads to a disturbance of the immune system and a shuffling of the genes. Tr. 18-19. Chronic activation and secretion of Type I interferons in the absence of infection can promote autoimmune diseases, and marked overproduction of Type I interferon-inducible transcripts and proteins in muscle is unique to dermatomyositis as compared to other muscle diseases. Resp. Ex. E at 1; Resp. Ex. F at 3.

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[27] Endoplasmic Reticulum is the membrane network of cells within the cytoplasm. *See Dorland's* 1632.

[28] Kanneboyina Nagaraju et al., *Activation of the Endoplasmic Reticulum Stress Response in Autoimmune Myositis*, 52 Arthritis & Rheumatism 1824 (2005).

[29] Stephen A. Greenberg et al., *Interferon-α/β–Mediated Innate Immune Mechanisms in Dermatomyositis*, 57 Annals of Neurology 664 (2005).

Dr. Rose agreed that JDM is a disease that is identified by autoantibodies and dysregulation involving both the innate and adaptive immune systems. Dr. Rose noted that the division of the immune system is artificial due to the intense cross talk and sharing of cells, cytokines, receptors and intracellular mediators between the "two" systems. Dr. Rose submitted that JDM is more akin to Lupus in which adaptive immunity is a central (yet not the only) player, as opposed to an innate system disease associated with mutation or polymorphysms of typical innate pathways. Dr. Rose also agreed that in JDM there is a genetic component. Dr. Rose acknowledged that, due to the rarity of the disease, research is limited, but he submitted that literature supports the importance of adaptive immunity in JDM given the finding (as in rheumatoid arthritis and systemic lupus) that the histocompatibility region in Chromosome 6 is the most important locus in terms of genetic susceptibility. Resp. Ex. A at 6.

Dr. Rose stated that Dr. Gershwin provided an accurate and up-to-date explanation of dermatomyositis and a great summary of what is known about the disease. Tr. 121; Resp. Ex. A at 11. According to Dr. Rose, the immune system exists to fight infections. Tr. 121. Dr. Rose stated that when you look at tissue, blood cells, and genes in autoimmune diseases, there is an overlap among infectious processes in that the 40 cytokines in the system all utilize the same mechanisms to do different things. Tr. 212.

Dr. Rose agreed that, beyond a doubt, interferon is one important component of the inflammatory infiltrate in the muscle of patients affected by dermatomyositis. Dr. Rose also agreed that there are reliable studies on muscle tissue from dermatomyositis showing interferon involvement. In particular, Dr. Rose explained that the findings of gene expression research in the muscle tissue of individuals with dermatomyositis and polymyositis are called interferonopathies. In these diseases, interferon is important. Systemic lupus is another interferonopathy. Dr. Rose agreed that interferon is a major mediator of myositis, particularly the muscle involvement in dermatomyositis. Tr. 122-23; Resp. Ex. A at 7; *see* Resp. Ex. D at 2.

Dr. Rose added that interferon is also the main way that infectious viruses are killed. "If you kill the cell that is holding the virus, you kill the virus because the virus leaves out of the cell. It does not have its own system." Tr. 124. Interferon is the important mediator in the process usually through apoptosis,[30] which is an ordered form of dying without producing too much damage. Tr. 124.

b. JDM and Vaccinations

In Dr. Gershwin's opinion, C.R. was exposed to multiple vaccines that activated his innate immune response either by direct antigen and/or enhanced by adjuvants in the vaccine. Having received multiple vaccinations, it would be difficult to pinpoint the exact vaccine that triggered the response, but C.R. was exposed to nine different proteins, each containing multiple antigens/epitopes and adjuvants. Pet. Ex. 7 at 8. Thus, according to Dr. Gershwin, C.R.'s "unique genetic response would have included an altered Type I interferon signature, activation of plasmacytoid dendritic cells, and this intense innate response, which was manifest as the

[30] Programmed cell death, commonly called *apoptosis* (from the Greek word meaning "falling off," like leaves from a tree), occurs when cells are no longer needed; they commit suicide by activating an intracellular death program. *See* Bruce Alberts et al., *Molecular Biology of the Cell* 1021 (6th ed. 2015).

inflammation of his muscles and skin. Thereafter this response would become amplified based on the promiscuous immune nature of plasmacytoid dendritic cells, leading to his chronic muscle and dermal inflammatory disease." *Id*.; *see* Tr. 16-18, 52.

Dr. Gershwin pointed out that JDM is so rare that conventional epidemiological studies, based upon current cohort sizes, "would make the disease invisible and below the line." Pet. Ex. 7 at 7. He relied upon *Ernste*[31] (Pet. Ex. 78) to emphasize how rare the disease is, noting that there is no appropriate epidemiology that studies the relationship of vaccines and JDM. The article does emphasize genetic diversity, stating that research over the last decade shows that Type I interferon genes and related proteins "are upregulated in JDM, and they serve as biomarkers for JDM disease activity and organ damage." Pet. Ex. 78 at 674; *see* Tr. 61.

Dr. Gershwin also discussed *Dhiman*[32] (Pet. Ex. 49), a study using the measles vaccination to illustrate the upregulation of genes and the diversity in responses among individuals who received the vaccine. Tr. 62-63; Pet. Ex. 7 at 7. In that study, a measles vaccine induced upregulation of 80 different genes; some involved immunity, others signaled transduction, apoptosis, proliferation of cells, and metabolic pathways. An additional 34 genes underwent downregulation. Dr. Gershwin pointed out that such diversity in response to a vaccine is extraordinary. Pet. Ex. 7 at 8. Further, recent estimates of human T cell receptor diversity suggests that there are approximately 100 million different antigen receptors. *Id.* at 8. The shuffling of all these genes can lead to a very rare event. Tr. 63. Again, the study dealt with interferon genes, and within seven days after measles vaccination the whole network of signaling pathways changed. Tr. 64; *see* Pet. Ex. 49 at 8-9. C.R. received an MMR vaccination.

Further, Dr. Gershwin discussed *Stübgen*[33] (Pet. Ex. 57), an article that provides case histories of the onset of post-vaccination dermatomyositis and polymyositis in patients with probable genetic predisposition, to show immunological plausibility and to note the latency period between onset and disease can be relatively short, as in one to two days. *See* Tr. 66-67. That article concludes that the "phenomenon of post-vaccination [inflammatory myopathies] likely exists, but occurrence is rare." Pet. Ex. 57 at 7.

Dr. Rose cautioned that looking to specific vaccines creates the risk of selection bias. He noted that *Stübgen* stated that any connection between immunizations and autoimmune reaction was temporal and not causal, adding that anything past two months is dubious. Tr. 130-31; Pet. Ex. 57 at 2. *Stübgen* then looked at case reports of JDM after vaccinations, but noted that no pattern was found and the anecdotal nature of the reports creates selection bias and limited potential for cause and effect. Tr. 132. According to Dr. Rose, the bias is choosing the event and trying to attribute causality to it. Tr. 133. *Stübgen* then did a retrospective study of case reports and concluded that there was only temporal relationship. Tr. 133; Pet. Ex. 57 at 4. Dr.

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[31] Floranne C. Ernste & Ann M. Reed, *Recent Advances in Juvenile Idiopathic Inflammatory Myopathies*, 26 Current Opinion in Rheumatology 671 (2014).

[32] Neelam Dhiman et al., *Immune Activation at Effector and Gene Expression Levels After Measles Vaccination in Healthy Individuals: A Pilot Study*, 66 Human Immunology 1125 (2005).

[33] Joerg-Patrick Stübgen, *A Review on the Association Between Inflammatory Myopathies and Vaccination*, 13 Autoimmunity Reviews 31 (2014).

Rose pointed out that the study showed that in 43 million people who received flu vaccine, there was no dermatomyositis. Tr. 136. For those who already had dermatomyositis, there was no worsening of symptoms, which is why all patients with dermatomyositis are vaccinated, unless they are on immunosuppressant drugs; then live vaccines are avoided. Tr. 135-36.

However, Dr. Rose conceded that influenza virus was previously reported to be associated with polymyositis and dermatomyositis. Cases of dermatomyositis have been reported following influenza vaccines, raising the possibility that immune responses to antigens shared by the virus and vaccine could be implicated in the development of myositis. Resp. Ex. G at 8.

Finally, both Dr. Gershwin and Dr. Rose agreed that Dr. Frederick Miller is a leading authority on JDM.[34] Dr. Miller is a proponent of the view that both innate immunity and the environment can precipitate inflammatory muscle diseases (such as JDM) and that vaccines can produce dermatomyositis. Tr. 50; Resp. Ex. G at 7-10; *see also Doherty v. Sec'y of Health & Human Servs.*, No. 01-0393V, 2005 WL 6114560, at *2 (Fed. Cl. Spec. Mstr. Aug. 17, 2005) ("Medical literature—endorsed by Dr. Miller, one of the world's leading authorities on myositis—confirms the biological plausibility of a proposition that vaccines can cause [JDM]."). Dr. Rose stated that he had no reason to disagree with anything Dr. Miller says. Tr. 141-42; *see* Tr. 166 ("I could not disagree with Dr. Miller [o]n anything."). Importantly, he agreed with Dr. Miller's opinions that inflammatory myopathies are likely autoimmune or immune-mediated diseases; that "vaccines are a possible environmental trigger for inflammatory myopathies"; and that the "biological plausibility of polymyositis and dermatomyositis following . . . immunizations suggest[s] that vaccines may serve as an environmental risk factor for the development of myositis in genetically susceptible individuals." Tr. 165-66. He also agreed that "the phenomenon of post vaccination [interferon myopathies] likely exists but the occurrence is rare." Tr. 292; *see* Pet. Ex. 57 at 7.

In sum, both experts agreed, as does the supporting medical literature proffered by both parties, that environmental and genetic factors are intricately interwoven in the initiation and progression of JDM. To that end, both experts agreed that vaccines can result in an abnormal and dysregulated innate immunity response involving Type I interferons, which can produce not only a normal response to the vaccines but an abnormal immune effector mechanism as well. Thus, based on the medical records and reports, medical literature, and testimony submitted, I find that petitioners have provided sufficient evidence of a reputable medical theory to satisfy their burden under *Althen*'s first prong.

2. Logical Sequence of Cause and Effect

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). In other words, even if the

[34] Respondent filed two articles authored by Dr. Miller as exhibits in this case. *See* Resp. Ex. D (Frederick W. Miller et al., *Genome-Wide Association Study of Dermatomyositis Reveals Genetic Overlap with Other Autoimmune Disorders*, 65 Arthritis & Rheumatism 3239 (2013)); Resp. Ex. G (Lu Gan & Frederick W. Miller, *State of the Art: What We Know About Infectious Agents and Myositis*, 23 Current Opinion Rheumatology 585 (2011)).

vaccinations can cause the injury, petitioners must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Human Servs.*, 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *id.* at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury," *Paluck v. Sec'y of Health & Human Servs.*, 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375). Petitioners are not, however, required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Human Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Human Servs.*, 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes").

Although both experts agreed on the mechanism by which JDM can be activated by vaccine or infection, they initially disagreed about whether C.R.'s JDM was triggered by vaccine or infection. Dr. Rose came to the hearing convinced that C.R.'s JDM was more likely the result of a viral infection than his vaccinations. Tr. 138. He explained that Type I interferons are central to killing virally-infected cells and serve to amplify the immune response in general. A viral infection is capable of setting off an interferon reaction in a predisposed individual. Resp. Ex. A at 7. Dr. Rose explained that these cells are important responders of the immune system in dealing with all infections, and in particular viral infections like those suffered by C.R. Dr. Rose added that it is not understood why these cells remain in the tissue of those with dermatomyositis instead of leaving in response to the normal down-regulating mechanisms that occur after the infectious stimulus is over. Tr. 117.

According to Dr. Rose, the relationship between infectious agents and rheumatic disorders have been known since the 1930s. "Because most rheumatic inflammatory diseases involve abnormalities in immune pathways and since the immune system['s] main function is to protect us against infections, it follows that infections have always been suspected as cause and triggers of rheumatic disease." Resp. Ex. A at 8. Dr. Rose also noted that enterovirus studies have been compelling showing viral RNA in the muscle of children with JDM, but he admitted that a seroprevalence control study of coxsackievirus—which is the enterovirus that causes HFMD—showed negative results. Resp. Ex. A at 10; *see also* Resp. Ex. G at 8 (noting that "enteroviral infection might simply be a common environmental exposure rather than a trigger for JDM").[35]

Up until the time of the hearing, Dr. Rose had concluded that he could not attribute C.R.'s JDM to his vaccinations with any more certainty than he could to any unidentified cause or to the infections C.R. suffered prior to the disease. Resp. Ex. A at 12. According to Dr. Rose, "environmental triggers are both ubiquitous and protean, and except for UV light we will be dealing with more speculation than proof anytime we wish to impute causality to an environmental stimulus . . . [I]n a theoretical hierarchical scale of plausibility the more overt

[35] Dr. Rose admitted that, when C.R. was diagnosed with HFMD in 2011, "viral panel kits" were not available, so the diagnosis of HFMD was based on clinical findings. Resp. Ex. H at 2. He agreed that the lab work in October 2011, showed sedimentation rate and C-reactive protein elevation, which according to Dr. Rose was compatible with infections. Dr. Rose admitted that it can be seen in autoimmune diseases like dermatomyositis, adding that it is not specific to either of these two conditions.

clinically (as [C.R's] infectious events were) and the closer to the onset of JDM[,] the more a putative trigger can be considered plausible. Still one should be reminded that temporal association is only one criterion for causality, and one cannot categorically make attributions just based on chronology." Resp. Ex. H at 2.

Although Dr. Gershwin could not say which of the various vaccinations given to C.R. was the cause of C.R.'s JDM or if it was a combination of all of them, he noted that C.R. had an MMR vaccine and that there is a scientific basis for the activation of the innate immune system within seven days after receipt of the MMR vaccine and resulting myositis. Tr. 65-66. According to Dr. Gershwin, C.R. had no evidence of an infection prior to August 30, 2011. Tr. 21; *see generally* Pet. Ex. 2.

The onset of C.R.'s JDM is thus the linchpin to this analysis, with an in-depth inquiry of the medical records coupled with the testimony of the parents being the pivotal determinant.

Dr. Gershwin noted that, according to Mrs. Rodriguez, C.R. developed a rash on his knuckles following his August 30, 2011, vaccinations and at the September 12, 2011, doctor's visit, he was noted to have a sore throat, fever, and a "patch of erythema" on his skin. The nurse practitioner documented it as "skin was dry and there was a patch with erythema with redness." Tr. 25-26; Pet. Ex. 1 at 11-12; Pet. Ex. 81. Dr. Gershwin stated that it would be unusual to document a patch if there were a whole body rash. Tr. 27.

Important to Dr. Gershwin's opinion was the fact that C.R. was sick enough at the September 12, 2011, visit to warrant a test of his sedimentation rate and C-reactive protein. Tr. 28. According to Dr. Gershwin, a sedimentation rate test is a non-diagnostic test for non-specific inflammation in which blood is collected, placed in a tube, and observed to determine how fast it falls. C-reactive protein is produced by the liver in response to the interleukin-6 cytokine, which is a marker for inflammation. Tr. 21-23; *see supra* notes 6, 9. Ordering sedimentation rate and C-reactive protein testing is a typical rheumatology practice, but it is not a typical pediatric practice for a child who is vomiting all night with pharyngitis and a fever. Tr. 23-24. According to Dr. Gershwin, one would not test sedimentation rate for a sore throat; it implies a systemic infection. Tests were also run for typhus fever, Lyme disease, Rocky Mountain Spotted Fever, and strep throat, and all were negative. Tr. 30.

C.R.'s sedimentation rate was 38, and his C-reactive protein level was 3.8—both elevated. Pet. Ex. 1 at 38. According to Dr. Gershwin, the sedimentation rate will increase every six hours; thus, for it to be at 38 on September 12, 2011, it would have started increasing approximately 18 hours before. Because the rate will generally level off, and because C.R.'s sedimentation rate was higher in September than it was in January, Dr. Gershwin opined that in September it was in the crescendo phase. This led Dr. Gershwin to conclude that C.R.'s JDM began before September 12, 2011. Tr. 29-31.

Dr. Gershwin also noted C.R.'s elevated white cell count of 17.7. He explained that this was likely caused by acute inflammation of dermatomyositis. He conceded that an elevated white count could also indicate infection, but he noted that the blood cultures taken to test for

infection were negative. This led him to conclude that the elevated white cell count was due to an acute stress situation. Tr. 74-75.

Dr. Gershwin also noted C.R.'s complaints of sun sensitivity. He cited *Mukamel*[36] (Pet. Ex. 82), an article that documents photosensitivity, as well as the characteristic rash which more commonly precedes the muscle weakness in JDM. Tr. 62; Pet. Ex. 82 at 973.

Dr. Gershwin noted that C.R. had enlarged tonsils and lymphadenopathy at his September 12, 2011, medical visit and that Mrs. Rodriguez reported that C.R. had been sick since the vaccinations. Tr. 32. According to Dr. Gershwin, this could have been a hyper-intense immune response due to an increased susceptibility to infections; the fact that C.R. had JDM did not mean he could not have a sore throat. Tr. 32. Alternatively, Dr. Gershwin stated that the vomiting the night before could have caused the sore throat. Tr. 33. Dr. Gershwin admitted on cross examination that in his review of the mother's affidavit, he believed that C.R. probably had a viral infection on September 12, 2011, and that it was not the dermatomyositis that caused the sore throat. Tr. 33, 68. He noted that, but for the rash and the sedimentation rate, he would not have been able to point to the vaccines as the cause at that point. He believes that there were two separate processes occurring. Tr. 69-70.

From the October 4, 2011, visit, Dr. Gershwin focused on a notation of patches on the knuckles and abdomen which are historically a sign of JDM. There was no prior history of atopic dermatitis. Tr. 33-35; Pet. Ex. 1 at 9-10, 23-24. The rash was significant enough that the nurse practitioner referred C.R. to a dermatologist. The record also refers to Fifth disease, a contagious childhood face rash that starts with a fever and respiratory symptoms but eventually goes away. This did not go away. Tr. 35-36.

With regard to HFMD, Dr. Gershwin stated that it is caused by the coxsackievirus and appears as lesions on the hands, feet, and mouth. *See supra* note 4. He would not expect a doctor to refer to ulcers or lesions as a "rash." Tr. 38. He also noted that, in fairness to the doctors, until there is a heliotropic rash over the eyelids and changes in the nail beds, there is not a lot to distinguish atopic dermatitis from JDM. Additionally, this was a child who lived on a farm with animals and had dry skin and flea bites. Tr. 37.

Dr. Gershwin addressed the dermatologist's record stating that C.R. had eczema from the chest down to the extremities with cheek erythema on the head and face. The dermatologist noted a fever a few weeks before and prescribed topical steroids. Tr. 39; Pet. Ex. 1 at 24. Dr. Gershwin submitted that all you have at this point is a rash and elevated sedimentation rate. There was really nothing to alert anyone to JDM. Tr. 40.

Dr. Gershwin then highlighted the persistence of the rash and viral symptoms. At the October 11, 2011, visit, the rash and other non-specific skin eruptions were noted, along with a red eye, enlarged tonsils, and vomiting. Tr. 40-41; Pet. Ex. 1 at 7. The record from the January 9, 2012, visit, states that C.R. still had a rash on his face, knuckles, and the top of his hands; and

[36] Masza Mukamel, *Amyopathic Dermatomyositis in Children: A Diagnostic and Therapeutic Dilemma*, 7 J. Clinical Rheumatology 191 (2001).

that Mrs. Rodriguez continued to report that C.R. was continually sick with a low-grade fever, body aches, and muscle weakness. Tr. 41-43; Pet. Ex. 1 at 4. At this point, Dr. Gershwin indicated that he would suspect a rheumatic disease or autoimmune disease, with JDM at the top of the list. Tr. 42-43. Finally, at the January 16, 2012, visit, Mr. Rodriguez provided the same medical history that had been documented over the previous months, adding that C.R. was having difficulty getting out of bed and snapping buttons. Bloodwork showed a positive ANA, which implied the existence of a systemic inflammatory autoimmune disease. After a thorough workup, C.R. was diagnosed with JDM. Tr. 45-46; Pet. Ex. 4 at 41-43.

With respect to the September 12, 2011, visit, Dr. Rose initially opined that C.R. had a viral infection, noting that C.R. presented with headache, fever, six episodes of vomiting, and a sore throat. Dr. Rose added that a sore throat is not a symptom of JDM, and that while JDM has gastrointestinal complications, those are quite serious and would be seen only in advanced stages of the disease. Tr. 92-94; Pet. Ex. 1 at 11-12. He pointed out that C.R. had exudate (pus) on examination of his throat, which suggested infection. He had enlarged lymph nodes, a symptom compatible with pharyngitis, either viral or bacterial. Tr. 94-95. According to Dr. Rose, the "dry patch with erythema" noted in the record could mean anything. Tr. 95; Pet. Ex. 1 at 12. It was not a symptom of JDM. Tr. 96. In Dr. Rose's opinion, the viral infection would be the more significant trigger to the immune system than the vaccine. Dr. Rose added that he did not know the cause of JDM, but if there were a trigger, he would say that an infection would push the Type I interferon as much or more than a vaccine. Tr. 117. He argued that C.R.'s pharyngitis and six episodes of vomiting could have "rubbed out" the interferon such that, if there were something wrong with C.R.'s interferon pathway, the viral infection was an equally likely culprit, sufficient to trigger his JDM. Tr. 124-25.

Dr. Rose pointed out that he placed far less weight on the blood work results than Dr. Gershwin did, stating that a child who is vomiting and has exudative pharyngitis can easily have elevated C-reactive protein and sedimentation rates. He added that the testing would be ordered to make sure the child did not have appendicitis or a bacterial infection with those symptoms. Tr. 112. He did not find ordering blood work to be unusual. Tr. 113.

When pressed at hearing about his initial opinions regarding the September 12, 2011, visit, Dr. Rose admitted to having misread the record as stating "eczema" rather than "erythema." Tr. 156-57, 174; Pet. Ex. 1 at 12; Resp. Ex. A at 2. Dr. Rose also agreed that a child with a full body rash, what he considers elevated sedimentation rate, and C-reactive protein with a patch of erythema required further testing. Tr. 157. Although Dr. Rose stated that none of the treating physicians (including a dermatologist) thought that the rash noted between September 12, 2011, and October 11, 2011, was suggestive of JDM, he conceded that the records describe the presence of a rash on the visit 13 days after vaccinations. Pet. Ex. 1 at 12; Resp. Ex. A at 4.

With respect to the October 4, 2011, visit, Dr. Rose noted the cough and rash on C.R.'s face, but stated that it was just another viral infection. Tr. 99-100; Pet. Ex. 1 at 9-10. Dr. Rose was directed to the reference on page 10 of Pet. Ex. 1, which documented dry, red patches over knuckles and abdomen, chest scratching and itching. In response, Dr. Rose stated that JDM rash does not itch, and therefore it was atopic dermatitis. Tr. 100.

During the hearing, Dr. Rose was presented with pictures of C.R. which were taken between August 2011, and December 2011. Pet. Ex. 74 at 1-7. In his observation, the pictures showed C.R. to have rosy cheeks, not the malar rash indicative of JDM. Tr. 100-03. He pointed to C.R.'s knuckles and stated that there was no redness on September 29, 2011, although he admitted that the hand was in flexion which could make the redness disappear. Tr. 103. In the November 14, 2011, picture, Dr. Rose noted a little red streak between the large and small knuckle. Tr. 104. In the December 4, 2011, picture, Dr. Rose testified that there was a rash that goes to the lower eyelid and across the nasal bridge; however, at that point it was just before his diagnosis. Tr. 104. To clarify, Dr. Rose stated that in his opinion these photographs showed "blush" rather than rash, essentially healthy rosy cheeks for children. Tr. 104. Dr. Rose also noted the reference to Fifth disease, a rash that takes over the whole face, as opposed to a dermatomyositis rash, which would predominate only the sun exposed areas. Tr. 105. Dr. Rose concluded that he would not be concerned with any of the pictures until December 4, 2011. Tr. 106. He did admit, however, that the doctors were giving a lot of viral diagnoses in the fall of 2011. Tr. 107. Other than the pharyngitis on September 12, 2011, he could not determine what other viral infection there may have been from the record. Dr. Rose added that he was not in the room to exam C.R., but from what he saw in the pictures, he did not believe that C.R. had a dermatomyositis rash. Tr. 108.

Dr. Rose explained that he relied heavily on the dermatologist's opinion that C.R. had atopic dermatitis and eczema in October 2011, because a dermatologist should be very familiar with dermatomyositis rash. Tr. 109-11, 166. According to Dr. Rose, dermatologists might mistake psoriasis or lupus as atopic dermatitis, but he has never seen a doctor misdiagnose dermatomyositis as atopic dermatitis. Tr. 111-12, 120. He conceded, however, that given a healthy child who started school with a host of viruses going around the community, when presented with C.R.'s complaints, the dermatologist might have assumed that he had one of those viruses. Tr. 118-20. Dr. Rose also agreed that JDM is very rare, and that a dermatologist in a rural area may never see JDM in his practice. Tr. 166. Dr. Rose defended his position by stating that a dermatologist would know what JDM was when he saw it, but at the very least would not mistake it for eczema or atopic dermatitis. He added that a dermatologist would have learned about JDM because it is tested on the Board examination; therefore, he was certain that the dermatologist would not have missed it, only to later add that he may have missed it—"[w]e all do." Tr. 120, 167.[37]

Dr. Rose had also pointed out that the October 11, 2011, visit, was important to his opinion because the skin exam was noted to be normal. Dr. Rose queried, if it was dermatomyositis at that time, where did it go? C.R. had bronchitis and sinusitis with purulent discharge from his nose, according to Dr. Rose, proof of another infection or severe allergies. Tr. 113-14; Pet. Ex. 1 at 7. Dr. Rose stated that JDM rash on the knuckles are Gottron's papules, elevated lesions that are sometimes scaly, and they do not go away without treatment. He then

[37] On this point, Dr. Gershwin responded that it is unknown whether the dermatologist had boards, where he was trained, or how long he spent examining the patient. The office form used for the visit does not even have a space for vitals, such as temperature or pulse. In his opinion, it is possible that the dermatologist had never before seen JDM. Tr. 170-71.

admitted that the topical steroid previously prescribed by the dermatologist, could have made it appear better. Tr. 106-07.[38]

In Dr. Rose's opinion, the January 4, 2012, visit—which noted a rash on C.R.'s hands, knees, and face, and Mrs. Rodriguez's report of C.R. having difficulty turning on the electric toothbrush—was more suggestive of something. Tr. 115-16; Pet. Ex. 1 at 4. According to Dr. Rose, based on the history and the photographs, in his opinion the rash was established in December 2011, and was unlikely to have started in October 2011, but added, "anything is possible." Tr. 116.

On cross examination, Dr. Rose conceded that a rash was documented on C.R.'s knuckles on October 4, 2011, as well as in January 2012, both at the pediatrician's office and at Emory. Tr. 176. Dr. Rose conceded that the only evidence he had to support his opinion that the signs of JDM did not appear until December 2011, was the one office note that did not mention any skin condition. He also admitted that the photographs were not of good quality but that he was looking for something objective that could help with a "very contradictory insufficient chart," noting, "I see a photograph as an opportunity, but it's not a perfect evidence of anything." Tr. 177. He further stated that for him it all boiled down to the dermatologist's examination and what he believed from that examination. Tr. 173.

After listening to petitioners' testimony, Dr. Rose was asked whether, assuming the rash on C.R.'s knuckles in September 2015 (when C.R. had a relapse), was the same as the one in September 2011 (shortly after C.R. received his vaccinations), he would agree that September 2011, was the onset of JDM. His response was "absolutely." Tr. 291-92.

There is no dispute that C.R. has JDM. By the time the hearing had concluded, there was no dispute that C.R. developed the characteristics of the disorder which included erythema, Gottron's papules over the skin of the joints, sensitivity to sun and ultimately muscle involvement. Pet. Ex. 82 at 2. Further, there was no dispute that the manifestations of JDM involving the skin were documented on September 12, 2011, along with what may have been an unrelated viral illness. There is no dispute that C.R.'s rash never went away. By the end of the hearing, Dr. Rose agreed that accepting petitioners' testimony would support onset of C.R.'s JDM shortly after his August 30, 2011, vaccinations. As stated above, the parents were sequestered during each other's testimony, and their testimony was consistent with each other as well as with the contemporaneous medical records. Based upon the testimony of the two experts and petitioners, along with the literature and medical records in this matter, petitioners have proven by preponderant evidence a logical sequence of cause and effect between the vaccinations that C.R. received on August 30, 2011, and the onset of his JDM in early September 2011. Petitioners have therefore satisfied their burden on the second *Althen* prong.

[38] The medical literature also indicates that the waxing and waning of symptoms is consistent with the progression of JDM. *See infra* note 39.

3. Proximate Temporal Relationship

To satisfy the third *Althen* prong, petitioners must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof 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*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Human Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

The literature supports an onset of JDM from 24 hours to 2 months. *See* Pet. Ex. 57 at 2. This timeframe is undisputed by the experts.

Dr. Gershwin explained that the clinical symptoms of JDM are unrelenting inflammation of the skin and the muscles, which leads to dysfunction of the muscles. The muscles get flabby and then weak. Tr. 20. Muscle weakness is absolutely required for JDM, though in the beginning of the disease it is almost always skin-based. There is waxing and waning as the immune system evolves and changes. Tr. 21. Dr. Gershwin cited *Patwardhan*[39] to note that the age of onset (whether the child is under or over three years old) dictates the symptoms and course of the disease, with the younger group (under three years old) faring better in the long run. Pet. Ex. 7 at 6. This was important, because C.R. was age four at the time of onset. Dr. Gershwin also pointed out that *Patwardhan* discusses the mean time between onset of symptoms to diagnosis as being 5.6 months for the younger group and 4.5 months for the older group.

The above analysis of onset is also pertinent here. C.R. received several vaccinations on August 30, 2011, and within three days he had a rash over his entire body, and shortly thereafter he developed patches of erythema on his knuckles, then elbows and knees. He later developed muscle weakness. This period of onset is consistent with an innate and then adaptive response to the vaccinations. Moreover, four months elapsed between the September 2011 onset and the January 2012 diagnosis, which is consistent with the onset-to-diagnosis timeframe discussed in *Patwardhan*. Tr. 59; Pet. Ex. 18 at 1. Thus, the timing and natural progression of C.R.'s symptoms, which were initially skin based but later included muscle dysfunction, are consistent with the etiology of vaccine-induced JDM. Petitioners have therefore satisfied their burden on the third and final *Althen* prong.

[39] Anjali Patwardhan et al., *Juvenile Dermatomyositis Is a Different Disease in Children up to Three Years of Age at Onset than in Children Above Three Years at Onset*, 10 Pediatric Rheumatology A63 (2012).

B. Respondent's Burden to Show Unrelated Factors

Because petitioners have established a prima facie case of causation under *Althen*, they are entitled to compensation unless respondent can show by a preponderance of the evidence that C.R.'s injury was in fact caused by a factor unrelated to the vaccines. *Deribeaux*, 717 F.3d at 1367; *see* § 13(a)(1)(B). To meet this standard, respondent must "present sufficient evidence to prove that the alternative factor was the sole substantial factor in bringing about the injury." *Deribeaux*, 717 F.3d at 1367. The Vaccine Act limits the scope of unrelated factors by excluding any "idiopathic, unexplained, unknown, hypothetical or undocumentable cause, factor, injury, illness or condition." § 13(a)(2)(A). "In other words, alternative causes that are 'idiopathic, unexplained, unknown, hypothetical or undocumentable' cannot overcome a petitioner's prima facie case." *Doe*, 601 F.3d at 1357 (quoting § 13(a)(2)(A)).

Initially, Dr. Rose opined that a viral infection was the more significant trigger to the immune system than the vaccines. *See* Tr. 117. Following the testimony of Dr. Gershwin, however, Dr. Rose admitted that he misread the September 12, 2011, record as patches of "eczema" rather than patches of "erythema." He then agreed that C.R. had the presence of a JDM rash at that visit—thirteen days after the vaccinations at issue. *See* Tr. 156-57; Resp. Ex. A at 2, 4. It became clear by the end of the hearing that any symptoms of a viral infection that were present on September 12, 2011, were subsequent, and unrelated to the full body rash and patches of erythema on C.R.'s knuckles, that developed shortly after his vaccinations on August 30, 2011. The onset of C.R.'s skin manifestations of JDM thus predated the development of any viral infection. Because no other evidence of alternative causation was offered, respondent has failed to show by preponderant evidence that an alternative cause was the sole substantial factor in causing C.R.'s JDM.

IV. Conclusion

Upon careful evaluation of all of the evidence submitted in this matter—including the medical records and tests, the testimony of petitioners, and the experts' opinions and medical literature—the undersigned concludes that petitioners have shown that they are entitled to compensation under the Vaccine Act. Petitioners have put forth preponderant evidence that the vaccinations C.R. received on August 30, 2011, triggered his development of JDM, and respondent has failed to rebut that showing. Accordingly, this case shall proceed to damages.

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master