

**In the United States Court of Federal Claims**

**OFFICE OF SPECIAL MASTERS**

Filed: June 23, 2021

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MATTHEW JIMENEZ,

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PUBLISHED

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Petitioner,

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No. 17-1190V

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v.

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Special Master Nora Beth Dorsey

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SECRETARY OF HEALTH  
AND HUMAN SERVICES,

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Ruling on Entitlement; Causation-in-Fact;

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Hepatitis A (“Hep A”) Vaccine; Human

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Papillomavirus (“HPV”) Vaccine; Systemic

Respondent.

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Juvenile Idiopathic Arthritis (“sJIA”); Still’s

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Disease; Cryopyrin-Associated Periodic

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Syndrome (“CAPS”); Muckle-Wells

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Syndrome (“MWS”).

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Richard Gage, Richard Gage, P.C., Cheyenne, WY, for petitioner.

Traci R. Patton, U.S. Department of Justice, Washington, DC, for respondent.

**RULING ON ENTITLEMENT**<sup>1</sup>

**I. INTRODUCTION**

On September 5, 2017, Matthew Jimenez (“petitioner”) filed a petition for compensation under the National Vaccine Injury Compensation Program (“Vaccine Act” or “the Program”), 42 U.S.C. § 300aa-10 *et seq.* (2012).<sup>2</sup> Petitioner alleges that he suffered from juvenile rheumatoid arthritis (“JRA”) as the result of Hepatitis A (“Hep A”) and human papillomavirus (“HPV”)

<sup>1</sup> The undersigned intends to post this Ruling on the United States Court of Federal Claims’ website. **This means the Ruling will be available to anyone with access to the internet.** In accordance with Vaccine Rule 18(b), petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access. Because this Ruling contains a reasoned explanation for the action in this case, undersigned is required to post it on the United States Court of Federal Claims’ website in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2012) (Federal Management and Promotion of Electronic Government Services).

<sup>2</sup> The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2012). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

vaccinations he received on September 18, 2014. Amended (“Am.”) Petition at 1 (ECF No. 16). Petitioner later characterized his injury as systemic juvenile idiopathic arthritis (“sJIA”). See Petitioner’s Pre-Hearing Brief (“Pet. Pre-Hearing Br.”), filed Aug. 3, 2020, at 1 (ECF No. 51).

After carefully analyzing and weighing the evidence in accordance with the applicable legal standards, the undersigned finds that petitioner has provided preponderant evidence as to diagnosis, and that the HPV and/or the Hep A vaccines he received caused him to develop sJIA, which satisfies his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, petitioner is entitled to compensation.

## **II. PROCEDURAL HISTORY**

Petitioner filed his petition requesting compensation under the Vaccine Act on September 5, 2017. Petition (ECF No. 1). On September 13, 2017, petitioner filed a motion to Amend/Correct the caption, which was granted that same day. Motion (“Mot.”) to Amend/Correct Caption, filed Sept. 13, 2017 (ECF No. 7); Order Granting Mot. to Amend/Correct Caption dated Sept. 13, 2017 (ECF No. 8). Petitioner filed medical records from December 2017 to April 2018. Pet. Exhibits (“Exs.”) 1-8.

On April 2, 2018, petitioner filed an Amended Petition correcting the type of vaccines petitioner received on September 18, 2014. Am. Petition. Respondent filed his Rule 4(c) Report on June 15, 2018, arguing against compensation. Respondent’s Report (“Resp. Rept.”), filed June 15, 2018 (ECF No. 19). In December 2018, petitioner filed affidavits and an expert report from Dr. M. Eric Gershwin with accompanying medical literature. Pet. Exs. 9-31.

The Court issued an Order to Show Cause to petitioner for failure to comply with previous orders on January 7, 2019. Order to Show Cause dated Jan. 7, 2019 (ECF No. 29). The parties scheduled a status conference on February 11, 2019 and respondent was ordered to file an expert report. Non-PDF Scheduling Order dated Feb. 11, 2019. Respondent filed expert reports from Dr. Carlos Rosé and Dr. Craig Platt with accompanying medical literature on June 28, 2019. Resp. Exs. A-D. Petitioner filed a supplemental expert report on August 2, 2019. Pet. Exs. 32-36.

The special master at the time held a status conference on August 20, 2019 to discuss scheduling an entitlement hearing and respondent’s request for petitioner to obtain genetic testing. See Non-PDF Order dated Aug. 29, 2019. On September 17, 2019, the parties filed a Joint Status Report requesting an entitlement hearing for September 2020 and the Court issued a Pre-Hearing Order the next day setting an entitlement hearing for September 16, 2020. Joint Status Rept., filed Sept. 17, 2019 (ECF No. 36); Pre-Hearing Order dated Sept. 18, 2019 (ECF No. 37). On September 23, 2019, petitioner filed a memorandum objecting to genetic testing. Pet. Memorandum (“Mem.”), filed Sept. 23, 2019 (ECF No. 38).

The case was reassigned to the undersigned on October 4, 2019. Order Reassigning Case dated Oct. 4, 2019 (ECF No. 39). On November 1, 2019, the undersigned issued an Amended Pre-Hearing Order changing the start of the hearing from September 16 to September 23, 2020. Am. Pre-Hearing Order dated Nov. 1, 2019 (ECF No. 42). Petitioner filed pre-hearing

submissions, medical literature, and medical records from July to September 2020. Pet. Pre-Hearing Submissions, filed July 29, 2020 (ECF No. 50); Pet. Pre-Hearing Submissions, filed Aug. 3, 2020 (ECF No. 51); Pet. Exs. 37-46. Respondent filed pre-hearing submissions and medical literature in August and September 2020. Resp. Pre-Hearing Submissions, filed Aug. 25, 2020 (ECF No. 59); Resp. Exs. E-G.

An entitlement hearing was held on September 23 and 24, 2020. Order dated Sept. 24, 2020 (ECF No. 68). Additional documents and post-hearing briefs were requested from both parties during the hearing, and were filed from September 2020 to January 2021. Resp. Ex. H; Pet. Exs. 47-53; Pet. Post-Hearing Brief (“Br.”), filed Dec. 4, 2020 (ECF No. 81); Resp. Post-Hearing Br., filed Jan. 22, 2021 (ECF No. 85).

The matter is now ripe for adjudication.

### **III. ISSUES TO BE DECIDED**

The parties dispute diagnosis and causation. Petitioner alleged he suffered from a variety of symptoms related to sJIA, beginning with the onset of a rash following his vaccinations. See Pet. Post-Hearing Br. at 2, 7. Petitioner stated he is entitled to compensation as outlined by the evidence presented in the relevant medical records and expert reports. Id. at 7.

Respondent argued petitioner has not established by preponderant evidence that he suffers from sJIA, or any other injury, caused by the HPV and/or Hep A vaccines he received on September 18, 2014. While petitioner’s treating physicians diagnosed him with sJIA, respondent stated that his symptoms are not consistent with this condition. Resp. Pre-Hearing Br. at 12. Instead of sJIA, respondent asserted that petitioner suffers from Cryopyrin-Associated Periodic Syndrome (“CAPS”), specifically a form known as Muckle-Wells Syndrome (“MWS”), which was not caused by petitioner’s vaccinations. Id. at 21; Resp. Ex. A at 11. Furthermore, respondent asserted that even if petitioner suffers from sJIA, he did not meet his burden of proof to show vaccine-related causation. Resp. Pre-Hearing Br. at 14. Therefore, respondent stated that petitioner is not entitled to compensation under the Vaccine Act. Id.

### **IV. MEDICAL TERMINOLOGY**

sJIA “is a heterogeneous and multifactorial autoimmune disease characterized by chronic joint inflammation in children” who are younger than age sixteen. Pet. Ex. 31 at 2.<sup>3</sup> It is the “most common cause of chronic arthritis in children.” Pet. Ex. 30 at 1.<sup>4</sup> The condition is characterized by “arthritis with spiking fever persisting for more than 2 weeks and at least one of the following clinical features of systemic inflammation: skin rash, lymphadenopathy, hepatosplenomegaly[,] or serositis (pleuritis or pericarditis).” Pet. Ex. 31 at 2. “[D]aily spiking

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<sup>3</sup> Yu-Tsan Lin et al., The Pathogenesis of Oligoarticular/Polyarticular vs Systemic Juvenile Idiopathic Arthritis, 10 *Autoimmunity Rev.* 482 (2011).

<sup>4</sup> Sheila Angeles-Han & Sampath Prahalad, The Genetics of Juvenile Idiopathic Arthritis: What Is New in 2010?, 12 *Current Rheumatology Rep.* 87 (2010).

fevers, [and a] fleeting salmon-colored macular rash” are notable signs of the illness. Pet. Ex. 44 at 1-2.<sup>5</sup> Laboratory findings evidencing systemic inflammation include elevated erythrocyte sedimentation rate, C-reactive protein, neutrophils, and platelets. *Id.* at 2.

“As in all complex disease, the underlying factors influencing susceptibility are thought to include a combination of genes and environmental interactions.” Pet. Ex. 29 at 1.<sup>6</sup> There are several subtypes and different classifications of the illness, but for purposes of this Ruling, the term sJIA will be used.<sup>7</sup> See Pet. Ex. 30 at 1. Historically, the condition was called Still’s disease, named after the physician who initially described it. Resp. Ex. A, Tab A1 at 1.<sup>8</sup>

The International League of Associations for Rheumatology (“ILAR”) diagnostic criteria for sJIA is set forth in the table below:

<b>BOX 16-1 Systemic Juvenile Idiopathic Arthritis: International League of Associations for Rheumatology (ILAR) Diagnostic Criteria</b>
Arthritis in any number of joints together with a fever of at least 2 weeks' duration that is documented to be daily (quotidian) for at least 3 days and is accompanied by one or more of the following: <ul style="list-style-type: none"><li>• Evanescent rash</li><li>• Generalized lymphadenopathy</li><li>• Enlargement of liver or spleen</li><li>• Serositis</li></ul> Exclusions: <ul style="list-style-type: none"><li>• Psoriasis or a history of psoriasis in the patient or a first-degree relative</li><li>• Arthritis in an HLA-B27–positive male beginning after his sixth birthday</li><li>• Ankylosing spondylitis, enthesitis-related arthritis, sacroiliitis with inflammatory bowel disease, reactive arthritis, or acute anterior uveitis, or a history of one of these disorders in a first-degree relative</li><li>• The presence of IgM RF on at least two occasions at least 3 months apart</li></ul>

Resp. Ex. A, Tab A1 at 2 Box16-1.

<sup>5</sup> Elizabeth D. Mellins et al., Pathogenesis of Systemic Juvenile Idiopathic Arthritis: Some Answers, More Questions, 7 *Nature Rev. Rheumatology* 416 (2011).

<sup>6</sup> Emma M. Ogilvie et al., The -174G Allele of the Interleukin-6 Gene Confers Susceptibility to Systemic Arthritis in Children: A Multicenter Study Using Simplex and Multiplex Juvenile Idiopathic Arthritis Families, 48 *Arthritis & Rheumatism* 3202 (2003).

<sup>7</sup> Dr. Gershwin primarily used the word Still or Still’s disease instead of sJIA throughout his reports and testimony. To avoid confusion, and for simplicity, the undersigned will use the phrase systemic Juvenile Idiopathic Arthritis or sJIA in this Ruling.

<sup>8</sup> Fabrizio De Benedetti & Rayfel Schneider, Systemic Juvenile Idiopathic Arthritis, in *Textbook of Pediatric Rheumatology* 205-16 (Ross E. Petty et al. eds., 7th ed. 2016).

CAPS “is a rare inherited inflammatory disease associated with overproduction of interleukin-1.” Resp. Ex. A, Tab A6 at 1.<sup>9</sup> The syndrome is caused by “dominantly inherited abnormalities in cryopyrin (NLRP3), which results from mutations in the *NLRP3* gene.” Resp. Ex. A, Tab A4 at 8.<sup>10</sup> There are three subtypes: (1) familiar cold autoinflammatory syndrome (“FCAS”), (2) MWS, and (3) neonatal-onset multisystem inflammatory disorder (“NOMIC”). Pet. Ex. 47 at 1.<sup>11</sup> All three “arise from mutations in a single gene, *NLRP3*, at chromosome 1a44, encoding a protein called cryopyrin.” *Id.* Cryopyrin is required for the “assembly of the NALP3 inflammasome, one of multiple distinct inflammasome complexes . . . responsible . . . for activation of the potent proinflammatory cytokines interleukin (IL) 1 beta and IL-18.” *Id.* at 2.

CAPS diagnostic criteria include elevated markers of inflammation, plus at least two out of the following six signs/symptoms: “(1) urticaria-like rash, (2) cold-trigger episodes, (3) sensorineural hearing loss, (4) musculoskeletal symptoms (arthralgia/arthritis/myalgia), (5) chronic aseptic meningitis[,] and (6) skeletal abnormalities (epiphyseal overgrowth/frontal bossing).” Resp. Ex. A, Tab A3 at 4.<sup>12</sup>

The rash in CAPS is “usually the first notable manifestation and develops shortly after birth or in early infancy.” Resp. Ex. A, Tab A4 at 9. Histologically, the rash is “a predominant[ly] perivascular neutrophilic infiltrate.” *Id.*

Clinical features of MWS include “[i]ntermittent episodes of fever, headache, urticarial rash, and joint pain (arthralgias or arthritis); progressive sensorineural hearing loss; [and] secondary (AA) amyloidosis with nephropathy.” Pet. Ex. 47 at 3. “Febrile episodes occur at irregular intervals every few weeks, lasting 12 to 36 hours before resolving spontaneously. Age of onset is variable.” *Id.* “Precipitating factors cannot usually be identified.” Resp. Ex. A, Tab A4 at 9.

“Sensorineural hearing loss is seen in approximately 70% of cases” of MWS and “usually begins in later childhood or early adulthood.” Resp. Ex. A, Tab A4 at 10. In addition to hearing loss, the disease may cause “long-standing uncontrolled inflammation [which] results in irreversible organ damage,” not only causing hearing loss, but also “amyloidosis, vision loss,

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<sup>9</sup> Helen J. Lachman et al., Use of Canakinumab in the Cryopyrin-Associated Periodic Syndrome, 360 *New Eng. J. Med.* 2416 (2009).

<sup>10</sup> Karyl S. Barron & Daniel L. Kastner, Periodic Fever Syndromes and Other Inherited Autoinflammatory Diseases, in Textbook of Pediatric Rheumatology 609-26 (Ross E. Petty et al. eds., 7th ed. 2016).

<sup>11</sup> Peter A. Nigrovic, Cryopyrin-Associated Periodic Syndromes and Related Disorders, UpToDate (2020), <https://www.uptodate.com/contents/cryopyrin-associated-periodic-syndromes-and-related-disorders>.

<sup>12</sup> Jasmin B. Kuemmerle-Deschner et al., Diagnostic Criteria for Cryopyrin-Associated Periodic Syndrome (CAPS), 76 *Annals Rheumatic Diseases* 942 (2017).

skeletal deformities and cognitive disability.” Resp. Ex. A, Tab A3 at 1. Chronic aseptic meningitis, which can cause severe headaches, may also be associated with the illness. Id. at 4; Resp. Ex. A at 12.

Macrophage activation syndrome (“MAS”) “is a clinical syndrome caused by excessive activation and proliferation of well differentiated macrophages.” Pet. Ex. 26 at 1.<sup>13</sup> It may occur as a complication in a wide range of systemic inflammatory conditions, including sJIA. Resp. Ex. A, Tab A7 at 2.<sup>14</sup> “Clinical findings of MAS are dramatic.” Pet. Ex. 26 at 1. Typically, a patient with a chronic disease becomes acutely ill with persistent fever, lymphadenopathy, hepatosplenomegaly, central nervous dysfunction, and hemorrhagic manifestations. Id.; Resp. Ex. A, Tab A7 at 2. Bone marrow biopsy reveals numerous well differentiated macrophages “exhibiting hemophagocytic activity.” Resp. Ex. A, Tab A7 at 2. If unrecognized and untreated, MAS can “result in progressive multi-organ failure and eventually a fatal outcome.” Id.

## V. FACTUAL SUMMARY

### A. Summary of Relevant Facts

On September 18, 2014, petitioner received his first HPV and first Hep A vaccinations from his primary care provider (“PCP”), Dr. Adam Cutler. Pet. Ex. 1 at 2, 42. At the time, he was a fifteen-year-old high school student with an unremarkable medical history. Id. at 41-42, 78; Pet. Ex. 8 at 1. Dr. Cutler did not document any rash, lesions, nighttime sweating, or other abnormal conditions, on the date of vaccination. Pet. Ex. 1 at 42-44.

Approximately one week after vaccination, petitioner noticed a rash on his arms and legs. Transcript (“Tr.”) 9. Petitioner characterized his rash as light pink salmon color, randomly splattered across his skin—like paint splatter. Id.; see also Pet. Exs. 49-51.<sup>15</sup> The rash was flat in appearance when petitioner first noticed it. Tr. 19. He did not think the rash was serious, so he ignored it until his mother saw it on his arms a few weeks later. Tr. 10. The rash became progressively worse and itchy, and petitioner developed other symptoms, including fevers, night sweats, joint pain, and body aches. Tr. 12-13, 19.

On November 20, 2014, petitioner presented to Dr. Cutler for evaluation of a rash on his arms, legs, and buttocks. Pet. Ex. 1 at 46-48. The rash was described as “intermittent, resolves spontaneously, over several weeks” and slightly itchy. Id. at 46. Dr. Cutler noted that petitioner took no prescribed or over-the-counter medications. Id. Petitioner was diagnosed with urticaria hives, and Dr. Cutler recommended daily Zyrtec, and advised petitioner to keep a diary to identify potential causes of his rash. Id. at 48.

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<sup>13</sup> S. Sawhney et al., Macrophage Activation Syndrome: A Potentially Fatal Complication of Rheumatic Disorders, 85 *Archives Disease Childhood* 421 (2001).

<sup>14</sup> Angelo Ravelli et al., 2016 Classification Criteria for Macrophage Activation Syndrome Complicating Systemic Juvenile Idiopathic Arthritis, 68 *Arthritis & Rheumatology* 566 (2016).

<sup>15</sup> Exhibits 49-51 are photographs of petitioner’s rash on his arms and legs.

On December 1, 2014, petitioner presented to MedExpress, an urgent care facility, complaining of a rash, sore throat, and fever. Pet. Ex. 4 at 1. The rash was noted to be present for one month. Id. Rapid flu and strep tests were negative. Id. at 3. Petitioner was diagnosed with acute pharyngitis and contact dermatitis. Id. He was given prescriptions for oral amoxicillin and a topical steroid. Id.

Petitioner presented to Dr. Karimu Smith-Barron the next day, on December 2, 2014. Pet. Ex. 1 at 49. He had a fever of 101.8°F, down from 104°F the previous evening. Id. at 49-50. Dr. Smith-Barron noted petitioner had a “fleeting” rash for two weeks and his mother stated the rash was still present after two months. Id. at 50; Tr. 41. He was suspected to have a bacterial infection and was assessed with acute pharyngitis, presumed streptococcus, and hyperpyrexia. Pet. Ex. 1 at 52. Lab work revealed an elevated white blood count, increased granulocytes, and he had a negative rapid strep test. Id. at 12, 51, 89.

Petitioner was next seen on December 4, 2014 by Dr. Gary Lieberman, with continued fever, joint pains, sore throat, headache, and rash. Pet. Ex. 1 at 55-57. Dr. Lieberman documented, “[t]his rash seems unlike prior rash.” Id. at 55. Physical exam was notable for a red rash around petitioner’s eyes and a macular rash on his extremities. Id. at 56. Petitioner also had limited range of motion (“ROM”) of one knee, and mild pain in his fingers and neck. Id. Throat cultures and mono spot test for Epstein-Barr virus were negative. Id. at 13, 56. He had an elevated white blood count, granulocytes, and lymphocytes. Id. at 56. Dr. Lieberman’s diagnoses were hyperpyrexia and unspecified multiple arthropathy. Id. at 57.

On December 6, 2014, petitioner returned to his PCP due to six days of persistent fevers, sore throat, and migratory joint pains. Pet. Ex. 1 at 59. He was diagnosed with hyperpyrexia and unspecified polyarthritis. Id. at 61.

Later that day, petitioner was admitted to West Boca Medical Center, where he received an extensive work up. Pet. Ex. 1 at 98, 111, 147-57. He tested negative for ASO titer,<sup>16</sup>

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<sup>16</sup> ASO titer is a blood test used to measure antibodies against group A streptococcus bacteria. Antistreptolysin, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=3511> (last visited May 27, 2021); Streptolysin, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=47431> (last visited May 27, 2021).

Bartonella studies,<sup>17</sup> CMV,<sup>18</sup> rheumatoid factor,<sup>19</sup> rotavirus,<sup>20</sup> ANA screen,<sup>21</sup> and Epstein-Barr virus.<sup>22</sup> Pet. Ex. 1 at 101; Pet. Ex. 3 at 168, 176. Stool, blood, and urine microbiology tests were negative, and no evidence of bacteria was found. Pet. Ex. 1 at 154-55; Pet. Ex. 3 at 168.

He was transferred to Miami Children’s Hospital (“Miami Children’s”) on December 10, 2014 for rheumatological evaluation with a presumed diagnosis of sJIA. Pet. Ex. 1 at 118; Pet. Ex. 3 at 168. Upon admission to Miami Children’s, it was noted that petitioner had nine days of fever of 104°F, sore throat, headache, migratory symmetric arthralgias, and macular blanchable nonpruritic, nonpainful rash on his upper and lower extremities. Pet. Ex. 2 at 102. He also had an elevated C-reactive protein of 18.3 (normal range is 0.0-1.0) and a high erythrocyte sedimentation rate of 55 (normal range is 0-30). Pet. Ex. 3 at 156, 160, 168.<sup>23</sup>

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<sup>17</sup> Bartonella is a Gram-negative bacteria. Bartonella, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=5564&searchterm=Bartonella> (last visited May 27, 2021).

<sup>18</sup> CMV, or cytomegalovirus, is a virus that infects humans and includes the herpesvirus 5. Cytomegalovirus, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=12438> (last visited May 27, 2021).

<sup>19</sup> Rheumatoid factor is “antibodies directed against antigenic determinants.” Rheumatoid Factor, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=74591> (last visited May 27, 2021). “[T]hese are found in the serum of about 80 percent of persons with classical or definite rheumatoid arthritis but only about 20 percent of those with juvenile rheumatoid arthritis. Rheumatoid factors may be of the IgM, IgG, or IgA classes of immunoglobulins . . . . Id.

<sup>20</sup> Rotaviruses are transmitted by the fecal-oral route and cause acute infantile gastroenteritis and diarrhea in young children. Rotavirus, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=44147> (last visited May 27, 2021).

<sup>21</sup> ANA test screens for antinuclear antibodies, which are antibodies directed against nuclear antigens; ones against a variety of different antigens are almost invariably found in systemic lupus erythematosus and are frequently found in rheumatoid arthritis. Antinuclear Antibodies, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=56804> (last visited May 27, 2021).

<sup>22</sup> Epstein-Barr virus, also known as human herpesvirus 4, is a member of the herpes virus family and can cause infectious mononucleosis. Epstein-Barr Virus, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=80849> (last visited May 27, 2021).

<sup>23</sup> The erythrocyte sedimentation rate and the level of C-reactive proteins are indications of inflammation in the body. Erythrocyte Sedimentation Rate, Dorland’s Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=102146> (last visited Mar. 17, 2021); C-



Upon admission to Miami Children's, petitioner was seen by rheumatologist Dr. Rafael Rivas-Chacon who noted a three-week history of an intermittent, erythematous, non-itchy rash, arthritis in two fingers, and arthralgias in the right wrist. Pet. Ex. 1 at 138. Petitioner was also seen by infectious disease expert Dr. Carolina Sanchez-Vegas, who noted that petitioner's symptoms reportedly began on November 30, 2014. Pet. Ex. 2 at 102-08. Petitioner was discharged on December 11, 2014, with diagnoses of prolonged fever, possible juvenile idiopathic arthritis ("JIA"), and rash. Id. at 122. The discharge summary indicated the providers were "concerned about possible JIA given the history of migratory arthritis with the rash." Id. Petitioner was prescribed naproxen and instructed to follow up with rheumatology. Id. at 123.

Petitioner was seen by his PCP in a follow-up on December 15, 2014, at which time he was still having twice daily fevers, along with daily arthritis, arthralgias, and rash. Pet. Ex. 1 at 63. He was assessed with viral disease, rash, hyperpyrexia, and polyarthritis. Id. at 65.

On January 7, 2015, petitioner had a follow-up exam with Dr. Sanchez-Vegas, his infectious disease specialist. Pet. Ex. 1 at 125-30. Dr. Sanchez-Vegas reported that after petitioner was discharged, he continued to have fevers up to 104°F with accompanying headache, migratory symmetric arthralgias, and macular rash. Id. All symptoms were stated to recur during a fever and resolve daily when petitioner was afebrile. Id. at 125. Dr. Sanchez-Vargas noted symptoms began on November 30, 2014. Id. She noted that petitioner had a positive Immunoglobulin G ("IgG") for parvovirus, though he was negative for Immunoglobulin M ("IgM").<sup>24</sup> Id. Petitioner was diagnosed with rash, joint pain, prolonged fever, and human parvovirus infection, though Dr. Sanchez-Vargas thought that parvovirus was unlikely. Id. at 127-28.

Petitioner was seen by Dr. Angela Weatherall, a dermatologist, on January 28, 2015, due to an itchy, red rash on his back, arms, and legs, which he reported had been present for three months. Pet. Ex. 1 at 184-85. He was assessed with urticaria, and a punch biopsy was performed. Id. at 188. Dr. Weatherall recommended that petitioner take Zyrtec. Pet. Ex. 3 at 2.

Two weeks later, on February 12, 2015, petitioner returned to see Dr. Weatherall who informed him that his biopsy was consistent with chronic urticaria. Pet. Ex. 1 at 178-79, 188. At

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reactive Protein, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=100489> (last visited Mar. 17, 2021).

<sup>24</sup> Immunoglobulins are "structurally related glycoproteins that function as antibodies." Immunoglobulin, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=24894> (last visited Mar. 17, 2021). IgG antibodies "are produced during an initial infection or other antigen exposure, rising a few weeks after it begins, then decreasing and stabilizing." Immunoglobulins (IgG, IgM), Merck Manual, <https://www.merckmanuals.com/-/media/Manual/LabTests/ImmunoglobulinsIgAIgGIgM> (last visited Mar. 17, 2021). IgM antibodies "are produced as a body's first response to a new infection," and "increase for several weeks and then decline as IgG production begins." Id.

a follow-up visit on March 12, 2015, Dr. Weatherall noted that petitioner's urticaria had improved. Pet. Ex. 3 at 11.

On June 2, 2015, petitioner was seen at his PCP's office, reporting that he had been fever-free for a while, but his fever and joint aches had returned over the past week, along with some headaches, and his rash had not gone away. Pet. Ex. 1 at 66-68. The records indicate that petitioner had not yet been tested for sJIA. Id. Lab results from blood drawn at this visit revealed elevated ferritin, erythrocyte sedimentation rate, C-reactive protein, and white blood cell count.<sup>25</sup> Id. at 24-25, 29; Pet. Ex. 3 at 191-200.

Petitioner presented to rheumatologist, Dr. Kristina Weirs-Shamir, on June 9, 2015. Pet. Ex. 1 at 70, 166. He reported that he had a rash that worsened with fevers, along with migratory joint pain in both knees and ankles, which caused difficulty walking. Id. He tested negative for Celiac disease and Lyme disease. Id. at 24.

On June 16, 2015, petitioner's mother called his PCP, reporting that petitioner was getting worse, and she wanted a referral to see an infectious disease expert. Pet. Ex. 1 at 31. Petitioner was seen at Miami Children's the following day by Dr. Vargas-Sanchez. Id. at 191. Chest X-rays were normal. Id. at 192. However, an ultrasound showed hepatosplenomegaly.<sup>26</sup> Id. at 194; Pet. Ex. 2 at 14-15.

On June 19, 2015, petitioner's mother called the PCP again, stating that petitioner's fever had returned and the lymph nodes under his arm were swollen. Pet. Ex. 1 at 73. He was seen by Dr. Jerome Sigua, an allergist/immunologist, on June 22, 2015. Id. at 199. Dr. Sigua ordered extensive bloodwork and recommended that petitioner see a pediatric hematology oncologist for a bone marrow biopsy. Id. at 199-201.

Petitioner was seen by hematology oncologist, Dr. Melissa Singer, on June 29, 2015. Pet. Ex. 1 at 204-07. Dr. Singer noted that petitioner had no diagnosis, but had multiple labs pending from various providers. Id. at 204. Bone marrow biopsy was performed on July 1, 2015. Pet. Ex. 6 at 32. It showed no overt immunophenotypic<sup>27</sup> evidence of non-Hodgkin B-cell lymphoproliferative disorder, aberrant T-cells, or acute leukemia. Id.

On December 1, 2015, petitioner presented to pediatric neurologist Dr. Farjam Farzam. Pet. Ex. 1 at 209. Dr. Farzam noted fevers worse at night than in the mornings, night sweats,

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<sup>25</sup> Ferritin was 534 (normal range is 13-83), erythrocyte sedimentation rate was 49 (normal range is 0-30), and C-reactive protein was 8.54 (normal range is 0.0-1.0). Pet. Ex. 3 at 192-93, 199.

<sup>26</sup> Hepatosplenomegaly is the enlargement of the liver and spleen. Hepatosplenomegaly, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=22274> (last visited Mar. 17, 2021).

<sup>27</sup> Immunophenotype is the characterization of a set of cells according to the antigens expressed. Immunophenotype, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=24914> (last visited May 27, 2021).

headaches, and fatigue, but there was no rash present. Id. Petitioner was neurologically normal. Id. at 209-10. He assessed petitioner with headaches and dizziness. Id. at 210. Petitioner had a follow-up with Dr. Farzam on January 12, 2016. Id. at 213. Dr. Farzam noted petitioner's electroencephalogram ("EEG") study was normal. Id.

Petitioner returned to Dr. Weatherall on June 29, 2016. Pet. Ex. 3 at 15. He reported his urticaria had continually reoccurred over the year in cycles with fever, migratory joint pain, and occasional nausea and dizziness. Id. Dr. Weatherall observed that petitioner had been seen by multiple specialists and all his workups were negative, except for elevated erythrocyte sedimentation rate and C-reactive protein. Id. Dr. Weatherall performed another punch biopsy and administered a Kenalog injection. Id. at 15-16. The punch biopsy showed "mild superficial/mid-dermal polymorphous inflammatory infiltrate with predominance of neutrophils, edema, and no epidermal changes." Id. at 188. The comment section of the biopsy stated, "findings may be consistent with urticaria . . . Similar changes can be seen in skin lesions of Still disease (sJIA). Clinical correlation is recommended." Id.

On July 11, 2016, petitioner presented to rheumatologist, Dr. Korey Ullrich, on referral from Dr. Weatherall. Pet. Ex. 1 at 219. Dr. Ullrich noted petitioner developed a rash on his arms and legs mid/late 2014. Pet. Ex. 5 at 1. In his "first cycle of symptoms," petitioner had night sweats, fevers, rash, joint pain, and fatigue. Id. These symptoms lasted for approximately six months and then resolved. Id. Petitioner's "second cycle" occurred in May 2015, and since then he had similar flare ups of symptoms every three months. Id. Dr. Ullrich noted extensive testing confirmed etiologies for infection and malignancy were negative. Id. at 2-3. After review of petitioner's biopsies, and extensive work ups, Dr. Ullrich concluded that petitioner's symptoms were consistent with sJIA. Id. at 1-2; Pet. Ex. 1 at 219. Dr. Ullrich diagnosed petitioner with sJIA. Pet. Ex. 1 at 221; Pet. Ex. 5 at 3. Dr. Ullrich prescribed a trial of prednisone. Pet. Ex. 5 at 3.

On July 13, 2016, petitioner presented to rheumatologist, Dr. Steven Goodman. Pet. Ex. 1 at 216. Petitioner had moderate to severe pain in both wrists, arms, and knees. Id. Dr. Goodman opined petitioner had a classical presentation of sJIA characterized by chronic arthritis with active synovitis, associated with rash and fever. Id. Dr. Goodman ordered additional laboratory tests and prescribed a Medrol dose pack. Id.

Petitioner returned to see Dr. Ullrich on August 4, 2016. Pet. Ex. 5 at 4. Dr. Ullrich noted petitioner was improving, but experiencing side effects on prednisone. Id. at 6. "Extensive [work ups] for infection, malignancy, and other potential etiologies [are] negative." Id. On September 12, 2016, Dr. Ullrich recommended a sparing steroid agent, Actemra. Id. at 9. Petitioner stated he wanted to try homeopathic treatment before trying Actemra. Id. On September 23, 2016, petitioner returned to Dr. Ullrich reporting his rash had worsened and he had decided to proceed with Actemra. Id. at 10. On November 9, 2016, Dr. Ullrich noted petitioner's rash was present, but stable. Id. at 16, 18.

Moving forward to 2017, on March 27, 2017, petitioner presented to Dr. Weatherall for hives. Pet. Ex. 3 at 19. Dr. Weatherall noted petitioner had been diagnosed with sJIA, was taking Actemra, and doing well. Id. Dr. Weatherall noted petitioner's only persistent symptom

was flare ups of the rash. Id. Petitioner returned to Dr. Weatherall on April 6, 2017, and his disease was in remission, except for the persistent urticarial rash. Id. at 24.

Petitioner followed up with Dr. Ullrich on April 11, 2017 for a hives flare up. Pet. Ex. 5 at 25. Dr. Ullrich stated the cause of the flare was unclear, but it may have been triggered by medication or an infection. Id. at 27. Petitioner improved on high-dose prednisone. Id. at 28, 30. Dr. Ullrich noted petitioner's urticaria was likely related to his sJIA. Id. at 33.

On July 14, 2017, Dr. Ullrich noted petitioner's rash had resolved. Pet. Ex. 5 at 37. Petitioner's medications listed prednisone, Actemra, and Dapsone. Id. Then on August 11, 2017, petitioner's rash reoccurred. Id. at 40. The assessment was sJIA, improved overall, with mild reoccurrence of rash. Id. at 42.

Petitioner continued with Actemra until September 4, 2018, when he switched to Anakinra, an IL-1 inhibitor. Pet. Ex. 38 at 17, 19. When petitioner saw Dr. Ullrich on November 16, 2018, his rash had resolved, and he had no joint pain or fever. Id. at 23.

On September 27, 2019, petitioner was involved in an accident that resulted in third-degree burns over 53% of his body. Pet. Ex. 37 at 7, 15. Petitioner spent over seventy days at the Jackson Memorial burn unit and underwent at least six graft surgeries. Id.; Tr. 15.

Petitioner continues to see his rheumatologist on a regular basis, every 1-3 months. See Pet. Ex. 38 at 30-43. He is currently taking the medication Ilaris, an IL-1 inhibitor, for his sJIA. Tr. 14, 84.

## **B. Affidavits and Testimony**

### **1. Petitioner**

Petitioner stated he did not have a rash prior to his sports physical in September 2014. Pet. Ex. 9 at ¶ 2; Tr. 8. At his sports physical, his doctor administered HPV and Hep A and vaccinations. Pet. Ex. 9 at ¶ 2; Tr. 9. Petitioner recalled a week after vaccination he noticed a salmon-colored rash, like paint splatter, over his chest, back, buttocks, legs, and arms. Pet. Ex. 9 at ¶ 3; Tr. 9. Petitioner stated there was noticeable raising of the skin, but it did not hurt, so he did not mention the rash to anyone. Pet. Ex. 9 at ¶¶ 3-4; Tr. 9-10. He also stated the rash was persistent in the evening and would fade in the morning. Tr. 9.

After about two weeks with the rash, petitioner's mother noticed the rash and expressed concern. Tr. 11-12. At that point, petitioner's rash became worse and began to itch. Tr. 12. He then started having daily fevers and sore throats. Pet. Ex. 9 at ¶ 5; Tr. 12-13. His sore throat would make it difficult to eat and his fever led to night sweats. Pet. Ex. 9 at ¶ 5; Tr. 12. His joints also began to hurt, and he had muscle aches. Pet. Ex. 9 at ¶ 5; Tr. 12-13. Petitioner's joints would swell and be warm to the touch. Tr. 25. When he tried to move there was a pinching sensation. Id. He stated he was eventually diagnosed with sJIA disease and is currently in treatment. Pet. Ex. 9 at ¶ 6.

Petitioner testified that he does not have any hearing loss, and there is no family history of hearing loss, although his maternal grandfather uses a hearing aid. Tr. 27-28. He is currently taking Ilaris for his sJIA and is symptom free. Tr. 14. Prednisone, a corticosteroid, Actemra, an anti-IL-6 biological, and Kineret,<sup>28</sup> an anti-IL-1 biological, also helped petitioner's symptoms for a while. Tr. 14-15, 83, 172. Prednisone could not be taken for long periods of time and Actemra helped with fevers, joint pain, muscle aches, and sore throat, but not his rash. Tr. 23. Petitioner switched from Kineret to Ilaris after his accident in September 2019. Id.

## **2. Petitioner's Mother – Jennifer Ansaroff**

Ms. Ansaroff, petitioner's mother, stated petitioner had no health problems prior to his September 2014 sports physical. Pet. Ex. 10 at ¶ 2. Petitioner received HPV and Hep A vaccinations at that visit. Tr. 30. In early October, Ms. Ansaroff stated she saw a rash on petitioner's arms, but he told her it was nothing. Pet. Ex. 10 at ¶ 3; Tr. 31-32. Approximately one month later, Ms. Ansaroff noticed petitioner's rash was still there and that his arm was completely covered by the rash. Pet. Ex. 10 at ¶ 4; Tr. 33-34. She called his pediatrician the next morning and made an appointment. Tr. 35.

On December 2, 2014, Ms. Ansaroff brought petitioner to Dr. Cutler and she asked Dr. Cutler what could cause a rash to last two months. Pet. Ex. 10 at ¶ 5; Tr. 41. Dr. Cutler was unsure what was causing petitioner's rash and so he ordered a strep test which was negative. Pet. Ex. 10 at ¶ 5. Petitioner had high fevers, sore throats, and painful body aches, and anytime the fever spiked his rash got worse. Tr. 37-38, 40. As time went by, petitioner's rash became worse and was more noticeable in the evening than in the morning. Tr. 50.

Ms. Ansaroff testified that she does not have hearing loss, but her father uses a hearing aid. Tr. 52.

## **C. Expert Reports and Hearing Testimony**

### **1. Petitioner – Dr. M. Eric Gershwin**

#### **a. Background and Qualifications**

Dr. Gershwin is a Distinguished Professor of Medicine with the University of California, Davis, where he currently holds a chaired professorship in honor of Jack and Donald Chia. Pet. Ex. 15 at 2. Dr. Gershwin received his undergraduate degree, summa cum laude, from Syracuse University and his medical degree from Stanford. Id. He has an honorary doctorate from the University of Athens, in recognition for his lifetime contribution in immunology and medicine. Id. He has also been awarded the AESKU prize in Autoimmunity in 2008, in recognition of his lifetime contribution in immunology. Id. He is also fellow with the American Association for

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<sup>28</sup> Kineret is “a recombinant, nonglycosylated form of the human interleukin-1 receptor antagonist, used as an anti-inflammatory in the treatment of rheumatoid arthritis” and the brand name of anakinra. Anakinra, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=2533> (last visited June 2, 2021).

the Advancement of Science. Id. He is board-certified in internal medicine, rheumatology, and allergy and clinical immunology. Id. at 3.

**b. Opinion**

**i. Diagnosis**

Dr. Gershwin opined that petitioner was correctly diagnosed with adult onset Still's disease, or sJIA. Pet. Ex. 11 at 1; Pet. Ex. 32 at 1; see also supra note 7. At the time of diagnosis, petitioner was seventeen years old; however, petitioner was approximately fifteen years old when the rash began, one week after vaccination. Pet. Ex. 11 at 1. Dr. Gershwin opined petitioner developed juvenile idiopathic arthritis ("JIA") and, "in particular, the Still's variation of JIA." Id.

JIA has different subtypes that are defined based on the number of joints involved in the first six months of disease and the extra-articular involvement. Pet. Ex. 11 at 2. Dr. Gershwin stated the subtypes "include oligoarticular JIA, polyarticular JIA (2:5 joints) and [s]JIA." Id. sJIA<sup>29</sup> is defined by "arthritis with spiking fever persisting for more than 2 weeks and at least one of the following clinical features of systemic inflammation: skin rash, lymphadenopathy, [and] hepatosplenomegaly or serositis (pleuritis or pericarditis)." Id. Dr. Gershwin opined that sJIA has a distinct pathogenesis and immunologic abnormality. Pet. Ex. 11 at 2.

Dr. Gershwin initially opined petitioner suffered from acute MAS. Pet. Ex. 11 at 2. According to Dr. Gershwin, immunologically, sJIA is a form of MAS. Id. On the spectrum of MAS, macrophages become dysregulated and over-produce pro-inflammatory cytokines. Tr. 63. Dr. Gershwin stated none of petitioner's physicians felt he was suffering from an acute MAS, however, "to understand [sJIA], one has to understand the spectrum of macrophage activation." Pet. Ex. 11 at 3. While Dr. Gershwin initially suggested that petitioner suffered from MAS, he later retreated from that position. He opined that petitioner did not meet the criteria for MAS, but that he did have aberrant macrophage activation. Tr. 107-08.

Dr. Gershwin disagreed with Dr. Rosé's suggestion that petitioner's diagnosis could be a form of CAPS. First, Dr. Gershwin stated CAPS patients would have an abnormality in a serum protein electrophoresis. Tr. 80. He explained in CAPS, patients would have an IgM monoclonal antibody<sup>30</sup> or monoclonal IgM dysplasia. Id. In petitioner's case, these abnormalities were not present. Id. In July 2016, petitioner's doctors ordered serum protein electrophoresis and no aberrant proteins were seen in those results. Id.

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<sup>29</sup> Dr. Gershwin also stated that sJIA is a generic term that could be used to describe both adult onset and pediatric onset Still's disease. Tr. 102. However, petitioner has adult onset Still's disease. Id.

<sup>30</sup> On January 7, 2015, petitioner had a positive IgG test for parvovirus, but tested negative for IgM. Pet. Ex. 1 at 125-30.

Second, Dr. Gershwin explained that while headaches can also be symptomatic of CAPS, the nature of petitioner's headaches were not consistent with what is usually seen in patients with CAPS. Tr. 93. Sterile meningitis, which may occur as a symptom of CAPS, produces chronic, unremitting, and painful headaches. Tr. 94. Petitioner's headaches would last for a short duration and were not associated with nausea, vomiting, loss of vision or hearing, and he had no motor, sensory, or gait problems. Tr. 95. Additionally, petitioner's EEG was normal. Tr. 96. Therefore, Dr. Gershwin did not find petitioner's headaches to be consistent with CAPS-type meningitis headaches. Id.

Third, Dr. Gershwin characterized petitioner's rash as lymphocytic in nature. Tr. 92. Lymphocytic rashes are characteristic of sJIA, whereas CAPS rashes are predominantly neutrophilic. Id. Since petitioner's rash improved with steroids, this suggests petitioner's rash was lymphocytic rather than neutrophilic. Id.

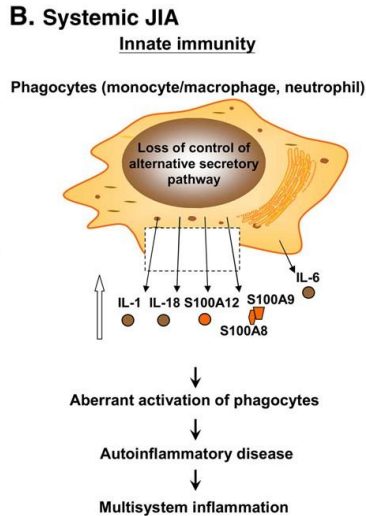
Fourth, Dr. Gershwin stated that hearing loss, recurrent noninfectious eye inflammation, and skeletal abnormalities, which are all classical symptoms of CAPS, were not present in petitioner's case. Tr. 96-97. Dr. Gershwin agreed with petitioner's doctor that petitioner's "presentation [was] quite classical for [sJIA]" and there was not "any possibility" that petitioner had CAPS. Tr. 82, 97.

## ii. Althen Prong One: Medical Theory of Causation

sJIA is an inflammatory disease with an abnormality of the innate immune system that causes the body to over-produce cytokines. Tr. 60-61, 64. Dr. Gershwin explained that macrophages also become overly activated and dysregulated as part of the disease process and begin producing cytokines in excess. Tr. 63. As a consequence of significant over-production of pro-inflammatory cytokines, lymphocytic infiltrates may appear on the skin, fever occurs, enlargement of the liver or spleen may occur, as well as significant arthritis. Tr. 63-64.

Dr. Gershwin opined that genetic susceptibility and environmental factors both play an important role in the cause of sJIA. Pet. Ex. 11 at 3-4; Tr. 65. Angles-Han and Prahalad provide, "[t]he subtypes of JIA share genetic and phenotypic features with other autoimmune disorders, which are believed to result from the interplay of genetic and environmental factors." Pet. Ex. 30 at 1; see also Tr. 66-67. The article stated that "[a]lthough JIA is believed to be influenced by genetic and environmental factors, twin and family studies strongly support a substantial role for genetic factors in JIA susceptibility." Pet. Ex. 30 at 1. The prevalence of sJIA in siblings is "15- to 30-fold greater than that of the general population." Id. sJIA is associated with certain genes encoding for specific cytokines/chemokines, in particular, a single nucleotide polymorphism within the region of the IL-6 gene. Pet. Ex. 11 at 3-4 (citing Pet. Ex. 29). The Ogilvie article provided that a dominant environmental etiology for sJIA has not been discovered; however, the IL-6-174 nucleotide variant is significantly associated with sJIA. Pet. Ex. 29 at 1.

The causal theory proposed by Dr. Gershwin implicates the innate immune system and is described in Lin et al. in the figure below.



Pet. Ex. 31 at 2 fig.1-B.

The above diagram (from an article co-authored by Dr. Gershwin) illustrates the causal mechanism proposed by Dr. Gershwin. sJIA results from a “loss of control of the alternative secretory pathway leading to aberrant activation of phagocytes including monocytes, macrophages, and neutrophils seems to be involved in the release of pro-inflammatory cytokines IL-1, IL-6, IL-18 and pro-inflammatory S100-proteins.” Pet. Ex. 31 at 2 fig.1. Therefore, in a genetically susceptible host, abnormal activation of mononuclear cells leads directly to the production of pro-inflammatory cytokines. Pet. Ex. 11 at 4; Tr. 58, 71. These cytokines produce inflammation, which can lead to the fever, the skin manifestations, and the rheumatic manifestations of sJIA. Pet. Ex. 11 at 4.

Immunologically, Dr. Gershwin opined that petitioner’s sJIA is a form of MAS. Pet. Ex. 11 at 2. In cases of sJIA, MAS is not a distinct disorder, but comprises one end of a spectrum of disease activity. Id. at 3 (citing Pet. Ex. 19).<sup>31</sup> Shimizu and Yachie investigated the role of alternative macrophage activation in sJIA. Pet. Ex. 36 at 1.<sup>32</sup> Abnormalities involved in macrophage regulation and function are apparent in patients suffering from sJIA in both the active and inactive states. Id. at 4. The inactive phase of sJIA may represent “a state of compensated inflammation rather than an absence of immune activity.” Id. at 5. Inadequate down-regulation of immune activation might be central to sJIA. Id. They also “suggest that macrophage activation may be integral to the pathogenesis of sJIA.” Id.

According to Dr. Gershwin, persons with sJIA have a genetic abnormality which predisposes them to have dysregulated activation of their macrophages. Tr. 68. When

<sup>31</sup> Edward M. Behrens et al., Occult Macrophage Activation Syndrome in Patients with Systemic Juvenile Idiopathic Arthritis, 34 J. Rheumatology 1133 (2007).

<sup>32</sup> Masaki Shimizu & Akihiro Yachie, Compensated Inflammation in Systemic Juvenile Idiopathic Arthritis: Role of Alternatively Activated Macrophages, 60 Cytokine 226 (2012).



influenced by cytokines, like those produced by the vaccines, their macrophages can become dysregulated, leading to aberrant cytokines. Tr. 70-71. This is especially true for the HPV vaccine, because of its ability to upregulate cytokines. Tr. 70; 120-21; see also Pet. Ex. 45 at 1.<sup>33</sup> Dr. Gershwin opined that the Hep A vaccine also produces cytokines, but not as intensely as the HPV vaccine. Tr. 120.

In support of his theory, Dr. Gershwin, cited Pinto et al., which reported significant increases in inflammatory cytokines following the HPV vaccination. Pet. Ex. 45 at 1. Pinto evaluated the innate and adaptive immune systems' cytokine responses induced by HPV-16 L1 virus-like particles ("VLP") in whole blood cultures from individuals receiving the vaccine or placebo before and after vaccination. Id. The study found the HPV vaccine activated both the innate and the adaptive immune systems and stimulated a broad spectrum of cytokine production, including IL-6 (26- and 11-fold at months 2 and 7). Id. at 5. Evans et al.<sup>34</sup> also performed a similar study to measure the immune response from HPV vaccination in healthy adult volunteers. Pet. Ex. 43 at 1. Evans concluded "that HPV 11 VLP vaccines are highly immunogenic and induce brisk B cell and T cell responses." Id. at 20. Thus, Dr. Gershwin opined the cytokine release from an environmental trigger, such as the HPV vaccine, is sufficient to activate a genetically predisposed macrophage. Tr. 121.

Dr. Gershwin opined sJIA is too uncommon to be detectable by current epidemiology. Pet. Ex. 32 at 1. Besides one case report, Korematsu,<sup>35</sup> detailing an sJIA relapse following rubella vaccination, Dr. Gershwin agreed there are no other case reports of sJIA following vaccination. Tr. 120; see Resp. Ex. G at 1.

Dr. Gershwin also stated that biologics that are IL-1 and IL-6 inhibitors are consistent with the treatment of sJIA. Tr. 83-84.

### iii. Althen Prong Two: Logical Sequence of Events

Dr. Gershwin opined that the petitioner was a genetically susceptible host, and the environmental stimulus, the HPV vaccination, was the precipitating factor that activated his

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<sup>33</sup> Ligia A. Pinto et al., HPV-16 L 1 VLP Vaccine Elicits a Broad-Spectrum of Cytokine Responses in Whole Blood, 23 Vaccine 3555 (2005).

<sup>34</sup> Thomas G. Evans et al., A Phase 1 Study of a Recombinant Viruslike Particle Vaccine Against Human Papillomavirus Type 11 in Healthy Adult Volunteers, 183 J. Infectious Diseases 1485 (2001).

<sup>35</sup> Seigo Korematsu et al., A Relapse of Systemic Type Juvenile Idiopathic Arthritis After a Rubella Vaccination in a Patient During a Long-Term Remission Period, 27 Vaccine 5041 (2009).

innate immune system and led to prolonged innate immune dysregulation.<sup>36</sup> Pet. Ex. 11 at 4. As a result, petitioner suffers from sJIA. Id.

Due to petitioner's rare genetic predisposition, the cytokines produced by the vaccinations activated dysregulated monocytes, precipitating his disease. Tr. 73. In petitioner, this led to "unremitting, severe systemic inflammation." Tr. 69. As evidence of markers of increased inflammation, Dr. Gershwin pointed to the medical records. Tr. 137. Petitioner had an elevated erythrocyte sedimentation rate and an elevated C-reactive protein. Id. His platelet count increased from the 300,000s to almost 500,000. Tr. 138; Pet. Ex. 3 at 168. Additionally, petitioner had some toxic granulations<sup>37</sup> and elevated ferritin<sup>38</sup> levels. Tr. 139-40; Pet. Ex. 1 at 66-68. Dr. Gershwin explained that the "HPV vaccine produced a normal cytokine response, but [petitioner's] macrophages are [] abnormal." Tr. 276.

Petitioner's initial clinical manifestation of sJIA, his rash, began one week following his vaccination. Pet. Ex. 32 at 2. As petitioner's condition progressed, other clinical manifestations of sJIA appeared: his rash was pinkish and worsened in the late afternoon and evening, he had high, unremitting fevers, sore throat, and significant pain and inflammation of the joints. Tr. 61-62. Petitioner also had enlargement of his spleen and liver, which is consistent with sJIA. Tr. 62. Further, Dr. Gershwin explained that sJIA has two typical "peaks" for age of onset. Tr. 87. The first is from age 15-25 and the second is age 35-45. Id. Petitioner's onset was at age fifteen. Id.

During the entitlement hearing, Dr. Gershwin conceded that petitioner did not meet the criteria for MAS and did not have that syndrome. Tr. 107-08. However, he stated petitioner did have aberrant macrophage activation, which is "found in virtually every patient with [sJIA]." Id. Dr. Gershwin stated 50% of patients with systemic onset disease have evidence of macrophage activation seen in their bone marrow. Tr. 107. Petitioner's bone marrow aspiration did not reveal this evidence. Tr. 147; Pet. Ex. 6 at 32. However, the bone marrow aspiration was searching for leukemia cells and did not look at the activation of macrophages. Tr. 146-47. Dr. Gershwin stated this did not change his opinion. Tr. 143.

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<sup>36</sup> During the hearing, Dr. Gershwin stated, "my opinions are predominantly based on HPV. . . . [I]n theory, [the Hep] A vaccine also produces cytokines," however, "[t]hey're not as intense as HPV." Tr. 120.

<sup>37</sup> Granulation is any granular material on the surface of a tissue, membrane, or organ. Granulation, Dorland's Med. Dictionary Online, <https://www.dorlandsonline.com/dorland/definition?id=20919> (last visited June 2, 2021).

<sup>38</sup> "Ferritin is a ubiquitous and specialized protein involved in the intracellular storage of iron." Pet. Ex. 17 at 1 (Stefania Recalcati et al., New Functions for an Iron Storage Protein: The Role of Ferritin in Immunity and Autoimmunity, 30 J. Autoimmunity 84 (2008)). Additionally, "macrophages play a role in the production and secretion of extracellular ferritin." Id. "[F]erritin acts as an immuno-suppressor" and has a possible role in the pathogenesis of autoimmune diseases. Id.

Lastly, Dr. Gershwin opined that there were no other environmental factors that led to petitioner's immune system activation. Pet. Ex. 11 at 4. Petitioner's extensive medical workups demonstrated that he did not have any causal viral infections, bacterial infections, or other environmental or chemical exposures. Id. Thus, Dr. Gershwin concluded that the most likely and plausible event that led to petitioner's immune system activation was his vaccines. Id.

**iv. Althen Prong Three: Proximate Temporal Relationship**

Dr. Gershwin opined that while the onset of petitioner's illness began within hours of his vaccinations, the clinical manifestations of the disease was the rash, which began approximately one week after vaccination. Tr. 141; Pet. Ex. 11 at 1. Dr. Gershwin testified that the onset of the rash was appropriate given the response of the innate immune system to vaccination. Tr. 72. "Although temporal association is not, by itself; evidence for causation, the onset of the rash within a week of the vaccination would be very consistent with immune activation secondary to the HPV vaccine." Pet. Ex. 11 at 4.

Following the onset of rash, there was subsequent progression to fever, myalgias, arthralgias, and fatigue. Pet. Ex. 32 at 2. These symptoms and their time course were diagnostic of JIA and reflective of the very high pro-inflammatory markers that one would expect of sJIA. Id.

In support of his opinion as to the time frame for appropriate onset, Dr. Gershwin cited the Hervé<sup>39</sup> and Herrin<sup>40</sup> articles. Herve reports that after injection of a vaccine that contains an adjuvant, chemokines and cytokines can be detected in animal muscle within three hours. Pet. Ex. 46 at 2. They return to baseline at 72 hours. Id. The "products of inflammation at the localised site (vaccine injection site) . . . may spill into the circulation . . . causing systemic side-effects." Id. at 3. Vaccines containing adjuvants induce "transient systemic innate responses, including IL-6 and C-reactive protein (CRP), mostly peaking at 24 h[ours] post administration and subsiding to baseline within 1 to 3 days." Id.

Specific as to the HPV vaccine, the authors of Herrin evaluated and compared circulating chemokine and cytokine responses after administration of two HPV vaccines (Cervarix and Gardasil). Pet. Ex. 48 at 1. After Gardasil vaccination, there was an increase in certain chemokines and cytokines after the first vaccination that peaked at five days and extended to 14 days. Id. at 7.

**2. Respondent – Dr. Carlos D. Rosé**

**a. Background and Qualifications**

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<sup>39</sup> Caroline Hervé et al., The How's and What's of Vaccine Reactogenicity, 4 Nature Partner J. Vaccines 1 (2019).

<sup>40</sup> Douglas M. Herrin et al., Comparison of Adaptive and Innate Immune Responses Induced by Licensed Vaccines for Human Papillomavirus, 10 Hum. Vaccines & Immunotherapeutics 3446 (2014).

Dr. Rosé graduated in 1977 from the University of Buenos Aires School of Medicine in Argentina, completing his residency in internal medicine at the University's hospital. Resp. Ex. B at 1. He held an adult rheumatology fellowship in the National Institute of Rehabilitation, Department of Medicine, Rheumatology Division, Buenos Aires. Id. He also completed a pediatric residency at Thomas Jefferson University in Philadelphia, Pennsylvania, which was followed by a fellowship in pediatric rheumatology at Children's Hospital of Philadelphia. Id. at 5. Dr. Rosé is board-certified in pediatrics as well as pediatric and adult rheumatology. Id. at 3-4. Dr. Rosé has been treating rheumatology patients for over forty years. Resp. Ex. A at 1. Since 1989, he has practiced at the DuPont Hospital for Children in Wilmington, Delaware. Id.

## **b. Opinion**

### **i. Diagnosis**

Dr. Rosé opined primarily as to petitioner's diagnosis of sJIA.

Dr. Rosé characterized petitioner's condition as "a rash with systemic features rather than a systemic disease with rash." Resp. Ex. A at 9. At the hearing, Dr. Rosé initially testified that petitioner's diagnosis of sJIA was unlikely given the clinical presentation. Tr. 159. However, he later clarified his opinion as follows: "So more likely than not—this is atypical enough for systemic JIA not to consider [MWS] and not to test. That's as far as I go." Tr. 191.

Instead of sJIA, Dr. Rosé questioned whether petitioner's diagnosis may be a variant of CAPS, known as MWS. Id.

To illustrate the issues with petitioner's diagnosis, Dr. Rosé cited De Benedetti and Schneider's ILAR Diagnostic Criteria for sJIA. Tr. 162 (citing Resp. Ex. A, Tab A1 at 2 Box16-1). The criteria stated sJIA is diagnosed with "arthritis in any number of joints together with a fever of at least two weeks' duration that is documented to be daily for at least three days, and is accompanied by one or more of the following: Evanescent rash, generalized lymphadenopathy, enlargement of liver or spleen, serositis." Resp. Ex. A, Tab A1 at 2 Box16-1. Dr. Rosé conceded that petitioner met the ILAR criteria for sJIA. Tr. 163. However, Dr. Rosé stated the diagnosis of sJIA is a "diagnosis of exclusion." Tr. 164-65; see also Resp. Ex. A, Tab A1 at 6. To confirm the diagnosis, Dr. Rosé stated that other diseases, like MWS, should be ruled out. Tr. 164.

Dr. Rosé opined that petitioner's rash/neutrophilic urticaria was not typical of sJIA. Resp. Ex. A at 10. First, he opined that petitioner's rash was not "evanescent." Tr. 162-63. The typical sJIA is a salmon-colored rash, which is "streaky," and found in the distal portion of extremities. Resp. Ex. A at 10. In contrast, he opined that petitioner's rash was often described as circular and popular.<sup>41</sup> Id.

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<sup>41</sup> After the hearing, petitioner submitted photographs of his rash. See Pet. Exs. 49-51. Subsequently, Dr. Rosé submitted a supplemental report wherein he opined that the rash shown

Second, Dr. Rosé opined that petitioner’s rash was neutrophilic, as supported by the two punch biopsies, and not lymphocytic as Dr. Gershwin asserted. Tr. 169. Dr. Rosé opined that neutrophilic urticaria is not typical of sJIA. Id. Dr. Rosé also mentioned the fact that petitioner needed skin biopsies was unusual for a typical sJIA rash. Tr. 168.

Dr. Rosé next explained that traditional sJIA rash tends to fluctuate with fever and resolve with treatment and seldom required corticosteroid therapy. Tr. 167. He opined that it was unusual for the rash to precede the fever by more than a few days. Id. Dr. Rosé testified that he was not sure when petitioner’s rash began, but it could have been October, mid-October, or November 1. Id. He opined that petitioner’s rash preceded his fevers by approximately four to six weeks, which he characterized as unusual. Id. He issued a caveat in that there were times that petitioner’s rash was treated with corticosteroids. Resp. Ex. A at 10; Tr. 167.

Dr. Rosé found what he described as petitioner’s daily headaches atypical of sJIA. Resp. Ex. A at 10-11; Tr. 173. Headaches are classically seen in CAPS. See Resp. Ex. A at 11. Further, petitioner was never tested to see if he had low-grade meningitis.<sup>42</sup> Tr. 174. Therefore, he questioned whether petitioner’s headaches could have been CAPS related. Id.

Dr. Rosé also found petitioner’s “transient” arthritis to be atypical for sJIA. Resp. Ex. A at 9. He opined that in sJIA, the arthritis may “take a while to show up,” and it “may be intermittent in the beginning,” but once it starts, it is chronic. Tr. 170-71. Petitioner had bouts of intense inflammatory disease of the joints, lasting short periods of time. Tr. 170. Dr. Rosé was surprised that petitioner did not have some permanent limitations of his joints after six years of disease. Tr. 171.

Dr. Rosé stated petitioner’s arthritis was “easily controlled with Actemra (Tocilizumab), a specific IL-6 blocker in 2016-2017,” but that his rash remained. Resp. Ex. A at 10-11. According to Dr. Rosé, after six months on Actemra, petitioner’s rash should have resolved. Tr. 178. He opined that this meant that petitioner had an incomplete response to Actemra. Resp. Ex. A at 11. During the hearing, Dr. Rosé stated petitioner “failed IL-6” treatment but responded well to Ilaris, which is an IL-1 cytokine inhibitor. Id.; Tr. 178. Actemra only inhibits IL-6 cytokines. Tr. 172. Dr. Rosé found this unusual and suggested that petitioner’s disease is predominantly IL-1-mediated. Id.

Dr. Rosé opined that CAPS, specifically the MWS phenotype, captures petitioner’s clinical presentation, evolution, and incomplete response to IL-6 inhibition better than sJIA.

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in the photographs was “compatible with neutrophilic urticaria.” Resp. Ex. H at 4. He also opined that the appearance and the “severity and histologic features” support his belief that petitioner has a genetic illness rather than sJIA. Id.

<sup>42</sup> At the hearing, Dr. Gershwin stated that meningitis, which occurs in CAPS, can produce chronic, unremitting, and painful headaches. Tr. 94. However, petitioner’s headaches would last for a short duration and were not associated with nausea, vomiting, loss of vision or hearing, and he had no motor, sensory, or gait problems. Tr. 95.

Resp. Ex. A at 11; Tr. 179. In addition, Ilaris is used to treat MWS, and petitioner has responded well to it. Tr. 186.

CAPS is a single point mutation of the NALP3 gene which can lead to spontaneous self-activation and activation of Caspase-1, which in turn releases IL-1 $\beta$  cytokines into the blood stream. Resp. Ex. A at 11-12 (citing Resp. Ex. A, Tab A5 at 1);<sup>43</sup> Tr. 181. This causes fever, chills, sweats, transient arthritis, neutrophilic urticaria, headaches, and hepatosplenomegaly. Id. at 12 (citing Resp. Ex. A, Tab A6 at 1). Dr. Rosé opined that MWS is often confused with sJIA due to their similar presentations. Tr. 181. Genetic testing is required to confirm the diagnosis. Tr. 183, 187.

The CAPS diagnostic criteria established by Kuemmerle-Deschner et al. consists of “amyloidosis, recurrent episodes of systemic symptoms, urticaria-like rash, chronic aseptic meningitis, recurrent eye inflammation, sensorineural hearing loss, musculoskeletal signs and symptoms, [and] skeletal abnormalities.” Resp. Ex. A, Tab A3 at 4 tbl.1; see also Tr. 182. Dr. Rosé conceded that over the last six years, petitioner’s physicians have not documented any recurrent, noninfectious eye inflammation, hearing loss, skeletal abnormalities, or chronic aseptic meningitis. Tr. 209-10. Dr. Rosé clarified, however, that hearing loss can appear later in life. Tr. 215.

Of note, Dr. Rosé suggested that the possibility of an autoinflammatory periodic syndrome was not lost on the petitioner’s treating physicians. Resp. Ex. A at 14; Tr. 189-90. Drs. Sigua and Weatherall ran limited genetic tests, though did not run tests for CAPS genetic abnormalities. Resp. Ex. A at 14. Dr. Rosé also thought that the finding of neutrophilic urticaria on biopsy was unusual in sJIA. Id.; Tr. 189-90.

In response to Dr. Gershwin’s expert reports and hearing testimony, Dr. Rosé agreed with Dr. Gershwin there is no question that petitioner’s macrophages were activated. Tr. 195. He attributed this to either a “mutation on the IL-3 gene” or his “[s]JIA.” Id. Further, Dr. Rosé agreed that there is a “strong association” between sJIA and MAS, and that many of the laboratory abnormalities are common to both conditions. Resp. Ex. A at 6, 16. Dr. Rosé opined that petitioner was, however, “never sick enough to be considered to have” macrophage activation syndrome. Id. at 15-16; see also Tr. 195-96. However, he did agree that there was an “overlap of some of the biological mechanism.” Resp. Ex. A at 16.

### **3. Respondent – Dr. Craig D. Platt**

#### **a. Background and Qualifications**

Dr. Platt is a clinical immunologist with board certification in Allergy and Clinical Immunology working at Boston Children’s Hospital. Resp. Ex. C at 1. He earned his M.D. and Ph.D. in Immunobiology from the Yale School of Medicine. Resp. Ex. D at 1. Dr. Platt

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<sup>43</sup> Hal M. Hoffman et al., Mutation of a New Gene Encoding Putative Pyrin-Like Protein Causes Familial Cold Autoinflammatory Syndrome and Muckle-Wells Syndrome, 29 Nature Genetics 301 (2001).

performed residency training in Pediatrics and fellowship training in Allergy/Immunology at Boston Children's Hospital. Resp. Ex. C at 1. His Ph.D. research was on the cellular biology of dendritic cells, which are required for the initiation of adaptive immune responses. Id. His current research focuses on the genetics of immunodeficiency and immune dysregulation syndromes. Id. Dr. Platt has co-authored 20 peer reviewed articles, three book chapters, and has authored an article on vaccine use in patients with immunodeficiency disorders for UpToDate. Id.

**b. Opinion**

**i. Diagnosis**

Dr. Platt did not offer an opinion regarding the proper diagnosis of petitioner's condition. He deferred to Dr. Rosé on that issue. Resp. Ex. C at 2; Tr. 225.

**ii. Althen Prong One: Medical Theory of Causation**

Although Dr. Platt did not offer an opinion as to the petitioner's proper diagnosis, he did opine that CAPS is due to a genetic defect, and is not a condition that can be caused by vaccination. Tr. 226.

With respect to sJIA, Dr. Platt disagreed with Dr. Gershwin's contention that vaccinations could cause sJIA. Resp. Ex. C at 3; Tr. 226. Specifically, Dr. Platt disagreed that sJIA could be triggered by an upregulation of cytokines post-vaccination. Tr. 226. He stated that there is no consensus in the medical community that vaccines can cause sJIA. Id. He further testified that the mechanism postulated by Dr. Gershwin is overly broad and could apply to anything that activates the immune system. Tr. 226, 231.

Dr. Platt did agree that cytokines are involved in the pathology of sJIA. Tr. 229-30. However, he did not agree that vaccines could serve as a trigger for the mechanism. Id. Dr. Platt also agreed that it was "reasonable to postulate, as Dr. Gershwin does, that there is a genetic basis" for the illness. Tr. 231.

Dr. Platt agreed with Dr. Gershwin that sJIA is systemic inflammatory disorder characterized by fevers and a variety of other systemic manifestations. Tr. 227. But he did not agree with Dr. Gershwin's statement that there is a consensus sJIA is virus induced. Pet. Ex. 11 at 3; Tr. 228. Dr. Platt emphasized that in most cases, the cause of sJIA is never determined. Tr. 228.

In support of his opinions, Dr. Platt cited the article by Berkun and Padeh.<sup>44</sup> They postulated that viral agents are one potential trigger for sJIA, but additionally they noted bacterial infection, genetic factors, stress and psychological factors, maternal smoking, and weather changes are also proposed triggers. Resp. Ex. C, Tab C1 at 1; see also Tr. 228, 235-36.

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<sup>44</sup> Yackov Berkun & Shai Padeh, Environmental Factors and the Geoeidemiology of Juvenile Idiopathic Arthritis, 9 Autoimmunity Rev. A319 (2009).

Berkun and Padeh concluded that while the role of viral infections has been questioned, their pathogenicity has not been proven. Resp. Ex. C, Tab C1 at 3.

Even if viral infection was a well-established cause of sJIA, Dr. Platt did not agree that the HPV and Hep A vaccines were potential triggers. Resp. Ex. C at 3. He explained that the HPV vaccine is a recombinant vaccine and not a live infectious agent. *Id.* Dr. Platt also explained that while it is generally accurate to say cytokines produce inflammation, which can lead to the fever, skin manifestations, and the rheumatic manifestations of sJIA, there is no evidence that the cytokines produced are the trigger for sJIA. *Id.* at 4; Tr. 229. He stated, “[i]mportantly, there is no data supporting a causal relationship between vaccines and JIA.” Resp. Ex. C at 3. He also cited Lin et al. (also cited by petitioner), which stated that “there is no evidence that vaccination is associated with the onset or exacerbation of oligo/polyarticular JIA so far.”<sup>45</sup> Pet. Ex. 31 at 3. According to Dr. Platt, the Lin article does not “shed any light at all on the pathogenesis or . . . the etiology or initial trigger” of sJIA. Tr. 230. That is, Dr. Platt did not agree that the article suggests that initial production of IL-1 cytokines can cause aberrant activation of phagocytes and autoinflammatory disease. *See id.* Overall, Dr. Platt asserted that it is unclear what causes the abnormal activation of phagocytes (macrophages), though a genetic predisposition, as postulated by Dr. Gershwin, is reasonable. Tr. 230-31.

Dr. Platt testified about the lack of epidemiological evidence showing an association between vaccination and sJIA. Resp. Ex. C at 4. He stated that “it is notable that Dr. Gershwin fails to mention that a number of epidemiological studies have been conducted and have concluded that there is not a relationship between certain vaccines and the development of JIA.” *Id.*; *see also* Tr. 232. Dr. Platt cited several studies that show a lack of relationship between several vaccines and sJIA. Resp. Ex. C, Tab C6;<sup>46</sup> Resp. Ex. C, Tab C7;<sup>47</sup> Resp. Ex. C, Tab C8.<sup>48</sup> However, Dr. Platt agreed with Dr. Gershwin that it is difficult to use epidemiology to determine whether vaccines can cause sJIA. Tr. 233.

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<sup>45</sup> This citation is somewhat misleading, as the reference was pulled from the section in the article that discussed oligo/polyarticular JIA, not sJIA. In the section about triggering factors in sJIA, the authors reference vaccination, and state “[o]nly a single case of an exacerbation of systemic JIA following live-attenuated rubella vaccination has been reported.” Pet. Ex. 31 at 6. The authors also emphasize the “markedly distinct pathogenesis” of the two conditions. *Id.*

<sup>46</sup> Marloes W. Heijstek et al., Safety of Measles, Mumps, and Rubella Vaccination in Juvenile Idiopathic Arthritis, 66 *Annals Rheumatic Disease* 1384 (2007) (finding no aggravation of sJIA within a six-month period after measles, mumps, and rubella vaccinations).

<sup>47</sup> Ö. Kasapçopur et al., Hepatitis B Vaccination in Children with Juvenile Idiopathic Arthritis, 63 *Annals Rheumatic Disease* 1128 (2004) (finding only one child out of 39 children developed an antibody response to Hepatitis B vaccination).

<sup>48</sup> Evelien Zonneveld-Huijssoon et al., Safety and Efficacy of Meningococcal C Vaccination in Juvenile Idiopathic Arthritis, 56 *Arthritis Rheumatology* 639 (2007) (concluding that meningococcal vaccination did not aggravate JIA in 234 patients).



Dr Platt next discussed the HPV vaccine, Gardasil, insert filed by petitioner. Pet. Ex. 42 at 8. The insert stated that 2.2% (351/15,703) of GARDASIL 9 recipients and 3.3% (240/7,378) of GARDASIL recipients reported new medical conditions potentially indicative of systemic autoimmune disorders. Tr. 236-37. This result was similar to rates reported following controls or placebos in clinical trials. Id. Dr. Platt opined this shows that patients may develop rare autoimmune disorders and immune dysregulation disorders regardless of whether or not they receive a vaccination. Tr. 237. Instead of a causal relationship, Dr. Platt opined that the development of autoimmunity in the follow up period after vaccination represents coincidence rather than causation. Resp. Ex. C at 5.

### iii. Althen Prong Two: Logical Sequence of Events

Dr. Platt opined that while the model of immune activation in the pathogenesis of rheumatologic disease postulated by Dr. Gershwin is generally accurate, he did not believe it was connected to any of the relevant facts in petitioner's case. Resp. Ex. C at 4. Dr. Platt testified that Dr. Gershwin's theory invokes the adaptive immune system, not the innate immune system. Tr. 248-49. Overall, Dr. Platt opined that Dr. Gershwin's mechanism was overly broad and not consistent with the facts in petitioner's case. Id.

Dr. Platt agreed that the cytokine IL-6 was involved with the pathology of sJIA, but he disagreed that there was any proof that the "initial production of IL-6 from the vaccine was involved in setting off any kind of reprogramming or anything along those lines" Tr. 245. He also agreed that vaccination caused a transient upregulation of cytokines, but disagreed that there was any fever or evidence of "cytokine storm" after vaccination.<sup>49</sup> Tr. 246-47. If there was an aberrant cytokine production described by Dr. Gershwin's mechanism, Dr. Platt would have expected petitioner to have had a fever within hours of the vaccination. Tr. 247.

Regarding alternative causes, Dr. Platt stated that it is impossible to know whether petitioner did not have a viral or bacterial infection in the weeks prior to his disease onset. Resp. Ex. C at 5; Tr. 246.

Dr. Platt cited Lin et al. to establish that there is no single precipitating cause that has been identified in sJIA. Resp. Ex. C at 6. Per Lin et al., "[n]ot one single triggering factor is responsible for the onset or exacerbation of JIA. The causal relation between these triggering factors and JIA pathogenesis remains not well proven and further studies are needed." Resp. Ex. C, Tab C5 at 3. Therefore, Dr. Platt asserted that the absence of other potential triggers does not imply that vaccination must be responsible simply because of the presence of a biologically plausible timeframe. Resp. Ex. C at 6. In most cases of sJIA, no single trigger is thought to be responsible. Tr. 228.

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<sup>49</sup> In Dr. Gershwin's supplemental expert report, he stated that "[a] vaccination is a potent environmental stimulus that activates mononuclear cells and based on [petitioner's] genetic predisposition, would have led to vaccine-induced abnormal monocyte regulatory elements and thence a pro-inflammatory cytokine storm." Pet. Ex. 32 at 2. Dr. Gershwin also testified MAS is like a cytokine storm. Tr. 63, 108.

Further, Dr. Platt noted that none of petitioner's treating physicians attributed his condition to vaccination. Tr. 246.

Dr. Platt agreed that rashes can be a reaction to a vaccination. Tr. 247. He testified that "the most common reaction . . . [is] hives developing after a vaccine." Id. But he disagreed that petitioner's rash was evidence of vaccine reaction. Id. He testified that rashes are common reactions and occur for all kinds of reasons. Id. Also, Dr. Platt opined that petitioner's rash was not immediate after vaccination. Id. Thus, he opined that petitioner's rash was an "early sign" of petitioner's diagnosis, but not related to the vaccine. Tr. 248.

#### iv. Althen Prong Three: Proximate Temporal Relationship

Dr. Platt opined that it did not matter when onset occurred because he does not consider timing alone when analyzing the issue of vaccine causation. Tr. 252. He opined that petitioner's rash one week after vaccination does not support a speculative mechanism with no precedent in the medical literature. Resp. Ex. C at 6. Further, he opined that if petitioner did have elevated cytokines in the blood from vaccination, it would be expected that petitioner would have a fever within 72 hours, not a rash one week later. Tr. 251.

As discussed above, Dr. Gershwin cited the Pinto article to illustrate the occurrence of inflammatory cytokines post-vaccination. Pet. Ex. 45 at 1. However, Dr. Platt opined that because Pinto was an in vitro study, it did not address whether there were elevated cytokines at more distant time points. Tr. 250-51. Further, he opined that cytokine response would be expected to return to baseline within 72 hours. Tr. 251. Therefore, Dr. Platt did not find there to be a plausible temporal association between the HPV vaccination and the rash one week later. Resp. Ex. C at 6.

## VI. DISCUSSION

### A. Diagnosis

As Federal Circuit precedent establishes, in certain cases it is appropriate to determine the nature of an injury before engaging in the Althen analysis. Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1346 (Fed. Cir. 2010). Since "each prong of the Althen test is decided relative to the injury[.]" determining facts relating to the claimed injury can be significant in a case like this, where petitioner has an evolving course of symptoms, resulting in a diagnosis of sJIA. Id. Thus, before determining if petitioner has met each prong of Althen, the undersigned addresses whether he has established, by a preponderance of the evidence, that he suffers from sJIA.

The undersigned finds that petitioner has proven by preponderant evidence that his correct diagnosis is sJIA. There are three reasons for this finding.

First, Dr. Gershwin opined that petitioner's correct diagnosis was sJIA. And Dr. Rosé conceded that petitioner met the ILAR criteria for the illness. At the hearing, Dr. Rosé testified, "[s]o there is no question that [petitioner] did meet the criteria for [s]JIA by having the first two,

arthritis with a fever, and at least one, to be absolutely certain, enlarged liver and spleen.” Tr. 163.

Dr. Rosé’s position was not that petitioner did not meet the diagnostic criteria for sJIA, but that other conditions, such as CAPS/MWS, had not been properly excluded, which to his mind was necessary. However, Dr. Rosé did not opine that, more likely than not, petitioner had CAPS/MWS. Instead, he testified that these alternative diagnoses should be considered. An opinion based on a recommendation that one consider an alternative diagnosis does not reach the evidentiary standard of preponderance.

The second reason the undersigned finds that petitioner has proven that sJIA is the proper diagnosis is that sJIA was the diagnosis given to his condition by his treating physicians. Petitioner underwent thorough rheumatological and infectious disease evaluations at Miami Children’s in December 2014. At that point he was diagnosed with possible sJIA. When petitioner continued to have flares, he saw a number of different specialists. Ultimately, after seeing several physicians and having many diagnostic studies in July and August 2016, Dr. Ullrich and Dr. Goodman diagnosed petitioner with sJIA. Since that time, sJIA has been petitioner’s diagnosis.

During the course of petitioner’s extensive diagnostic workup, a number of conditions were excluded. Rapid flu and strep tests were negative. Throat cultures were negative and a mono spot test for Epstein-Barr virus was also negative. Petitioner tested negative for ASO titer, Bartonella, CMV, rheumatoid factor, and rotavirus. Stool, blood, and urine were negative for bacteria. Parvovirus was thought to be unlikely. Celiac and Lyme disease tests were negative. Tests for lymphoproliferative malignant disorders were negative. He was seen by numerous physicians in many different specialties.

Once petitioner was diagnosed with sJIA and began therapy with steroids, and later with Ilaris, which is designed to control cytokines, his condition stabilized.

In evaluating petitioner’s claim, the opinions and views of the vaccinee’s treating physicians are entitled to some weight. Andreu v. Sec’y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Capizzano v. Sec’y of Health & Hum. Servs., 440 F.3d 1317, 1326 (Fed. Cir. 2006) (“[M]edical records and medical opinion testimony are favored in vaccine cases, as 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.’” (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence since they are created contemporaneously with the treatment of the vaccinee. Cucuras v. Sec’y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). The petitioner need not make a specific type of evidentiary showing, i.e., “epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect.” Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Here, petitioner's treating physicians appear to have conducted a very thorough diagnostic workup. As stated by Dr. Goodman, "[t]he presentation is quite classical for [sJIA]." Pet. Ex. 1 at 216-17.

The third reason for the undersigned's finding as to diagnosis is that petitioner's signs and symptoms are not consistent with CAPS. While on occasion petitioner reported a headache, headache was not a central feature of his condition. He did not have hearing loss, amyloidosis, aseptic meningitis, or skeletal abnormalities. Additionally, he did not have abnormal proteins when his serum protein electrophoresis was performed.

In summary, petitioner has proffered preponderant evidence which establishes he suffers from sJIA.

### **B. Standards for Adjudication – Causation**

The Vaccine Act was established to compensate vaccine-related injuries and deaths. 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." 13(a)(1)(B).

To receive compensation through the Program, petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 13(a)(1)(A), 11(c)(1); Capizzano, 440 F.3d at 1319-20. Because petitioner's claim is not a Table claim, he must prove his claim by showing that her injury was caused-in-fact by the vaccination in question. § 11(c)(1)(C)(ii). To do so, petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be “legally probable, not medically or scientifically certain.” Knudsen v. Sec’y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material in the record, including “any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation.” § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties’ proffered experts and rule in petitioner’s favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 (“Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence.”); Althen, 418 F.3d at 1280 (noting that “close calls” are resolved in petitioner’s favor).

### C. Causation Analysis

#### 1. Althen Prong One: Medical Theory of Causation

Under Althen Prong One, petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner’s theory of causation need not be medically or scientifically certain, but it must be informed by a “sound and reliable” medical or scientific explanation. Boatmon v. Sec’y of Health & Hum. Servs., 941 F.3d 1351, 1359 (Fed. Cir. 2019); see also Knudsen, 35 F.3d at 548; Veryzer v. Sec’y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both “relevant” and “reliable”). If petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen, 618 F.3d at 1347 (“The special master’s decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories.”); Perreira v. Sec’y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an “expert opinion is no better than the soundness of the reasons supporting it” (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

Here, the undersigned finds that petitioner has shown by preponderant that the HPV and Hep A vaccines<sup>50</sup> can cause sJIA and that the proffered mechanism implicating the innate immune system is a sound and reliable theory.

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<sup>50</sup> It is not entirely clear whether Dr. Gershwin implicated the Hep A vaccine along with the HPV vaccine in his causal mechanism. However, he opined that the Hep A vaccine produced cytokines, albeit to a lesser degree. Therefore, the undersigned finds that the HPV vaccine is the primary causal vaccine, but that the Hep A vaccine may have also played a causal role.

Both Dr. Gershwin and Dr. Platt agree that cytokines produce inflammation which contribute to pathology. They both agree that there is a genetic basis for the illness. And, lastly, they both recognize the important of environmental triggers. Dr. Platt cited the Berkun and Padeh article, which discusses environmental factors (bacterial infection, stress, cold weather, and smoking) thought to act as triggers for sJIA. Thus, the experts agree on key elements relevant to the pathogenesis of the disease. However, Dr. Platt does not agree that post-vaccination cytokines can serve as a trigger or otherwise lead to the reprogramming of cells resulting in persistent activation of the cytokines.

In response, Dr. Gershwin testified that once the macrophages are “turned on” the process persists because the macrophages are abnormal. Tr. 276. Once the macrophages become “aberrant and turned on . . . the process will persist, just as in other autoinflammatory diseases.” Id. This is the “loss of control of alternative secretory pathway” aspect of the mechanism as it relates to sJIA, which is described in the Lin article. Tr. 118. Thus, there is medical literature support for this aspect of Dr. Gershwin’s proffered causal theory.

The undersigned is also mindful of the guidance of the Federal Circuit in Koehn v. Secretary of Health & Human Services, a case involving the same vaccine (HPV), the same illness (sJIA), and the same theory proposed here (genetic predisposition/environmental trigger/dysregulation of cytokines). 773 F.3d 1239 (Fed. Cir. 2014). The Federal Circuit found that denial of entitlement was proper because the petitioner failed to prove by preponderant evidence that there was an appropriate temporal association between vaccination and onset of sJIA.<sup>51</sup>

However, in a footnote, the Federal Circuit suggested that the petitioner in Koehn would have likely met her burden as to Althen Prong One. Koehn, 773 F.3d at 1244 fn.1. Without undertaking an analysis of the significance of the footnote, or the differences between the facts or expert opinions in the two cases, the undersigned simply notes that the Circuit suggested that the theory at issue was sufficient to carry the day.

Moreover, the undersigned’s Ruling is consistent with Ramsay v. Secretary of Health & Human Services, where the special master found the petitioner was entitled to compensation when she developed sJIA following the HPV vaccine. No. 11-549V, 2017 WL 1150796 (Fed. Cl. Spec. Mstr. Feb. 28, 2017).

The lack of supportive epidemiological evidence is not dispositive. The cases relied on by respondent did not study the HPV vaccine. Further, Dr. Platt agreed with Dr. Gershwin that it is difficult to use epidemiology to determine whether a vaccine is implicated in causation. Moreover, “[r]equiring epidemiologic studies . . . or general acceptance in the scientific or

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<sup>51</sup> See also Pafford, where the Federal Circuit affirmed denial of entitlement where evidence did not support a temporal association between vaccination and onset of illness. Further, evidence showed that petitioner had other contemporaneous events including bacterial (mycoplasma) infection, sinus infection, tonsillitis, cold and diarrhea. 451 F.3d 1352.

medical communities . . . impermissibly raises a claimant’s burden under the Vaccine Act and hinders the system created by Congress, in which close calls regarding causation are resolved in favor of injured claimants.” Andreu, 569 F.3d at 1378 (citing Capizzano, 440 F.3d at 132-26 (citations and internal quotation marks omitted)); see also Althen, 418 F.3d at 1280 (noting that “close calls” are resolved in petitioner’s favor).

For these reasons, undersigned finds that petitioner has provided preponderant evidence of a sound and reliable causal theory, satisfying Althen Prong One.

## **2. Althen Prong Two: Logical Sequence of Cause and Effect**

Under Althen Prong Two, petitioner must prove by a preponderance of the evidence that there is a “logical sequence of cause and effect showing that the vaccination was the reason for the injury.” Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). “Petitioner must show that the vaccine was the ‘but for’ cause of the harm . . . or in other words, that the vaccine was the ‘reason for the injury.’” Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee’s treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 (“[M]edical records and medical opinion testimony are favored in vaccine cases, as 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.’” (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. The petitioner need not make a specific type of evidentiary showing, i.e., “epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect.” Capizzano, 440 F.3d at 1325. Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In regard to Althen Prong Two, the undersigned finds petitioner provided preponderant evidence of a logical sequence of cause and effect showing that his vaccinations were the cause of his sJIA. Although his treating physicians did not provide any opinions that support or negate a finding that petitioner’s vaccines were causal, his medical records show circumstantial evidence of a clinical course that is consistent with the causal mechanism at issue.

As described above, sJIA is an autoinflammatory illness. There is “a loss of control of the alternative secretory pathway leading to aberrant activation of phagocytes (monocytes, macrophages, neutrophils),” which results in the “release of pro-inflammatory cytokines” that cause “multisystem inflammation.” Pet. Ex. 31 at 2. Generally, the experts agree that rash and fever are consistent with cytokine upregulation. Petitioner had rash and fever. Additionally, petitioner had laboratory markers consistent with systemic inflammation, including elevated C-reactive protein, elevated erythrocyte sedimentation rate, and elevated platelet counts. He also had enlargement of his liver and spleen, and arthritis—systemic manifestations of the inflammatory process.

Moreover, petitioner's illness improved once he began taking cytokine inhibitors. He improved on Actemra, Kineret, and Ilaris at various times during his treatment.

Lastly, petitioner underwent several extensive workups and there was no alternative cause found for his illness. Rapid flu and strep tests were negative. Throat cultures were negative. Testing for the Epstein-Barr virus was negative. Petitioner tested negative for ASO titer, Bartonella, CMV, rheumatoid factor, and rotavirus. Stool, blood, and urine were negative for bacteria. Parvovirus was thought to be unlikely. Celiac and Lyme disease tests were negative. Tests for lymphoproliferative malignant disorders were negative. In summary, there was no evidence of a viral, bacterial, or other triggering factor for his sJIA.

For all of the reasons described above, the undersigned finds that petitioner provided preponderant evidence of a logical sequence of cause and effect, satisfying Althen Prong Two.

### 3. Althen Prong Three: Proximate Temporal Relationship

Althen Prong Three requires petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been equated to mean a "medically acceptable temporal relationship." Id. The petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disease's etiology, it is medically acceptable to infer causation-in-fact." De Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn, 773 F.3d at 1243; Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 542 (2011), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

In regard to onset, a petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at \*19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy v. Sec'y of Health & Hum. Servs., 23 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379



(Fed. Cir. 2009); Bradley v. Sec’y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

Despite the weight afforded medical records, special masters are not bound rigidly by those records in determining onset of a petitioner’s symptoms. Valenzuela v. Sec’y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at \*3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec’y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at \*3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) “must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them”); see also Kirby v. Sec’y of Health & Hum. Servs., No. 2020-2064, 2021 WL 2006226, at \*4 (Fed. Cir. May 20, 2021) (finding the presumption that oral testimony does not conflict with medical records is reasonable when medical records are silent as to the nonexistence of symptoms.)

Petitioner received his vaccines on September 18, 2014. He testified that he had a rash about one week following his vaccinations. Petitioner was a credible witness, understated in most of his responses.

On November 20, 2014, Dr. Cutler documented that the rash was “intermittent, resolves spontaneously over several weeks.” Pet. Ex. 1 at 46. This note implies that the rash had been present long enough to come and go over a period of several weeks. The note also suggests that the rash had come and gone more than once.

Subsequent records put the onset of the rash at either approximately October 1 or November 1, and as time progresses, and as the petitioner saw a number of different physicians, the references to onset of the rash become somewhat inconsistent.

When read in concert, the petitioner’s testimony that the onset of the rash was about a week after vaccination is not inconsistent with Dr. Cutler’s note, which was the first note by a health care provider. Dr. Cutler’s note suggests the passage of time. The rash was intermittent. It would appear and then resolve over the period of several weeks. This had occurred for a period of time. As such, the note corroborates the testimony of petitioner, that onset of the rash occurred about one week after vaccination.

Onset one week after vaccination is an appropriate time frame given petitioner’s proffered theory. In Herrin, proinflammatory cytokines were found to be elevated for up to 14 days following vaccination. Herrin supports Dr. Gershwin’s opinion that the time frame here is appropriate given the causal mechanism.

Dr. Platt did not offer an opinion as to what an appropriate time frame would be because he rejected the causal mechanism. He did not, however, offer rebuttable evidence in response to Herrin.

Therefore, the undersigned finds that the onset of petitioner's rash was approximately one week after vaccination and that this time frame is appropriate given petitioner's proffered causal mechanism. Petitioner provided preponderant evidence satisfying Althen Prong Three.

## **VII. CONCLUSION**

Based on the record as a whole, the undersigned finds there is preponderant evidence to satisfy all three Althen prongs and to establish petitioner's vaccination caused his sJIA. Thus, the undersigned finds petitioner has established by preponderant evidence that he is entitled to compensation. A separate damages order will issue.

**IT IS SO ORDERED.**

**s/Nora Beth Dorsey**

Nora Beth Dorsey

Special Master