In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: November 20, 2025

DEVEN LOMAGO,	*	
Petitioner,	*	No. 19-1092V
v.	*	Special Master Young
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Gary A. Butler, Massa Butler Giglione, Pittsburgh, PA, for Petitioners.
Jay Travis Williamson, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION ON ENTITLEMENT[1]

On July 29, 2019, Careen Lomago 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. (2018),[2] on behalf of her then minor child, Deven Lomago ("Petitioner").[3] Pet., ECF No. 1. She alleged Petitioner developed Crohn's disease ("CD")[4] as the result of a human papillomavirus ("HPV") vaccine he received on August 3, 2016. *Id.* at 1. Respondent argued against compensation, asserting that Petitioner could not establish a causation-in-fact claim by a preponderance of the evidence. Resp't's Rept. at 12, ECF No. 15.

[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision 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, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755. Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

[3] On December 29, 2022, I issued an order to amend the case caption to be Deven Lomago to reflect that he had reached the age of majority. ECF No. 30.

[4] Crohn's disease is "a chronic granulomatous disease of the gastrointestinal tract of unknown etiology." *Crohn Disease*, DORLAND'S ONLINE MED. DICTIONARY, https://www.dorlandsonline.com/dorland/definition?id=70226 (hereinafter, "*Dorland's*").

After carefully analyzing and weighing all the evidence presented in this case in accordance with the applicable legal standards,[5] I find that Petitioner has not provided preponderant evidence that the HPV vaccine he received on August 3, 2016, caused him to suffer from CD. Accordingly, Petitioner is not entitled to compensation.

I. Procedural History

Petitioner filed a petition for compensation, medical records, and an affidavit on July 29, 2019. Pet.; Pet'r's Exs. 1–10, ECF No. 1. On July 31, 2019, I issued an initial order directing Petitioner to file requested medical records and a statement of completion. ECF No. 5. Petitioner filed these records and a statement of completion on October 10, 2019. Pet'r's Ex. 11, ECF Nos. 9–10.

Respondent filed his Rule 4(c) report, opposing compensation, on June 19, 2020. Resp't's Rept. Specifically, Respondent argued that Petitioner's treating physicians explicitly ruled out the HPV vaccine as being implicated in the development of his condition, in addition to Petitioner failing to meet his burden under *Althen*. *Id.* at 12–13.

At the parties' request, on September 3, 2020, I held a Rule 5 status conference. Min. Entry, docketed Sept. 3, 2020. Following the conference, on December 2, 2020, Petitioner filed an expert report from Dr. John Cromwell, along with supporting medical literature. Pet'r's Exs. 12–20, ECF No. 17. Respondent then filed a responsive expert report from Dr. Chris Liacouras on April 2, 2021, and supporting medical literature on April 30, 2021. Resp't's Exs. A–B, D, ECF No. 20; Resp't's Ex. A, Tabs 1–17, ECF No. 22. Respondent also filed an expert report from Dr. Stephen McGeady, as well as supporting medical literature, on May 3, 2021. Resp't's Ex. C, Resp't's Ex. C, Tabs 1–10, ECF No. 23. Ms. Lomago filed a response from Dr. Cromwell and additional literature on August 12, 2021. Pet'r's Exs. 21–24, ECF No. 25. Respondent filed responses from both Dr. Liacouras and Dr. McGeady on November 8, 2021, and supporting medical literature on November 19, 2021. Resp't's Exs. E–F, ECF No. 26; Resp't's Ex. E, Tabs 1–3, Resp't's Ex. F, Tabs 1–3, ECF No. 27.

On October 25, 2023, I held a status conference where the parties agreed to resolve this case via a ruling on the record. Min. Entry, docketed Oct. 25, 2023; ECF No. 31. Specifically, I requested Petitioner to clarify his expert's explanation as to the role of cytokines in his molecular mimicry[6] biological mechanism. *Id.* On December 4, 2023, Petitioner filed his motion for a ruling on the record, a supplemental report from Dr. Cromwell, and additional medical literature. Pet'r's Exs. 25–27, ECF No. 33; Pet'r's Mot., ECF No. 34. Respondent filed a responsive expert report

[5] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the Decision will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

[6] Molecular mimicry is "a model of autoimmunity in which an immune response to a foreign antigen containing a peptide region that mimics a self epitope provokes cross-reactivity to a self protein." *Dorland's*.

and a response to Petitioner's motion on February 22, 2024. Resp't's Ex. G, Resp't's Ex. G, Tabs 1–7, Resp't's Resp., ECF No. 36. Petitioner filed his reply, as well as a responsive expert report and medical literature, on April 1, 2024. Pet'r's Exs. 28–32, ECF No. 38; Pet'r's Reply, ECF No. 39.

This matter is now ripe for consideration.

II. Medical History

Petitioner was born on April 4, 2004, and was 12 years old at the time of vaccination. Pet'r's Ex. 1 at 5. He had a prior history of seasonal allergies, a nut allergy, and anxiety. *Id.* On August 3, 2016, Petitioner presented to his primary care physician ("PCP"), Gary Smith, for his 12-year-old wellness exam. *Id.* He reported that he had a well-balanced diet, and his bowel movements were normal with regular consistency. *Id.* On examination, Dr. Smith noted his abdomen was "soft, nontender, [and] nondistended," with "no masses palpable." *Id.* at 6. Dr. Smith administered the Gardasil-9 HPV vaccine to Petitioner at this visit. *Id.*

Twelve days later, on August 15, 2016, Petitioner returned to Dr. Smith with complaints of a boil on his buttock. Pet'r's Ex. 1 at 3. His father reported the boil had been growing for one week and was painful. *Id.* On examination, Dr. Smith observed a one centimeter "area of induration and mild erythema and tenderness [on the] medial right buttock just lateral to [the] anus[, with] no pus or fluctuance." *Id.* Dr. Smith diagnosed Petitioner with an abscess and ordered him to start antibiotics for 10 days. *Id.*

Two days after this visit, on August 17, 2016, Petitioner presented to the Children's Hospital of Pittsburgh ("CHOP") emergency department ("ED") with complaints of a perirectal abscess and a fever of 102º F. Pet'r's Ex. 2 at 22. He reported his symptoms began "as a 'boil' on his right buttock 10 days ago; four days ago it became more enlarged, red, and painful." *Id.* On examination his general gastrointestinal systems appeared normal, but his treating physician noted a "[p]erianal abscess at the 9:00 position immediately adjacent to this external anal sphincter; [two centimeters], indurated, with a pustule but not actively draining." Petitioner was admitted that day to undergo an examination under anesthesia the next morning. *Id.* at 23.

The next day, on August 18, 2016, Dr. Aviva Katz performed an incision and drainage of Petitioner's abscess. Pet'r's Ex. 2 at 64. His surgery notes indicated that his pain had started "1.5 weeks ago" and that "[a]t first, there was no visible lesion. His discomfort became worse and mom started to notice[an] erythema on the [right] buttocks near the anus." *Id.* The notes further indicated that he had no family history of inflammatory bowel disease ("IBD"). *Id.* He was discharged home the same day following the procedure. *Id.* at 9.

Petitioner returned to Dr. Smith on September 22, 2016, with complaints that his wound was not healing properly after surgery. Pet'r's Ex. 1 at 1. He reported that his wound had improved but was still open, with discharge "like boogers." *Id.* Dr. Smith restarted Petitioner on antibiotics and referred him to pediatric surgery at CHOP. *Id.* at 2.

Petitioner presented to Dr. Katz for his follow-up referral on September 27, 2016. Pet'r's Ex. 3 at 203. On examination Dr. Katz observed "moist granulation tissue visible at the site of incision and drainage at approximately 7 o'clock," as well as "a small skin tag present at approximately 6 o'clock . . . without any other obvious changes consistent with abscess or fistulae." *Id.* Due to a lack of surrounding cellulitis, Dr. Katz recommended Petitioner cease antibiotic treatment and explained to his family that the appearance of Petitioner's wound, in addition to the skin tag, raised a concern of IBD and recommended Petitioner follow up with a gastroenterologist. *Id.* at 203–04.

Petitioner returned to Dr. Katz for a follow-up on October 25, 2016, where he reported "intermittent crampy abdominal pain and frequent loose stools, which [were] not alternating with significant constipation." Pet'r's Ex. 3 at 218. Dr. Katz again noted a significant concern for IBD, but observed that Petitioner's incision was improving and did not feel it would require more intervention. *Id.* She also observed that his small skin tag remained, but with no perianal or perirectal inflammatory changes. *Id.* Petitioner's family also asked about the HPV vaccine and its potential role in the development of Petitioner's injury. *Id.* at 219. Dr. Katz noted in her report that she "strongly reassure[d] the mother that the perirectal abscess and the potential for [IBD were] in no way related to the HPV vaccine." *Id.*

On November 22, 2016, Petitioner presented to the Pediatric Gastroenterology department at CHOP for "an esophagogastroduodenoscopy and colonoscopy for further evaluation of abdominal pain and history of perirectal abscess." Pet'r's Ex. 2 at 264. Nurse Practitioner ("NP") Leslie Coda reviewed Petitioner's results, which revealed mild inflammation in the antrum of the stomach and "an area of mucosa at the ileocecal valve that was moderately ulcerated." *Id.* "Biopsies were significant at the ileocecal valve for severe active chronic ileocolitis[7] with ulceration. . . . the gastric antral biopsy showed mild active chronic gastritis with epithelioid granulomas. [The t]erminal ileum biopsy showed epithelioid granulomas and mild architectural distortion." *Id.* Petitioner's transverse, descending, and sigmoid colon showed "increased lamina propria cellularity with multiple epithelioid granulomas," and similar findings were shown in his rectal biopsy. *Id.* These findings "strongly support[ed] the diagnosis of [CD]." *Id.* at 264–65. Petitioner was directed to follow up with gastroenterologist Sandra Kim for further evaluation of CD versus chronic granulomatous disease. *Id.* at 265.

Petitioner presented to Dr. Kim on December 5, 2016, and reported a slight decrease in appetite over the previous few months, coinciding with the onset of his perianal abscess and pain. Pet'r's Ex. 4 at 40. Petitioner also reported "intermittent periumbilical and lower abdominal pain that he describe[d] as being more cramping . . . occasionally alleviated by defecation." *Id.* Dr. Kim also observed that Petitioner had not had issues with weight gain or wound healing prior to August 2016, and that Petitioner had only gained one pound "in the past few months." *Id.* at 41. Dr. Kim noted Petitioner's probable diagnosis to be "inflammatory ileocolonic [CD] with possible early upper [gastrointestinal ("GI")] tract involvement and perianal involvement as well." *Id.* at 43. Dr. Kim also ordered a neutrophil oxidative burst to rule out chronic granulomatous disease, which came back negative. *Id.* at 5, 43.

[7] Ileocolitis is "inflammation of the ileum and colon." *Dorland's*.

Petitioner saw Dr. Kim again on March 6, 2017, to follow up on his CD diagnosis. Pet'r's Ex. 4 at 67. Since his December 2016 visit, Petitioner had been overall stable but continued to experience mild abdominal discomfort about once or twice a week, and denied pain when defecating or any recurrence of an abscess. *Id.* Although his appetite had been stable, he had not gained any weight since his last visit in December and only grown one centimeter. *Id.* Dr. Kim attributed this to a specific carbohydrate diet his parents switched him to at the advice of a dietitian and not his CD symptoms. *Id.* Petitioner further reported that he had experienced occasional low-grade fevers over the past month and Dr. Kim observed that he appeared to present with swollen lymph nodes in the femoral region bilaterally. *Id.* Petitioner also had one episode of "gastroenteritis type symptoms including diarrhea, which he had approximately [six] times during the course of the day without any blood[,] and fevers to 102." *Id.* at 69.

In the history section of her report, Dr. Kim wrote that Petitioner's parents had extensive questions about his diagnosis at the December 2016 visit, "including a question of whether [the] HPV vaccine could have induced [an] inflammatory response mimicking [CD]." Pet'r's Ex. 4 at 67. Dr. Kim continued that she "discussed the fact that there is nothing in the literature that suggests a connection between the HPV vaccine and [CD]. Furthermore, the evaluation, and clinical presentation and biopsies were indeed consistent with [CD]." *Id.* At the present visit, Petitioner's parents again reiterated their belief that the HPV vaccine may have contributed to his disease, given that all his symptoms had developed after receipt of the vaccine. *Id.* at 70. Dr. Kim offered to give Petitioner and his family a second opinion at Children's Hospital of Philadelphia, but they declined. *Id.* Dr. Kim referred Petitioner to a GI dietitian to review his specific carbohydrate diet, as well as the possibility of a CD specific diet to help alleviate his symptoms, and ordered additional labs to check his metabolic panel and inflammatory markers. *Id.* On March 13, 2017, Dr. Kim emailed Petitioner's family to inform them his stool markers for inflammation were significantly inflated, with a calprotectin level of 672.1. *Id.* at 75.

Petitioner underwent an endoscopy and colonoscopy on June 2, 2017, at CHOP. Pet'r's Ex. 7 at 556–58. His endoscopy findings were normal, however, his colonoscopy revealed scattered aphthae throughout the colon and mildly congested mucosa in the rectum. *Id.* at 558.

Petitioner presented to pediatrician Stacey Bregman on December 21, 2017, with "a perianal fistula and abscesses." Pet'r's Ex. 6 at 143. He reported that his symptoms began with "'irritation' both on [the] outside and inside rectum on the [left] side" that was worse internally, though not as severe as his prior abscess, and his primary GI physician recommended he undergo magnetic resonance imaging ("MRI"). *Id.* Dr. Bregman performed an MRI of Petitioner's pelvis that day which revealed "intersphincteric perianal fistula with abscesses at about 2 o'clock measuring up to 21 and 14 mm in diameter." *Id.* Petitioner was admitted to CHOP the same day for surgical drainage of his abscesses and to place a seton[8] in his fistula. Pet'r's Ex. 7 at 107. He was discharged the following day on December 22, 2017, with a recommendation to start Remicade or Humira and a two-week course of antibiotics. *Id.* at 107–08.

[8] A seton is "a thread of silk, linen, or other finely drawn material for passage through a sinus, fistula, or epithelial tract, often to serve as a guide for subsequent dilatation with instruments of a larger diameter." *Dorland's*.

Petitioner returned to Dr. Kim on January 8, 2018, for a follow-up at the CHOP IBD center. Pet'r's Ex. 6 at 45. Dr. Kim noted that Petitioner began Remicade following his December 2017 surgery due to "perianal involvement being notoriously difficult to control." *Id.* Petitioner reported no abdominal pain and that his stools were normal without blood or mucus. *Id.* Dr. Kim advised Petitioner to restart methotrexate and his antibiotics. *Id.* at 48.

On March 26, 2018, Petitioner presented to pediatric surgeon Kevin Mollen with complaints of irritation and bloody drainage from his fistula site, beginning about three weeks ago. Pet'r's Ex. 7 at 537. "[Petitioner was] convinced that this [was] due to [the knots] in the seton itself." *Id.* Dr. Mollen noted that Petitioner's CD "appear[ed] to be completely asymptomatic." *Id.* On examination, Dr. Mollen observed that Petitioner's seton was "in place [and] the skin surrounding it [was] completely healthy" without any drainage or fluctuance. *Id.* After discussion with his family, Dr. Mollen decided to remove Petitioner's seton, with the understanding that "it is possible he will now redevelop a perianal abscess." *Id.* at 538.

Petitioner returned to Dr. Kim for routine follow-up appointments on April 16, 2018, and June 11, 2018. Pet'r's Ex. 6 at 227; Pet'r's Ex. 7 at 328. At both visits, Petitioner reported feeling well with minimal abdominal pain, and Dr. Kim recommended that he continue taking Remicade and methotrexate. Pet'r's Ex. 6 at 228; Pet'r's Ex. 7 at 330. On December 3, 2018, Petitioner presented to Dr. Kim with complaints of a symptom flare consisting of bad gas pain in his lower abdomen four weeks earlier. Pet'r's Ex. 7 at 29. Dr. Kim was also concerned for mild leukopenia[9] she suspected was due to the discontinuance of methotrexate, but otherwise was pleased with Petitioner's progress. *Id.* at 31. "His endoscopic evaluation overall was quite unremarkable, with only signs of mild chronic inactive inflammation in the terminal ileum and some scattered granulomas without significant inflammation in the colon." *Id.*

On February 26, 2019, Petitioner presented to pediatric hematologist-oncologist Steven Allen for evaluation of persistent leukopenia, which began when he started Remicade and continued after discontinuation of methotrexate. Pet'r's Ex. 11 at 287. Dr. Allen opined that Petitioner's leukopenia was a side-effect of his Remicade treatment, but decided not to wean him off medication due to the mild nature of the condition with a note to continue monitoring his blood counts. *Id.* at 288–89. As of July 2019, Petitioner was still receiving Remicade infusions that were well-tolerated. *Id.* at 233.

No other relevant medical records were filed.

III. Affidavit of Petitioner's Mother, Careen Lomago

On July 29, 2019, Petitioner's mother, Careen Lomago, filed a brief affidavit. Pet'r's Ex. 10. She stated that prior to Petitioner's August 3, 2016 HPV vaccination Petitioner had no personal or family history of CD. *Id.* at ¶¶ 6–7. Petitioner had seen his PCP, Dr. Smith, on August 15, 2016, for a boil on his buttock that appeared approximately a week prior. *Id.* at ¶¶ 8–9. This was later diagnosed as CD by Dr. Kim at CHOP, who had been treating Petitioner from December 2016 to the filing of this affidavit. *Id.* at ¶¶ 13–15. Ms. Lomago further stated that Petitioner has continued

[9] Leukopenia is a "reduction in the number of leukocytes in the blood." *Dorland's*.

to suffer "the residual complications, damages and effects of [CD] for more than six months after the administration of the vaccination; and, he continues to suffer from the effects of the disease." *Id.* at ¶ 23.

IV. Experts

A. Expert Qualifications

1. Petitioner's Expert, Dr. John W. Cromwell, M.D.

Dr. Cromwell is a Professor of Surgery and Director of Gastrointestinal, Minimally Invasive, and Bariatric Surgery at the University of Iowa, and Associate Chief Medical Officer and Director of Surgical Quality & Safety for UI Healthcare. Pet'r's Ex. 12 at 1. He received his M.D. from the University of Minnesota Medical School, and completed his internship and residency in general surgery, as well as a surgical infectious disease fellowship at University of Minnesota Hospitals & Clinics. Pet'r's Ex. 13 at 1. He also completed a research fellowship with the University of Minnesota Cancer Center and a colon and rectal surgery fellowship with University of Texas Affiliated Hospitals. *Id.* He is board certified in general surgery and colorectal surgery. Pet'r's Ex. 1. Dr. Cromwell has practiced for over 17 years "in a practice that has focused on the surgical treatment of [IBD] and colorectal cancer." *Id.* at 1. He also has "a background in transplant immunology research dealing with both innate and adaptive immunity in humans and primates." *Id.*

2. Respondent's Expert, Dr. Chris A. Liacouras, M.D.

Dr. Liacouras is a board-certified pediatric gastroenterologist with over 30 years of clinical practice. Resp't's Ex. A at 2. He is currently a Professor of Pediatrics at the Perelman School of Medicine at the University of Pennsylvania and the Children's Hospital of Philadelphia in the Division of Gastroenterology, Hepatology, and Nutrition. *Id.* He is also the Medical Director of the Children's Hospital of Philadelphia's Center for Gastrointestinal Endoscopy and Chairman of Pediatric Endoscopy for the North American Society for Pediatric Gastroenterology, Hepatology, and Nutrition. *Id.* Dr. Liacouras received his M.D. from Harvard University. Resp't's Ex. B at 1. He completed his internship in pediatrics, as well as his residency and fellowship in pediatric gastroenterology at the Children's Hospital of Philadelphia. *Id.*

Dr. Liacouras has "extensively published in the field of pediatric gastroenterology" and has evaluated between 2,000 to 3,000 pediatric patients "every year since 1991." Resp't's Ex. A at 2. He has "personally evaluated or consulted on more than [1,000] pediatric patients who have had [CD]." *Id.*

3. Respondent's Expert, Dr. Stephen J. McGeady, M.D.

Dr. McGeady is a Professor of Pediatrics at Thomas Jefferson University and the Emeritus Chief of the Allergy, Asthma & Immunology Division of duPont Hospital for Children, and has been "trained as both a Pediatrician and as an Allergist/Immunologist." Resp't's Ex. C at 1; Resp't's Ex. D at 1. He received his M.D. from Creighton University, and completed his pediatric

residency at St. Christopher's Hospital for Children in Philadelphia, PA. Resp't's Ex. C at 1; Resp't's Ex. D at 1. He also completed his allergy and immunology fellowship at Duke University Medical Center. Resp't's Ex. C at 1. For eight years he was the Medical Director of the Children's Rehabilitation Hospital in Philadelphia, and "oversaw the provision of therapy services to children with chronic illnesses of many types" in that capacity. *Id.* at 2. Dr. McGeady is "the author or co-author of approximately 70 peer reviewed papers" in the field of allergy and immunology. *Id.* at 1.

B. Expert Reports

1. Reports of Dr. Cromwell

a. Initial Report

Dr. Cromwell structured his initial report to reflect the three *Althen* prongs and began his explanation of his medical theory by describing CD as an autoimmune disease. Pet'r's Ex. 12 at 3. "The pathophysiology of CD is widely accepted to be autoimmune in nature, in which pro- and anti-inflammatory mediators within the intestinal mucosa become imbalanced and favor inflammation." *Id.* He cited to Khor et al.,[10] which explained that this process involves "an inadequate [] response in the face of an overly exuberant response" from T-cells. *Id.* at 4 (citing Pet'r's Ex. 14 at 7). Khor et al. explained that the intestinal epithelium "is complemented by a well-evolved mucosal innate immune system, which is populated by cells poised to defend against pathogenic incursions and curtail inflammatory responses." Pet'r's Ex. 14 at 5. "[P]atients with [CD] have defective innate immune responses," which disrupts intestinal homeostasis. *Id.* This homeostasis is "maintained in part by the actions of resident macrophages that have enhanced phagocytic and bactericidal activity and decreased production of pro-inflammatory cytokines." *Id.* "Intestinal inflammation resulting from a failure to maintain this balance is exemplified by patients . . . who have deficient [T-cell] function." *Id.* at 7. Dr. Cromwell supported his argument that CD is autoimmune in nature by pointing to the successful use of immune-suppressant medications to treat CD, such as corticosteroids, thiopurines, and anti-TNF agents. Pet'r's Ex. 12 at 4 (citing Pet'r's Ex. 15).[11]

Dr. Cromwell next turned to the non-specific effects of vaccines, arguing that "both beneficial and adverse non-specific effects [("NSEs")] of vaccines have been well-documented in epidemiologic studies" in both live and inactivated vaccines. Pet'r's Ex. 12 at 4 (citing Pet'r's Ex. 16).[12] Jensen et al. explained that "NSEs may be beneficial or detrimental depending on the type of vaccine; live attenuated vaccines . . . have beneficial NSEs reducing all-cause mortality, whereas inactivated vaccines . . . though protective against the target diseases, appear to have deleterious effects, increasing susceptibility to other pathogens." Pet'r's Ex. 16 at 2. The study found that the live Bacillus Calmette-Guérin ("BCG") vaccine against tuberculosis "showed increased pro-

[10] Bernard Khor et al., *Genetics and Pathogenesis of Inflammatory Bowel Disease*, 474 NATURE 307 (2011).
[11] Gary R. Lichtenstein et al., *ACG Clinical Guideline: Management of Crohn's Disease in Adults*, 113 AM. J. GASTROENTEROLOGY 481 (2018).
[12] Kristoffer Jarlov Jensen et al., *Unravelling the Nature of Non-Specific Effects of Vaccines – A Challenge for Innate Immunologists*, 28 SEMINARS IMMUNOLOGY 377 (2016).

inflammatory cytokine production in response to non-related pathogens . . . [two] weeks and [three] months after vaccination." *Id.* at 4. The authors also acknowledged that "there are no reports of a sustained imprinting effect of the inactivated vaccines . . . on innate immunity," but that findings indicated inactivated vaccines "impair resistance to other infectious diseases than the targeted disease." *Id.* at 5. Specific to the HPV vaccine, Dr. Cromwell pointed to Benn et al.[13] to show that the HPV vaccine "has been used to trigger panniculitis in rodents through non-specific effects." Pet'r's Ex. 12 at 4 (citing Pet'r's Ex. 17 at 4). He also cited to Aaby et al.,[14] which explained that "all vaccines tested in epidemiological studies have shown important NSEs on child survival in low-income countries." *Id*. (citing Pet'r's Ex. 18 at 1.) The review found that live vaccines increased resistance to vaccine-unrelated infections such as pneumonia and sepsis, while "non-live vaccines . . . seem to increase susceptibility to vaccine-unrelated infections." Pet'r's Ex. 18 at 1. Dr. Cromwell acknowledged that "[w]hile the presence of [NSEs] of vaccines on the innate immune system are clear, the study of these mechanisms is currently immature." Pet'r's Ex. 12 at 4.

In addressing how vaccines can be implicated in the development of CD, Dr. Cromwell explained that imbalances with the adaptive immune system have been implicated in the development of CD, which are driven by alterations in non-specific immunity. Pet'r's Ex. 12 at 4. He argued that activation of innate immunity "can alter homeostasis between Th1/Th2 cells and between Th17/Treg [cells] within the adaptive immune system . . . creating a molecular milieu favorable for disruption of the mucosal barrier and inflammation in the lining of the large intestine." *Id.* Huang & Chen[15] described this process as the abnormal activation of the innate immune system, which can "include adaptive immunity imbalance, inflammatory cytokines produced by which increase innate immune damages, abate intestinal barrier functions and aggravate inflammation." Pet'r's Ex. 19 at 6. Dr. Cromwell argued that this process, combined with "the broadly documented non-specific effects of vaccines on recipients and the connection of vaccines to alterations in innate immunity in experimental models of [IBD], it is a logical conclusion that HPV vaccines may drive the known imbalances in adaptive immunity . . . leading to [CD] in susceptible individuals." Pet'r's Ex. 12 at 4–5.

Dr. Cromwell next addressed the logical sequence of cause and effect connecting his medical theory to the injury sustained by Petitioner, arguing that

> it is more likely than not that [Petitioner] was an individual susceptible to the non-specific immune effects of the vaccines. When he received the HPV [vaccine], this led to a cascade of molecular events including the priming of the innate immune system via non-specific effects of the vaccines. This, in turn, led to alteration in the adaptive immunity within the intestine through a known mechanism causing T-cell imbalances . . . this led to the disruption of the mucosal barrier function and inflammation characteristic of [CD].

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[13] Christine S. Benn et al., *A Small Jab – A Big Effect: Nonspecific Immunomodulation by Vaccines*, 34 TRENDS IN IMMUNOLOGY 431 (2013).

[14] Peter Aaby et al., *The Non-Specific and Sex-Differential Effects of Vaccines*, 20 NATURE REVS.: IMMUNOLOGY 464 (2020).

[15] Yuan Huang & Zhonge Chen, *Inflammatory Bowel Disease Related Innate Immunity and Adaptive Immunity*, 8 AM. J. TRANSLATIONAL RSCH. 2490 (2016).

Pet'r's Ex. 12 at 5.

With regard to the temporal relationship between vaccination and the onset of Petitioner's injury, Dr. Cromwell argued that Petitioner's development of a perianal abscess approximately five days after vaccination "is perfectly consistent with [NSEs] from vaccination being the causative event" and that Petitioner did not have any symptoms of IBD prior to vaccination. Pet'r's Ex. 12 at 5. He pointed to an article by Frank Shann[16] to support his timeframe, which noted that "[t]he [NSEs] of vaccines appear to be maximal in the first [six] months after administration of the vaccine." *Id.*; Pet'r's Ex. 20 at 1.

b. First Supplemental Report

In his first supplemental report, Dr. Cromwell explained that while he did not provide an "exhaustive explanation of the pathophysiology of [CD]," he "focused on the elements of the immune defects in [CD] that are relevant to [his] conclusion." Pet'r's Ex. 21 at 1. Dr. Cromwell noted that although "direct experimental evidence of causation [of CD] has not been demonstrated outside of genetic abnormalities, there has been linkage to environmental triggers, including drugs." *Id.* at 2. He argued that it is "common for direct experimental evidence to lag behind epidemiologic links; thus, the absence of direct experimental evidence does not serve as proof of no linkage." *Id.* He also argued that the causative relationship between genetic abnormalities and the development of CD supports his proposed biological theory of causation that the HPV vaccine "can serve as a trigger in a patient with a susceptible genetic phenotype." *Id.*

Dr. Cromwell admitted that he is "not aware of experimental evidence in humans regarding the timing from exposure to a [CD] trigger until the time of measurable alterations in the intestinal mucosal integrity." Pet'r's Ex. 21 at 2. "However, this information may be inferred from accepted experimental models of [CD] in animals." *Id.* He cited to LeBlanc et al.,[17] which studied the preventative effects of lactic acid bacteria on IBD in mice. Pet'r's Ex. 22. In order to induce CD-like symptoms in mice, the authors administered 100 µl of trinitrobenzenesulfonic acid ("TNBS") dissolved in phosphate buffered saline with an equal volume of ethanol directly into the rectal cavity of the mice. *Id.* at 2. The authors used a zero to six grading scale to measure the extent of inflammation in the mice, with zero indicating normal findings and six indicating extensive ulceration and necrosis. *Id.* Three days after administration of TNBS, the authors noted that the mice had an average inflammation score of 4.3 (±.3), which would indicate 50% of the specimen showed "[p]rominent inflammatory infiltrate and oedema . . . frequently with deeper areas of ulceration extending through the muscularis mucosae into the submucosa," but without necrosis. *Id.* at 2, 4. Dr. Cromwell argued that these findings were "the best available evidence demonstrating that progression from a Crohn's trigger to mucosal damage can occur in a very short period of time, in this case less than 72 hours." Pet'r's Ex. 21 at 2. He referenced his own experience as a colorectal surgeon to support the theory that "the time from a break in mucosal integrity to a palpable and visible anal abscess may happen in less than [two] days time." *Id.* Dr.

[16] Frank Shann, *The Non-Specific Effects of Vaccines*, 95 ARCHIVES DISEASES CHILDHOOD 662 (2010).
[17] Jean Guy LeBlanc et al., *Use of Superoxide Dismutase and Catalase Producing Lactic Acid Bacteria in TNBS Induced Crohn's Disease in Mice*, 151 J. BIOTECHNOLOGY 287 (2011).

Cromwell also cited to a review of perianal CD by Lightner,[18] which found that a perianal fistula is the initial manifestation of CD in approximately 10% of patients. Pet'r's Ex. 24 at 1.

Dr. Cromwell "acknowledge[d] that there is no direct finding of [CD] in the published clinical trials [of the HPV vaccine] to date," but insisted that such events are anticipated to be rare and thus the lack of evidence found in clinical trials does not refute his theory under the *Althen* standard. Pet'r's Ex. 21 at 3. He argued that the theory of causation he presented in this case was analogous to the theory he presented in *Morgan v. Sec'y of Health & Hum. Servs.*, where the special master accepted his theory. *Id.*; *See* No. 13-529V, 2015 WL 9694667 (Fed. Cl. Spec. Mstr. Dec. 10, 2015).

c. Second Supplemental Report

Dr. Cromwell's second supplemental report focused on clarifying the difference between cytokine upregulation in a "'standard' immune response to vaccination compared to that seen in autoimmune 'pathologic' responses." Pet'r's Ex. 25 at 1. In a normal cytokine response, cytokines are "responsible for homing appropriate cells from the circulation to the site of injection and activating the initial normal immune response." *Id.* (citing Pet'r's Ex. 26).[19] He explained that while not all vaccine recipients exhibit cytokine-related symptoms from vaccination, those that do, "experience flu-like symptoms" which are listed as potential side-effects of the HPV vaccine. *Id.* at 1–2. This response typically dissipates within days and "is not enough to lead to a pathological response such as an autoimmune syndrome." *Id.* at 2.

Instead, Dr. Cromwell relied on the theory of molecular mimicry to explain how the HPV vaccine could induce CD. Pet'r's Ex. 25 at 2. He explained this process as where a foreign antigen shares structural or sequential similarities with self-antigens, which may cause an immune response against the bodies' own tissues, which typically takes longer than the initial cytokine response associated with vaccine administration. *Id.* In the context of CD following HPV vaccination, Dr. Cromwell explained his theory as follows:

> The HPV vaccine contains viral proteins that are intended to stimulate an immune response against HPV. However, these proteins share structural similarities with proteins present in the human gastrointestinal tract. In a susceptible individual, the immune system mistakenly recognizes these "self" proteins as foreign and generates an immune response against them. The recognition of intestinal proteins as foreign would be expected to elicit a separate cytokine response independent of the initial vaccination response. This pathological immune response could lead to acute followed by chronic inflammation in the gut, characteristic of [CD].

Id. He supported his theory by citing to a study by Kanduc,[20] which identified "numerous peptide regions on HPV16 that are identical to human peptides, some of which are found exclusively in

[18] Amy L. Lightner, *Perianal Crohn's Disease*, 63 DISEASES COLON RECTUM 1023 (2020).
[19] Gillie A. Roth et al., *Designing Spatial and Temporal Control of Vaccine Responses*, 7 NATURE REVS. 174 (2022).
[20] Darja Kanduc, *Quantifying the Possible Cross-Reactivity Risk of an HPV16 Vaccine*, 8 J. EXPERIMENTAL THERAPEUTICS ONCOLOGY 65 (2009).

the human intestine." *Id.* (citing Pet'r's Ex. 27). The study, which found 82 perfect heptapeptide matches between the human proteome and HPV16, concluded that "[t]he number of viral matches and their locations make the occurrence of side autoimmune cross-reactions in the human host following HPV16-based vaccination almost unavoidable." Pet'r's Ex. 27 at 1. Dr. Cromwell concluded by asserting that Petitioner's development of CD was "due to the separate molecular mimicry mechanism as described." Pet'r's Ex. 25 at 3.

d. Third Supplemental Report

In his final expert report, Dr. Cromwell focused on further clarifying this theory of molecular mimicry. He argued that studies which show "massive peptide sharing between viruses/bacteria and humans" did not undermine his theory, but rather "because these shared epitopes are so prevalent [] opportunities for pathological cross-reactivity and autoimmune consequences manifest in some cases." Pet'r's Ex. 28 at 1. He acknowledged that such reactions are rare, but that a lack of proven examples "does not negate its biological plausibility." *Id.* Dr. Cromwell next addressed the unique folding patterns of viral proteins, which may alter their three-dimensional shape, and thus make the proteins unrecognizable to human receptors, and argued that this phenomenon "would both support the selectivity for molecular mimicry to occur and support the expectation that autoimmune diseases arising from this would be rare occurrences." *Id.*

Dr. Cromwell supported the plausibility of the theory of molecular mimicry by citing to Bjornevik et al.,[21] which found that Epstein Barr virus ("EBV") peptide exposure was a cause for multiple sclerosis ("MS") in animal and human models. Pet'r's Ex. 28 at 2 (citing Pet'r's Ex. 29). The authors, who examined the effects of EBV on the later development of MS, concluded that "the occurrence of EBV infection, detectable by the elicited immune response, is a cause and not a consequence of MS," finding a 32-fold increase in the development of MS approximately 10 years after EBV infection. Pet'r's Ex. 29 at 4. Lanz et al.[22] later demonstrated this to be caused by molecular mimicry occurring between EBV nuclear antigen 1 and the central nervous system protein glial cell adhesion molecule. Pet'r's Ex. 30 at 1.

Dr. Cromwell next turned to the lack of an abnormal immune response documented in Petitioner's medical records. Pet'r's Ex. 28 at 3. He argued that his theory did not require the presence of a cytokine storm, and instead "[m]ore subtle dysfunction of the innate immune system is sufficient to prime autoreactive T cells through mechanisms like molecular mimicry." *Id.* Dr. Cromwell also asserted that "the lack of overt symptoms immediately after vaccination does not negate an adverse innate immune system impact." *Id.* He explained that "[m]any autoimmune processes have preclinical periods before pathologic effects manifest," particularly CD, which "frequently has an indolent, insidious onset without obvious symptoms initially." *Id.* Dr. Cromwell also reiterated his point that Petitioner's five-day onset was consistent with LeBlanc et al. and his own clinical experience. *Id.* at 4.

2. Reports of Dr. Liacouras

[21] Kjetil Bjornevik et al., *Longitudinal Analysis Reveals High Prevalence of Epstein-Barr Virus Associated with Multiple Sclerosis*, 375 SCIENCE 296 (2022).
[22] Tobias V. Lanz et al., *Clonally Expanded B Cells in Multiple Sclerosis Bind EBNA1 and GlialCAM*, 603 NATURE 321 (2022).

a. Initial Report

Dr. Liacouras began his report by explaining the pathophysiology of CD, which he described as "chronic, idiopathic inflammation of the gastrointestinal tract." Resp't's Ex. A at 2. Although the specific etiology of CD remains unknown, Dr. Liacouras attributed it to "specific genetic abnormalities, alterations in the intestinal microbiome, use of antibiotics, environmental factors and a defective intestinal inflammatory response." *Id.* at 3 (citing Resp't's Ex. A, Tab 1).[23] "Other than specific genetic abnormalities, there are no known accepted triggers that have been directly linked to the cause of [IBD]." *Id.*

"The symptoms of [CD] represent a spectrum. . . . [T]he disorder often begins with insidious and unappreciated symptoms that are mild and intermittent . . . in retrospect, [many adolescents] have had unrecognized symptoms of infrequent abdominal pain, rare bouts of diarrhea, and occasional anorexia." Resp't's Ex. A at 3. Other patients develop much more severe symptoms, such as gastrointestinal bleeding, intestinal strictures, and fistulas, "which can lead [to] malnutrition and intestinal obstruction." *Id.* (citing Resp't's Ex. A, Tab 3).[24] "Between 25-50% of patients with [CD] have perianal disease," including skin tags, abscess formation, and fistulas. *Id.* (citing Resp't's Ex. A, Tab 4).[25] Dr. Liacouras noted that a diagnosis of CD is often "delayed and may take years to formally diagnose" due to the fact that "mucosal disease activity is typically present even when there are no clinical symptoms." *Id.* (citing Resp't's Ex. A, Tab 9).[26] Schoepfer et al. detailed a study wherein they observed the diagnostic delay in pediatric CD patients, finding the average delay to be approximately four months following initial symptom onset, and an average of one month from onset to the initial physician's visit. Resp't's Ex. A, Tab 9 at 2.

Dr. Liacouras noted that "there are no reports identifying that vaccinations, specifically the HPV vaccine, causes or exacerbates [IBD]." Resp't's Ex. A at 4. He cited to Skufca et al.,[27] which reviewed the effects of the HPV vaccine on over 130,000 girls in Finland and found that the HPV vaccine "was not significantly associated with an increased risk for any of the 38 outcomes," including IBD. Resp't's Ex. A, Tab 12 at 3. Dr. Liacouras also observed that "the medical literature and medical guideline advocate that patients with active IBD receive routine vaccinations, including the HPV vaccine." Resp't's Ex. A at 4 (citing Resp't's Ex. A, Tab 15).[28]

Turning to Dr. Cromwell's report, Dr. Liacouras asserted that Dr. Cromwell "did not provide adequate details on the current pathophysiology of [CD]," including that current thinking

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[23] Jennifer L. Dotson & Brendan Boyle, *Crohn's Disease*, *in* Pediatric Gastrointestinal and Liver Disease 461 (Robert Wyllie et al., eds., 6th ed. 2021).
[24] Joana Torres et al., *Crohn's Disease*, 389 LANCET 1741 (2017).
[25] Jeremy Adler et al., *Perianal Crohn Disease in a Large Multicenter Pediatric Collaborative*, 64 J. PEDIATRIC GASTROENTEROLOGY NUTRITION 117 (2017).
[26] Alain M. Schoepfer et al., *Systemic Evaluation of Diagnostic Delay in Pediatric Inflammatory Bowel Disease*, 64 J. PEDIATRIC GASTROENTEROLOGY NUTRITION 245 (2017).
[27] Jozica Skufca et al., *The Association of Adverse Events with Bivalent Human Papilloma Virus Vaccination: A Nationwide Register-Based Cohort Study in Finland*, 36 VACCINE 5926 (2018).
[28] Francis A. Farraye et al., *ACG Clinical Guideline: Preventative Care in Inflammatory Bowel Disease*, 112 AM. J. GASTROENTEROLOGY 241 (2017).

around the etiology of CD pertains to genetic abnormalities and environmental factors. Resp't's Ex. A at 4. Dr. Liacouras also contended that Dr. Cromwell had not explained "how, within [five] days of receiving the HPV vaccine, [Petitioner's] immune system was not only altered to the degree that caused intestinal abnormalities but also then resulted in the formation of a perianal abscess." *Id.* Instead, Dr. Liacouras opined that the rapidity of these events "could not occur within that short a time frame and are much more likely to be coincidental." *Id.* at 4–5. He also argued that Dr. Cromwell had failed to account for the fact that "the HPV vaccine has been vigorously studied and has demonstrated multiple years of safety," with no links between the HPV vaccine and the development or worsening of CD. *Id.* at 5. Instead, Dr. Liacouras opined that Petitioner's CD and his "buttock inflammation began some time before he noticed pain or irritation," and thus was not the result of the HPV vaccine. *Id.*

b. Supplemental Report

Dr. Liacouras' supplemental report initially focused on his points of agreement with Dr. Cromwell, namely that there have been no direct findings of HPV vaccine-induced CD in a clinical trial setting and that underlying genetic abnormalities are an important factor in the development of CD. Resp't's Ex. E at 1. He also asserted that Dr. Cromwell's points of disagreement are not cited to "any specific error, nor any published human evidence contradicting any of my conclusions." *Id.* at 2.

Next, Dr. Liacouras addressed the specific issue of onset, arguing that Dr. Cromwell's hypothesis was unproven, and that "the medical literature demonstrates that the diagnosis of [CD] is typically delayed. In other words, the majority of patients have an underlying intestinal inflammatory condition which begins months to years before symptoms occur or a diagnosis of [CD] is made." Resp't's Ex. E at 2. Dr. Liacouras noted that most patients with a definitive CD diagnosis have an average diagnostic delay of "six to seven months. In children, a decrease in weight gain is frequently a subtle early manifestation of [CD]. [Petitioner's] growth curve demonstrated a decrease in weight gain over several years." *Id.* at 2–3. Ricciuto et al.[29] documented the diagnostic delay in children with IBD, specifically noting CD to be associated "greater linear growth impairment at presentation, as reflected by significantly lower" height-for-age Z-score ("HAZ") percentiles. Resp't's Ex. E, Tab 3 at 4–5. These discrepancies persisted even one year after diagnosis, with the authors noting that for every standard deviation decrease in HAZ, the risk of delayed CD diagnosis increased twofold. *Id.* at 5–6.

Dr. Liacouras also specifically addressed the timing of the development of a perianal abscess, which he stated "takes a significant amount of time to develop," inconsistent with Petitioner's presentation. Resp't's Ex. E at 3. Although Dr. Cromwell stated that such an abscess can occur within two days of a break in mucosal integrity, Dr. Liacouras argued that "[t]his fails to address the underlying cause of the failed mucosal integrity." *Id.* He continued that "an abscess secondary to [CD] develops as a result of chronic intestinal inflammation, which . . . takes significant time to progress." *Id.* Accordingly, Dr. Liacouras opined that Petitioner developed CD "significantly earlier, long before he developed a perianal abscess," as "[t]here has been no

[29] Amanda Ricciuto et al., *Diagnostic Delay in Canadian Children With Inflammatory Bowel Disease is More Common in Crohn's Disease and Associated With Decreased Height*, 103 Archives Disease Childhood 319 (2018).

documented evidence in the medical literature that a perianal abscess can develop within a few days of the very start of [CD]." *Id.* He cited to Park et al.[30] to support this conclusion, a study which found that the average time from CD diagnosis to development of a perianal fistula to be five months, with 40% of patients (34 observations) developing "their first perianal . . . fistula before or at the time of CD diagnosis." Resp't's Ex. E, Tab 2 at 2. Excluding these findings, the study found the average time for development of a perianal fistula to be 4.4 years, with a range of six days to 22.6 years across 380 patients. *Id.*

3. Reports of Dr. McGeary

a. Initial Report

Dr. McGeary began his report by characterizing CD has an autoinflammatory disease instead of an autoimmune disease. Resp't's Ex. C at 2.

> The distinction reflects the finding that host tissues are directly targeted by antibodies or T cells in autoimmune diseases (i.e. primarily by adaptive immune mechanisms), whereas the host tissue injury in autoinflammatory diseases, results from proximity to inflammation resulting from disordered immune responses, usually originating from dysfunctions of the innate immune system.

Id. Dr. McGeary cited to both Arakelyan et al.[31] and Ciccarelli et al.[32] to support this theory, studies that both considered CD to be autoinflammatory diseases due to the widespread nature of its inflammation in the intestinal tract. *Id.* (citing Resp't's Ex. C, Tab 1; Resp't's Ex. C, Tab 2). He also generally agreed with Dr. Liacouras' description of the immunopathology of CD, specifically with regard to the contribution of genetic and environmental factors. *Id.*

Turning to Petitioner's case, Dr. McGeary opined that "there is no evidence in the clinical record to suggest that [] a dysregulated immune response to the vaccine actually occurred." Resp't's Ex. C at 2. He argued that Petitioner's four-to-five day timeline from vaccination to development of a perianal abscess was "impossible" with what is known of the formation. *Id.* He further contested Dr. Cromwell's medical theory, agreeing with him that the vaccine was designed to activate the immune system, but stating that he "offer[ed] no evidence that excessive or inappropriate activation actually occurred." *Id.* Dr. McGeary explained that due to the highly toxic nature of proinflammatory cytokines, their production is "tightly regulated by the innate and adaptive immune cells that produce them." *Id.* (citing Resp't's Ex. C, Tab 7). He argued that if such an inappropriate immune response did occur after vaccination, Petitioner would have presented with "acute symptoms, including fever, chills, respiratory distress and hypotension . . . almost immediately." *Id.* Dr. McGeary argued that in order for Petitioner's symptoms to progress

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[30] Sang Hyoung Park et al., *Update on the Natural Course of Fistulizing Perianal Crohn's Disease in a Population-Based Cohort*, 25 INFLAMMATORY BOWEL DISEASE 1054 (2019).
[31] Arsen Arakelyan et al., *Autoimmunity and Autoinflammation: A Systems View on Signaling Pathway Dysregulations Profiles*, 12 PLOS ONE 187572 (2017).
[32] F. Ciccarelli et al., *An Update on Autoinflammatory Diseases*, 21 CURRENT MEDICINAL CHEMISTRY 261 (2014).

to an abscess within four-to-five days, "one would have to hypothesize a rapid, aggressive process directly impacting [Petitioner's] GI tract," but no evidence of such a process was present. *Id.*

Dr. McGeary explained that chronic inflammation resulting from an innate immune system response only occurs "if acute inflammation fails to eliminate the originating stimulus." Resp't's Ex. C at 2. Such inflammation appears several days later, and signs of adaptive immunity even later. *Id.* at 3. Dr. Cromwell continued by explaining the process of chronic inflammation, stating that it "is characterized by infiltration of monocytes . . . and activated lymphocytes. This phase of inflammation may lead to the remodeling of tissues, and the activated lymphocytes become active participants in chronic inflammation by secreting additional pro-inflammatory cytokines." *Id.* When chronic inflammation fails to eliminate the stimulus, the adaptive immune system is activated through the production of T cells and B cells, which may result in further inflammation. *Id.* (citing Resp't's Ex. C, Tab 8).[33] Given these mechanisms, Dr. McGeary opined that "as a chronic inflammatory disease that presented in [Petitioner] . . . could not have developed to this point in the [four-to-five] day period between the receipt of [the vaccine] on [August 3, 2016,] and the appearance of signs of the abscess." *Id.*

Dr. McGeary next addressed Dr. Cromwell's specific medical theory, noting that the NSEs documented in Dr. Cromwell's cited studies "are from non-Gardasil 9 epidemiological studies, or from work in experimental animals or from vaccines unrelated to Gardasil 9." Resp't's Ex. C at 3. He continued by arguing that Dr. Cromwell had not explained how these studies were relevant to Petitioner's case and that Petitioner's medical record "does not support any untoward reaction to the vaccine, either at the injection site or in systemic symptoms." *Id.* "The onset of CD [four-to-five] days following the immunization is inconsistent with the kinetics of immune responses." *Id.*

b. First Supplemental Report

Dr. McGeary began his first supplemental report by addressing Dr. Cromwell's assertion that he had failed to provide any evidence discussing the timeline progression of CD from the exposure to a CD trigger to "development of the ensuing suppurative response." Resp't's Ex. F at 1. Dr. McGeary explained that there are no known triggers that have been directly linked to IBD, and thus, "there is no medical evidence to pinpoint the time between exposure to a theoretical CD trigger and the symptomology seen here." *Id.* "While the time for development of CD with abscess formation is not known . . . CD often develops over months or years before symptoms appear." *Id.* Dr. McGeary also reiterated that Petitioner's presentation was inconsistent with the kinetics of the development of an immune response and rejected Dr. Cromwell's argument that "factors previously associated with [IBD] may be shown in the future to cause it" as conjecture. *Id.* at 1, n.1.

Dr. McGeary next addressed Dr. Cromwell's reliance on LeBlanc et al., which Dr. McGeary argued had "significant differences between that experimental system and the proposed immunopathology that led to [Petitioner's] development of CD." Resp't's Ex. F at 1. He explained that in that study TNBS, not the HPV vaccine, was administered directly into the rectum of mice, and the ensuing inflammation produced a mouse model of CD. *Id.* As described by the authors,

[33] ABUL K. ABBAS, ANDREW H. H. LICHTMAN & SHIV PILLAI, CELLULAR AND MOLECULAR IMMUNOLOGY INTERNATIONAL EDITION 82 (9th ed. 2018)

"TNBS is a covalently reactive compound, and its administration in mice results in acute necrosis of the wall of the distal colon due to oxidative damage." *Id.* (citing Pet'r's Ex. 22). Dr. McGeary argued that

> [i]t is impossible to reliably extrapolate the rate of development of inflammation in a mouse model, in which a chemical insult was delivered directly to the mucosa of the distal colon, to the expected kinetics of an immunologically-based injury in humans from a vaccine injected as a site remote from the gastrointestinal tract.

Id.

Dr. McGeary also reiterated his original argument that activation of the adaptive immune system by a vaccine not previously encountered would not expect to generate a reaction sufficient enough to cause a tissue injury via T-cells or B-cell antibodies. Resp't's Ex. F at 2. He clarified his original discussion of cytokines, explaining that a "cytokine storm" was not required for the innate immune system to be implicated in the development of CD; rather, cytokines are generally toxic, and he would expect Petitioner to have suffered from flu-like symptoms such as fever "if cytokines were to have been released systemically from a site remote from the gastrointestinal tract." *Id.* "Since that did not happen, [he] would opine that the activation of the innate immune system . . . was contained locally within [Petitioner,] . . . and did not elicit any systemic symptoms, as would be necessary to cause his CD." *Id.*

c. Second Supplemental Report

Dr. McGeary began his second supplemental report by addressing Dr. Cromwell's theory of molecular mimicry and casted doubt on the theory as a viable mechanism for autoimmune injuries in humans. Resp't's Ex. G at 1. He cited to Oldstone,[34] who summarized the state of molecular mimicry research in 2005 as follows:

> In many instances, hard data derived in experimental systems clearly indicate molecular mimicry as a mechanism for disease causation. For others, especially human disorders, the evidence can be strongly suggestive, but additional information is required before molecular mimicry can be accepted or rejected as biological reality.

Resp't's Ex. G, Tab 1 at 13. He also cited to Rose,[35] who, in a paper addressing potential molecular mimicry in myocarditis, noted that apart from a select few instances, the vast majority of instances still showed no evidence "that the antigenic mimic is capable of inducing an autoimmune disease." Resp't's Ex. G, Tab 2 at 4. Although Dr. Cromwell cited to Kanduc et al. to show potential mimics between HPV16 and proteins located in the human gastrointestinal tract, Dr. McGeary contended that there is "no evidence that these 'mimics' are capable of causing disease." Resp't's Ex. G at 1. Dr. McGeary also reiterated his contention that CD is an autoinflammatory condition as opposed

[34] M. B. A. Oldstone, *Molecular Mimicry, Microbial Infection, and Autoimmune Disease: Evolution of the Concept*, 296 CURRENT TOPICS MICROBIOLOGY IMMUNOLOGY 1 (2005).
[35] Noel R. Rose, *Learning from Myocarditis: Mimicry, Chaos and Black Holes*, 6 F1000 PRIME REP. 25 (2014).

to an autoimmune condition, and that "[i]t would be a novel construct to apply [molecular mimicry] to autoinflammation, and such an application has not been made for CD." *Id.* at 2.

Dr. McGeary noted that the authors of Kanduc et al. had also studied the prevalence of mimics in nature in other areas and found "massive" peptide sharing between 30 viral proteomes and the human proteome. Resp't's Ex. G at 2 (citing Resp't's Ex. G, Tab 3).[36] The study "call[ed] into question the possibility of a direct causal association between virus-host sharing of amino acid motifs and incitement of autoimmune reactions" due to the high prevalence of mimics between the viral and human proteomes. Resp't's Ex. G, Tab 3 at 19. Specifically,

> the molecular mimicry hypothesis implies that viral infections should be a practically infinite source of autoimmunity diseases since this study demonstrates that viral 5-mer matches are disseminated throughout practically all the human proteome and each viral match is repeated almost more than 10 times . . . . Consequently, autoimmune diseases should theoretically approach a 100% real incidence, since the 30 viruses [] examined practically are more or less disseminated throughout the entire human species.

Id. Accordingly, Dr. McGeary argued that the authors clearly did not believe mimics lead to autoimmune disease, as proposed by Dr. Cromwell. Resp't's Ex. G at 2. Dr. McGeary explained this may be due to the difference in three-dimensional configuration of the proteins within different species, as "[r]esearch has shown that similarity in three dimensional structure may be more important in ligation of the T-cell receptor than amino acid sequence." *Id.* (citing Resp't's Ex. G, Tab 5).[37] Dr. McGeary also reiterated his previous argument that Petitioner did not suffer from an abnormal cytokine response which led to his CD, as evidenced by his lack of other symptoms following vaccination. *Id.* at 2–3.

V. Applicable Legal Standards

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) that the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of her receiving a covered vaccine. *See* § 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). In this case, CD is not a Table injury associated with the HPV vaccine, and thus Petitioner must prove by preponderant evidence that his injury was caused-in-fact by a Table vaccine.

A. Factual Issues

[36] Daria Kanduc et al., *Massive Peptide Sharing Between Viral and Human Proteomes*, 29 PEPTIDES 1755 (2008).

[37] Bernhard Hemmer et al., *Cutting Edge: Predictable TCR Antigen Recognition Based on Peptide Scans Leads to the Identification of Agonist Ligands with No Sequence Homology*, 160 J. IMMUNOLOGY 3631 (1998).

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). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993); *but see Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejection the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 538 (2001) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance").

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 the Dep't of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (Fed. Cl. 1991)). Ultimately, a determination regarding a witness' 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.").

B. Causation-In-Fact

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999).

In the seminal case of *Althen v. Sec'y of Health & Hum. Servs.*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). The *Althen* test requires petitioners to set forth: "(1) a medical theory causally connecting the vaccination and the

injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano*, 440 F.3d at 1326.

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. den'd*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278 – 81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1360 (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322; *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde*, 746 F.3d at 1339.

Furthermore, establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). The Supreme Court's opinion in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579 (1993) requires that courts determine the reliability of an expert opinion before it may be considered as evidence. However, in the Vaccine Program, the *Daubert* factors are used in the weighing of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (2010) ("[U]niquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to the persuasiveness of expert testimony already admitted."); *see also Cedillo v. Sec'y of health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). Under *Daubert*, the

> Factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.

Terran, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors are "meant to be helpful, not definitive." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 151 (1999). The factors do not "constitute a 'definitive checklist or test'" and may be applied differently depending on the facts of a particular case. *Id.* at 150 (quoting *Daubert*, 509 U.S. at 593).

"In short, the requirement that an expert's testimony pertain to 'scientific knowledge' establish a standard of evidentiary reliability." *Daubert*, 509 U.S. at 590 (citation omitted). Thus, for Vaccine Act claims, a "special master is entitled to require some indicia of reliability to support the assertion of the expert witness." *Moberly*, 592 F.3d at 1324. Nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Synder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); *see also D'Tiole v. Sec'y of Health & Hum. Servs.*, No. 15-085V, 2016 WL 7664475, at *24 (Fed. Cl. Spec. Mstr. Nov. 28, 2016) (stating that the Vaccine Act "require[s] a chain of reliable propositions supporting [a] petitioner's theory[]").

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed. Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibily raises a claimant's burden under the Vaccine Act and hinders the system created by Congress . . . ." *Capizzano*, 440 F.3d at 1325–26. "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen* 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians.

Capizzano, 440 F.3d at 1326. This is because "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.'" *Id.* In addition, "[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events." *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). However, there is no "presumption that medical records are accurate and complete as to all of the patient's physical conditions." *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021) (finding that a special master must consider the context of a medical encounter before concluding that it constitutes evidence regarding the absence of a condition). While a special master must consider these opinions and records, they are not "binding on the special master or court." § 300aa-13(b)(1). Rather, when "evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . ." *Id.*

In determining the accuracy and completeness of medical records, special masters will consider various explanations for inconsistencies between contemporaneously created medical records and later given testimony. The Court of Federal Claims has identified four such explanations for explaining inconsistencies: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting with testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *LaLonde v. Sec'y of health & Hum. Servs.*, 110 Fed. Cl. 184, 203 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014).

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to finger causation-in-fact." *de Bazan v. Sec'y of health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Hum. Servs.*, 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that she suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen*, 35 F.3d at 547. In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Kundsen*, 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden the shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan*, 539 F.3d at 1354; *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

VI. Analysis

A. Symptom Onset

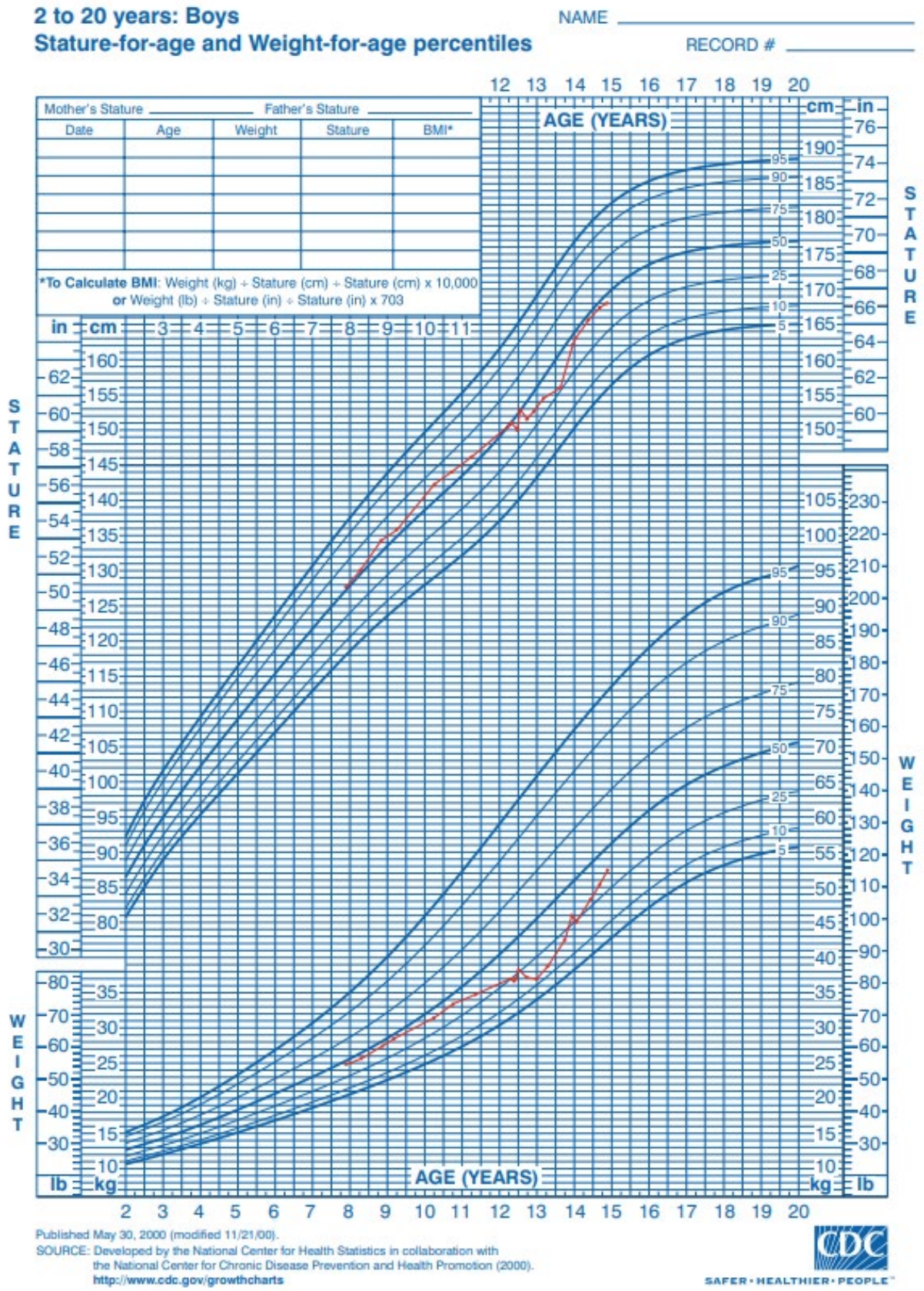
The parties agree that Petitioner suffered from CD, but they dispute whether his symptoms likely began before or after vaccination. Petitioner's medical records prior to and including his August 3, 2016 12-year-old well-child visit noted no significant problems from his providers, and Petitioner did not report any symptoms of CD to his providers until his August 15, 2016 PCP visit. Petitioner contends that this temporal association is supportive of a vaccine-induced CD injury, whereas Respondent argues that such an injury is not physically possible in such a short time after vaccination. Respondent's expert, Dr. Liacouras, specifically notes that Petitioner's growth trends prior to vaccination showed he was trending downward, which he argues is indicative of CD manifestation prior to vaccination.

A thorough review of the case record reveals Petitioner has not provided preponderant evidence that his CD symptoms began after his August 3, 2016 HPV vaccination. Although Petitioner had no noted symptoms in his records prior to his August 15, 2016 PCP visit, I find Dr. Liacouras' argument that Petitioner's slow growth prior to vaccination was indicative of CD to be persuasive, especially when taken in the context of his prior growth trends. Petitioner's submitted medical records document healthcare visits dating back to January 10, 2012, at which his height and weight were regularly measured. Analyzing these trends in accordance with the Centers for Disease Control's ("CDC") Height and Weight for Age and Body Mass Index ("BMI") for Age charts shows a significant change in Petitioner's growth curve that was demonstrated prior to his receipt of the relevant HPV vaccine. From January 10, 2012, to December 30, 2014, Petitioner consistently held between the 49th and 44th percentiles in weight, the 64th and 59th percentiles for height, and the 35th and 27th percentiles for BMI.[38] *See generally* Pet'r's Ex. 1. At Petitioner's

[38] All percentile references were made using the CDC metrics for boys aged two to 20, which can be found here: https://www.cdc.gov/growthcharts/cdc-growth-charts.htm.

11-year-old well-child visit on July 28, 2015, he weighed 34.9kg (36th percentile), at a height of 146.05cm (55th percentile) with a BMI of 16.3 (30th percentile). *Id.* at 9. This is a pre-vaccination, notable deviation from his prior growth trajectory records. Petitioner's growth had slowed even more by the date of vaccination on August 3, 2016, where he weighed 37kg (24th percentile), at a height of 150.5cm (50th percentile) with a BMI of 16.3 (20th percentile). *Id.* at 5. As Dr. Liacouras noted in his opinion, "a decrease in weight gain is frequently a subtle early manifestation of [CD]." Resp't's Ex. E at 2. If, as Petitioner asserts, his CD developed *after* vaccination, his most drastic growth decline should have manifested as of his August 15, 2016 visit, the first one post vaccination and coinciding with his initial report of symptoms. However, this visit's records noted a continuation of his already declining growth and are not as drastic as the first significant drop recorded on July 28, 2015. Below are Petitioner's growth trends documented from January 10, 2012, to February 26, 2019, as well as this data plotted on the CDC's Height for Age, Weight for Age, and BMI for Age charts.

Date	**Weight (kg)**	**Weight %**	**Height (cm)**	**Height %**	**BMI**	**BMI %**	**Pet'r's Ex.**
1/10/2012	24.9	49	128.27	63	15.1	35	1 at 97
6/8/2012	25.85	48	130.81	63	15.1	31.4	1 at 90
2/12/2013	27.66	47	134.62	63	15.3	30.4	1 at 86
6/11/2013	28.57	46	135.89	59	15.5	32.6	1 at 81
7/9/2014	31.75	42	142.24	64	15.7	27.8	1 at 18
12/30/2014	33.56	44	144.15	61	16.2	33.3	1 at 14
7/28/2015	34.9	36	146.05	55	16.4	30.6	1 at 10
8/3/2016 (Vaccination)	**37**	**24**	**150.5**	**46**	**16.3**	**20.4**	**1 at 6**
8/15/2016	36.74	23	151.13	50	16.1	16.4	1 at 4
9/22/2016	37.1	23	149.86	40	16.5	22.7	1 at 1
10/25/2016	38.4	26	153	50	16.4	19.3	3 at 218
12/5/2016	37.3	19	151.5	39	16.3	16.2	4 at 40
3/6/2017	36.7	13	152.5	35	15.8	8.5	4 at 67
6/2/2017	38.8	16	154.5	36	16.3	12.4	7 at 556
12/21/2017	42.6	22	156	25	17.5	26.8	6 at 143
3/26/2018	46.35	30	162.2	42	17.6	25.2	7 at 537
4/16/2018	45	25	163	46	16.9	15.1	6 at 227
8/9/2018	48.4	32	165.6	47	17.6	22.4	7 at 463
12/3/2018	50.6	34	167.2	45	18.1	26.1	7 at 29
2/26/2019	52.4	36	168	42	18.6	30.9	11 at 287


2 to 20 years: Boys
Stature-for-age and Weight-for-age percentiles
NAME
RECORD #
Mother's Stature
Father's Stature
Date
Age
Weight
Stature
BMI*
*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703
AGE (YEARS)
STATURE
WEIGHT
95
90
75
50
25
10
5
Published May 30, 2000 (modified 11/21/00).
SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts
SAFER · HEALTHIER · PEOPLE

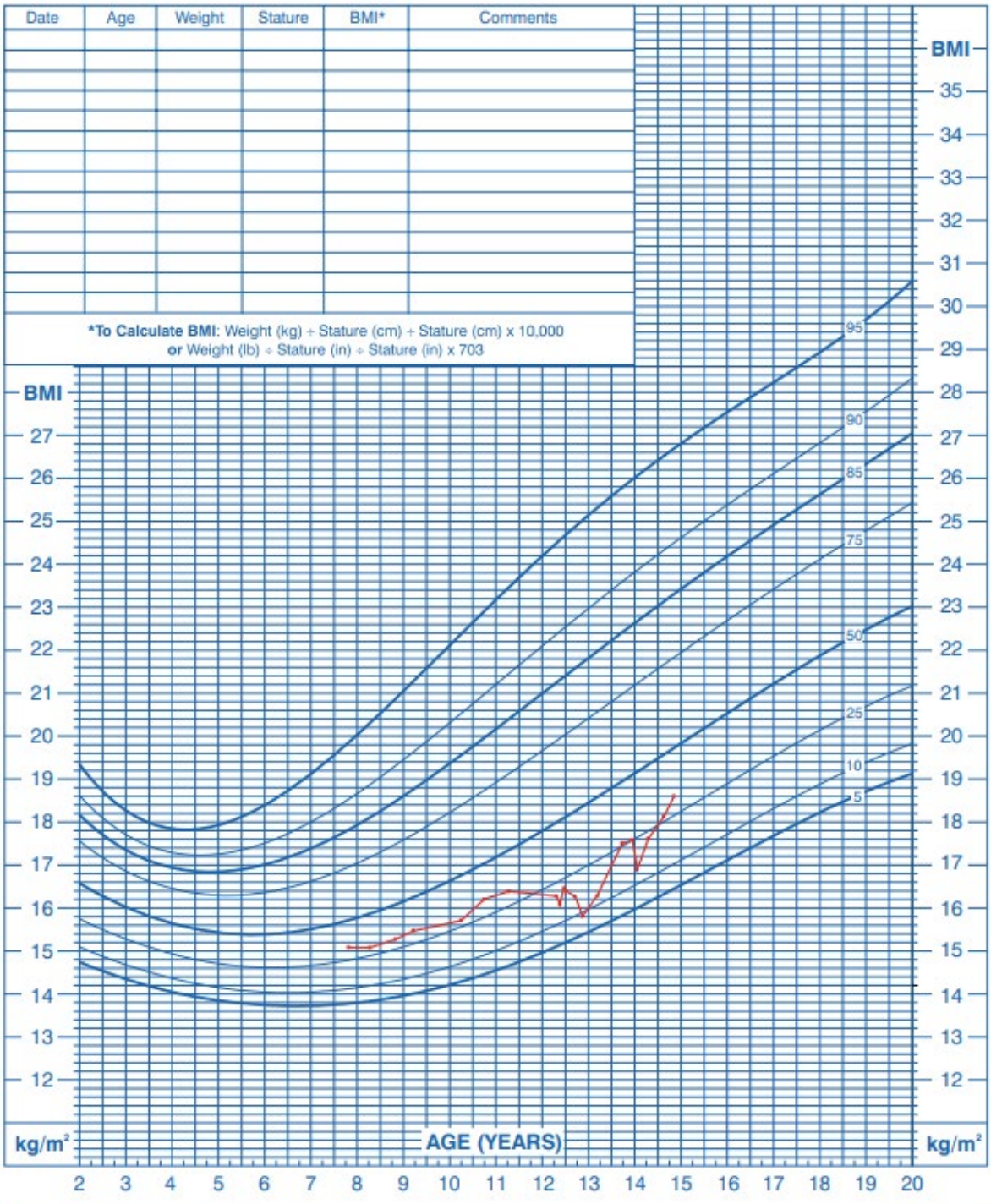
2 to 20 years: Boys
Body mass index-for-age percentiles

NAME _____

RECORD # _____

Date	Age	Weight	Stature	BMI*	Comments

*To Calculate BMI: Weight (kg) ÷ Stature (cm) ÷ Stature (cm) x 10,000
or Weight (lb) ÷ Stature (in) ÷ Stature (in) x 703



Published May 30, 2000 (modified 10/16/00).

SOURCE: Developed by the National Center for Health Statistics in collaboration with the National Center for Chronic Disease Prevention and Health Promotion (2000).
http://www.cdc.gov/growthcharts

CDC
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These findings are consistent with the Ricciuto et al. article which found that individuals suffering from delayed diagnosis CD presented with significantly impaired linear growth curves, reflected by significantly lower height-for-age scores. Resp't's Ex. E, Tab 3 at 4. Petitioner demonstrated a noticeable decline in his growth rates prior to receiving the vaccination, beginning with his July 28, 2015 well-visit and continuing at his August 3, 2016 well-visit. Although Dr. Liacouras briefly addressed this point in his supplemental report by stating that Petitioner's already declining growth trends were indicative of pre-existing CD, he did not file much supporting medical literature to explain his reasoning and did not provide a specific analysis of Petitioner's individual growth patterns. It is not the Court's obligation to accept unsupported conclusions by an expert. *See Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 743 (2009) (explaining that nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert") (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997); *see also Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d at 1328, 1339 (Fed. Cir. 2010); *Hughes v. Sec'y of Health & Hum. Servs.*, No. 16-930V, 2021 WL 839092, at *23 (Fed. Cl. Spec. Mstr. Jan. 4, 2021). Although Dr. Liacouras did not explain the significance of Petitioner's growth trends as well as he should have, the evidentiary record establishes Petitioner's growth trends. Further, the CDC Growth Charts carry substantial authority when analyzing this data, as evidenced by authors such as Ricciuto et al. relying on them to form the basis of their analysis.

Here, the medical record clearly reflects that Petitioner was suffering from weight loss and slowed growth prior to his receipt of the HPV vaccine. The CDC Growth Charts hold I also find it particularly persuasive that following Petitioner's receipt of the vaccine, his growth trends did not substantially worsen from the already significant drop-off he had been experiencing; and his eventual improvement returned him to growth levels recorded prior to his July 28, 2015 PCP visit.[39] This is consistent with the theories proposed by Dr. Liacouras and Dr. McGeary that Petitioner had been suffering from ongoing, undetected inflammation in his intestine prior to the receipt of the HPV vaccine, which then later developed into a perianal abscess.

Accordingly, there is not preponderant evidence that Petitioner's symptoms began after vaccination, and I find Petitioner's arguments to the contrary unpersuasive.

B. *Althen* Prong One

Under *Althen* prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused or 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*, 941 F.3d at 1359; *see also Knudsen*, 35 F.3c 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"), *aff'd* 475 F. App'x 765 (Fed. Cir. 2012). If Petitioner relies upon a medical opinion to support her theory, the basis for the

[39] Petitioner's weight, height, and BMI percentiles on August 3, 2016 were recorded in the 24th, 46th, and 20th percentiles, respectively. Petitioner did not significantly deviate from these percentiles until December 5, 2016, when he was recorded in the 19th, 39th, and 16th percentiles, and then later in the 13th, 35th, and 8th percentiles. Petitioner's growth then improved, returning him to his prior healthy growth trends in the 36th, 42nd, and 30th percentiles by February 26, 2019.

opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. *See Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision oftentimes is based on the credibility of the experts and the relative persuasiveness of their competing theories"); *Perriera 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 (Ct. Cl. 1980))).

Although Petitioner's inability to establish that his symptoms began post vaccination precludes any entitlement to compensation, Petitioner presented evidence in support of his argument for vaccine causation that will also be considered for completeness. Notably, Dr. Cromwell's medical theory shifted over the course of his opinions, first arguing that the NSEs of vaccines caused a dysregulated immune response, and then arguing that molecular mimicry caused Petitioner's injury through a secondary cytokine reaction. Dr. Cromwell noted that his initial theory of CD resulting from the NSEs of vaccines was accepted by another special master in a case alleging ulcerative colitis. *See Morgan*, 2015 WL 9694667. However, Dr. Cromwell's reliance on the acceptance of his theory in *Morgan* is misplaced here. Although both ulcerative colitis and CD are forms of IBD, they are not the same condition. Further, the special master in *Morgan* went to great lengths to explain how other literature specific to the development of ulcerative colitis, in addition to Dr. Cromwell's NSE argument, were persuasive in satisfying the first *Althen* prong. *See Morgan*, 2015 WL 9694667, at *10–11 (explaining that the medical literature found that "non-genetic factors may have an even more important role in ulcerative colitis than [CD]" and relying on "several other reports of ulcerative colitis after Gardasil vaccines in the VAERS database"). It cannot be that Dr. Cromwell can simply wave the wand of 'NSEs resulting from vaccines' and create a viable medical theory with nothing more to link the specific vaccine to the alleged injury. Although the special master's decision in *Morgan* reveals that Dr. Cromwell filed much of the same literature in that case as here, his argument is not specific to CD and is therefore much less persuasive. His expert reports place too much reliance on the acceptance of his theory in *Morgan*, without explanation of why the two claims, and more specifically the alleged conditions, are analogous to each other with respect to pathology. Accordingly, Dr. Cromwell's theory of vaccine-induced NSEs causing Petitioner's CD is insufficient to satisfy the first *Althen* prong.

Similarly, I find Dr. Cromwell's theory of molecular mimicry to be insufficient to satisfy the first *Althen* prong. Although I do not find Dr. McGeary's arguments refuting molecular mimicry generally to be determinative under the standard used in the Program, Dr. Cromwell does not present sufficient prima facie evidence to connect the HPV vaccine to CD via a molecular mimicry mechanism. Dr. Cromwell cites to specific sources identifying specific peptide similarities between HPV16 and the human intestine but does not explain how this homology could lead to pathogenesis beyond the mere statement of molecular mimicry. Further, Dr. Cromwell's explanation of the three-dimensional shape problem raised by Dr. McGeary does not adequately explain the lack of incidence rate or even a single case study involving the HPV vaccine and CD, and he relies solely on his own explanations to support his claim. Accordingly, I find Petitioner has not presented a sound and reliable medical explanation and thus has not satisfied the first *Althen* prong.

C. *Althen* Prong Two & Prong Three

Although these prongs traditionally warrant separate analysis, I will address the second and third *Althen* prongs together for the sake of brevity. 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). *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 defined as a "medically acceptable temporal relationships." *Id.* Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. 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 v. Sec'y of Health & Hum. Servs.*, 773 F.3d at 1239, 1243 (Fed. Cir. 2014); *Shapiro*, 101 Fed. Cl. at 542; *see Pafford*, 451 F.3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does constitute preponderant evidence of vaccine causation. *See, e.g., Veryzer*, 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that [P]etitioner must posit a medical theory causally connecting the vaccine and injury").

In the present case, as discussed above, Petitioner has failed to provide preponderant evidence that the onset of his symptoms began after vaccination. Accordingly, it is not possible for Petitioner to present a logical sequence of cause and effect, as evidenced by his clinical presentation or medical records, that he was developing vaccine-caused CD. Similarly, the evidence in this case illustrates a timeframe for Petitioner's symptom onset that cannot be consistent with but-for vaccine-causation, as I found his symptoms began to develop prior to vaccination. Accordingly, Petitioner fails both the second and third *Althen* prongs.

VII. Conclusion

After a careful review of the record, Petitioner has failed to provide preponderant evidence that his August 3, 206 HPV vaccine caused or significantly aggravated his CD. Accordingly, Petitioner's claim is **DENIED.** Absent a timely motion for review, the Clerk is directed to enter judgment dismissing this case for insufficient proof in accordance with Vaccine Rule 11(a).[40]

[40] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.

IT IS SO ORDERED.

s/Herbrina D. S. Young
Herbrina D. S. Young
Special Master