In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-1144V

Filed: October 2, 2025

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KATHRYN NELSON,

Petitioner,

v.

SECRETARY OF HEALTH AND HUMAN SERVICES,

Respondent.

* *

Kathleen Loucks, Lommen Abdo Law Firm, Minneapolis, MN, for Petitioner;
James Lopez, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION DENYING ENTITLEMENT[1]

Shah, Special Master:

On August 6, 2018, Kathryn Nelson ("Petitioner" or "Ms. Nelson") filed a petition for compensation under the National Vaccine Injury Compensation Program, 42 U.S.C. § 300aa-10, *et seq*.[2] (the "Vaccine Act" or "Program"), alleging that she suffered from Guillain-Barré syndrome ("GBS") as a result of the influenza ("flu") vaccine she received on October 21, 2015. Pet. at 1.

[1] Because this Decision contains a reasoned explanation for the action 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 (2012).

For the reasons discussed in this decision, I conclude that Petitioner has failed to establish entitlement to compensation. First, Petitioner has not proven a Table injury, because the evidence shows the onset of her GBS was the same day as the subject flu vaccination. Second, Petitioner has not met her burden of preponderantly demonstrating the flu vaccine was a cause-in-fact of her GBS. Her GBS was more likely caused by a *Campylobacter jejuni* ("*C. jejuni*") infection, and its onset was too soon to causally implicate the vaccine. Accordingly, the petition is dismissed.

I. PROCEDURAL HISTORY

Petitioner filed medical records and an affidavit on August 6 and 10, 2018, and January 17, 2019. ECF Nos. 1.2, 8-11, 29, 31. On April 26, 2019, Respondent filed a status report identifying additional outstanding medical records. ECF No. 36. Petitioner filed additional records on June 7 and July 25, 2019. ECF No. 38.

On October 28, 2019, Respondent filed his Rule 4(c) report, arguing that this case is not appropriate for compensation because preponderant evidence supports *C. jejuni* infection as the alternative cause of Petitioner's GBS. ECF No. 50 ("Report") at 19.

Petitioner filed additional medical records on November 20, 2019. ECF No. 51. Respondent filed a status report on January 10, 2020, stating that he was unwilling to engage in informal settlement negotiations. ECF No. 56.

On April 14, 2020, Petitioner filed an expert report from Praful Kelkar, M.D., along with his curriculum vitae and supporting medical literature. Exs. 40-49. Respondent filed an expert report, curriculum vitae, and literature from Kathleen Collins, M.D., Ph.D., on November 20, 2020. Exs. A, Tabs 1-17, Ex. B. Dr. Kelkar submitted a supplemental expert report and literature on March 9, 2021, and a letter further summarizing his opinion on November 3, 2021. Exs. 50-57. Dr. Collins submitted a supplemental expert report on July 6, 2021. Ex. C. On February 15, 2022, Petitioner filed a second supplemental report and additional literature from Dr. Kelkar. Exs. 58-70.

On October 5, 2021, former Special Master Katherine E. Oler held a status conference with the parties at which she stated her preliminary opinion on the case. ECF No. 76. Special Master Oler stated that two major issues with Petitioner's claim were (1) she tested positive for *C. jejuni*; and (2) Petitioner's back pain appeared to have started within just 48 hours of receiving the allegedly causal flu vaccine. *Id.* She recommended that Petitioner consider dismissing her case. *Id.*

On November 4, 2021, Petitioner filed a status report requesting a fact ruling limited to "the sole issue as to whether the *C. jejuni* infection and the vaccine both contributed to cause GBS." ECF No. 79. Special Master Oler held another status conference on November 10, 2021, at which she explained that a fact ruling would not resolve the question of the cause(s) of Petitioner's GBS. ECF No. 80. Petitioner's counsel stated that Petitioner wished to file an additional expert report, which she did on February 15, 2022. *Id.*; Ex. 58. Respondent filed an additional expert report on June 17, 2022. Ex. D, Tabs 1-3.

On August 22, 2022, Petitioner filed a motion for a ruling on the record ("Motion"). ECF No. 87. Respondent filed a response ("Response") on October 21, 2022, and Petitioner filed a reply ("Reply") on December 5, 2022. ECF Nos. 88, 89. Respondent filed a sur-reply ("Resp't's Sur-Reply") on January 20, 2023, and Petitioner filed her own sur-reply ("Pet'r's Sur-Reply") on February 16, 2023. ECF Nos. 91, 92. On March 2, 2023, the parties filed a joint status report indicating that the record was complete. ECF No. 93.

After further review of the record, on March 29, 2024, Special Master Oler issued an order directing the parties to file supplemental expert reports addressing several questions relating to the issues of onset and alternative causation. ECF No. 95. She also directed Petitioner to file a supplemental declaration. *Id.*

On April 29, 2024, Petitioner filed her declaration. Ex. 71. On June 27, 2024, Respondent filed a report and literature from Dr. Collins addressing Special Master Oler's questions; the next day, Petitioner filed a report from Dr. Kelkar responding to those questions. Ex. E, Tabs 1-7; Ex. 72.

On August 13, 2024, this case was reassigned to my docket. ECF No. 102. The case is now ripe for adjudication.

II. FACT EVIDENCE

A. Pre-Vaccination Medical History

Petitioner was 80 years old at the time of the subject flu vaccination. She had an extensive pre-vaccination medical history that was significant for gastroesophageal reflux disease ("GERD"), hypertension, gout, hyperlipidemia, multinodular goiter, macular degeneration, osteoarthritis of the knees, hips, and left foot, juvenile rheumatoid arthritis, Sjogren's syndrome with parotitis, cervical spinal stenosis, osteoporosis, diverticulosis, carpal tunnel syndrome requiring surgery, Lyme disease, and left retinal artery occlusion. Ex. 16 at 10-15; Ex. 21 at 78-80, 85-86, 89; Ex. 27 at 2; Ex. 29 at 63; *see also* Ex. 71 at 1 (Petitioner's Affidavit).

In September 2015, Petitioner sustained a left toe fracture after falling down a bluff. *See* Ex. 19 at 9; Ex. 36 at 116. She had surgery to reduce the fracture on September 23, 2015. Ex. 37 at 1-2. At discharge, she was given prescriptions for Vicodin, Vistaril,[3] ibuprofen, and Senokot. Ex. 19 at 44.

On October 8, 2015, Petitioner had a follow-up visit with her foot surgeon. Ex. 37 at 3. She reported no pain but had some swelling and tenderness with motion of the injured foot. *Id.* A

[3] Vistraril: trademark for preparations of hydroxyzine. DORLAND'S MEDICAL DICTIONARY ONLINE ("DORLAND'S"), https://www.dorlandsonline.com/dorland/definition?id=53275 (last accessed on September 18, 2025); Hydroxyzine: a piperazine derivative with central nervous system depressant, antispasmodic, antihistaminic, and antifibrillatory actions. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=23563 (last accessed on September 18, 2025).

peripheral neurovascular exam revealed "intact sensation to light touch and intact gross motor function." *Id.* Her sutures were removed, and she was referred to physical therapy ("PT"). *Id.*

On October 21, 2015, Petitioner saw podiatrist Christina Knutson, D.P.M., of the Stillwater Medical Group, for bilateral foot numbness. Ex. 36 at 137-39. At the time of the visit, she was wearing a surgical shoe following her surgery. *Id.* at 137. Dr. Knutson recorded a history of midfoot arthritis for which Petitioner had received past corticosteroid injections. *Id.* Petitioner reported that she had experienced numbness in all toes on both feet, particularly the central toes. *Id.* She relayed that an earlier EMG had showed latency in the peroneal nerve,[4] but she did not have numbness in that region. *Id.* She described the numbness in her feet as "bothersome" but not particularly painful. *Id.* It had not worsened. *Id.* She was concerned she might have a neuroma.[5] *Id.*

On exam, Petitioner had normal strength but had pain in the second and third intermetatarsal spaces of both feet, with the left foot more painful. Ex. 36 at 137. She had bilateral hammertoe deformities of the second toe. *Id.* She had no pain with palpation but had restricted range of motion of the midfoot. *Id.* She had a negative "Mulder click."[6] *Id.* Dr. Knutson's differential included neuroma and peripheral neuropathy. *Id.* at 138. Possible causes of Petitioner's numbness included hypothyroidism, mechanical or hereditary neuropathy, vitamin deficiency, or unknown etiology. *Id.* Dr. Knutson administered injections of Marcaine and dexamethasone into the feet. *Id.* The goal was to rule out neuroma as the cause of the numbness. *Id.* She advised Petitioner to monitor her symptoms for 10 days and, if her numbness improved, consider receiving an injection into the second intermetatarsal space. *Id.*

Petitioner received the subject flu vaccination during her visit with Dr. Knutson on October 21, 2015. Ex. 1 at 4.

B. Post-Vaccination Medical History

According to Petitioner's supplemental declaration, she drove from Minnesota to Florida on about October 27-28, 2015. Ex. 71 at 1.

[4] Peroneal nerve: The peroneal nerve is on the outside of the fibula just below the knee. Pressure to the peroneal nerve, as you might experience if you sit with your legs crossed for too long, can trigger temporary foot drop. MAYO CLINIC, https://www.mayoclinic.org/diseases-conditions/foot-drop/multimedia/peroneal-nerve/img-20008172 (last accessed September 18, 2025).

[5] Neuroma: a tumor growing from a nerve or made up largely of nerve cells and nerve fibers. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=33770 (last accessed September 17, 2025).

[6] Mulder sign: when the foot is compressed from the sides and the involved distal metatarsal space is digitally compressed, if a Morton neuroma is present the involved nerve will snap below the metatarsal heads, sometimes making an audible click. Called also Mulder click. DORLAND'S, https://www.dorlandsonline.com/dorland/definition?id=106378 (last accessed September 17, 2025).

Eight days after the vaccination, on October 29, 2015, at 4:15 a.m., Petitioner was seen in the emergency room ("ER") of Venice Regional Medical Center ("VRMC") in Florida, for nausea and sharp, non-radiating pain in her left mid-back since 3 a.m. the previous night, October 28. Ex. 29 at 6. She had taken an oxycodone the previous evening for the pain. *Id.* She denied vomiting, abdominal pain, or diarrhea. *Id.* at 11. She reported that she "[j]ust drove here from Minnesota." *Id.*

Petitioner's examination was unremarkable, and the ER physician suspected her symptoms were musculoskeletal in origin. Ex. 29 at 13. A CT scan of the lumbar spine showed a possible large hemangioma in the T12 vertebral body with a "corduroy appearance."[7] *Id.* at 22. Petitioner was discharged home with a diagnosis of back pain and nausea/vomiting. *Id.* at 15. She was given prescriptions for Zofran for nausea, Percocet for pain, and Colace, a laxative. *Id.*

At 2:45 p.m. on October 30, 2015, Petitioner saw her Florida primary care physician ("PCP"), Christopher Jefferson, M.D., in the office. Ex. 21 at 87. Dr. Jefferson's notes reflect the following:

> Patient is here today after follow-up in the emergency room. When she was there she was nauseous she was vomiting she had a significant pain in her back and radiated to her back I thought maybe it was pancreatitis her (sic) UTI at that time so we sent her over to the emergency room to get looked at the urine had a couple of cells in it but was minimally affected but it did not look like a urine infection. And she had a pancreatic enzymes which were negative she also had a CAT scan that was done as well that did not show anything significant as well. She's here today to have a follow-up exam done and to see if there is anything else that could be done to improve her symptoms. ***Pain in the back was first. Had 1 day of dry heaves for 24 hours then gone. It was about a month ago. 3 am a couple of days ago, took a [pain] med as well, Pepcid AC in the car getting down here. Again in the morning Prilosec and Amoxicillin as well had taken for the stomach as well. Took something else for the back pain and took her Prednisone***[8] ***as well for 6 days. Everything seemed to get worse when she did the prednisone about 4 days later which makes me think she may have an ulcer there.*** Organ [sic] put her on Dexilant twice a day Carafate 4 times a day and give her ondansetron pills to take and as a Phenergan backup as well at this time and I will give her a call on Monday and see how she [is] doing at that time. We'll also be able to get the urine culture that was done in the emergency room and should have it's [sic] final report back by then as well.

Ex. 21 at 87 (emphasis added). Dr. Jefferson noted that Petitioner had received "[a] recent immunization for flu[;] Doctor up north." *Id.* at 88. A Review of Systems ("ROS") indicated that she had nausea and abdominal pain but no diarrhea or chronic constipation. *Id.* at 90.

[7] A CT scan of the abdomen and pelvis showed chronic diverticular disease in the colon but was otherwise normal. Ex. 29 at 23.

[8] It is unclear when or for what purpose Petitioner was prescribed prednisone.

On exam, Petitioner had a temperature of 99.7°F. Ex. 21 at 90. She did not have motor dysfunction or paralysis, and her balance, gait, and stance were normal. *Id.* at 92. Dr. Jefferson's impressions were gastric ulcer, abdominal pain, nausea with vomiting, and dehydration. *Id.*

On October 31, 2015, Petitioner called 911 and EMS arrived at her home. Ex. 29 at 37. She complained of weakness for four days since arriving in Florida. *Id.* She reported that she had seen her doctor for back pain and that the pain medication he prescribed made her sick to her stomach and caused vomiting. *Id.* Her echocardiogram showed atrial fibrillation, and EMS transported her to the VRMC ER. *Id.*

At VRMC, Petitioner was seen by Dr. Jefferson again. Ex. 29 at 50. He recounted that Petitioner had been seen in the ER the morning of October 29, 2015, and provided a more detailed history of her course:

> She was seen [in the ER] about 3 o'clock in the morning as she woke up with significant abdominal pain that was radiating to the back was actually more back pain than anything else. The abdominal pain got better. The radiating pain got worse and it felt like it was in the pit of her stomach radiating to the back. She went to the emergency room for evaluation and had a CAT scan that was done that was negative and had blood tests that were done showing creatinine was at 1.6. Potassium was normal. Sodium was normal. Magnesium was normal. Urinalysis was normal as well and she was sent home after IV fluids and hydration at that time. [S]he came into our office yesterday and I saw her at the end of the day. She is still complaining of the same pain radiating to her back and again thought it was either pancreatitis or UTI, which were both negative with negative enzymes and negative urine cultures, she had not made any improvement[.] ***[L]ooking back at the history before this happened is (sic) started about a week before, she had a day of dry heaves for 24 hours and then it was gone and then it happened again a couple of days later and it was more of the acute onset of symptoms at that point. O[f] interest, she had been having some back pain prior to that and had taken a course of prednisone for 6 days she says and then had stopped it a couple of days after that is when she started to get more of the abdominal symptoms radiating to the back, thought it could possibly be an ulcer, so yesterday we put her on Dexilant twice a day, [C]arafate liquid 4 times a day, ondansetron pills, Phenergan as a backup and she was supposed to follow up with this on Monday.*** [S]he went home and as the night went on, she got more miserable got to the point where her legs got weak and she tried to get up with a walker and as the night went on, she had a couple of falls and decided it was time to come into emergency room to get evaluated. [W]hen she came in this time, her sodium was down to 129. Her potassium was normal, creatinine was up to 2.2. Her white count was over 14,000 and it looks like she was some hemoconcentration as well with dehydration and her legs were so weak that she could not bear weight on them. She is being given IV fluids, she is feeling somewhat better. The nausea that she had before is completely gone at this time, which is good. [S]he complains more that her back hurts than anything else at this point, but the nausea part is gone so hopefully with the fluids

> and hydration that she got hopefully that is going to make a difference and get her back up, but she is needing management of the electrolyte imbalance and a workup of the leukocytosis at this time and we will do that workup during her hospitalization, which will take 2 midnights and we will see how she does with the electrolyte replacement.

Id. (emphasis added).

A ROS was positive for weakness and fatigue but negative for nausea, vomiting, or abdominal pain at that time. Ex. 29 at 51. A physical exam was unremarkable. *Id.* Dr. Jefferson's assessment was that Petitioner had abdominal pain of uncertain etiology, severe dehydration, and nausea and vomiting that was improved with Zofran. *Id.* His plan was to admit her to the hospital to do blood cultures to look for infection and other tests. *Id.* at 52.

That same day, Petitioner saw gastroenterologist Peter Dumas, M.D., for "recalcitrant nausea and vomiting." Ex. 29 at 53. On exam, Dr. Dumas was unable to assess Petitioner's gait because she was too weak to walk. *Id.* at 54. His impression was acute, severe epigastric pain, acute nausea and vomiting, dehydration, and hyponatremia. *Id.* He suspected gastric ulcer or gastric carcinoma, recommended continuing IV fluids, and ordered an esophagogastroduodenoscopy ("EGD"). *Id.* The EGD was not performed because of Petitioner's weakness and electrolyte imbalances. *Id.* at 80.

The next day, November 1, 2015, Petitioner saw Tonya Stephenson, M.D., for a neurology consultation. Ex. 29 at 56. The history noted that Petitioner developed abdominal pain that radiated to her back beginning early Thursday, October 29. *Id.* She was "on prednisone for the back pain." *Id.* After being discharged from the ER, she developed 24 hours of dry heaves. *Id.* Then, either late Friday or early Saturday, she experienced a fall due to weakness. *Id.* Petitioner also reported that, "prior to her illness, she had a little bit of tingling and numbness in the left foot, predominantly the top of her foot." *Id.* At the time of the examination, she was complaining of tingling and numbness in the feet and hands. *Id.*

Dr. Stephenson observed that Petitioner had diffuse weakness with an abrupt onset, absent reflexes, and paresthesias. Ex. 29 at 57. Her assessment was possible GBS and possible myopathy. *Id.* She ordered an MRI of Petitioner's cervical spine and a head CT and left open the possibility of a lumbar puncture. *Id.*

That same day, Petitioner saw William Woolverton, M.D., for a cardiology consultation. Ex. 29 at 59. Dr. Woolverton's impression was progressive weakness suggesting a possible primary neuromuscular disorder such as GBS, abnormal cardiac enzymes likely of a skeletal muscle source, tachycardia secondary to dehydration with gastrointestinal losses, and hyponatremia likely due to dehydration. *Id.* at 60. Dr. Woolverton ordered an echocardiogram and continuing IV fluids. *Id.*

Later that day, Petitioner saw Ahmed Farooq, M.D., for an infectious disease consultation. Ex. 29 at 62. Petitioner reported to Dr. Farooq that, on her drive to Florida, she ate spaghetti and spinach salad at a local restaurant in Illinois. *Id.* That night, she began to "feel worse," with

abdominal pain and nausea, but no diarrhea. *Id.* "Since they were traveling and driving, they decided to continue [to Florida]." *Id.* The nausea got worse after arriving in Florida, prompting her initial ER visit early Thursday morning. *Id.* After being discharged from the ER, she became progressively weaker "to the point that she was unable to walk." *Id.* She went back to the hospital on Saturday. *Id.* at 63.

Petitioner told Dr. Farooq that that about a week earlier, "she stopped taking her methotrexate" for juvenile rheumatoid arthritis. Ex. 29 at 62. Also, she "was recently given some steroids" for back pain. *Id.* On exam, she exhibited significant weakness and no reflexes. *Id.* at 64. Dr. Farooq suspected GBS. *Id.* He explained that she had "profound muscle weakness" with abdominal pain, nausea, and vomiting. *Id.* at 65. He commented that "[t]he only gastrointestinal infection that can actually present like that is Campylobacter jejuni infection that will present as [GBS]." *Id.* Food poisoning such as botulism was another possibility, but based on Petitioner's clinical presentation, Dr. Farooq did not believe that was the correct diagnosis. *Id.* Dr. Farooq noted that Petitioner was likely experiencing an "autoimmune phenomenon" for which antibiotics would not be helpful. *Id.* Nonetheless, he said he would order Campylobacter stool studies. *Id.*

The results of a lumbar puncture showed elevated protein in the cerebrospinal fluid ("CSF"). Ex. 29 at 68. Lyme disease testing was negative. *Id.* Petitioner began an eight-day course of IVIG. *Id.*

On November 2, 2015, Petitioner was transferred out of intensive care. Ex. 29 at 82. Dr. Dumas noted that Petitioner's GBS could have been caused by the flu vaccine, but more testing was needed to be certain. *Id.* at 103-04.

On November 3, 2015, Petitioner was seen by an infectious disease specialist who commented that she reported receiving the "senior" dose of the flu vaccine in Minnesota in October.[9] Ex. 29 at 103. The physician noted that GBS "has always been a questionable side effect from flu vaccine," but, if no other cause was found, the case should be reported to VAERS. *Id.*

On November 5, 2015, Petitioner went into respiratory arrest and was intubated for one week. Ex. 29 at 68, 83.

On November 6, 2015, the infectious disease physician attempted to report Petitioner's case to VAERS but was told the clinic administering the vaccine would have to do so.[10] Ex. 29 at 114. On November 7, 2015, the physician commented that serology for *C. jejuni* was positive. *Id.* at 115, 279. The physician remarked that Petitioner's GBS could possibly be related to *C. jejuni* if the infection was recent. *Id.* On November 9, 2015, the physician noted that the cause of

[9] These notes and the notes from November 7 and 9, 2015 were handwritten, apparently by the same physician, whose name was illegible.

[10] Although the records indicate the infectious disease specialist contacted the Minnesota clinic at which Petitioner was vaccinated to request that it file a VAERS report, it is unclear whether the clinic did so. Ex. 29 at 114. No such report has been produced in this case.

Petitioner's GBS was "not clear" and could have been either the recent flu vaccination or *C. jejuni* infection. *Id.* at 120.

On November 16, 2015, a neurologist, Dr. Coleman, noted that Petitioner had GBS "likely triggered by influenza vaccine." Ex. 29 at 135. That same day, Petitioner was discharged from the hospital to HealthSouth Sarasota Rehabilitation Hospital, an inpatient rehabilitation center. *Id.* at 68-69.

On November 19, 2015, Petitioner was discharged back to VRMC. Ex. 30 at 110-12. Her EKG showed atrial fibrillation with a rapid ventricular rate. *Id.* She was discharged from the hospital on December 4, 2015, with a diagnosis of acute perforated diverticulitis. Ex. 39 at 22. She underwent surgery for the diverticulitis that month, requiring a colostomy. *See* Ex. 5. She returned to the rehabilitation facility, where she received inpatient treatment until her discharge on December 30, 2015. Ex. 30 at 613-14. Petitioner went from there to a skilled nursing facility, where she remained until March 9, 2016. Ex. 26 at 1-18.

In 2016, after returning to Minnesota, Petitioner continued to follow up for her diverticulitis. Ex. 6 at 2. On December 6, 2016, she was seen in Florida by a cancer specialist for severe anemia. Ex. 11 at 27. She received a blood transfusion, which returned her hemoglobin levels to normal. *Id.* at 67.

On September 12, 2018, nearly three years after the vaccination, Petitioner saw neurologist Eric Ahlskog, M.D., Ph.D., at the Mayo Clinic, for an evaluation of sequelae of her GBS. Ex. 32 at 212. Dr. Ahlskog noted that she developed GBS "after a flu shot." *Id.* He also noted that her recovery was slow, requiring six months of rehabilitation, and that it was complicated by "heart failure," diverticulosis with colon rupture, and an upper GI ulcer. *Id.* Before her illness, she was active and "very athletic," but afterward she required a forearm cane for balance. *Id.* She used a motorized scooter. *Id.* She suffered paresthesias and dysesthesias distally in all of her limbs. *Id.* She had more recently developed "extreme fatigue" and dyspnea on exertion. *Id.*

On exam, Petitioner had reduced touch sensation in her lower legs and forearms. Ex. 32 at 213. Her vibration sense was absent in her big toes and ankles. *Id.* She had marked tenderness distally in her arms. *Id.* She had impaired strength in both hands, but "normal or close to normal" strength in her wrists and proximal arms. *Id.* She had marked weakness of the right iliopsoas and hamstring. *Id.* Her toe extensors were also weak. *Id.*

Dr. Ahlskog's assessment was marked but incomplete improvement of GBS. Ex. 32 at 213. He ordered an EMG, which was done on September 18, 2018. *Id.* at 214. The EMG revealed "minimally abnormal" results, with no evidence of neuropathic disease activity. *Id.* This surprised Dr. Ahlskog, given Petitioner's clinical presentation, so he consulted with another neurologist. *Id.* She opined that the likely explanation was "the demyelinating attack was predominantly proximal at nerve root entry zones[.]" *Id.* Both neurologists agreed that further evaluation of the contemporaneous hospital records would be helpful to confirm the GBS diagnosis. *Id.* The records produced in this case, however, do not indicate this evaluation was completed.

III. EXPERT EVIDENCE

A. Praful Kelkar, M.D.: First Expert Report

Dr. Kelkar received his medical degree from the University of Bombay in 1983. Ex. 41 ("Kelkar CV") at 1. He completed his residency in internal medicine in 1987 and additional residencies in internal medicine and neurology in the United States between 1988 and 1992. *Id.* He is board certified in neurology, neuromuscular and electrodiagnostic medicine with a subspecialty in neuromuscular medicine, and clinical neurophysiology. *Id.* Since 2007, he has been an adjunct associate professor in the department of neurology at the University of Minnesota. *Id.* He previously taught as an associate professor at the University of Iowa. *Id.*; Ex. 40 ("First Kelkar Rep.") at 1. He has authored or co-authored 26 peer-reviewed journal articles and three book chapters in neurology, and he has received several research grants. Kelkar CV at 2-3, 7.

In his first expert report, Dr. Kelkar noted that Petitioner had a complex history with multiple chronic conditions. First Kelkar Rep. at 2. He opined that Petitioner's GBS diagnosis was correct, based on her clinical symptoms of pain and weakness, the elevated protein level in her CSF, and her improvement following treatment with IVIG. *Id*. at 3.

Turning to causation, Dr. Kelkar stated that *C. jejuni* infections are "common" and that "GBS can be triggered by a variety of infections and the most common infection is campylobacter jejuni." First Kelkar Rep. at 3. Up to 30% of GBS patients are positive for *C. jejuni*, and one in 1,000 cases of *C. jejuni* are estimated to result in GBS. *Id.* (citing Israeli et al., *Guillain-Barré Syndrome – A Classical Autoimmune Disease Triggered by Infection or Vaccination*, 42 CLINICAL REVS. ALLERGY & IMMUNOLOGY 121-30 (2012) (Ex. 45) ("Israeli")).

Dr. Kelkar acknowledged that Petitioner tested positive for *C. jejuni* antibody. First Kelkar Rep. at 3. He noted that the test was run by Quest Diagnostics. *Id.* He reported that he contacted the medical director for Quest and was advised that their test did not distinguish between IgM, IgA, and IgG antibodies for *C. jejuni*, making it impossible to determine whether Petitioner had an active, recent, or remote *C. jejuni* infection. *Id.*

Dr. Kelkar also stated that flu vaccination is causally linked to GBS. First Kelkar Rep. at 3. Citing the Israeli paper, he pointed out that the typical onset of GBS is 3-6 weeks following flu vaccination, but he also said that here, the onset was "one week after the vaccination, which is typical for vaccine related GBS." *Id.* (citing Israeli).

Petitioner therefore had two risk factors for GBS: flu vaccination and positive *C. jejuni* serology. First Kelkar Rep. at 3. Dr. Kelkar noted that "[h]ow to ascertain relative risk of developing GBS given multiple risk factors is the question," given the scant literature "about dual trigger factors inducing GBS." *Id.* He cited literature reporting GBS after administration of both H1N1 and seasonal flu vaccinations. *Id.* (citing Greene et al., *Risk of Confirmed Guillain-Barré Syndrome Following Receipt of Monovalent Inactivated Influenza A (H1N1) and Seasonal Influenza Vaccines in the Vaccine Safety Datalink Project, 2009-2010*, 175(11) AM. J. EPIDEMIOLOGY 1100-09 (2012) (Ex. 46) ("Greene 2012")). He also referenced reports of GBS after dual infection with Zika and dengue virus or with *C. jejuni* and *Yersinia pseudotuberculosis*.

Id. (citing Parra et al., *Guillain-Barré Syndrome Associated with Zika Virus Infection in Colombia*, NEW ENGLAND J. MED. 375:16 (2016) (Ex. 47) ("Parra"); Amereller et al., *A horse and a zebra: an atypical clinical picture including Guillain-Barré syndrome, recurrent fever and mesenteric lymphadenopathy caused by two concomitant infections*, INFECTION (2020) (Ex. 48) ("Amereller")).

According to Dr. Kelkar, *C. jejuni* infection is associated with the induction of anti-GM1 antibodies, which are believed to be involved in causing GBS. First Kelkar Rep. at 3. The flu vaccine also induces anti-GM1 antibodies. *Id.* (citing Nachamkin et al., *Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome*, 198 J. INFECTIOUS DISEASES 226-33 (2008) (Ex. 44) ("Nachamkim")). "Mechanisms such as this may explain [the] synergistic effect of two concomitant risk factors inducing GBS." *Id.*

Dr. Kelkar opined that the subject flu vaccine "probably" caused Petitioner's GBS. First Kelkar Rep. at 4. He considered the vaccine to be a more significant factor than a *C. jejuni* infection because the infection "was not entirely proven" with the serology results. *Id.* Additionally, he opined that Petitioner did not develop the type of GBS most often associated with a *C. jejuni* infection. *Id.* *C. jejuni* usually causes axonal GBS, which involves a much slower recovery and a much poorer outcome than the demyelinating form of GBS, acute inflammatory demyelinating polyneuropathy ("AIDP"). *Id.* (citing D. Chowdhury & A. Arora, *Axonal Guillain-Barré syndrome: a critical review*, 103 ACTA NEUROLOGICA SCANDINAVICA 267-77 (2001) (Ex. 42) ("Chowdhury & Arora")). In Dr. Kelkar's view, Petitioner more likely had AIDP, based on her quick response to IVIG therapy and her "nearly normal" EMG after her GBS, which are not consistent with axonal GBS. *Id.*

B. Kathleen Collins, M.D., Ph.D.: First Expert Report

Dr. Collins earned her medical degree and doctorate from Johns Hopkins University in 1993. Ex. B ("Collins CV") at 1. She completed her residency in internal medicine in 1995 and clinical and research fellowships in infectious disease from 1995-1998. *Id.* Since 1998, she has held clinical and academic professorships at the University of Michigan in internal medicine and microbiology and immunology. *Id.* She is the recipient of numerous research grants. *Id.* at 3-6. She has authored or co-authored 40 peer-reviewed journal articles and five book chapters, with a particular focus on immunology and HIV. *Id.* at 14-20.

Dr. Collins did not dispute that Petitioner developed GBS. She took issue, however, with the claim that seasonal flu vaccination can cause GBS. First Collins Rep. at 5-6. She cited several epidemiologic studies reporting no increased risk of GBS following flu vaccination. *Id.* at 5-7. First, she noted that the Israeli article cited by Dr. Kelkar did not "actually support [Dr. Kelkar's] claim that influenza vaccination has been linked to [GBS]." *Id.* at 5 (citing Israeli). That paper *did* support a link between *C. jejuni* infection and GBS. *Id.* The Israeli paper also cited Stowe et al., in which the authors found no increased risk of GBS after seasonal flu vaccination. *Id.* at 6 (citing Julia Stowe et al., *Investigation of the Temporal Association of Guillain-Barré Syndrome With Influenza Vaccine and Influenzalike Illness Using the United Kingdom General Practice Research Database*, 169 AM. J. EPIDEMIOLOGY 382-88 (2008) (Ex. A, Tab 2) ("Stowe")).

Dr. Collins also referenced an article by Grimaldi-Bensouda, which reported an association between flu infection (or infection with flu-like symptoms) and GBS, but no association between the flu vaccine and GBS. First Collins Rep. at 6 (citing Grimaldi-Bensouda et al., *Guillain-Barré Syndrome, Influenza-like Illnesses, and Influenza Vaccination During Seasons With and Without Circulating A/H1N1 Viruses*, 174 AM. J. EPIDEMIOLOGY 326-35 (2011) (Ex. A, Tab 3) ("Grimaldi-Bensouda")). Similarly, the Greene study found that the 2009 and 2010 seasonal flu vaccines did not pose an elevated risk of GBS. *Id.* at 6-7 (citing Greene et al., *Guillain-Barré Syndrome, Influenza Vaccination, and Antecedent Respiratory and Gastrointestinal Infections: A Case-Centered Analysis in the Vaccine Safety Datalink, 2009-2011*, 8 PLOS One 1-10 (2013) (Ex. A, Tab 1) ("Greene 2013")). The Vellozzi study reported a lower risk of GBS following H1H1 influenza vaccination than in unvaccinated populations. *Id.* at 8 (citing Vellozzi et al., *Cumulative Risk of Guillain-Barré Syndrome Among Vaccinated and Unvaccinated Populations During the 2009 H1N1 Influenza Pandemic*, 104(4) AM. J. PUB. HEALTH 696-701 (2014) (Ex. A, Tab 4) ("Vellozzi")). Finally, the Baxter study found no evidence of increased risk of GBS following any type of vaccination, including flu. *Id.* (citing Baxter et al., *Lack of Association of Guillain-Barré Syndrome With Vaccinations*, 57(2) CLINICAL INFECTIOUS DISEASES 197-204 (2013) (Ex. A, Tab 7) ("Baxter")).

Next, Dr. Collins pointed out that *C. jejuni* infection is "strongly associated with [GBS]." First Collins Rep. at 8. She noted that about 70% of GBS patients reported a "prior history of diarrhea[.]" *Id.* The mean time between onset of diarrhea and GBS symptoms was nine days. *Id.* (citing Rees et al.). A history of *C. jejuni* infection "has been reported in 26-60% of [GBS] cases." *Id. C. jejuni* infection is associated with an up to 60-fold increased risk of GBS. *Id.* (citing Tam et al., *Guillain-Barré Syndrome and Preceding Infection with Campylobacter, Influenza and Epstein-Barr Virus in the General Practice Research Database*, 4(344) PLOS ONE 1-6 (2007) (Ex. A, Tab 9) ("Tam")). The Tam authors reported up to a 38-fold increased risk of GBS in the two months following *C. jejuni* infection, but a non-statistically significant lowered risk of GBS following flu vaccination. *Id.* (citing Tam at 2 (Table 1)).

Dr. Collins explained that one type of GBS associated with *C. jejuni* infection is acute motor axonal neuropathy ("AMAN"). First Collins Rep. at 8. AMAN is believed to be caused by molecular mimicry between components of nerve axons and the antibody response to the infection. *Id.* The Nodes of Ranvier, which are gaps in the myelin sheath covering the nerve, "are a major target of the antibody response." *Id.* at 9 (citing Hahn et al., *Guillain-Barré syndrome*, 352 THE LANCET 635-41 (1998) (Ex. A, Tab 11) ("Hahn"); van Doorn, et al., *Clinical features, pathogenesis, and treatment of Guillain-Barré syndrome*, 7 LANCET NEUROLOGY 939-50 (2008) (Ex. A, Tab 12) ("van Doorn")). Rabbits sensitized to *C. jejuni* developed anti-GM1 antibodies, limb weakness, and nerve changes mimicking AMAN. *Id.* (citing Yuki et al., *Guillain-Barré Syndrome* (2004)). The animals had more pronounced degeneration at the nerve roots, "which have less blood-nerve barrier and greater access of neurons to antibodies." *Id.* (citing Yuki et al., *Carbohydrate mimicry between human ganglioside GM and Campylobacter jejuni lipoligosaccharide causes Guillain-Barré syndrome*, Proceedings of the National Academy of Sciences, 2004. 101(31): p. 11404-11409 (2004) (Ex. A, Tab 13) ("Yuki 2004")).

Dr. Collins disagreed with Dr. Kelkar's theory that the flu vaccine and a *C. jejuni* infection could have caused Petitioner's GBS together. First Collins Rep. at 10. She opined that Dr. Kelkar presented no evidence to support the interaction between the vaccine and infection and that his reliance on the Greene article was "misleading" because the authors did not find that vaccination increased the risk of developing GBS. *Id.* (citing Greene 2012). She likewise criticized Dr. Kelkar's reliance on Nachamkin because that paper's authors did "not believe modern influenza vaccines are a risk factor for [GBS]." *Id.* (citing Nachamkin). Furthermore, in Sivadon-Tardy, GBS patients with preceding influenza A infection were found not to have antiganglioside antibodies. *Id.* at 10-11.[11] Dr. Collins commented that "[i]f the natural infection does not produce these antibodies, it is very unlikely that the vaccine does." *Id.* at 11.

Dr. Collins critiqued Dr. Kelkar's claim that Petitioner's GBS was less likely caused by *C. jejuni* because she did not have a presentation consistent with that cause. First Collins Rep. at 11. She noted that Sivadon-Tardy found *C. jejuni*-associated GBS was more likely to require mechanical ventilation, as happened with Petitioner, than GBS caused by flu infection or vaccination. *Id.* She also disagreed with Dr. Kelkar's view that *C. jejuni* was an unlikely cause of Petitioner's GBS because that would have produced a slower recovery and a worse outcome. *Id.* The article Dr. Kelkar cited, Chowdhury & Arora, referenced a different paper, Ho et al., which found a "comparable" rate of recovery in AMAN and AIDP patients.[12] *Id.* Furthermore, a study by Hiraga measured rates of recovery of AMAN and AIDP patients and concluded that "AMAN electrodiagnosis is not always a marker of poor recovery." *Id.* (citing Hiraga et al., *Recovery patterns and long term prognosis for axonal Guillain-Barre syndrome*, 75 J. NEUROLOGY NEUROSURGERY & PSYCHIATRY 719-22 (2005) (Ex. A, Tab 16) ("Hiraga")). Moreover, Petitioner met the criteria for "slow recovery" as defined in the Hiraga study, undermining Dr. Kelkar's claim that she had a milder disease than would be expected in *C. jejuni*-caused GBS. *Id.*

Dr. Collins further explained that *C. jejuni* infection can also be associated with AIDP. First Collins Rep. at 9, 11. The Chowdhury & Arora paper acknowledged that both axonal and AIDP cases have been described in association with *C. jejuni* infection. *Id.* at 11 (citing Chowdhury & Arora).

Lastly, Dr. Collins opined that "the timing of the vaccination relative to the onset of symptoms does not support the vaccine causing [GBS]." First Collins Rep. at 12. Petitioner's reported use of prednisone for six days, plus approximately two days before she first went to the ER, indicated that her symptoms started at least eight days prior to October 31, which would have been only two days after vaccination. *Id.* Onset of GBS within 48 hours is too short a period to infer vaccine causation. *Id.*

[11] Respondent did not file the Sivadon-Tardy paper.

[12] Dr. Collins explained that Ho investigators also conducted laboratory experiments suggesting quick recovery in AMAN could be due to anti-GM1 antibodies, such as those thought to be produced in response to *C. jejuni* infection, disrupting nerve conduction at the Nodes of Ranvier without causing permanent damage. Respondent did not file the Ho paper into the record.

C. Dr. Kelkar's Second Report

Dr. Kelkar first addressed the onset of Petitioner's GBS symptoms following vaccination. Ex. 50 ("Second Kelkar Rep.") at 1. He pointed out that, although GBS typically begins within the first three to six weeks after vaccination, "[o]nset of GBS within the first week after an inciting event (such as infection or vaccination) is perfectly within the expected range." *Id.* (referencing Israeli).

Second, Dr. Kelkar argued Petitioner's symptoms did not suggest she had a *C. jejuni* infection. Second Kelkar Rep. at 2. She did not develop diarrhea, a fever, an elevated white blood count, or gastroenteritis. *Id.* The Nandkishor case report of *C. jejuni* involved a patient with abdominal pain without diarrhea, but, unlike Petitioner, that patient had a fever and required antibiotic treatment. *Id.* (citing Nandkishor et al., *Enterocolitis without diarrhoea in an adult patient: a clinical dilemma*, BMJ CASE REPS. (2014) (Ex. 56) ("Nandkishor")). Furthermore, Petitioner "did not have positive blood or stool culture for Campylobacter." *Id.*

Third, Dr. Kelkar commented that pain is "often associated with GBS and can be a presenting symptom of the GBS." Second Kelkar Rep. at 3 (citing Yao et al., *Pain during the acute phase of Guillain-Barré syndrome*, 97 MEDICINE 34-38 (2018) (Ex. 54) ("Yao"); L. Ruts et al., *Pain in Guillain-Barré syndrome: A long-term follow-up study*, 75 NEUROLOGY 1439-47 (2010) (Ex. 52) ("Ruts"); D.E. Moulin et al., *Pain in Guillain-Barré syndrome*, 48 NEUROLOGY 328-31 (1997) (Ex. 51) ("Moulin")). In particular, pain radiating to the back is "extremely common with GBS" and is often the presenting symptom. *Id.*

Fourth, Dr. Kelkar maintained that axonal GBS is more commonly associated with *C. jejuni* infection and statistically results in poorer outcomes than AIDP. Second Kelkar Rep. at 4 (citing Rees et al., *Campylobacter Jejuni Infection and Guillain-Barré Syndrome*, NEW ENGLAND J. MEDICINE (1995) (Ex. 55) ("Rees")). He disagreed that Petitioner developed axonal GBS/AMAN, instead arguing that she developed AIDP. *Id.* This is because she recovered quickly with IVIG therapy, "with successful extubation within [one] week," and had a nearly normal EMG "one year later." *Id.* at 5. Furthermore, her blood work did not confirm an acute *C. jejuni* infection. *Id.*

Dr. Kelkar concluded: "It is my opinion that the vaccination played an important role in development of GBS (AIDP) and that it is more likely than not, to a reasonable degree of medical certainty, that the influenza vaccine served as a trigger for [Petitioner's] GBS." Second Kelkar Rep. at 5.

D. Dr. Kelkar's Supplemental Letter

In a supplemental letter clarifying his opinions, Dr. Kelkar stated that Petitioner developed a severe case of AIDP, which would not have occurred "but for the flu vaccine." Ex. 57 ("Kelkar

Letter") at 1. A *C. jejuni* infection might have also contributed to her GBS, but, in his opinion, "the flu vaccine played a greater role in causing GBS than did the infection and was a substantial contributing factor in causing Petitioner's GBS." *Id.*

E. Dr. Collins's Second Report

Dr. Collins disputed Dr. Kelkar's claim that Petitioner developed GBS one week after the vaccination. Ex. C ("Second Collins Rep.") at 1. Instead, the records "strongly support[ed] the conclusion that symptoms actually began too soon after vaccination to have been caused by vaccination." *Id.* "[D]ays or even weeks are needed for the immune system to respond to an antigen." *Id.*

With respect to *C. jejuni*-related GBS, Dr. Collins also commented that the latency period between exposure to *C. jejuni* and development of GBS symptoms "must be longer than [two] days" because the infection has an incubation period prior to the development of diarrhea. Second Collins Rep. at 2. In humans, the median interval between the onset of diarrheal illness and neuropathic symptoms was nine days. *Id.* (citing Rees).

Dr. Collins further opined that Dr. Kelkar's theory that the flu vaccine caused Petitioner's GBS did not account for the abdominal symptoms she reported, including "recalcitrant nausea and vomiting." Second Collins Rep. at 2-3. She stated that Dr. Kelkar had not provided any evidence that these symptoms are associated with vaccine-induced GBS. *Id.* at 3. Conversely, the fact that Petitioner did not develop diarrhea did not rule out an acute or recent *C. jejuni* infection in her case. *Id.* at 2 (citing Rees; Nandkishor). Also, there is evidence that intestinal infections other than *C. jejuni* can cause GBS. *Id.* at 4 (citing Tam at 2 (Table 1)). Thus, even if Petitioner did not have *C. jejuni*-caused GBS, "other microbes causing an indistinguishable symptom complex of 'intractable nausea and vomiting' could also cause [GBS] and this would be more likely than the vaccine [to cause] GBS." *Id.*

F. Dr. Kelkar's Third Report

Dr. Kelkar explained that he has treated more than 100-150 GBS patients during his 25-year career, of which about 20-30 had *C. jejuni* infections. Ex. 58 ("Third Kelkar Rep.") at 1. He pointed out that GBS is generally understood as a post-infectious or post-inflammatory autoimmune reaction affecting the peripheral nerves, likely produced by molecular mimicry. *Id.* at 5. Studies have confirmed that "certain formulations of the influenza vaccination, most notably the 1975 to 1976 swine flu and 2009 H1-N1 vaccines, have been clearly associated with increased incidents [sic] of GBS." *Id.* Seasonal flu vaccines have also been associated with increased risk of GBS. *Id.* at 5-6 (citing Arias et al., *Guillain-Barré syndrome and influenza vaccines: A meta-analysis*, 33 VACCINE 3773-3778 (2015) (Ex. 65) ("Arias")).

Dr. Kelkar opined that "[a]utoimmune disorders sometimes are related to two or more triggers resulting in synergist[ic] effects," such as two unrelated infections or infection and

vaccination. Third Kelkar Rep. at 8 (citing Westall et al., *Cause and prevention of postvaccinal neuropathies in light of a new theory of autoimmunity*, LANCET 8501: 251-52 (1986) (Ex. 67) ("Westall")). He added that the first trigger (the flu vaccine in Petitioner's case) primes the immune system, and the second ("possible" *C. jejuni* "or other infection") "results in immune activation resulting in GBS. *Id.* (citing Wirguin et al., *Induction of anti-GM1 ganglioside antibodies by* Campylobacter jejuni *lipopolysaccharides*, 78 J. NEUROIMMUNOLOGY 138-42 (1997) (Ex. 68) ("Wirguin")). He argued that this priming and triggering relationship could explain why no particular wild infection has been conclusively linked to autoimmune diseases such as GBS. *Id.* (citing Cusick et al., *Molecular Mimicry as a Mechanism of Autoimmune Disease*, 42 CLINICAL REV. ALLERGY & IMMUNOLOGY 102-11 (2011) (Ex. 69) ("Cusick")).

Dr. Kelkar acknowledged that Petitioner complained of nausea and abdominal pain on October 29 and 30, 2015, had a low-grade fever on October 30, and had a positive serology test for *C. jejuni* on November 1. Third Kelkar Rep. at 7. He nevertheless did not feel that Petitioner had an active *C. jejuni* infection during this time, based on the absence of diarrheal illness, the opinions of Drs. Farooq and Dumas, the fact that Petitioner was never treated with antibiotics, and the lack of specificity of the serology testing as to whether Petitioner's exposure to *C. jejuni* was recent or remote. *Id.* He added that "[e]ven if she had a minor or inactive *c. jejuni* infection, more likely than not it was a combination of both – vaccine and infection – that led to GBS." *Id.* at 8.

Dr. Kelkar opined that, if Petitioner did not have an active *C. jejuni* infection preceding the onset of GBS, her symptoms, such as back and abdominal pain, could have been caused by her preexisting rheumatoid arthritis, peptic ulcer disease, and/or the "long car ride" she underwent just before her hospitalization, during which she ate at a restaurant. Third Kelkar Rep. at 6-7. Alternatively, she could have had another type of infection. *Id.* at 7. He noted that the evidence as to when Petitioner suffered from "dry heaves" is inconsistent, in that one medical record stated that occurred one month prior to GBS onset, but another placed that event around the same time as GBS onset. *Id.* (citing Ex. 29 at 30, 56). He opined that in any event, this symptom "does not indicate an active infection of any kind." *Id.*

Dr. Kelkar maintained that the onset of Petitioner's GBS was about three or four days prior to her second ER visit on October 31, 2015. Third Kelkar Rep. at 6. This was when her weakness/motor symptoms began and was an appropriate timeframe for onset of GBS following vaccination. *Id.* In a footnote, Dr. Kelkar acknowledged that the medical history pertaining to onset "is quite confusing." *Id.* at 6 n.1. He stated that given the lack of clarity concerning the timing of Petitioner's use of prednisone, he focused on weakness as the symptom signifying the onset of Petitioner's GBS. *Id.* He opined that

> [i]f back pain is considered the onset of GBS the shorter timing is supported in the literature. If we work backwards from 10/21 and the self-reported use of Prednisone for 6 days then off for 3-4 and symptoms got worse, this could mean that onset of back pain was within a day or two following the flu vaccination on 10/21.

Id. He further stated that the medical literature supports a shorter onset period in cases where GBS is caused by both an infection and vaccination.[13] *Id.* (citing Stratton et al., *Adverse Effects of Vaccines: Evidence & Causality*, INST. OF MED. (2012)).[14]

G. Dr. Collins's Third Report

In her third report, Dr. Collins addressed the timeline of Petitioner's symptom onset. Ex. D ("Third Collins Rep.") at 1. She explained that medical professionals are taught that, where medical records are not wholly consistent, greater weight should be given to a history that is obtained directly from the patient close in time to the events being described. *Id.* Dr. Collins thus concluded that the October 30, 2015 record from Petitioner's PCP was the most reliable source of information about the timeline of events. *Id.* This record placed the onset of Petitioner's back pain "around the same time as vaccination," which is "too short [a timeframe] to invoke the vaccine as a cause of her back pain." *Id.* at 2.

Dr. Collins disagreed with Dr. Kelkar's view that the back pain Petitioner experienced was not her initial GBS symptom and was instead related to her other medical conditions. Third Collins Rep. at 2. She also argued that Dr. Kelkar incorrectly minimized Petitioner's gastrointestinal symptoms "even though they were a very prominent part of her presentation and the reason she sought medical attention on October 29." *Id.* She commented that, by focusing on Petitioner's weakness as the presenting GBS symptom, Dr. Kelkar failed to provide a "compelling explanation for the abdominal symptoms that brought [Petitioner] to medical attention." *Id.*

Dr. Collins reviewed additional medical literature submitted by Dr. Kelkar relating to the question of whether flu vaccination can cause GBS. Third Collins Rep. at 3-5. This literature, she argued, supports the contention that gastrointestinal infection is a much more likely cause of GBS than vaccination. *Id.* (citing van den Berg et al., *Guillain-Barré syndrome: pathogenesis, diagnosis, treatment and prognosis*, 10 NATURE REVS. NEUROLOGY 469-82 (2014) (Ex. 61) ("van den Berg"); Willison et al., *Guillain-Barré syndrome*, 388 LANCET 717-27 (2016) (Ex. 62) ("Willison"); Yu et al., *Ganglioside Molecular Mimicry and Its Pathological Roles in Guillain-Barré Syndrome and Related Diseases*, 74(12) INFECTION & IMMUNITY 6517-27 (2006) (Ex. 63) ("Yu")). She acknowledged that the Fadrique meta-analysis described two studies that found a very small increased risk of GBS after vaccination. *Id.* at 4 (citing Fadrique et al., *Guillain-Barré syndrome and influenza vaccines: current evidence*, 32(4) OFFICIAL J. SPANISH SOC'Y CHEMOTHERAPY 288-95 (2019) (Ex. 64) ("Fadrique")). She noted, however, that "the apparent small risk observed in some studies was not supported by other observations." *Id.* She explained that "the very small apparent increased risk due to vaccination is confounded by the fact that influenza vaccination is administered while seasonal influenza is circulating in the population," and that "because of these confounding parameters, one cannot be confident of even a minimal role for the vaccine in causing GBS." *Id.* at 5.

[13] The expert report reads "caused by both an infection and *GBS*," which presumably was an error. Third Kelkar Rep. at 6 n.1 (emphasis added).

[14] Petitioner did not file this into the record.

Dr. Collins next responded to Dr. Kelkar's opinion that, despite Petitioner's positive *C. jejuni* serology, that infection was not the likely cause of her GBS. Third Collins Rep. at 7. First, even if the infection were no longer active at the time of her blood test, GBS is a post-infectious condition, so *C. jejuni* could have caused it if it occurred within the appropriate timeframe. *Id.* at 3. Furthermore, it is uncommon to know precisely when a patient was exposed to a wild pathogen. *Id.* "What is clear is that [Petitioner] had a gastrointestinal illness prior to the development of [GBS] along with a test showing prior exposure to a pathogen associated with high risk of developing [GBS]." *Id.* at 7. In sum, the medical records and literature support the proposition that Petitioner's GBS was caused by *C. jejuni* or a different intestinal infection. *Id.*

Next, Dr. Collins opined that the medical literature Dr. Kelkar submitted does not support his dual causation theory. Third Collins Rep. at 8. The Westall article, published in 1986, relied on outdated theories of GBS causation and did not provide any evidence "that the combination of influenza vaccination plus a gastrointestinal infection accelerate autoimmune disease or GBS." *Id.* (citing Westall). Similarly, none of the other papers cited by Dr. Kelkar even addressed the question of whether concomitant vaccination and infection could cause GBS, much less concluded as such. *Id.* (citing Wirguin, Cusick, Steiner et al., *Transient immunosuppression: a bridge between infection and the atypical autoimmunity of Guillain–Barré syndrome,* 162 CLINICAL AND EXPERIMENTAL IMMUNOLOGY 32–40 (2010) (Ex. 70) ("Steiner")).

Dr. Collins concluded that, if back pain were the first GBS symptom, its onset was too soon after the subject vaccination to make a causal attribution. Third Collins Rep. at 8. Alternatively, regardless of whether back pain or weakness were considered the first GBS symptom, the gastrointestinal illness Petitioner experienced was the more likely cause of her GBS than was the vaccine. *Id.* at 8-9.

H. Dr. Kelkar's Fourth Report

The fourth expert report from Dr. Kelkar addressed the questions posed by Special Master Oler. First, Dr. Kelkar opined that Petitioner's back pain was not her initial GBS symptom but was instead "related to her pre-existing history." Ex. 72 ("Fourth Kelkar Rep.") at 1. He explained that Petitioner had experienced previous flares of back pain and had stopped taking her rheumatoid arthritis medication. *Id.*

Second, Dr. Kelkar opined that Petitioner's October 29, 2015 lumbar CT showed a large hemangioma at the T12 vertebral body. Fourth Kelkar Rep. at 1. He did not believe this finding was significant or explained Petitioner's back pain. *Id.*

Third, Dr. Kelkar confirmed that, in his view, Petitioner's leg tingling and weakness were the symptoms signifying the onset of GBS. Fourth Kelkar Rep. at 1. These symptoms began six days after the vaccination. *Id.*

Fourth, Dr. Kelkar stated that typically, the symptoms of a *C. jejuni* infection would appear before the onset of GBS symptoms. Fourth Kelkar Rep. at 2. Neurological symptoms of GBS following such an infection usually occur 1-3 weeks after the onset of diarrheal illness, with an incubation period of 2-5 days before symptoms. Thus, the "*C. jejuni* infection likely predated the vaccine." *Id.* Because Petitioner's weakness began on about October 27, 2015, "the infection would have predated the vaccine received on 10/21/15 by 4 days." *Id.*

Fifth, Dr. Kelkar opined that, although Petitioner had a "high" level of *C. jejuni* antibodies in her blood, that did "not necessarily lead to the conclusion that she had an active or recent infection." Fourth Kelkar Rep. at 3. The testing method used did not distinguish between antibodies indicating recent/acute infection and those indicating remote exposure. *Id.* Dr. Kelkar also opined that Petitioner had AIDP, which is associated with flu vaccination, not axonal GBS, which is associated with *C. jejuni*. *Id.*[15]

I. Dr. Collins's Fourth Report

Responding to Special Master Oler's questions, Dr. Collins first opined that "it is more likely than not that the onset of Petitioner's back pain on or before October 23 constitutes the onset of her GBS." Ex. E ("Fourth Collins Rep.") at 1. This was based on Petitioner's self-reported medical history to Dr. Jefferson on October 31, 2015. *Id.* The peer-reviewed literature provides "extensive description of back pain as the presenting symptom of GBS." *Id.* For instance, the Moulin paper stated that moderate to severe pain, including back pain, is "a common and early symptom of GBS and requires aggressive treatment." *Id.* (citing Moulin). That study reported pain as a symptom in 85.5% of GBS patients. *Id.* Additionally, Petitioner's pain was improved by her use of prednisone; "[a]s prednisone inhibits the immune response and GBS is immune mediated, a partial response from prednisone therapy is consistent with GBS-induced pain." *Id.*

Second, Dr. Collins agreed that the hemangioma seen on Petitioner's lumbar CT was not the cause of her back pain. Fourth Collins Rep. at 2. Most hemangiomas are asymptomatic and would not cause a sudden onset of pain. *Id.* Also, Petitioner's pain resolved without treatment of the hemangioma. *Id.*

Third, Dr. Collins opined that *C. jejuni* symptoms and GBS symptoms can appear concurrently. Fourth Collins Rep. at 2 (citing Taylor et al., *Sensitivity and specificity of serology in determining recent acute Campylobacter infection*, 34(5) INTERNAL MEDICINE J. 250-58 (2004) (Ex. E, Tab 4) ("Taylor")). Studies looking at the onset of GBS following *C. jejuni* symptoms have provided "highly variable" results, with a median interval of nine days. *Id.* at 3 (citing Rees).

[15] Although Dr. Kelkar referenced several papers in his fourth report that were not previously filed, Petitioner did not file any of them with the report.

Furthermore, "[i]t takes time following exposure to a pathogen for antibody to become detectable." Fourth Collins Rep. at 3. Thus, the fact that Petitioner had detectable *C. jejuni* antibodies in her blood during her hospitalization "indicates that her infection was ongoing for long enough for an immune response to develop and for the antibody levels to be very high." *Id.* Dr. Collins referenced studies on the antibody response to coronavirus infection that showed detectable IgM antibodies about four days after infection, with the peak level at day 20. *Id.* (citing Hou et al., *Detection of IgM and IgG antibodies in patients with coronavirus disease 2019*, 9(5) CLINICAL & TRANSLATIONAL IMMUNOLOGY (2020) (Ex. E, Tab 6) ("Hou"); Liu et al., *Patterns of IgG and IgM antibody response in COVID-19 patients*, 9(1) EMERGING MICROBES & INFECTIONS 1269-74 (2020) (Ex. E, Tab 7) ("Liu")). IgG antibodies to coronavirus were detectable about seven days post-infection and peaked at 25 days. *Id.* "Given that Ms. Nelson's antibody levels were described as 'markedly elevated[,'] antibody response was more likely than not near its peak on November 1st when the sample was collected." *Id.* Dr. Collins concluded, based on the November 1, 2015 test results, that Petitioner's exposure to *C. jejuni* "could have been approximately three weeks prior to the sample date (approximately October 10th)." *Id.*

Lastly, Dr. Collins opined that Petitioner's November 1, 2015 *C. jejuni* test result indicated she had an active infection. Fourth Collins Rep. at 3. A study measuring antibody responses in *C. jejuni* patients showed that the peak antibody response was generally three weeks following infection as defined by symptom development. *Id.* (citing Taylor). Dr. Collins argued that "high levels of antibody are indicative of a recent immune response with a peak of antibody production about three weeks following development of symptoms. Over time, following the peak, the antibody response typically declines." *Id.*

IV. LEGAL FRAMEWORK

A. Petitioner's Burden

Under the Vaccine Act, a petitioner may prevail in one of two ways. First, a petitioner may demonstrate that she suffered a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the time period provided in the Table. § 11(c)(1)(C)(i). "In such a case, causation is presumed." *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006); *see* § 13(a)(1)(B). Second, where the alleged injury is not listed in the Vaccine Injury Table, a petitioner may demonstrate that she suffered an "off-Table" injury. § 11(c)(1)(C)(ii).

For both Table and non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1324 (Fed. Cir. 2010); *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873* (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a

substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records of by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*. *Althen* requires that petitioner establish by preponderant evidence that the vaccinations he received caused her injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citations omitted). 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 a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioner may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378-79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325-26). Special Masters, despite their expertise, are not empowered by statute to conclusively resolve what are complex scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras v. Sec'y of Health & Hum. Servs*., 121 Fed. Cl. 230, 245 (2015) ("[p]lausibility … in many cases may be enough to satisfy *Althen* prong one" (emphasis in original)), *vacated on other grounds*, 844 F.3d 1363 (Fed. Cir. 2017). But this does not negate or reduce a petitioner's ultimate burden to establish her overall entitlement to damages by preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs*., 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted).

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; *Andreu*, 569 F.3d at 1375-77; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, because they are created contemporaneously with the

treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

However, medical records and/or statements of a treating physician's views do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing … that mandates that the testimony of a treating physician is sacrosanct -- that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should also be weighed against other, contrary evidence also present in the record -- including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs*., 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Caves v. Sec'y of Health & Hum. Servs*., No. 06-522V 2011 WL 1935813 at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without op.*, 475 Fed. App'x 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *de Bazan v. Sec'y of Health & Hum. Servs*., 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must also coincide with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs*., 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand on other grounds*, 105 Fed. Cl. 353 (2012), *aff'd without op.,* 503 F. App'x 952 (Fed. Cir. 2013). *Koehn v. Sec'y of Health & Hum. Servs*., No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for review den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. Law Governing Analysis of Fact Evidence

The process for making factual determinations in Vaccine Program cases begins with analyzing the medical records, which are required to be filed with the petition. Section 11(c)(2). The special master is required to consider "all [] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 413, 417 (Fed. Cir. 1993) (it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral

testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records created contemporaneously with the events they describe are generally trustworthy because they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions," where "accuracy has an extra premium." *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378 (Fed. Cir. 2021) citing *Cucuras*, 993 F.2d at 1528. This presumption is based on the linked proposition that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11-685V, 2013 WL 1880825 at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) *mot. for rev. denied*, 142 Fed. Cl. 247, 251-52 (2019), *vacated on other grounds and remanded*, 809 Fed. Appx. 843 (Fed. Cir. Apr. 7, 2020).

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie v. Sec'y of Health & Hum. Servs.*, No. 03-1585V, 2005 WL 6117475 at *20 (Fed. Cl. Spec. Mstr. Dec. 12, 2005). Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony -- especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; see also *Murphy v. Sec'y of Health & Hum. Servs.*, 23 Cl. Ct. 726, 733 (1991), *aff'd per curiam*, 968 F.2d 1226 (Fed. Cir. 1992), *cert. den'd*, *Murphy v. Sullivan*, 506 U.S. 974 (1992) (citing *United States v. U.S. Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

However, there are situations in which compelling oral 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) ("like 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*, 2005 WL 6117475 at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent and compelling." *Sanchez*, 2013 WL 1880825 at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs.*, No. 90-2808V, 1998 WL 408611 at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (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 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, 203-04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory connecting the vaccine to the injury often requires a petitioner to present expert testimony in support of his or her claim. *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993). *See 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). "The *Daubert* 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 play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora. *Daubert* factors are employed by judges to exclude evidence that is unreliable and potentially confusing to a jury. In Vaccine Program cases, these factors are used in the weighing of the reliability of scientific evidence. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66-67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate persuasiveness and reliability of expert testimony has routinely been upheld. *See*, e.g., *Snyder*, 88 Fed. Cl. at 743. In this matter, (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)). 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. Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Id.* at 1325-26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously

explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

D. Consideration of Medical Literature

Finally, although this decision discusses some but not all of the medical literature in detail, I have reviewed and considered all of the medical records and literature submitted in this matter. *See 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 [s]he does not explicitly reference such evidence in h[er] decision."); *Simanski v. Sec'y of Health & Hum. Servs.*, 115 Fed. Cl. 407, 436 (2014) ("[A] Special Master is 'not required to discuss every piece of evidence or testimony in her decision.'" (citation omitted)), *aff'd*, 601 F. App'x 982 (Fed. Cir. 2015).

V. ANALYSIS

A. Issues Presented

Petitioner alleges two alternative theories of recovery. First, she alleges she has suffered the Table injury of GBS following flu vaccination. Motion at 10. Second, if the Table requirements are not found to be satisfied, Petitioner argues she has established GBS caused-in-fact by her flu vaccination, meeting the *Althen* prongs of causation. *Id.* at 11.

Respondent does not dispute that Petitioner developed GBS. Instead, he argues that Petitioner has not proven the Table injury of GBS following flu vaccination, because she did not establish the onset of her GBS fell within the requisite period after vaccination. Response at 8. Likewise, he contends that Petitioner has not proven any of the *Althen* prongs. *Id.* at 13-20. Finally, he argues Petitioner's GBS was more likely caused by *C. jejuni* infection than by her flu vaccination. *Id.* at 20.

B. Table Claim

To establish the Table injury of GBS following flu vaccination, Petitioner must preponderantly prove she developed GBS from three to 42 days after flu vaccination. 42 C.F.R. § 100.3(a)(XIV)(D). The parties here agree that Petitioner developed GBS. *See* First Kelkar Rep. at 3; First Collins Rep. at 12. I agree that this diagnosis is supported by the medical records.

The operative question for the Table claim, then, is whether the onset of Petitioner's GBS occurred within the 3-42-day timeframe following vaccination. That question, in turn, presents two subsidiary questions. The first is *when* Petitioner developed various symptoms that potentially signified the onset of GBS. The second is *which* of those symptoms were related to GBS, as opposed to other conditions.

1. When did potential GBS symptoms begin?

As described in more detail below, the parties' experts focused on back pain or weakness

as the symptoms most likely signifying the onset of GBS in Petitioner. Based on my review of the contemporaneous medical records, as well as Petitioner's affidavit and supplemental declaration, I conclude that she developed back pain on or before October 21, 2015. She most likely developed general weakness on October 27 and leg weakness on October 30.

Petitioner received the flu vaccination on October 21, 2015, during a visit to podiatrist Dr. Christina Knutson. Ex. 36 at 138. She saw Dr. Knutson that day for numbness in all of her toes on both feet. *Id.* at 137. At that visit, she had normal strength but had restricted range of motion of the midfoot and some pain in the second and third intermetatarsal spaces, which was worse on the left side. *Id.* Dr. Knutson included peripheral neuropathy on her differential diagnosis, suggesting a possible neurologic cause of these symptoms. *Id.* at 138. Neither expert in this case, however, addressed whether the foot symptoms Petitioner reported at that visit might have been an early sign of GBS.

Petitioner's supplemental declaration stated that, to the best of her recollection, she and her husband drove from Minnesota to Florida beginning six days later, on October 27, 2015. Ex. 71 at 1. They spent the night of October 27 in Georgia and arrived in Florida the night of October 28. *Id.* She first went to the ER early in the morning of October 29, 2015, eight days after the vaccination. Ex. 29 at 6. She complained of nausea and sharp, non-radiating pain in her left mid-back, which had begun the previous night, October 28. *Id.*

Petitioner saw her PCP, Dr. Jefferson, on October 30, 2015, after being discharged from the ER. Ex. 21 at 87. She still complained of nausea and abdominal pain. *Id.* At that time, she expanded on her history, reporting that she had experienced one day of "dry heaves" about one month earlier. *Id.* Perhaps at odds with this report, she also said that her back pain "was first," prompting her to take prednisone for six days, and then "everything seemed to get worse when she did the prednisone about 4 days later," leading Dr. Jefferson to suspect an ulcer. *Id.*

An exam done at the October 30 visit with Dr. Jefferson showed no signs of balance or gait problems, cranial nerve problems, paralysis, or other motor dysfunction. Ex. 21 at 92.

The next day, October 31, 2015, Petitioner was taken back to the hospital by EMS. Ex. 29 at 37. She reported to the paramedics that she had experienced four days of weakness. *Id.* She also said this weakness had been present "since arriving" in Florida. *Id.*

Later on October 31, Petitioner saw Dr. Jefferson again in the hospital. Ex. 29 at 50. He documented a detailed history. *Id.* Petitioner repeated her story that she had previously taken prednisone for six days. She also reported that after that period, she stopped taking the prednisone, and "a couple of days" after that began experiencing abdominal symptoms radiating to the back:

> [L]ooking back at the history before this happened [it] started about a week before, she had a day of dry heaves for 24 hours and then it was gone and then it happened again a couple of days later and it was more of the acute onset of symptoms at that point. O[f] interest, she had ***been having some back pain prior to that and had taken a course of prednisone for 6 days she says and then had stopped it a couple of days after that is when she started to get more of the abdominal symptoms***

> ***radiating to the back***, thought it could possibly be an ulcer, so yesterday we put her on Dexilant twice a day, [C]arafate liquid 4 times a day, ondansetron pills, Phenergan as a backup and she was supposed to follow up with this on Monday.

Id.

Petitioner further explained that her leg weakness began after her office visit with Dr. Jefferson the previous day, October 30. Ex. 29 at 50. After several falls, she "decided it was time to come into [the ER] to get evaluated." *Id.*

a. Back pain

I construe the medical records to indicate Petitioner's back pain likely began no later than October 21, 2015, the day of the vaccination. On October 30 and 31, 2015, Petitioner twice reported that, before she first went to the ER on October 29, she took a six-day course of prednisone for back pain. Ex. 29 at 30, 50. She also added that at least "a couple" days passed after she finished the prednisone before her acute, radiating abdominal pain began, prompting her to go to the ER. *Id.* This story puts the onset of back pain at least eight days prior to October 29, or October 21.[16]

These accounts are credible for several reasons. First, Petitioner made these reports during the first days of her treatment, when her recollection would have been clearest. As Dr. Collins persuasively explained, physicians are trained to give greatest credence to histories taken closest in time to the events in question and directly from the patient.[17] Third Collins Rep. at 1. Second, the detail about taking the six-day course of prednisone was quite specific, and the fact that Petitioner repeated it twice adds to its reliability. Third, this story was documented in some detail by Dr. Jefferson, who evidently believed it to be important to deciphering her condition.

b. Weakness

According to the October 31, 2015 EMS record, Petitioner reported four days of "weakness" prior to making the EMS call. Ex. 29 at 37.[18] Later that day, she explained to Dr.

[16] At the October 30 office visit with Dr. Jefferson, Petitioner suggested her symptoms got worse about four days after the prednisone, but on October 31, she said her acute symptoms began "a couple of days" after discontinuing prednisone. Ex. 29 at 30, 50. This puts the beginning of the acute symptoms from two to four days after the six-day course of prednisone, meaning the back pain started at least eight days before October 29, and perhaps started ten days earlier.

[17] Dr. Kelkar agreed that, based on the records, Petitioner's back pain "was within a day or two following the flu vaccination on 10/21." Third Kelkar Rep. at 6 n.1.

[18] Petitioner also told EMS had felt weakness "since" arriving in Florida. Ex. 29 at 37. As stated above, Petitioner's supplemental declaration stated that she arrived in Florida the night of October 28, only three days before she called EMS. Ex. 71 at 1. The timeline in the declaration was provided nine years after the

Jefferson that she experienced leg weakness beginning after her visit with him the day before, putting the onset of leg weakness on October 30. Ex. 29 at 50. These accounts were made close in time to the events in question and probably reflected Petitioner's clearest and best recollection of her experience. Third Collins Rep. at 1. Taken together, these reports indicate that more general symptoms of weakness began on or about October 27 (six days after vaccination), while leg weakness began October 30 (nine days post-vaccination).

2. Which symptom(s) signified the onset of GBS?

The question of which symptom marked the onset of GBS is determinative of Petitioner's Table injury claim. If back pain was the first GBS symptom, then the onset of GBS was on the day of vaccination, not between three to 42 days thereafter, as required by the Table. If weakness was the first GBS symptom, onset fell within the Table timeframe.

Dr. Kelkar stated that pain radiating to the back is "extremely common in GBS" and is often the presenting symptom of the condition. Second Kelkar Rep. at 3. He submitted several papers supporting this. *See* Yao at 4 (34.5% of GBS patients reported pain during the acute phase; low back pain was the second most common area of reported pain); Ruts at 1441 (66% of GBS patients had pain during the acute phase, and 36% had pain in the two weeks prior to the onset of weakness; "[l]ow-back or back pain was notably present in the acute phase."); Moulin at 328 (89.1% of GBS patients described pain during the course of their illness, with a majority of those patients reporting pain preceding the onset of weakness). Dr. Kelkar did not, however, believe back pain was the first symptom of *Petitioner's* GBS. He opined that Petitioner's back pain was instead "related to her pre-existing history." Fourth Kelkar Rep. at 1. He explained that Petitioner had experienced previous flares of back pain, for which she had been prescribed steroids. *Id.* Additionally, "[s]omewhere down the line" before her hospitalization she stopped taking her rheumatoid arthritis medication, methotrexate, suggesting her back pain might have been caused by a flare-up of her arthritis. *Id.* By contrast, in Dr. Kelkar's view, Petitioner's "tingling and weakness" were "absolutely indicative of onset of GBS." *Id.*

Dr. Kelkar also said that he focused on weakness as the presenting symptom of GBS in Petitioner's case because the medical records were "quite confusing" and had "conflicting reports" concerning her timing and use of prednisone for the back pain. Third Kelkar Rep. at 6 n.1.

Dr. Collins agreed with Dr. Kelkar that there is "extensive" literature supporting the proposition that back pain can be the presenting symptom of GBS. Fourth Collins Rep. at 1 (citing Moulin at 328). She opined that Petitioner's back pain did, indeed, constitute her first GBS symptom. *Id.* She noted that Petitioner's pain seemed to resolve with prednisone, suggesting it was immune-mediated and "consistent with GBS-induced pain." *Id.* She also pointed out that the

events, however, and by its own account only stated Petitioner's best recollection of what happened. *Id.* As such, I credit the story provided to EMS on October 31, 2015.

back pain must have been severe, as it prompted her to take her prescription prednisone for relief. Third Collins Rep. at 2.

I find Dr. Collins's opinion on this question to be more persuasive than Dr. Kelkar's, and thus I conclude that Petitioner's bout of back pain marked the onset of her GBS. Importantly, both experts agreed that back pain is very common in GBS and is commonly a presenting symptom. Additionally, I am persuaded by Dr. Collins's point that Petitioner's successful use of prednisone to treat her pain meant (1) it was severe; and (2) it was immune-mediated and responsive to steroids. Moreover, the records in this case demonstrate that both Petitioner and Dr. Jefferson viewed the back pain to be the beginning of an acute course of symptoms that eventually required her to seek emergency care. Petitioner twice reported, and Dr. Jefferson twice recorded, the onset of back pain, six-day course of prednisone, and subsequent onset of radiating abdominal pain several days later as a unified history of Petitioner's present illness. Ex. 29 at 30, 50. Had Dr. Jefferson considered the back pain to be an entirely distinct problem relating to Petitioner's pre-existing issues, he would not have emphasized it as part of the medical history culminating in her hospitalization.

Finally, I am not persuaded by Dr. Kelkar's argument that weakness marked the onset of GBS because the medical records were unclear about the timing or use of prednisone for back pain. As noted, the records are actually quite specific about Petitioner's reported use of prednisone for six days for back pain, a story she repeated twice in the first days of treatment. It is unpersuasive to dismiss these accounts as immaterial.

I therefore conclude that the onset of Petitioner's GBS was October 21, 2015, the day of vaccination. Accordingly, she does not meet the criteria for the Table injury of GBS following flu vaccination, which requires the onset of GBS to fall within three to 42 days after the vaccination.

C. Causation-In-Fact Claim

With respect to the causation-in-fact theory, the parties dispute the question posed by *Althen* prong one; namely, whether the flu vaccination can cause GBS. *See, e.g.*, First Kelkar Rep. at 3; First Collins Rep. at 5-6; Third Kelkar Rep. at 5; Third Collins Rep. at 3-5. I need not resolve that question here, because I conclude Petitioner has failed to satisfy *Althen* prongs two or three, requiring denial of her claim.

1. *Althen* Prong Two

Under *Althen* prong two, Petitioner must establish "a logical sequence of cause and effect showing that the vaccination was the reason for the injury." *Althen*, 418 F.3d at 1278. The sequence of cause and effect must be "'logical' and legally probable, not medically or scientifically certain." *Andreu*, 569 F.3d at 1380 (quoting *Knudsen*, 35 F.3d at 548-49). A petitioner is not required to show "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities to establish a

logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1325. Instead, circumstantial evidence and reliable medical opinions may be sufficient to satisfy the second *Althen* prong. *Id.*

Special masters are expected to consider the views of treating doctors. *Capizzano*, 440 F.3d at 1326. Such views are often persuasive because the doctors have direct experience with the patient whom they are diagnosing -- but they are not necessarily dispositive of the causation question. *See McCulloch v. Sec'y of Health & Hum. Servs.*, No. 09-293V, 2015 WL 3640610, at *20 (Fed. Cl. Spec. Mstr. May 22, 2015).

Although a petitioner does not bear the burden of eliminating all alternative causes for her injury, it is appropriate for a special master to consider evidence relating to such alternative causes in assessing the *Althen* prongs. *Winkler v. Sec'y of Health & Hum. Servs.*, 88 F.4th 958, 963 (Fed. Cir. 2023); *Doe 11 v. Sec'y of Health & Hum. Servs.*, 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *Walther v. Sec'y of Health & Hum. Servs.*, 485 F.3d 1146, 1151 (Fed. Cir. 2007).

a. Petitioner's GBS was more likely caused by a C. jejuni infection than her flu vaccination.

In this case, I am persuaded by Dr. Collins's opinion that Petitioner's GBS was likely caused by a *C. jejuni* infection, negating a logical connection between the vaccination and her injury. *See* First Collins Rep. at 12. As Dr. Kelkar acknowledged, "GBS can be triggered by a variety of infections and the most common infection is campylobacter jejuni." First Kelkar Rep. at 3. Up to 30% of GBS patients are positive for *C. jejuni*, and one in 1,000 cases of *C. jejuni* are estimated to result in GBS. *Id.* (citing Israeli at 122); *see also* First Collins Rep. at 8 (a history of *C. jejuni* infection is reported in 26-60% of GBS cases).

1) Petitioner's test results and clinical presentation were consistent with active or recent *C. jejuni* infection.

According to her blood test from November 1, 2015, Petitioner's blood had a high level of *C. jejuni* antibodies. Ex. 29 at 279. The test report explained that a level greater than 1.10 indicated detection of the antibody; Petitioner had a level of 2.34, which was designated in the report as "high." *Id.* The report further explained that "[m]arkedly elevated levels of antibodies recognizing *C. jejuni* typically indicate recent or ongoing infection." *Id.*

Despite this result, Dr. Kelkar maintained that it was unlikely Petitioner had a recent or active *C. jejuni* infection at the time of her hospitalization. *See* First Kelkar Rep. at 3. He pointed out that the blood test did not differentiate among IgM, IgA, and IgG antibodies, and thus he argued it did not reveal whether Petitioner's *C. jejuni* exposure was active, recent, or remote.[19] *Id.* He said the "high" designation did not necessarily indicate that she had an active or recent infection.

[19] According to Dr. Kelkar, he learned this information from a discussion with a medical director for Quest, which generated the report. First Kelkar Rep. at 3. The statements of the Quest representative are hearsay. Nonetheless, even assuming the truth of the statement that Quest's test did not differentiate among *C. jejuni* antibodies, as discussed, the test report *itself* supports the conclusion that Petitioner had an acute or recent infection.

Fourth Kelkar Rep. at 3. But this is contrary to the test report's characterization of the results. Moreover, as Dr. Collins pointed out, the Taylor study showed a significant rise in *all* classes of *C. jejuni* antibodies in patients with acute infections compared to controls, with the highest level at about three weeks post-symptom onset, after which antibody levels began to decline. Fourth Collins Rep. at 3; Taylor at 254 (Fig. 3). Thus, the "high" level identified in the test was consistent with acute or recent infection.

Dr. Kelkar also questioned whether Petitioner had symptoms indicative of an active or recent *C. jejuni* infection. Second Kelkar Rep. at 2. He said she did not have diarrhea, a fever, or an elevated white blood cell ("WBC") count[20] at any time during her course. *Id.* The fact that Petitioner did not have diarrhea did not rule out *C. jejuni*. In the Rees study of GBS in *C. jejuni*-infected patients, only 19 of 27 patients who tested positive for *C. jejuni* reported experiencing diarrhea in the 12 weeks before the onset of GBS. Rees at 1376. Also, the Nandkishor case report described a case of *C. jejuni* infection in which the patient did not have diarrhea, nausea, vomiting, *or* chills. Nandkishor at 1.

As Dr. Collins commented, Petitioner had significant gastrointestinal symptoms, including abdominal pain and "recalcitrant nausea and vomiting," which prompted her to seek medical attention on October 29, 2015. Second Collins Rep. at 2-4. A few days later, she was seen in the hospital by a gastroenterologist for these symptoms. Ex. 29 at 53 (consultation with gastroenterologist Dr. Dumas). Dr. Kelkar suggested the symptoms might have been caused by peptic ulcer disease or restaurant food. Third Kelkar Rep. at 6-7. This was not the view of Petitioner's treating physicians, however. Although they considered a number of possibilities, none of them concluded her gastrointestinal symptoms were due to an ulcer or a food-borne illness other than *C. jejuni*. After Petitioner was diagnosed with GBS, Dr. Dumas surmised that her gastrointestinal symptoms were related to that condition, not an ulcer or carcinoma as he originally considered. *Id.* at 82. Also, infectious disease specialist Dr. Farooq did not suspect food poisoning such as botulism explained Petitioner's presentation; he believed *C. jejuni* was the only infectious illness that fit her symptoms. *Id.* at 65.

2) Petitioner's GBS presentation was consistent with *C. jejuni* causation.

Dr. Kelkar did not believe Petitioner's symptoms were suggestive of the AMAN subtype of GBS, which is an axonal form of the disease primarily associated with *C. jejuni*. Second Kelkar Rep. at 4. Instead, he opined she developed AIDP, a demyelinating form of GBS. *Id.* He claimed Petitioner had a faster recovery and a better outcome than would be expected in AMAN. First Kelkar Rep. at 4.

Petitioner's treating physicians did not diagnose her with any particular subtype of GBS. Dr. Collins persuasively explained that her clinical course fit with *C. jejuni* causation. *See* First Collins Rep. at 11. With respect to her rate of recovery, Chowdhury & Arora noted that the Ho

[20] This is inaccurate. Dr. Jefferson noted on October 31, 2015, that Petitioner's white blood cell count was over 14,000 at the time of her admission. Ex. 29 at 50. Petitioner's hematology results showed high WBCs of 14,800 on October 31, 2015 and 15,700 on November 1, 2015, with the reference range between 3,600-11,200. *Id.* at 244.

study "found a comparable rate of recovery in AMAN and AIDP patients (average 40 days)." Chowdhury & Arora at 270. The Hiraga study found variability in the recovery rates among AMAN patients, noting that at least some patients with severe AMAN (as shown by EMG results) recovered rapidly. Hiraga at 721. Moreover, contrary to Dr. Kelkar's view, Petitioner's recovery was fairly slow. In Hiraga, a "slow" recovery from GBS was defined as being unable to walk independently six months after onset. *Id.* at 720. Petitioner met this definition: nearly three years after the vaccination, neurologist Dr. Ahlskog characterized Petitioner's recovery as slow and incomplete, remarking that after her illness she newly required a cane for balance and used a scooter. Ex. 32 at 212.

Also, Dr. Kelkar argued that Petitioner's EMG – taken "one year" after the onset of her GBS – showed "nearly normal" results, inconsistent with the expected recovery from AMAN. Second Kelkar Rep. at 5. But in fact, the EMG was performed by Dr. Ahlskog in September 2018, nearly *three* years after the vaccination. Ex. 32 at 214. Thus, Dr. Kelkar's opinion on the import of this study was unpersuasive.

Finally, Dr. Collins persuasively pointed out that both AMAN and AIDP are associated with *C. jejuni* infection. First Collins Rep. at 11; *see* Chowdhury & Arora at 270 ("Both axonal GBS and AIDP cases have been described in association with *C. jejuni*."). Thus, regardless of which subtype Petitioner had, it could have been caused by *C. jejuni.*

3) The onset of Petitioner's GBS was consistent with *C. jejuni* causation.

As discussed above, the records show Petitioner likely developed back pain on or about October 21, 2015, the day of vaccination. The records indicate she started having gastrointestinal symptoms about six days later, during her drive to Florida from October 27-28. Ex. 29 at 30. She told infectious disease specialist Dr. Farooq that the first night of the drive, she had abdominal pain and nausea, but no diarrhea. *Id.* at 62. She had to take Pepcid AC during the drive. *Id.* at 30. By the time she first presented to the ER on October 29, she was experiencing nausea and sharp, non-radiating left mid-back pain, for which she had taken oxycodone. *Id.* at 6. Thus, it appears that Petitioner's GBS symptoms predated the onset of her gastrointestinal symptoms.

However, Dr. Collins persuasively explained that Petitioner's *exposure* to *C. jejuni* probably predated her development of GBS symptoms. She pointed out that "[u]nlike vaccination where the date of exposure is precisely known, it is harder to determine when an individual initially became infected with a pathogen. This is because pathogens have an incubation period following exposure and prior to symptoms onset that is highly variable." Fourth Collins Rep. at 2-3. For example, in the Rees study, while the median interval between the onset of diarrhea and GBS symptoms was nine days, 30% of the *C. jejuni*-positive patients who developed GBS did not have *any* diarrheal illness preceding onset of their GBS. *Id.* at 3; *see* Rees at 1376.

Dr. Collins further explained that "[i]t takes time following exposure to a pathogen for antibody to become detectable." Fourth Collins Rep. at 3. Thus, the result of Petitioner's November 1, 2015 blood test "indicates that her infection was ongoing for long enough for an immune response to develop and for the antibody levels to be very high." *Id.* "Given that Ms.

Nelson's antibody levels were described as 'markedly elevated[,'] antibody response was more likely than not near its peak on November 1st when the sample was collected." *Id.* Thus, Petitioner's exposure to *C. jejuni* "could have been approximately three weeks prior to the sample date (approximately October 10th)." *Id.*

The literature supports Dr. Collins's opinion. In the Taylor study, all classes of *C. jejuni* antibodies peaked about three weeks after the onset of symptoms. Taylor at 254 (Fig. 3). Similarly, in Liu, IgM antibodies for Covid-19 peaked 20 days after the onset of symptoms, while IgG antibodies peaked at day 25. Liu at 2. In Hou, Covid-19 IgM levels peaked at 2-3 weeks after symptom onset, while IgG antibodies increased more slowly but stayed high for up to seven weeks. Hou at 5. Collectively, these studies show that exposure to a pathogen will precede the detectability of high levels of antibodies potentially by several weeks. Based on this, given her "high" test result on November 1, 2015, Petitioner's *C. jejuni* exposure may have occurred as early as October 10, about 11 days before the onset of back pain on October 21, 2015 (which marked the beginning of her GBS).

Notably, Dr. Kelkar agreed that Petitioner's *C. jejuni* exposure likely predated the vaccination. Based on his view that weakness marked the onset of Petitioner's GBS, he opined that Petitioner's *C. jejuni* exposure likely occurred about 10 days before her weakness began, on October 17, 2015. Fourth Kelkar Rep. at 2.

4) The evidence does not substantiate the claim that Petitioner's GBS was concurrently caused by the vaccine and *C. jejuni* infection.

Dr. Kelkar proposed that the flu vaccine and *C. jejuni* infection could have acted synergistically to cause Petitioner's GBS. First Kelkar Rep. at 3; Third Kelkar Rep. at 8.

I did not find Dr. Kelkar's opinion on this question to be persuasive. Aside from the fact that the onset of Petitioner's GBS was too soon after the vaccination to implicate it as a causal factor, which is discussed below, the literature Dr. Kelkar cited did not adequately substantiate his proposition.

Dr. Kelkar first cited the Greene study. First Kelkar Rep. at 3. Greene reported on six cases of GBS following both respiratory infection and flu vaccination. Greene at 1100. The authors did not attempt to measure or describe a possible synergistic effect of these factors, though they did mention this possibility. *Id.* at 1106. Notably, they considered antecedent respiratory infection a confounding factor in assessing the independent risk of the vaccine for GBS. *Id.*

Dr. Kelkar cited the Parra study. Parra reported an association of GBS with Zika virus infection based on a study done in Colombia. Parra at 1522. Many of the study subjects tested positive for antibodies indicating past exposure to dengue virus. *Id.* None of the subjects had active dengue virus infection. *Id.* Dr. Kelkar also cited Amereller, a case report of a patient who had concurrent *C. jejuni* and *Y. pseudotuberculosis* infections and developed GBS and mesenteric lymphadenopathy, as well as recurrent abdominal symptoms. Amereller at 1. These papers

addressed the potential role of concurrent or successive *infections* in GBS; neither suggests that GBS can be caused by a synergy between flu *vaccine* and *C. jejuni* infection.

Dr. Kelkar referenced Nachamkin, in which the authors reported the development of anti-ganglioside antibodies in mice who were immunized with seasonal flu vaccines. Nachamkin at 226. The authors did not comment on whether concurrent *C. jejuni* infection and flu vaccination could combine to cause GBS in humans.

Dr. Kelkar cited Westall, which proposed that post-vaccination neuropathy was caused by a combination of molecular mimicry and the body's reaction to vaccine adjuvants. Westall at 251. As Dr. Collins persuasively noted, this nearly 40-year-old paper was published "well before a substantive body enlightened our understanding of risk factors associated with [GBS]." Third Collins Rep. at 8. Moreover, the paper did not hypothesize or present any evidence that the flu vaccine can combine with *C. jejuni* to cause GBS.

Finally, Dr. Kelkar cited Wirguin, Cusick, and Steiner. In Wirguin, rats immunized with both *C. jejuni* lipopolysaccharides and keyhole limpet hemocyanin developed high titers of antiganglioside antibodies. Wirguin at 138. In Cusick, the authors discussed the hypothesis that the expression of dual T cell receptors on a single T cell could leave the host vulnerable to autoimmunity. Cusick at 102. In Steiner, the authors proposed that GBS could be caused by successive infections due to transient immunosuppression. Steiner at 32. None of these studies discussed a possible synergistic effect produced by flu vaccination and *C. jejuni* infection.

5) Conclusion

In *Winkler*, the Federal Circuit affirmed the denial of a petition alleging GBS caused-in-fact by a tetanus, diphtheria, and acellular pertussis ("Tdap") vaccination. 88 F.4th at 963. The special master determined that the petitioner had failed to satisfy *Althen* prong two, because the evidence showed he suffered a gastrointestinal illness before the onset of his GBS symptoms. *Id.* at 962. The illness was clinically consistent with *C. jejuni*, but that diagnosis was not confirmed. *Id.* The Federal Circuit noted that, even though the Vaccine Act did not require the petitioner to eliminate all possible alternative causes of the injury, the special master could consider potential alternative causes in determining whether a logical sequence of cause and effect existed between the vaccination and the injury. *Id.* Moreover, the special master was not required to make a definitive finding that an alternative cause was the more likely cause of the GBS; she could instead rely on evidence of a *possible* alternative cause to find *Althen* prong two unsatisfied. *Id.* at 963 (citing *Stone v. Sec'y of Health & Hum. Servs.*, 676 F.3d 1373, 1379 (Fed. Cir. 2012)).

Similarly, in this case the evidence shows Petitioner likely suffered from a *C. jejuni* infection, and that infection was a more likely cause of her GBS than the flu vaccination, particularly given the same-day onset of GBS symptoms and vaccination. Thus, *Althen* prong two is not satisfied here.

b. The medical records do not show Petitioner's treating physicians believed her GBS to be vaccine-caused.

Lastly, I did not find persuasive evidence in the medical records that Petitioner's treating physicians considered her GBS to be caused by the flu vaccine, though the vaccine was discussed as a possible cause. The only treating physician who explicitly attributed Petitioner's GBS to her flu vaccination was neurologist Dr. Coleman, who summarily characterized her case as "likely triggered by influenza vaccine," without further comment and weeks after the onset of Petitioner's GBS. Ex. 29 at 135.

The question of causation was considered primarily by the infectious disease specialist who saw Petitioner in the hospital several times. The first time that physician saw Petitioner, he or she noted that she had received the flu vaccine but stated that GBS "has always been a questionable side effect from flu vaccine." Ex. 29 at 103. After Petitioner's positive *C. jejuni* result came back, that physician remarked that her GBS could be related to either the flu vaccination or the infection and that its etiology was unclear. *Id.* at 120. Despite this equivocation, the physician did attempt to report Petitioner's case to VAERS as a vaccine injury. *Id.* at 114.

None of the other treating physicians robustly discussed Petitioner's flu vaccine as a possible cause of her GBS. Overall, the records do not provide persuasive evidence that the treating physicians believed Petitioner's GBS was caused by the vaccine.

2. *Althen* Prong Three

Althen prong three contains two parts. First, a petitioner must establish the "timeframe for which it is medically acceptable to infer causation," and second, he must demonstrate that the onset of the disease occurred in this period. *Shapiro,* 101 Fed. Cl. at 542-43. Having decided the onset of Petitioner's GBS was October 21, 2015, the day of vaccination, the remaining issue is whether the same-day onset of GBS symptoms following flu vaccination is medically acceptable.

Dr. Kelkar opined that, "[i]f back pain is considered the onset of GBS the shorter timing is supported in the literature." Third Kelkar Rep. at 6 n.1. Dr. Collins, by contrast, consistently opined that an onset of GBS less than 48 hours after flu vaccination would be too short to support vaccine causation. First Collins Rep. at 12; Second Collins Rep. at 1; Third Collins Rep. at 2.

I am not persuaded by Dr. Kelkar's opinion on this question. The only study he cited, Schonberger, did not report any cases of GBS starting the same day as flu vaccination, or even one or two days post-vaccination. Instead, the study reported some GBS cases occurring in the first *week* after flu vaccination, with the peak risk occurring two to three weeks after vaccination. Schonberger at 112 (Fig. 4). The fact that GBS occurred within a week of vaccination is entirely consistent with the 3–42-day onset timeframe set forth in the Vaccine Injury Table. *Id.* The Schonberger study lacks the specificity to adequately support the proposition that onset within *less* than three days would be medically acceptable.

Furthermore, onset of GBS less than three days following vaccination has consistently been found to be too short in the Vaccine Program. *See, e.g., Flowers v. Sec'y of Health & Hum. Servs.*, 173 Fed. Cl. 613 (2024) (upholding dismissal of an entire claim where it was determined that onset of the petitioner's GBS occurred too soon to qualify as on- or off-Table claim (one to two days following vaccination)); *Velasquez v. Sec'y of Health & Hum. Servs.*, No. 19-1703V, 2024 WL

829599, at *16 (Fed. Cl. Spec. Mstr. Jan. 31, 2024) (finding that a one-day onset of GBS is not medically acceptable, absent a showing specific to the claimant's circumstances that would justify stretching the Table timeframe for onset of GBS following receipt of a flu vaccine); *Rowan v. Sec'y of Health & Hum. Servs*., No. 17-760V, 2020 WL 2954954, at *16-19 (Fed. Cl. Spec. Mstr. Apr. 28, 2020) (finding a GBS onset sooner than three days post vaccination was not scientifically or medically supported by the record, given that GBS is known to be mediated by antibodies produced via the adaptive immune system, and this process takes longer than three days to result in symptoms); *Orton v. Sec'y of Health & Hum. Servs*., No. 13-631V, 2015 WL 1275459, at *3-4 (Fed. Cl. Spec. Mstr. Feb. 23, 2015) (finding a one-day onset of GBS following a flu vaccination was not substantiated by the evidence).[21] While I am not bound by other special masters' findings, I find the number of decisions concluding this persuasive. [22]

VI. CONCLUSION

Although I sympathize with Petitioner, after careful review of the record, I conclude that she has not met her burden to show that she is entitled to compensation under the Vaccine Act. **The petition is therefore dismissed. The clerk shall enter judgment accordingly.**[23]

IT IS SO ORDERED.

s/ Jennifer A. Shah
Jennifer A. Shah
Special Master

[21] Dr. Kelkar alternatively posited that the concurrent triggers of flu vaccination and infection could cause a more rapid onset of GBS following such exposures. Third Kelkar Rep. at 6 n.1. He cited the Stratton/IOM report, but he did not produce it. *Id.*

[22] Because Petitioner has not met her *prima facie* burden demonstrating either a Table injury or causation in fact, the burden has not shifted to Respondent to demonstrate that Petitioner's GBS was caused by a factor unrelated to the subject vaccination (e.g., the *C. jejuni* infection).

[23] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by each filing (either jointly or separately) a notice renouncing their right to seek review.