In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 18-1625V

Filed: March 25, 2026

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ROY ROMERO, *

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Petitioner, *

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v. *

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SECRETARY OF HEALTH *
AND HUMAN SERVICES, *

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Respondent. *

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Michael A. Baseluos, Esq., Baseluos Law Firm, PLLC, San Antonio, TX, for petitioner.
Naseem Kourosh, Esq., U.S. Department of Justice, Washington, DC, for respondent.

RULING ON ENTITLEMENT[1]

Roth, Special Master:

On October 19, 2018, Roy Romero ("petitioner") 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"). The petition alleged that Mr. Romero developed acute inflammatory demyelinating polyneuropathy ("AIDP") from the influenza ("flu") vaccination he received on October 21, 2016. Petition, ECF No. 1. He later amended the petition to clarify that AIDP is also known as "GBS," or Guillain-Barré syndrome. Amended Petition, ECF No. 13.

On December 18, 2023, petitioner filed the instant Motion for Ruling on the Record. ECF No. 125. Following careful review of all evidence, I find that petitioner has provided preponderant evidence that his flu vaccine was a substantial factor in causing his GBS and that but for the

[1] Because this Ruling contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Ruling will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), the parties have 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. Any changes will appear in the document posted on the website.

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

vaccination he would not have developed GBS.

I. Procedural History

The petition was filed on October 19, 2018, and assigned to the Special Processing Unit ("SPU"). Petition, ECF No. 1; ECF No. 5.

Petitioner filed medical records through December 12, 2021, ECF Nos. 1, 10, 12, 14, 15, 22, 25, 29, 30, 32, 33, 57, 59, 63, 67, and 68, when he filed his fourth and final statement of completion. ECF Nos. 9, 16, 34, 69. Respondent filed his Rule 4(c) Report on May 14, 2020, arguing that petitioner could not show a causal link between the flu vaccine and his GBS. ECF No. 41.

Thereafter, an Order to Show Cause issued for petitioner to show cause as to why his Table claim should not be dismissed. ECF No. 43. On March 11, 2021, the Chief Special Master issued his Findings of Fact and Conclusions of Law dismissing petitioner's Table claim. ECF No. 50. The case was permitted to proceed on causation in fact.

The claim was then reassigned to me. ECF No. 52. A status conference was held following which petitioner was ordered to file updated medical records, a supplemental affidavit, and all documentation supporting his alleged damages. ECF No. 54.

Petitioner then filed expert reports and supporting medical literature. Petitioner's Exhibits ("Pet. Ex.") 42-49, 52-54, ECF Nos. 74-80, 89-93, 107-08, 117-20, 122-23. Respondent also filed expert reports and medical literature. Respondent's Exhibits ("Resp. Ex.") A-G, ECF Nos. 82-86, 99, 101, 114-15.

At the July 26, 2023, status conference, the parties requested to file supplemental expert reports and agreed to proceed with a Motion for Ruling on the Record. ECF No. 113. Petitioner filed the instant Motion on December 18, 2023. ECF No. 125. Respondent filed his Response on March 18, 2024. ECF No. 130. Petitioner filed his Reply on April 18, 2024. ECF No. 131. Based on the evidence in the record and arguments of the parties, I determined that the parties have had a fair and complete opportunity to be heard.

This matter is now ripe for decision.

II. Factual Background

The detailed medical history that follows includes but is not limited to the factual findings made by the Chief Special Master in his Ruling on Onset, petitioner's Motion for Ruling on the Record, respondent's Response to the Motion for Ruling on the Record, petitioner's Reply and Respondent's Rule 4(c) report along with a thorough review of all the medical records filed.

A. Medical History Prior to Vaccination

Petitioner was born on June 26, 1967. Pet. Ex. 1 at 1.

Petitioner received medical care when needed at Ramos Family Medical Group and Texas MedClinic, an urgent care clinic, since 2014. He had no documented history of any prior health issues. Pet. Ex. 7 at 7.

On October 5, 2016, petitioner presented to Texas MedClinic reporting body aches, chills, eye burning, throat pain, hoarse voice, pain on inspiration, chest and stomach pain, excessive thirst, decreased appetite, and 102° fever. Pet. Ex. 7 at 1. Examination was normal but for nasal discharge. *Id*. A/B flu testing was negative. *Id*. at 5. *See* Pet. Ex. 8 at 5. He was diagnosed with flu-like symptoms and was prescribed Bentyl for stomach cramping with instructions to return if necessary. Pet. Ex. 7 at 1, 4.

Four days later, on October 9, 2016, petitioner presented to the emergency room ("ER") at Mission Trails Baptist Hospital ("Mission Trails") reporting abdominal pain that started on October 4, 2016, associated with consuming bad fast food. Pet. Ex. 15 at 14. He reported fever, chills, and diarrhea each time he ate or drank, four to five times a day, with stomach pain improved after bowel movements. *Id*. at 14, 22. Petitioner also reported a history of consuming alcohol, typically one to two beers weekly, but had binge drank over the weekend, approximately 12 cans of beer. *Id*. at 5, 22.

Petitioner presented because his stomach pain had worsened. There was no vomiting, but he did report some episodes of nausea. Pet. Ex. 15 at 14. He had a fever of 102° at a recent urgent care visit days before and was treated with fluids. *Id*. He had upper abdominal tenderness on examination but no fever. *Id*. at 15, 17, 24. A CT and ultrasound of the abdomen revealed a fatty liver and gallstones without inflammation. *Id*. at 18. Stool testing revealed fecal leukocytes[3] indicating infection or inflammation but no parasites, campylobacter infection, or shigella toxin. *Id*. at 122. Blood work revealed elevated AST,[4] CRP,[5] lipase,[6] glucose,[7] and A_{1C}.[8] *Id*. at 116-119. The diagnoses included diabetes mellitus ("DM") and acute pancreatitis. *Id*. at 2. It was unclear if petitioner's pancreatitis was the cause of his diarrhea, if his elevated lipase was due to viral gastroenteritis, or if his drinking was the cause. *Id*. at 5, 15. He was treated with ciprofloxacin[9] and

[3] A leukocyte is a type of "colorless blood cell." Leukocytes are also called "white blood cell[s]." "Leukocyte," Dorland's Illustrated Medical Dictionary 1015 (33rd ed. 2020).

[4] Aspartate transaminase, or "AST," is "an enzyme . . . present in most eukaryotic cells." "The serum level of [AST] . . . [is] frequently elevated in a variety of disorders causing tissue damage." "Aspartate Transaminase," Dorland's Illustrated Medical Dictionary 161 (33rd ed. 2020).

[5] A c-reactive protein, or "CRP," is "the most predominant of the acute-phase proteins." "C-reactive Protein," Dorland's Illustrated Medical Dictionary 1509 (33rd ed. 2020). A high-sensitivity CRP test is used to "determine the level of CRP in the blood." "[A]s the hs-CRP rises, the risk of cardiovascular disease increases." "High-sensitivity C-reactive Protein Test," Dorland's Illustrated Medical Dictionary 1862 (33rd ed. 2020).

[6] Elevated lipase levels in the blood "are seen in impaired liver function and pancreatitis." "Lipase Test," Dorland's Illustrated Medical Dictionary 1864 (33rd ed. 2020).

[7] Blood glucose testing is "a direct measurement of the blood glucose level. It is most commonly used in the evaluation of diabetic patients." Kathleen Deska Pagana & Timothy J. Pagana, Mosby's Manual of Diagnostic and Laboratory Tests 267 (3rd ed. 2006). "In general, true glucose elevations indicate diabetes mellitus (DM)." *Id*. at 268.

[8] Hemoglobin A_{1C} levels "are increased in persons with poorly controlled diabetes mellitus." "Hemoglobin A_{1C}," Dorland's Illustrated Medical Dictionary 830 (33rd ed. 2020).

[9] Also known as "Cipro," ciprofloxacin is an "antibacterial effective against many" bacteria, including many penicillin-resistant strains. "Ciprofloxacin," Dorland's Illustrated Medical Dictionary 357 (33rd ed. 2020).

Flagyl for possible C. Difficile ("C Diff") due to the presence of fecal leukocytes. C-diff testing was negative. *Id*. at 121. The elevated AST was thought to be due to a fatty liver. *Id*. at 15.

The diarrhea and stomach pain resolved by the next day. Pet. Ex. 15 at 101. Petitioner was discharged on October 11, 2016, with diagnoses of acute pancreatitis without necrosis or infection, likely secondary to binge drinking on the weekends; viral intestinal infection NOS (not otherwise specified); and DM for which Metformin and Glipizide were prescribed. Pet. Ex. 15 at 3-5, 14-15, 18, 98. He was to follow up with CentroMed as soon as possible since he did not have a primary care physician ("PCP"). *Id*. at 5.

Petitioner returned to Mission Trails on October 15, 2016, reporting chest pain. Pet. Ex. 15 at 198, 200. Troponin[10] levels were elevated. *Id*. at 447. The thought was recurrent pancreatitis, but petitioner reported that his pain was different and thought it was due to his new DM medication. *Id*. at 199. He was admitted. Cardiology evaluation revealed T wave changes on EKG. *Id*. at 201. Lipase was abnormal and amylase[11] was suggestive of pancreatic disease. *Id*. Echocardiogram was normal. There was no coronary artery disease seen during heart catheterization, only suspected microvascular disease. *Id*. at 295, 365. Some ischemia was noted on stress testing. *Id*. at 371, 472. Petitioner had severe stomach pain while hospitalized that resolved, but his discharge summary included "unusual" abdominal pain and bloating. He was to follow up with cardiology and for his pancreatitis. *Id*. at 181-82. His white blood cell count ("WBC")[12] was elevated at 14,000, and though his "readmission risk [was] high," he stated that he wanted to go home. *Id*. at 182. No antibiotic was prescribed but he needed to follow up with repeat labs in two days. *Id*. No CT changes of his pancreatitis were noted, and his lipase remained elevated. *Id*. The diagnosis on discharge was "Acute Pancreatitis." *Id*. at 183.

B. Medical History Post-Vaccination

Petitioner presented to a new primary to establish care on October 21, 2016. His recent diagnoses of diabetes, hypertension, pancreatitis, and his cardiac catheterization were noted. Pet. Ex. 17 at 3. He had no complaints. His examination was normal with normal strength and intact sensory. *Id*. at 3-4. He was taking DM medication and metoprolol.[13] *Id*. at 3-5. Blood work performed that that day was abnormal including but not limited to inflammatory/oxidation levels of myeloperoxidase[14] of 1083 (at risk > 995); CRP of 186.6 (at risk >3.0); fibrinogen[15] of 659 (>

[10] Troponin is a "complex of globular muscle proteins . . . that inhibits contraction" of the muscle. "Troponin," Dorland's Illustrated Medical Dictionary 1942 (33rd ed. 2020).

[11] Amylase is a type of enzyme. Together with lipase, elevated levels are suggestive of pancreatitis. "Amylase," Dorland's Illustrated Medical Dictionary 68 (33rd ed. 2020).

[12] WBC, or "leukocyte count," is the "determination of the number of leukocytes in the unit volume of blood." "Leukocyte Count," Dorland's Illustrated Medical Dictionary 420 (33rd ed. 2020).

[13] Metoprolol is "a cardioselective β1-adrenergic blocking agent" often used to treat chest pain or high blood pressure. "Metoprolol," Dorland's Illustrated Medical Dictionary 1138 (33rd ed. 2020).

[14] Myeloperoxidase is an "oxidoreductase . . . [that] play[s] a role in oxygen-dependent killing of microorganisms and tumor cells." "Peroxidase," Dorland's Illustrated Medical Dictionary 1399 (33rd ed. 2020).

[15] Fibrinogen is a coagulation factor and a plasma protein. "Fibrinogen," Dorland's Illustrated Medical Dictionary 694 (33rd ed. 2020).

at risk at 493); interleukin-6 ("IL-6")[16] at 39.6 (at risk at >13); interleukin-8 ("IL-8")[17] at 52.6 (at risk at >26); mildly elevated WBC at 11.9 (4.0-11.0); and elevated platelets[18] at 550 (130-400). *Id*. at 98-102. The subject flu vaccine was administered that day. *Id*. at 4.

Petitioner affirmed the onset of weakness in his hands at around 10:00 PM on October 23, 2016, which he believed was caused by the vaccination. By Tuesday, October 25, 2016, he could not walk and presented to the doctor's office that day. Pet. Ex. 4 at 5.[19] Petitioner was diagnosed with GBS on October 25, 2016, "while in the care of Dr. Neely. She was not the one to diagnose but attended him at same facility [sic] at University of San Antonio Health System." *Id*.

Petitioner presented to the ER at University Health System on October 26, 2016. Pet. Ex. 11 at 212. His recent hospitalization for pancreatitis on October 9, 2016, and prescriptions for diabetes, high cholesterol, hypertension, and gastroesophageal reflux disease ("GERD") medications were noted. *Id*. Petitioner reported that since taking the medications, he had experienced progressive weakness with difficulty performing fine motor tasks along with decreased energy. *Id*.

Multiple ER physician assessments followed. One ER physician documented numbness for five days, with bilateral lower extremity weakness and pain beginning two to three days prior. Pet. Ex. 11 at 210. Another ER physician documented a normal state of health until four days prior, numbness and weakness of the extremities beginning the day after, followed by fatigue and tingling of both lower extremities and weakness with an inability to walk. Petitioner's hands were involved, and he had difficulty writing over the previous one to two days. *Id*. at 205, 207. Recent gastrointestinal or upper respiratory infection was denied. *Id*. at 206. Examination revealed weakness and absent reflexes. *Id*. at 205. The assessment was "[l]ikely Guillain Barre vs. atypical ALS." *Id*. at 207. A third ER physician documented bilateral lower extremity weakness for two to three days that progressed upward to the upper extremities. *Id*. at 208. Neurology consultation was ordered. *Id*. at 209.

Petitioner was evaluated by the neurology resident that same day. Pet. Ex. 11 at 199. He reported a flu vaccine on Friday, October 21, 2016; weakness of his hands on Sunday, October 23, 2016; walking slowly on Monday, October 24, 2016; and inability to walk by Tuesday, October 25, 2016. *Id*. Examination revealed weakness and diminished/absent reflexes. *Id*. at 201. The impression was "Guillain-Barre syndrome following vaccination." *Id*. at 202. The attending neurologist documented "flue [sic] like illness" and acute pancreatitis on October 9, 2016, receipt of a flu vaccination a few days prior to this evaluation, weakness the following day, and inability to walk the day after that. *Id*. at 203. The assessment was that petitioner's symptoms were

[16] Interleukin is a "generic term for a group of multifunctional cytokines that are produced by a variety of lymphoid and nonlymphoid cells and have effects at least partly within the lymphopoietic system." "Interleukin," Dorland's Illustrated Medical Dictionary 937 (33rd ed. 2020). IL-6 is a lymphokine produced by certain cells, including T cells, that stimulates immunoglobulin production by B cells. "Interleukin-6," Dorland's Illustrated Medical Dictionary 937 (33rd ed. 2020).

[17] Interleukin-8 is a "chemokine" that may play a role in the discharge of certain white blood cells in inflammation. "Interleukin-8," Dorland's Illustrated Medical Dictionary 937 (33rd ed. 2020).

[18] A platelet is a "disk-shaped structure . . . found in the blood of all mammals and chiefly known for its role in blood coagulation." "Platelet," Dorland's Illustrated Medical Dictionary 1437 (33rd ed. 2020).

[19] No record of the October 25, 2016, visit has been filed.

"[u]nlikely to be related to the flue [sic] vaccine and [were] likely related to the flue [sic] like illness in eaely [sic] October which also caused acute pancreatitis." *Id*.

Blood work performed on October 27, 2016, included but was not limited to: strong positive GM1 antibody 180 and GD1b antibody 395 (≥101),[20] Pet. Ex. 11 at 69-70, and Pet. Ex. 3 at 691; A1C 9.0 [4.0-6.4], Pet. Ex. 11 at 71; elevated lipase on admission at 595 [350-373], *Id*. at 64; and ESR and CRP inflammatory markers elevated at 81 [0-20] and 62 [≥10 indicates inflammatory process] respectively, *Id*. at 70. These results "could all be explained by pancreatitis." *Id*. at 168. At discharge, ESR was still elevated at 97 [0-20] but CRP was normal. *Id*. at 78; Pet. Ex. 3 at 693. The clinical summary included GBS following vaccination. Pet. Ex. 11 at 2.

A lumbar puncture was consistent with AIDP, a variant of GBS. Pet. Ex. 11 at 168; Pet. Ex. 3 at 687-689. EMG/NCS studies revealed axonal and demyelinating neuropathy consistent with the axonal variant of AIDP. Pet. Ex. 11 at 155-156, 159. The EMG/NCS history included weakness of the arms and legs beginning "~8 days ago," October 23, 2016, with tingling that started in the feet now affecting the hands. *Id*. at 152. There was no noted recent diarrheal illness, but petitioner was noted to have received a flu shot two days before onset of symptoms. *Id*. The attending neurologist found that the sensory fibers were spared from peripheral neuropathy on the EMG/NCS but added that there could be abnormality in the future if AIDP was not of a pure motor form. *Id*. Upper quadrant ultrasound revealed gallstones without inflammation. *Id*. at 84, 169.

An inpatient rehabilitation consultation was performed on November 1, 2016, for treatment of upper and lower extremity weakness. Petitioner reported onset of symptoms about three days after receipt of a flu vaccination with no recent illness. Pet. Ex. 11 at 139. Gabapentin and nortriptyline were prescribed in turn for neuropathic pain. *Id*. at 135, 138. Petitioner received five days of IVIg with improvement. He was discharged to Reeves Inpatient Rehabilitation ("Reeves") from November 2, 2016, to November 18, 2016. *Id*. at 139-140.

Petitioner's history of flu vaccine two or three days prior to onset of symptoms was noted upon admission to Reeves. Pet. Ex. 3 at 72, 184-185. By November 9, 2016, he was ambulating with a walker. *Id*. at 154. On discharge, he had 4/5 strength throughout, was prescribed senna, Colace, Tylenol with codeine, and Lipitor, and was instructed to follow up with his PCP and neurology. Outpatient physical and occupational therapy was to continue. *Id*. at 240-241.

Petitioner presented to his PCP on November 23, 2016, with weakness, in a wheelchair, and with many medication changes. Pet. Ex. 17 at 6. The assessment was type 2 diabetes with hyperglycemia (primary); AIDP; hyponatremia;[21] chronic pancreatitis; and vitamin D deficiency. *Id*. His blood work continued to show elevated platelets, CRP, AST, and ALT. *Id*.

Petitioner presented for outpatient therapy on December 1, 2016, and received physical

[20] GM_1 autoantibodies "participate[] in the recognition of cells, compaction of myelin, signal transduction, and chemokine binding." They are "present in . . . [GBS], and other motor neuron diseases (MNDs), including [CIDP]." "GM_1 Autoantibodies," Julius M. Cruse & Robert E. Lewis, Illustrated Dictionary of Immunology 297 (3rd ed. 2009).
[21] Hyponatremia is a "deficiency of sodium in the blood." "Hyponatremia," Dorland's Illustrated Medical Dictionary 892 (33rd ed. 2020).

and occupational therapy throughout that month. Pet. Ex. 3 at 261, 267.

On December 21, 2016, petitioner presented for his first visit to UT Health San Antonio/MARC. Pet. Ex. 16 at 4. He consistently reported his history following his receipt of the flu vaccine. *Id*. He now had tingling and pain in his hands with cramping in his lower extremities which interfered with sleep. He was unable to walk. He had diminished strength with EMG/NCS testing suggestive of a motor variant of AIDP due to intact sensation. *Id*. at 5-6. His history included flu vaccination, DM, and pancreatitis at the beginning of October 2016. Lyrica and Tylenol #3 were prescribed, along with a follow-up with his PCP, physiatry, continued PT/OT and a return in six months. *Id*.

That same day, December 21, 2016, petitioner presented to the ER at University Hospital with right upper quadrant pain, nausea, and vomiting that began that afternoon. Pet. Ex. 3 at 277. Ultrasound revealed gallbladder sludge and fatty liver with fibrosis. *Id*. at 378. Liver enzymes were elevated and attributed to atorvastatin which had been prescribed in November. Lipase was normal. *Id*. at 285-286. He was prescribed Cipro for abnormal urinalysis. He was to follow up with his PCP. *Id*. at 287.

Petitioner presented to his PCP on December 23, 2016, for right upper quadrant pain and elevated LFTs. CT and ultrasound showed no acute findings. His statin was stopped. Pet. Ex. 17 at 8. He had pain and was in a wheelchair. *Id*. at 9. He had normal motor strength in his upper and lower extremities with intact sensory and cerebellar function. *Id*. at 10. He was referred to a gastroenterologist. *Id*. at 10.

A hepatobiliary scan on December 28, 2016, revealed obstruction of a cystic duct and petitioner was referred to general surgery. Pet. Ex. 2 at 92; Pet. Ex. 17 at 14-15. On December 30, 2016, he was admitted to Baptist Health for surgery of an obstructed cystic duct of the gallbladder. Pet. Ex. 14 at 5, 12, 132. Chronic inflammation was found, and only partial gallbladder removal could be performed with drain placement. *Id*. at 40-41. Flu vaccine was listed under allergies, with GBS as the reaction. *Id*. at 235. Petitioner was discharged but returned later that day due to concern about drainage from the drain. He was noted to be in a wheelchair due to GBS. *Id*. at 231, 234.

Petitioner's follow-up examination with his PCP on January 12, 2017, was normal. Pet. Ex. 17 at 17. The assessment included AIDP, type 2 diabetes without complication, long term current use of insulin, and acute and chronic cholecystitis. *Id*. at 18.

Outpatient therapies resumed on January 23, 2017, with improvement noted since his initial visit. However, petitioner still had significant deficits with fine motor control issues in his hands. Pet. Ex. 3 at 298, 303.

On February 9, 2017, petitioner presented to his PCP for follow-up and FMLA paperwork. Pet. Ex. 17 at 22-23. He attended the appointment in a wheelchair but was advised to walk for at least an hour with 15-minute intervals of power walking and to engage in other exercise routines. Neurological examination was normal. *Id*. Importance of glucose level monitoring was discussed. *Id*. PT was to continue. *Id*. In an addendum, the PCP documented his discussion with petitioner's neurologist who advised that petitioner "developed AIDP soon after getting the flu shot, EMG, did

have axonal loss which take[s] longer to recover from. . . ." *Id*. at 23.

A rehabilitation record from March 27, 2017, noted slow but ongoing progress with mobility resulting from PT/OT. Petitioner was ambulating while holding on to a manual wheelchair at home. He was able to walk 81 feet with a rolling walker while at PT with assistance. Pet. Ex. 3 at 372. He had not followed up with neurology or been compliant with AFOs due to poor fitting. The PT tried to adjust the AFOs with tape to make them more comfortable. *Id*. at 372-373.

Insurance discontinued petitioner's OT with his last visit on April 25, 2017. Pet. Ex. 3 at 462, 475. He had improved but he still had weakness. *Id*. at 462. His OT prescription was renewed but denied by insurance. *Id*. at 475-476. Petitioner continued with PT with slow progress. He was using a cane by July 2017 with continued weakness and imbalance by September 2017. Pet. Ex. 2 at 502; Pet. Ex. 3 at 554. He was discharged from PT on December 27, 2017, after 72 sessions. At that time, he was ambulating in the clinic with no assistive device for more than 300 feet and he used AFOs with improved balance. Pet. Ex. 3 at 637-638.

A rehabilitation examination on January 11, 2018, noted petitioner as being "better" with improved function. He was walking up to 300 feet with a cane but was unable to return to work because his employer had "no less physical options." Pet. Ex. 3 at 640. He still had fatigue and balance issues, but no pain. Lower extremities were near normal except for ankle flexion and great toe strength. *Id*. Petitioner received referrals to vocational rehabilitation, aqua therapy, OT for driving evaluation, mental health counseling for suspicion of depression, and a PCP. Sleep issues and tremor were thought to be fatigue related. *Id*. at 644-45.

At a new OT evaluation on January 30, 2018, petitioner reported shooting pain in his forearms and hands and decreased grip and pinching strength. Pet. Ex. 3 at 647. He received four OT sessions and was discharged with home exercises. *Id*. at 661.

Improvement was noted at a May 3, 2018, rehabilitation examination, but petitioner still had tremors causing incoordination, left hand cramps, and numbness and tingling in the ulnar nerve distribution. Pet. Ex. 3 at 644. He still could not lift 40 pounds, which was a requirement for him to return to his previous job. He brought FMLA paperwork for a six-month extension. He had not secured a PCP, attended a driving evaluation, or seen a mental health specialist. Examination was unchanged. New referrals were placed. He was started on Celexa for depression and melatonin for sleep. *Id*. at 669.

Petitioner was officially terminated from his job on June 1, 2018, and he lost insurance coverage. Pet. Ex. 4 at 2. His last OT visit was on June 4, 2018. Pet. Ex. 18 at 39. Insurance authorized seven PT visits. Petitioner attended only five, last presenting on June 13, 2018, with complaints of knees buckling and balance issues. He was discharged on October 15, 2018. *Id*. at 28.

A neurorehabilitation examination for functional recovery was conducted on October 25, 2018. The record included discharge from his job, receipt of social security, and attempt at job placement, but petitioner reported that the salaries offered were not "adequate." Pet. Ex. 18 at 20.

Petitioner had continued weakness and fatigability of both upper and lower extremities. *Id*. His tremor was stable and unchanged. He used bilateral dorsiflexion AFOs and a cane for ambulation. *Id*. He had occasional leg cramping and neuropathic pain in his hands and he took Tylenol. Other medications had proved ineffective and had undesirable side effects. His mood improved but he had continued depression due to loss of function. He did not take Celexa regularly. *Id*. He had fallen several times over the past six months. *Id*. at 22. The assessment was AIDP with residual weakness and impaired gait and balance two years post-diagnosis with the potential for continued improvement. *Id*. at 25. Petitioner did not have any flares or waxing and waning symptoms; he just had not returned to his prior state of health. Functional outcome was difficult to predict due to continued deficits. Outpatient therapies were recommended for maximum functional independence. *Id*. at 25.

At an April 25, 2019, rehabilitation medicine follow-up, petitioner was doing well. He was more active in the community and with family, but still unemployed with no goals of returning to work. His mood and strength improved with more outside activities. He was receiving Medicare and wanted to establish with a PCP. Pet. Ex. 18 at 9. He still had 4/5 strength bilaterally on ankle flexion and extension and hand tremors. *Id*. He received information on vocational rehabilitation. *Id*. He declined OT driving evaluation, however. *Id*. He also declined medication and counseling for his mood disorder. *Id*. He was referred for neurology evaluation. *Id*. at 14. At that time, he had no pain and walked with a cane. *Id*. at 15.

C. Petitioner's Affidavits

Petitioner submitted four affidavits. Pet. Exs. 4, 5, 21, and 35.

In his first affidavit, petitioner affirmed being previously healthy and the sole financial provider for his family, which included his wife, son, and three grandchildren. Pet. Ex. 4 at 1. He detailed his daily activities, which included working the night shift as a restaurant manager supervising eight to ten people, doing inventory, hiring, customer service, and supporting his team. *Id*. He would arrive home about 8:30 AM, attend to home tasks, sleep from 10:00 AM to 3:00 PM, pick up his grandchildren from school and his wife from work, take a nap, then go back to work. *Id*. He was 49 years old at the time of the flu vaccination.

Following the vaccination, petitioner experienced paralysis from his waist down as well as in his hands. Pet. Ex. 4 at 1-2. He required constant care and could do little for himself which greatly affected his family, marriage, and ability to work. *Id*. at 2. His illness placed a financial burden on his family. He lost his job and insurance, and suffered from depression, insomnia, mood swings, and outbursts. *Id*. Petitioner detailed all the activities in which he engaged prior to the vaccination and his work with an income of $50,000 per year. *Id*. at 3-4. Petitioner affirmed that he lost his job on June 1, 2018, due to his vaccine-related injuries. However, his company paid most of his medical bills. He detailed some out-of-pocket medical costs and affirmed that he was in the process of obtaining Medicaid. *Id*. at 4. The "Sequence of Events" he provided following receipt of the flu vaccination was consistent with what he reported to physicians upon presentation. *Id*. at 5-6.

Petitioner's second affidavit was an affirmation that no civil action was pending, and that

petitioner had not received compensation for his vaccine related injuries. Pet. Ex. 5.

In a third affidavit filed on September 27, 2020, petitioner provided a timeline of events starting with his receipt of the flu vaccine on Friday, October 21, 2016, between 4:00 and 5:00 PM and ending with his presentation to the hospital on October 25, 2016. *See generally* Pet. Ex. 21. This account included noticing weakness in his hands at around 10:00 PM on October 23, 2016, and having trouble unbuttoning his pants. *Id*. at 1. By the following morning, petitioner noted an inability to lift his feet and loss of strength in his hands. By Tuesday, he could not walk or stand and had no hand strength at all. He was paralyzed from the waist down. *Id*. at 2. He was carried to a vehicle and taken to the hospital. *Id*. at 2.

In a fourth affidavit, petitioner detailed his 15-year work history at Whataburger as of June 2016. Pet. Ex. 35 at 1. He worked ten hours a day, 50 or more hours a week, with 95% of his time spent on his feet. *Id*. He had no prior medical or health complications that prohibited him from performing his job. *Id*. Further, upon completing 15 years with the company in June 2016, he received recognition. Petitioner would have been eligible for retirement at 20 years, though he intended to keep working. *Id*. He dreamed of opening his own restaurant. *Id*.

Petitioner affirmed being diagnosed with GBS on October 25, 2016, following the flu vaccine. Pet. Ex. 35 at 1-2. He was "hospitalized for 25 days with paralysis from [his] waist down and in [his] hands." *Id*. at 2. He was "unable to return to work" and was "wheelchair-bound for 18 months. In 2017 [he] was unable to return to work due to [his] paralysis." In June 2017, he reached 16 years with his employer, and "was able to stay employed due to . . . accumulated sick time . . . and vacation time." *Id*. at 2. He was then put on FMLA and received long-term, but not short-term, disability. *Id*.

According to petitioner, by 2018, he was still in a wheelchair. Pet. Ex. 35 at 2. Petitioner underwent extensive physical therapy and progressed to using a walker. *Id*. He was terminated from his job on June 1, 2018, before completing 17 years. *Id*. His health issues increased by the end of that year. *Id*. He had a leg brace to maintain his balance and used a cane for support. *Id*. He started seeing a psychiatrist to help with insomnia, anxiety, and depression in 2019.[22] *Id*. As of 2020 he continued his therapy and visits to his doctor and psychiatrist. *Id*.

In 2021, he still had tremors, used an AFO on his foot and leg, was unable to walk without falling, and had chronic fatigue and depression. Pet. Ex. 35 at 2. He could not stand for more than 20 minutes before getting tired and needing to sit down. He often took naps. *Id*.

Petitioner affirmed that he has received Medicare, which covers 80% of his therapies, as of April 2019. Pet. Ex. 35 at 3. He received long term disability of $836 per month, which at the time of his affidavit had been extended for another year. *Id*. Lastly, he received social security benefits and long-term disability payments which made up the difference of his pay. *Id*. at 3.

[22] No psychiatry records were filed in this matter.

III. Expert Opinions

What follows is a broad overview of each expert's opinions and conclusions. The details of each expert's reports are discussed more fully in Section V below.

A. Petitioner's Experts

1. Dr. Marcel Kinsbourne[23]

Dr. Kinsbourne wrote five reports. Pet. Exs. 45, 47, 49, 53, and 54.[24]

Dr. Kinsbourne began by summarizing petitioner's medical history. He claimed that petitioner's diagnosis of GBS is undisputed, as is "the proposition that influenza vaccination can trigger GBS."[25] Pet. Ex. 45 at 4. For this reason, Dr. Kinsbourne did not address Prong I in his first report. *Id*. He generally deferred to petitioner's immunology expert, Dr. Akbari, for his analysis of how the flu vaccine can cause GBS. Dr. Kinsbourne agreed with Dr. Akbari that "molecular mimicry and induction of inflammasome are credible medical theories linking the flu vaccination to injuries that [petitioner] experienced." Pet. Ex. 49 at 4.

Dr. Kinsbourne acknowledged that the Vaccine Table provides for symptom onset of GBS following flu vaccination within three to 42 days but claimed that the three- to 42-day onset was an "interval [] imposed by a committee, for administrative rather than medical reasons." Pet. Ex. 45 at 4. Epidemiological studies impose a risk interval of zero to 42 days with no exclusions of onset within the first two days. *Id*.; Pet. Ex. 45 Ref No. 5;[26] Pet. Ex. 45 Ref No. 4.[27] Most importantly, *Park, et al.* found that "[m]ore than half the cases (54%) occurred within 2 days after the vaccination." Pet. Ex. 45 at 5 (citing Pet. Ex. 45 Ref No. 4[28] at Fig. 1). Thus, petitioner's onset of 54-58 hours post-vaccination satisfies Prong III.

Dr. Kinsbourne also addressed the issue of potential alternative causes. He first noted that,

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[23] Dr. Kinsbourne graduated from Oxford University in England with a B.M., B.Ch., the equivalent of an American M.D. He became licensed in the United States in 1967. Dr. Kinsbourne has served as an associate professor in pediatrics and neurology and a senior research associate at Duke University Medical Center before holding a series of academic positions, including professorships in pediatrics, neurology, and psychology. His clinical experience includes serving as a senior staff physician in Ontario from 1974-1980 and a clinical associate in neurology at Massachusetts General Hospital from 1981-1991. *See generally* Pet. Ex. 44.

[24] The National Vaccine Injury Compensation Program's Guidelines for Practice Section II, Chapter 3(B)(12) states that "[i]f medical literature accompanies the expert report, each article should be filed as a separate exhibit," rather than as attachments to the expert report. Petitioner's medical literature should have been filed with consecutive exhibit numbers.

[25] Not so. Respondent specifically notes that he "has not conceded *Althen* prong one is all flu/GBS cases because of respondent's decision to add flu/GBS to the Table or because respondent has decided not to defend certain flu/GBS cases." Resp. Br., ECF No. 130 at 29.

[26] Lawrence B. Schonberger, et al., *Guillain-Barre Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977*, 110 Am. J. of Epidemiology 105 (1979), filed as "Pet. Ex. 45 Ref No. 5" and "Resp. Ex. A-17."

[27] Yong-Shik Park, et al., *Clinical Features of Post-Vaccination Guillain-Barré Syndrome (GBS) in Korea*, 32 J. of Korean Med. Sci. 1154 (2017), filed as "Pet. Ex. 45 Ref No. 4," "Pet. Ex. 43 Ref No. 23," and "Pet. Ex. 48 Ref No. 3."

[28] *Id*.

while petitioner's records diagnose him with a flu-like illness on October 5, 2016, Pet. Ex. 7 at 2, petitioner tested negative for influenza A and B. Pet. Ex. 45 at 5. Petitioner also did not present with respiratory symptoms and did not have a cough, running nose, or shortness of breath. *Id*. Thus, "[t]here is no evidence supporting the presence of respiratory or gastrointestinal virus infection." *Id*. at 8.

Petitioner then presented on October 9, 2016, with what Dr. Kinsbourne argued was "plainly [] a gastrointestinal issue, with nausea, vomiting, cramps and diarrhea, as well as continued fever" which were symptoms of mild acute pancreatitis. *Id*. Dr. Kinsbourne distinguished two studies which observed cases of pancreatitis followed by GBS or polyneuritis, both of which presented as severe cases of pancreatitis with accompanying complications. *Id*.; Pet. Ex. 45 Ref No. 1;[29] Pet. Ex. 45 Ref No. 2.[30] He thus found that "[t]he antecedent acute pancreatitis has not been shown to trigger GBS." Pet. Ex. 45 at 8.

Dr. Kinsbourne concluded that petitioner's onset is "fully consistent with empirical evidence in the medical literature on GBS" and that there was no plausible alternative cause for petitioner's GBS other than the flu vaccine. Pet. Ex. 45 at 8.

2. Dr. Omid Akbari[31]

Dr. Akbari wrote four reports. Pet. Ex. 43; Pet. Ex. 46; Pet. Ex. 48; Pet. Ex. 52.

Dr. Akbari is a Ph.D. in Cellular and Molecular Immunology. Pet. Ex. 42.

After providing a brief summary of petitioner's medical history, Dr. Akbari detailed the "commonly implicated mechanism" of molecular mimicry to explain how the flu vaccine can cause GBS. Pet. Ex. 43 at 6. Molecular mimicry occurs when there is a "sharing of antigens between a host and a microorganism." *Id*. More specifically, this theory is implicated when "the host and the microbe share an immunologic epitope by either sequence or conformational homology." *Id*.; Pet. Ex. 43 Ref No. 3.[32] Dr. Akbari posited that significant protein peptide sequence similarity between the host and the microorganism is no longer recognized as necessary to induce molecular mimicry. Pet. Ex. 43 at 7. Rather, "conservation of only a few crucial residues can allow for cross-reactivity of immune cells." *Id*.

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[29] M.L.P. Gross, et al., *Peripheral Neuropathy Complicating Pancreatitis and Major Pancreatic Surgery*, 51 J. of Neurology, Neurosurgery, and Psychiatry 1341 (1988), filed as "Pet. Ex. 45 Ref No. 1."

[30] Aswathi Harikumar, et al., *An Interesting Case of Acute Polyneuropathy in the Context of Acute Pancreatitis*, 7 U. J. of Med. And Med. Specialties 1 (2021), filed as "Pet. Ex. 45 Ref No. 2."

[31] Dr. Omid Akbari received his undergraduate degree and his M.S. in Medical and General Microbiology at University College London. He received his Ph.D. in Cellular and Molecular Immunology from the National Institute for Medical Research in London and was a postdoctoral fellow at Stanford University. He is currently a tenured Professor of Immunology in the Department of Molecular Microbiology and Immunology and the Department of Medicine at the Keck School of Medicine at the University of Southern California, Los Angeles, an Adjunct Professor of Immunology and Medicine at the Department of Immunology at Chiba University, and an Adjunct Professor of Pediatrics-Medicine at the David Geffen School of Medicine at the University of California-Los Angeles-Harbor. *See generally* Pet. Ex. 42.

[32] Robert S. Fujinami, et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 Clinical Microbiology Rev. 80 (2006), filed as "Pet. Ex. 43 Ref No. 3."

Vaccination causes an inflammatory reaction that is mediated by the cells of the innate immune system. Pet. Ex. 43 at 7. The immune system's ability to recognize and eliminate foreign antigens, induce immunologic memory, and develop tolerance to self-antigens results in effective homeostasis. When homeostasis is disrupted and the immune system responds in favor of activation, as in response to vaccination, the host is susceptible to autoimmunity. *Id*. at 14. In GBS, research indicates that this balance is being disrupted leaving the host susceptible to an adverse autoimmune reaction upon stimulation of the immune system from infection or vaccination, mediated by activation of inflammasome.

Dr. Akbari identified six amino acids in the flu vaccine that have "high homology" to the "myelin basic protein sequence." Pet. Ex. 43 at 14. He also cited to studies which he argues "suggest that the influenza vaccine that [petitioner] received contains a protein that cross-reacts with myelin based antigens in humans who had demyelinating disease." *Id*. at 13; Pet. Ex. 43 Ref No. 34;[33] Pet. Ex. 43 Ref No. 35.[34]

Dr. Akbari also opined that petitioner's "history of inflammation, diarrhea and subsequent development of pancreatitis" sensitized petitioner prior to his flu vaccination which made him more likely to develop GBS as a result of the flu vaccine. Pet. Ex. 43 at 19.

Lastly, Dr. Akbari argued that highly activated T cells which secrete proinflammatory cytokines can, within as soon as 36 hours following the inciting event, cause injury and demyelination. Pet. Ex. 43 at 19; Pet. Ex. 43 Ref No. 56.[35] These T cells, together with innate like lymphocytes ("ILLs") which can produce cytokines and cause adverse effects within a few hours of stimulation, can cause fast-acting symptoms like those experienced by petitioner. Pet. Ex. 43 at 9-10, 19.

Thus, Dr. Akbari argued that petitioner met each of the three Althen prongs.

B. Respondent's Experts

1. Dr. Dara Jamieson[36]

Dr. Jamieson wrote three reports. Resp. Exs. A, C, and F.

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[33] Silva Markovic-Plese, et al., *High Level of Cross-Reactivity in Influenza Virus Hemagglutinin-Specific CD4+ T-Cell Response: Implications for the Initiation of Autoimmune Response in Multiple Sclerosis*, 169 J. of Neuroimmunology 31 (2005), filed as "Pet. Ex. 43 Ref No. 34."

[34] Kai W. Wucherpfennig and Jack L. Strominger, *Molecular Mimicry in T Cell-Mediated Autoimmunity: Viral Peptides Activate Human T Cell Clones Specific for Myelin Basic Protein*, 80 Cell 695 (1995), filed as "Pet. Ex. 43 Ref No. 35."

[35] Richard M. Ransohoff, et al., *Three or More Routes for Leukocyte Migration into the Central Nervous System*, 3 Nature Rev. Immunology 569 (2003), filed as "Pet. Ex. 43 Ref No. 56."

[36] Dr. Jamieson received her A.B. from George Washington University with honors in zoology and physics in 1976. In 1982, she received her M.D. from the University of Pennsylvania School of Medicine and underwent postgraduate study at the University of Pennsylvania's Department of Biochemistry and Biophysics. Dr. Jamieson worked as a neurologist at various hospitals between 1989 and 2018, and as the Director of the Section of Neurovascular Neurology and Cerebrovascular Neurosurgery at Wills Eye Hospital from 1997 to 1999, and the Co-Director of the Neurovascular Laboratory at New York Presbyterian Hospital from 2009 to 2018. Dr. Jamieson is currently licensed to practice in New York state. *See generally* Resp. Ex. A-1.

Dr. Jamieson summarized petitioner's medical history and detailed his presentation on October 5, 2016, as body aches, fever, chills, eye burning, and decreased appetite. Resp. Ex. A-2 at 2; Pet. Ex. 7 at 1-5. She added that a form filled out for that visit included fever, chills, pain with deep breath, weakness in petitioner's arms and legs, eye pain and pressure, watery or itchy eyes, throat pain, hoarseness, chest pain, muscle aches, change in appetite, abdominal pain, and excessive thirst. Resp. Ex. A-2 at 2; Pet. Ex. 7 at 1-2. Petitioner was febrile with a 102° fever, was tachycardic with decreased blood pressure, and was congested and notably sweating. A handwritten note included "flu-like symptoms" with a billing code for influenza. The evaluation was done by a nurse practitioner who prescribed Bentyl. An influenza A/B test was negative.

Continuing, petitioner was admitted to the hospital on October 9, 2016, for five to six days for abdominal pain and cramps, nausea, and diarrhea. He reported drinking six-packs of beer over the weekend. Examination revealed right upper quadrant tenderness. Lipase was elevated. Ultrasound of the gallbladder and CT scan of the abdomen and pelvis were unremarkable. Resp. Ex. A-2 at 2; Pet. Ex. 20 at 124-125. The assessment was acute pancreatitis with diarrhea, treated with bowel rest, morphine, and fluids. Resp. Ex. A-2 at 2; Pet. Ex. 20 at 13-14. The discharge summary included acute pancreatitis of uncertain etiology, possibly related to viral infection, resolved gastroenteritis, also suspected to be viral in etiology, newly discovered type 2 diabetes, and a history of binge drinking. Resp. Ex. A-2 at 2; Pet. Ex. 20 at 2-3.

Dr. Jamieson then described GBS as a group of autoimmune disorders with acute inflammatory demyelinating polyneuropathy ("AIDP") being the most common in the United States. Resp. Ex. A-2 at 7. GBS and AIDP are thus often referred to interchangeably. *Id*. GBS is often triggered by infection, presents with rapid and progressive onset, and is monophasic. *Id*. About two-thirds of patients report respiratory or gastrointestinal symptoms within four weeks of onset of weakness, and typically within 10 to 14 days. *Id*.; Resp. Ex. C at 4; Resp. Ex. F at 2; Resp. Ex. A-4;[37] Resp. Ex. A-5;[38] Resp. Ex. A-6.[39] One-third of patients have no identified antecedent event. Resp. Ex. A-2 at 7; Resp. Ex. C at 4.

Dr. Jamieson asserted that vaccination against respiratory infections has not been consistently proven to be associated with GBS, unlike respiratory infection itself. Resp. Ex. A-2 at 9. While the risk of GBS increased during the influenza pandemic of 1976, the increased risk was not associated with vaccination. *Id*.; Resp. Ex. A-11.[40] See Resp. Ex. A-12[41] and Resp. Ex. A-19[42] for studies finding slight or no increases in GBS risk upon vaccination. But see Resp. Ex.

[37] Hugh J. Willison, et al., *Guillain-Barré Syndrome*, 388 Lancet 717 (2016), filed as "Resp. Ex. A-4."
[38] Peter D. Donofrio, *Guillain-Barré Syndrome*, 23 Continuum 1295 (2017), filed as "Resp. Ex. A-5."
[39] Helmar C. Lehmann, et al., *Guillain-Barré Syndrome After Exposure to Influenza Virus*, 10 Lancet Infectious Diseases 643 (2010), filed as "Resp. Ex. A-6."
[40] Alexander D. Langmuir, et al., *An Epidemiologic and Clinical Evaluation of Guillain-Barré Syndrome Reported in Association with the Administration of Swine Influenza Vaccines*, 6 Am. J. of Epidemiology 841 (1984), filed as "Resp. Ex. A-11."
[41] J.O.T. Sipilä & M. Soilu-Hänninen, *The Incidence and Triggers of Adult-Onset Guillain-Barré Syndrome in Southwestern Finland 2004-2013*, 22 Eur. J. of Neurology 292 (2015), filed as "Resp. Ex. A-12."
[42] Daniel A. Salmon, et al., *Association Between Guillain-Barré Syndrome and Influenza A (H1N1) 2009 Monovalent Inactivated Vaccines in the USA: A Meta-Analysis*, 381 Lancet 1461 (2013), filed as "Resp. Ex. A-19," "Pet. Ex. 48 Ref No. 1," and "Pet. Ex. 43 Ref No. 22."

A-20[43] for a study finding a "small but statistically significant association," Resp. Ex. A-2 at 9, between influenza vaccination and GBS. Studies have found that the risk of developing GBS during the 42-day period following an acute gastrointestinal or acute respiratory infection was increased compared to that of an infection-free period. The risk of developing GBS was found to be comparable for both types of infection, like the infections that petitioner suffered prior to developing GBS. Resp. Ex. A-2 at 10; Resp. Ex. A-16.[44]

Dr. Jamieson added that the risk interval of GBS is between three to 42 days as provided by the Program. Resp. Ex. C at 1-2. She disagreed that 53 to 54 hours, or 2.2 days after vaccination, provided a causative correlation between vaccination and symptom onset. Resp. Ex. A-2 at 10-11; Resp. Ex. A-17.[45] "The consensus has been, based on logical and immunological arguments, that the period of presumed risk starts on day 3, not on day 2 as in the case of [petitioner]." *Id*. at 3.

Dr. Jamieson submitted that petitioner had "multiple systemic viral and bacterial infections, including a flu-like illness, gastroenteritis, pancreatitis, bacteriuria, and cholecystitis" in the weeks prior to vaccination with documented fever, tachycardia, low normal pulse oxygenation, sweating, and congestion. Resp. Ex. A-2 at 12. The records noted that this infection was more likely the triggering event for petitioner's GBS than the flu vaccination. Resp. Ex. A-2 at 12; Resp. Ex. C at 3-4; Pet. Ex. 7 at 1-5; Pet. Ex. 20 at 197-199; Pet. Ex. 20 at 2-3. Dr. Jamieson added that two months later, petitioner was treated with antibiotics for acute and chronic acalculous hemorrhagic cholecystitis. Resp. Ex. A-2 at 12; Pet. Ex. 4 at 178. She argued that petitioner had AMAN, or acute motor axonal neuropathy, a subset of GBS particularly associated with gastrointestinal infections as the triggers: "His multiple preceding infections, including gastrointestinal infections, are a more usual and accepted trigger for AMAN, than is vaccination." Resp. Ex. A-2 at 12.

Dr. Jamieson disagreed that petitioner's symptoms were attributed to an abundance of inflammation consistent with the inflammatory nature of acute pancreatitis. Resp. Ex. C at 3-4; Resp. Ex. F at 4. She asserted that petitioner "had multiple respiratory and systemic symptoms indicative of a flu-like infection and he was congested on examination, not indicative of pancreatitis." Resp. Ex. C at 3.

Dr. Jamieson submitted that petitioner suffered from a flu-like illness, pancreatitis, gastroenteritis, cholecystitis, and a non-ST-elevation myocardial infarction ("NSTEMI") in the three weeks prior to developing motor/axonal GBS which were the triggers for developing AMAN. The flu vaccination 53 to 54 hours, or two days, prior to onset was not the "plausible trigger for AMAN" or a cause of his neurological symptoms. Resp. Ex. A-2 at 13-14; Resp. Ex. F at 4, 14. Petitioner continues to have neurological deficits from AMAN with superimposed diabetic peripheral neuropathy diagnosed in 2021 and mainly sensory symptoms. His current disability is a combination of diabetic neuropathy, which will worsen with age, and the mild residual effects of AIDP that will improve with time. Resp. Ex. F at 2; Resp. Ex. A-2 at 13.

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[43] L.H. Martín Arias, et al., *Guillain-Barré Syndrome and Influenza Vaccines: A Meta-Analysis*, Vaccine (2015), filed as "Resp. Ex. A-20."

[44] Clémence Grave, et al., *Seasonal Influenza Vaccine and Guillain-Barré Syndrome: A Self-Controlled Case Series Study*, 94 Neurology 2168 (2020), filed as "Resp. Ex. A-16."

[45] Schonberger, et al., *supra*, note 26.

"To a reasonable degree of medical probability, if [petitioner] had not received the flu vaccine, he still would have developed GBS triggered by the preceding infections." Resp. Ex. A-2 at 13.

2. Dr. Stephen Tompkins[46]

Dr. Tompkins wrote three reports. Resp. Exs. B-1, D-2, and G.

Dr. Tompkins, like Dr. Akbari, is a Ph.D. with a specialty in immunology and molecular pathogenesis. Resp. Ex. B-1 at 1.

Dr. Tompkins did not dispute petitioner's diagnosis of GBS because he is not a clinician. Resp. Ex. B-1 at 2-3. Dr. Tompkins disagreed that any evidence exists to support molecular mimicry as the mechanism by which inactivated influenza vaccines can trigger GBS. *Id*. at 5-8. He further disagreed that an inactivated flu vaccine could activate inflammasomes which would in turn activate Th17 T cells triggering GBS. *Id*. at 8-10; Resp. Ex. D-2 at 7-13. Finally, Dr. Tompkins disagreed that an onset of GBS could occur in fewer than three days. Rather, there were alternative causes for petitioner's GBS. Resp. Ex. G at 2-3.

Dr. Tompkins asserted that most relevant to this case is the more recent surveillance of GBS conducted by *Arya, et. als*. which assessed risk windows of one to 42 days and eight to 21 days for all types of influenza vaccine during the 2015-16 and 2016-17 flu seasons and found no excess risk for more than 13 million Medicare recipients of flu vaccines per risk period. Resp. Ex. B-1 at 4; Resp. Ex. B-6.[47] The components of the flu vaccines during these seasons were similar to those Dr. Akbari suggested were responsible for triggering GBS in this case. Resp. Ex. B-1 at 4; Pet. Ex. 43 at 6, 13, 14, 18, 19. The proposed mechanisms by petitioner's experts, including molecular mimicry and inflammasome activation, that would accelerate vaccine-specific immune responses simply do not substantiate a causal link. Resp. Ex. G at 11.

In contrast, Dr. Tompkins argued that upper respiratory and gastrointestinal infections are strongly associated with GBS, with a peak interval of two to three weeks after antecedent event. Petitioner's medical records show that he suffered from both in the two to three weeks prior to the onset of his GBS. Resp. Ex. B-1 at 10-11; Resp. Ex. D-2 at 13; Resp. Ex. G at 8, 12; Pet. Ex. 15 at 14; Pet. Ex. 45 Ref No. 5.[48] Dr. Tompkins referenced the National Institute of Neurological Disorders and Strokes to show that most cases of GBS occur a few days or weeks following respiratory or gastrointestinal viral infection. Resp. Ex. B-1 at 10; Resp. Ex. B-18.[49] Therefore, petitioner's interval onset of GBS "falls squarely within the accepted interval for onset of GBS

[46] Dr. Tompkins received his B.S. in Microbiology from the University of Illinois in 1990 and his Ph.D. in Immunology from Emory University in 1997. He performed post-doc research in immunology and virology at Northwestern University Medical School and at CBER/FDA. Dr. Tompkins currently serves as the Director of the Center for Influenza Disease and Emergence Research and as the Assistant Department Head of the Department of Infectious Diseases in the College of Veterinary Medicine at the University of Georgia. *See generally* Resp. Ex. B-2.

[47] Deepa P. Arya, et al., *Surveillance for Guillain-Barré Syndrome After 2015-2016 and 2016-2017 Influenza Vaccination of Medicare Beneficiaries*, 37 Vaccine 6543 (2019), filed as "Resp. Ex. B-6."

[48] Schonberger, et al., *supra*, note 26.

[49] Guillain-Barré Syndrome Fact Sheet, Nat'l Inst. of Health (Last Reviewed Apr. 25, 2022), NIH Publication No. 18-NS-2902.

following an antecedent event," and "also falls within a reasonable timeline for an immune response to the infection to elicit clinical disease." Resp. Ex. B-1 at 10. The anti-ganglioside GD1b and GM1 antibodies detected on October 27, 2016, would have had the time necessary to be elicited by an infection beginning about 21 days before. *Id*.; Pet. Ex. 18 at 1520.

Rather than responding to the Court's question of whether "it [was] more probable that the combination of all of petitioner's health issues along with his receipt of the flu vaccine during that three-and-a-half-week period resulted in his development of GBS," Dr. Tompkins raised two questions of his own: 1) Could any of the events noted in petitioner's medical history result in the onset of GBS between two days and two to three weeks later without requiring an additional exacerbating event?, and 2) Could multiple infections, uncontrolled diabetes, and/or catheterization have been in the process of eliciting GBS such that receipt of the influenza vaccine rapidly, in less than three days, triggered petitioner's GBS? Resp. Ex. G at 2; ECF No. 110 at 2.

Dr. Tompkins, responding to his own first question, asserted that infections are the most common triggers of GBS in 75% of cases. Resp. Ex. G at 2. He added that influenza A/B testing is only 73% to 78% accurate and petitioner's test may have showed a false negative, failing to rule out his suffering an influenza illness two to three weeks before vaccination. Resp. Ex. B-1 at 10; Resp. Ex. B-20.[50]

Dr. Tompkins asserted that his own second question was difficult to answer because, while the mechanisms for how viral infections can cause GBS are generally accepted, how flu vaccination could trigger GBS is significantly less so. Resp. Ex. G at 3. He relied on *Baxter*, which reviewed the *recurrence* of GBS in 550 cases following vaccination and observed that "'[w]ith over 30 million person-years of follow-up over the study period and nearly 1,000 vaccines administered after a diagnosis of GBS (including over 400 influenza vaccinations), we did not observe a single recurrent case of GBS that could be considered associated with vaccination.'" *Id*. at 3; Resp. Ex. G-4.[51] He conceded that there were no other studies of recurrence for GBS after flu vaccination but cited to studies involving potential exacerbation of multiple sclerosis and myasthenia gravis. Resp. Ex. G at 4; Resp. Ex. G-6;[52] Resp. Ex. G-7;[53] Resp. Ex. G-8;[54] Resp. Ex. G-9;[55] Resp. Ex. G-11.[56] Dr. Tompkins added that the American Academy of Neurology recommends flu vaccination to MS patients. Resp. Ex. G at 4; Resp. Ex. G-10.[57] He concluded that, taken together, these publications support that inactivated flu vaccination does not exacerbate

[50] Sofia Influenza A+B FIA, Quidel.
[51] Roger Baxter, et al., *Recurrent Guillain-Barré Syndrome Following Vaccination*, 54 Clinical Infectious Diseases 800 (2012), filed as "Resp. Ex. G-4."
[52] A.E. Miller, et al., *A Multicenter, Randomized, Double-Blind, Placebo-Controlled Trial of Influenza Immunization in Multiple Sclerosis*, 48 Neurology 312 (1997), filed as "Resp. Ex. G-6."
[53] Christian Confavreaux, et al., *Vaccinations and the Risk of Relapse in Multiple Sclerosis*, 344 New Eng. J. of Med. 319 (2001), filed as "Resp. Ex. G-7."
[54] Eitan Auriel, et al., *Seasonal and H1N1v Influenza Vaccines in MS: Safety and Compliance*, 314 J. of the Neurological Sci. 102 (2012), filed as "Resp. Ex. G-8."
[55] Jacques De Keyser, et al., *Effects of Influenza Vaccination and Influenza Illness on Exacerbations in Multiple Sclerosis*, 159 J. of the Neurological Sci. 51 (1998), filed as "Resp. Ex. G-9."
[56] Lorne Zinman, et al., *Safety of Influenza Vaccination in Patients with Myasthenia Gravis: A Population-Based Study*, 40 Muscle & Nerve 947 (2009), filed as "Resp. Ex. G-11."
[57] Mauricio F. Farez, et al., *Practice Guideline Update Summary: Vaccine-Preventable Infectious and Immunization in Multiple Sclerosis*, 93 Neurology 584 (2019), filed as "Resp. Ex. G-10."

autoimmune disease. And more specifically, *Baxter* showed that flu vaccination does not trigger GBS. Resp. Ex. G at 4.

Dr. Tompkins concluded that respiratory and/or gastrointestinal infections are established causes of GBS without an additional triggering event. Infections are the most common cause of GBS. There is an absence of evidence to support relapse or exacerbation of GBS or other autoimmune disease following influenza vaccination. Therefore, it is not plausible that petitioner's flu vaccination was the final insult in the cause of [petitioner's] GBS. "Taken together, the infections reported in the medical records two to three weeks prior to onset of petitioner's GBS remain the most likely cause of his autoimmune disease and fall within a reasonable window to allow for the infection-elicited immune responses to trigger clinical symptoms of GBS." Resp. Ex. G at 4.

IV. Standards for Adjudication

A petitioner is required to establish their case by a preponderance of the evidence. § 13(1)(a). The preponderance of the evidence standard requires a "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [they] 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, 1322 n.2 (Fed. Cir. 2010) (citations omitted). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.,* 931 F.2d 867, 873 (Fed. Cir. 1991).

Distinguishing between "preponderant evidence" and "medical certainty" is important because a special master should not impose an evidentiary burden that is too high. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1379-80 (Fed. Cir. 2009) (reversing a special master's decision that petitioners were not entitled to compensation); *see also Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357 (Fed. Cir. 2000); *Hodges v. Sec'y of Health & Hum. Servs.*, 9 F.3d 958, 961 (Fed. Cir. 1993) (disagreeing with the dissenting judge's contention that the special master confused preponderance of the evidence with medical certainty). At the same time, mere conjecture or speculation is insufficient under a preponderance of evidence standard. *Snowbank Enters. v. United States*, 6 Cl. Ct. 476, 486 (1984).

The Vaccine Act provides two avenues for petitioners to receive compensation. First, a petitioner may demonstrate a "Table" injury—i.e., an injury listed on the Vaccine Injury Table that occurred within the provided time period. 42 U.S.C. § 300aa-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 on the Vaccine Injury Table, a petitioner may demonstrate an "off-Table" injury, which requires that the petitioner "prove by a preponderance of the evidence that the vaccine at issue caused the injury." *Capizzano*, 440 F.3d at 1320; *see* § 11(c)(1)(C)(ii); *see also Wright v. Sec'y of Health & Hum. Servs*., 22 F.4th 999, 1006 (Fed. Cir. 2022) (defining the term "residual effects" in the Act, as "detrimental conditions within the patient, such as lingering or recurring signs and symptoms" of the alleged vaccine injury, which are compensable). A petitioner need not show that the vaccination was the sole cause, or even the predominant cause, of the alleged injury; showing that the vaccination was

a "substantial factor" and a "but for" cause of the injury is sufficient for recovery. *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006); *Shyface*, 165 F.3d at 1352.

Petitioners are not required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe v. Sec'y of Health & Hum. Servs*., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); *see Walther v. Sec'y of Health & Hum. Servs*., 485 F.3d 1146, 1152 (Fed. Cir. 2007) (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes"). Once a petitioner has proven causation by preponderant evidence, "the burden then shifts to the respondent to show by a preponderance of the evidence that the injury is due to factors unrelated to the administration of the vaccine." *Deribeaux ex rel. Deribeaux v. Sec'y of Health & Hum. Servs*., 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing 42 U.S.C. § 300aa-13(a)(1)(B)).

To prove causation, a petitioner must satisfy the three-pronged test established in *Althen v. Sec'y of Health & Hum. Servs*., 418 F.3d 1274 (Fed. Cir. 2005). *Althen* requires that a petitioner show by preponderant evidence that a vaccination they received caused their injury "by providing: (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id*. at 1278. Together, these prongs must show "that the vaccine was 'not only a but-for cause of the injury but also a substantial factor in bringing about the injury.'" *Stone v. Sec'y of Health & Hum. Servs*., 676 F.3d 1373, 1379 (Fed. Cir. 2012) (quoting *Shyface*, 165 F.3d at 1352-53). Causation is determined on a case-by-case basis, with "no hard and fast per se scientific or medical rules." *Knudsen v. Sec'y of Health & Hum. Servs*., 35 F.3d 543, 548 (Fed. Cir. 1994). Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge, the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano*, 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). "[C]lose calls regarding causation are resolved in favor of injured claimants." *Althen*, 418 F.3d at 1280.

Each *Althen* prong requires a different showing. Under the first prong, a petitioner 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 (citation omitted). To satisfy this prong, a petitioner's "theory of causation must be supported by a 'reputable medical or scientific explanation.'" *Andreu*, 569 F.3d at 1379 (quoting *Althen*, 418 F.3d at 1278). This theory need only be "legally probable, not medically or scientifically certain." *Id*. at 1380 (emphasis omitted) (quoting *Knudsen*, 35 F.3d at 548). Nevertheless, "petitioners [must] proffer trustworthy testimony from experts who can find support for their theories in medical literature." *LaLonde v. Sec'y of Health & Hum. Servs*., 746 F.3d 1334, 1341 (Fed. Cir. 2014).

The second *Althen* prong requires proof of a "logical sequence of cause and effect." *Capizzano*, 440 F.3d at 1326 (quoting *Althen*, 418 F.3d at 1278). Even if the vaccination can cause the injury, a petitioner must show "that it did so in [this] particular case." *Hodges v. Sec'y of Health & Hum. Servs*., 9 F.3d 958, 962 n.4 (Fed. Cir. 1993) (citation omitted). "A reputable medical or scientific explanation must support this logical sequence of cause and effect," *Id*. at 961 (citation omitted), and "treating physicians are likely to be in the best position to determine whether a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury,"

Paluck v. Sec'y of Health & Hum. Servs., 786 F.3d 1373, 1385 (Fed. Cir. 2015) (quoting *Andreu*, 569 F.3d at 1375). A petitioner is not, however, required "to eliminate alternative causes as part of establishing [their] prima facie case." *Doe*, 601 F.3d at 1357-58; *see Walther*, 485 F.3d at 1152 (holding that a "petitioner does not bear the burden of eliminating alternative independent potential causes").

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

Finally, although this ruling discusses some but not all the literature in detail, I have reviewed and considered all of the medical records and literature submitted in this matter. *See Moriarty ex rel. 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. *Althen* Prong One: Reputable Medical Theory

The first Althen prong requires petitioner to provide a "reputable medical theory" demonstrating that the vaccine(s) received can cause the type of injury alleged. *Pafford*, 451 F.3d at 1355-56 (citation omitted). The mere fact that GBS resulting from flu vaccination is an on-Table injury in certain contexts does not control the Court on Prong One of an off-Table case. *Beckwith v. Sec'y of Health & Hum. Servs.*, 2026 WL 669236, at *10 (Fed. Cl. Feb. 20, 2026) (citing *de Bazan*, 539 F.3d at 1352) ("[T]he proposed 'medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement).'"). As always, "petitioner must by a preponderance of the evidence demonstrate . . . 'a medical theory causally connecting the vaccination and the injury.'" *Id*. (quoting *Althen*, 418 F.3d at 1278).

Dr. Akbari described molecular mimicry as a mechanism by which the host and a microbe share an immunologic epitope by either sequence or conformational homology. Pet. Ex. 43 at 7. For example, it has been established that autoantibodies from patients with rheumatic fever can cross react with cardiac myosin and induce autoimmune disease in mouse models. *Id*. at 7-8. The

scientific understanding of molecular mimicry has evolved, and significant sequence-specific requirements are no longer thought necessary for autoimmunity to result. Further, "molecular mimicry is accepted as a theory to explain causation of autoimmune disease and showing the direct sequence homology or cross-reactive protein involved in the process, especially in vivo, is the exception." *Id*. at 7; Pet. Ex. 43 Ref No. 4;[58] Pet. Ex. 43 Ref No. 5.[59]

Dr. Akbari contends that commercially available vaccines were developed "without much comprehension regarding how the vaccine activates the immune system." Pet. Ex. 43 at 7; Pet. Ex. 43 Ref No. 6.[60] He explained that receipt of a vaccine causes a local inflammatory reaction mediated by cells of the innate immune system. Nonspecific innate immune pathways recruit other immune cells to the area of the injection through cytokines. Dendritic cells that process foreign antigens display a piece of the foreign antigen on their surface to activate the adaptive immune system. Pet. Ex. 43 at 7. Once activated, the dendritic cell migrates to the lymph nodes and interacts with B and T cells that recognize the antigen. B cells, a type of white blood cell, function in the humoral immunity part of the adaptive immune system to produce antibody molecules which can directly bind to a foreign antigen for later destruction and removal from the body. *Id*. at 7-8. Antigens are large and complex, so antibodies can only bind to small specific areas of them.

The immune system has evolved to allow for the production of many different antibodies by many different B-cell clones against the same foreign antigen. Pet. Ex. 43 at 8. While this response increases the probability of reacting against pathogens, it also increases the chance of developing an autoimmune disease from an adverse reaction of the immune system against native self-molecules. *Id*. T cells are helper cells, subsets of which, Th1 and Th2, develop into effector T cells to help eliminate different types of pathogens. Th1 cells have a pathogenic role in GBS. *Id*.; Pet. Ex. 43 Ref No. 7.[61] T cells mature in the thymus and are also called CD4+ cells. Peripheral self-tolerance in CD4+ T cells is maintained by several mechanisms, most importantly suppression by regulatory T lymphocytes, or "Tregs," known for their active role in maintaining tolerance to self-antigens and combating autoimmune disease. Pet. Ex. 43 at 8-9. If the suppression function of Tregs in humans is altered, autoimmune diseases including GBS occur. *Id*. In patients with acute GBS, the number of Tregs is decreased. *Id*. at 9; Pet. Ex. 43 Ref No. 8.[62] This is likely the reason certain people are predisposed to develop autoimmune diseases when their immune system is activated, such as following vaccination.

Dr. Akbari explained that immune activation is required for the onset of autoimmune disease. Normally, self-reactivity is part of regeneration and healing through removal of cellular debris from damaged cells. The immune system plays a central role in the maintenance of these healing processes. However, the same process that promotes self-tolerance can promote autoimmunity. Environmental triggers are required for the induction of disease. Within hours of

[58] Adam P. Kohm, et al., *Mimicking the Way to Autoimmunity: An Evolving Theory of Sequence and Structural Homology*, 11 Trends in Microbiology 101 (2003), filed as "Pet. Ex. 43 Ref No. 4."
[59] Marinos C. Dalakas, *Future Perspectives in Target-Specific Immunotherapies of Myasthenia Gravis*, 8 Therapeutic Advances in Neurological Disorders 316 (2015), filed as "Pet. Ex. 43 Ref No. 5."
[60] Claire-Anne Siegrist, *Vaccine Immunology*, in *Plotkin's Vaccines* 16 (2018), filed as "Pet. Ex. 43 Ref No. 6."
[61] Shujuan Li, et al., *IL-17 and IL-22 in Cerebrospinal Fluid and Plasma Are Elevated in Guillain-Barré Syndrome*, 2012 Mediators of Inflammation 1 (2012), filed as "Pet. Ex. 43 Ref No. 7."
[62] Li-jun Chi, et al., *Abnormality of Circulating $CD4^+$ $CD25^+$ Regulatory T Cell in Patients with Guillain-Barré Syndrome*, 192 J. of Neuroimmunology 206 (2007), filed as "Pet. Ex. 43 Ref No. 8."

vaccination, immune regulatory mechanisms such as Tregs maintain peripheral tolerance actively suppressing autoreactive effector immune cells such as lymphocytes capable of causing injury. While vaccines are safe inducers of an immune response, with effective mechanisms maintaining immune homeostasis mounting immunological memory in response, adverse immune response may occur. Pet. Ex. 43 at 20.

Dr. Akbari detailed how toll-like receptors (TRLs) can be upregulated by flu vaccination whether adjuvanted or not through inflammasome activation and subsequent production of interleukins and related genes. Pet. Ex. 46 at 1-5; Pet. Ex. 46 Ref No. 1;[63] Pet. Ex. 46 Ref No. 2;[64] Pet. Ex. 46 Ref No. 3;[65] Pet. Ex. 46 Ref No. 4;[66] Pet. Ex. 48 at 12-14; Pet. Ex. 48 Ref No. 14;[67] Pet. Ex. 48 Ref No. 15;[68] Pet. Ex. 48 Ref No. 16;[69] Pet. Ex. 48 Ref No. 17;[70] Pet. Ex. 48 Ref No. 18.[71] Activation of the immune system following receipt of the flu vaccine causes complex molecules known as inflammasomes to form. These inflammasomes activate the production of active cytokines, including those of the IL-1 family, which are potent stimulators of the adaptive immune response. Petitioner's flu vaccine contained hemagglutinin from several strains of flu virus including H1N1 proteins which are primary targets for inflammasomes. Pet. Ex. 46 at 4-5; Pet. Ex. 46 Ref No. 5;[72] Pet. Ex. 46 Ref No. 6;[73] Pet. Ex. 46 Ref No. 7.[74] The activation of cytokines and TNF-a experimentally induce demyelination, elevate temperature, and increase lassitude,[75] providing strong evidence of the important role cytokines play in the production of Th17 cells. Pet. Ex. 48 at 14. Succinctly, flu infection and vaccination can produce inflammasomes along with T-cell activation that can cause demyelination. Pet. Ex. 46 at 6; Pet. Ex. 48 at 14. Emerging research has found increased levels of Th17 in patients with peripheral and central demyelinating

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[63] Stephen N. Crooke, et al., *Inflammasome Activity in Response to Influenza Vaccination is Maintained in Monocyte-Derived Peripheral Blood Macrophages in Older Adults*, 2 Frontiers in Aging 1 (2021), filed as "Pet. Ex. 46 Ref No. 1" and "Pet. Ex. 48 Ref No. 13."

[64] Pin Wan, et al., *AP-1 Signaling Pathway Promotes Pro-IL-1β Transcription to Facilitate NLRP3 Inflammasome Activation Upon Influenza A Virus Infection*, 12 Virulence 502 (2022), filed as "Pet. Ex. 46 Ref No. 2."

[65] Xing Yang, et al., *KSHV-Encoded ORG45 Activates Human NLRP1 Inflammasome*, 23 Nature Immunology 916 (2022), filed as "Pet. Ex. 46 Ref No. 3."

[66] Ella Hartenian & Petr Broz, *Viral Protein Activates the NLRP1 Inflammasome*, 23 Nature Immunology 818 (2022), filed as "Pet. Ex. 46 Ref No. 4."

[67] Rebecca E. Tweedell, et al., *A Comprehensive Guide to Studying Inflammasome Activation and Cell Death*, 15 Nature Protocols 3284 (2020), filed as "Pet. Ex. 48 Ref No. 14."

[68] Lisa M. Christian, et al., *Proinflammatory Cytokine Responses Correspond with Subjective Side Effects After Influenza Virus Vaccination*, 33 Vaccine 3360 (2015), filed as "Pet. Ex. 48 Ref No. 15."

[69] Weichun Tang, et al., *Post-Vaccination Serum Cytokines Levels Correlate with breakthrough Influenza Infections*, 13 Sci. Rep. 1174 (2023), filed as "Pet. Ex. 48 Ref No. 16."

[70] Si Ming Man & Thirumala-Devi Kanneganti, *Regulation of Inflammasome Activation*, 265 Immunological Revs. 6 (2015), filed as "Pet. Ex. 48 Ref No. 17."

[71] Taejoon Won, et al., *Increased Interleukin 18-Dependent Immune Respondent are Associated with Myopericarditis After COVID-19 mRNA Vaccination*, 13 Frontiers in Immunology (2022), filed as "Pet. Ex. 48 Ref No. 18."

[72] Alexander Khoruts, *IL-1 Acts on Antigen-Presenting Cells to Enhance the* In Vivo *Proliferation of Antigen-Stimulated Naïve CD4 Cells via a CD28-Dependent Mechanism That Does Not Involve Increased Expression of CD28 Ligands*, 34 European J., of Immunology 1085 (2004), filed as "Pet. Ex. 46 Ref No. 5" and "Pet. Ex. 43 Ref No. 25."

[73] Kiyoshi Takeda, et al., *Toll-Like Receptors*, 21 Ann. Rev. of Immunology 335 (2003), filed as "Pet. Ex. 46 Ref No. 6" and "Pet. Ex. 43 Ref No. 24."

[74] Virginie Pétrilli, et al., *The Inflammasome: A Danger Sensing Complex Triggering Innate Immunity*, 19 Current Opinion in Immunology 615 (2007), filed as "Pet. Ex. 46 Ref No. 7."

[75] Lassitude refers to "weakness" or "exhaustion." "Lassitude," Dorland's Illustrated Medical Dictionary 994 (33rd ed. 2020).

disease. Pet. Ex. 46 at 6; Pet. Ex. 46 Ref No. 8;[76] Pet. Ex. 48 at 14-15; Pet. Ex. 48 Ref No. 20;[77] Pet. Ex. 48 Ref No. 21;[78] Pet. Ex. 48 Ref No. 22.[79] *See also* Pet. Ex. 52 at 8-13.

Dr. Akbari continued that interleukins, or ILs, are fast-acting cells and produce cytokines and chemokine within hours to days of flu vaccination. Pet. Ex. 46 at 13-14. Petitioner's immune system was already activated and in an inflammatory state from acute pancreatitis and diarrhea prior to his vaccination. The production of proinflammatory cytokines from pancreatitis and diarrhea sensitized his immune system which was further exacerbated by an inflammatory response to the flu vaccination. The influx of proinflammatory cytokines played a substantial role in the faster onset of petitioner's GBS. *Id*. at 14, 24-25; Pet. Ex. 48 at 20-21, 23; Pet. Ex. 52 at 1-2.

Dr. Tompkins disagreed that the flu vaccine can cause GBS. He detailed several issues he took with studies cited by Dr. Akbari. He also provided a study which conducted surveillance for GBS during two recent flu seasons and found no excess risk of GBS in either flu vaccine for either risk window observed. Resp. Ex. B-1 at 4; Resp. Ex. B-6.[80] See also Resp. Exs. B-19[81] and B-22[82] for additional epidemiological studies finding no elevated risk of GBS following flu vaccination when adjusted for antecedent infections.[83]

Dr. Tompkins agreed that *Finsterer* recognized an association between flu vaccination and GBS. Resp. Ex. G at 2; Resp. Ex. G-1.[84] But he added that *Petras,* a meta-analysis, concluded there was no increased risk of GBS following vaccination, only following flu-like illness, based on 40 years of studies since 1981. Resp. Ex. G at 2-3; Resp. Ex. G-2.[85] According to Dr. Tompkins, highly purified inactivated influenza vaccines are safe with many studies showing no increased risk of GBS following vaccination. In the studies that do show a slightly increased risk of GBS following contemporary flu vaccination, flu and other infections were subsequently found to account for the risk. Resp. Ex. D-2 at 13. Dr. Tompkins disagreed that inactivated flu vaccine

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[76] Feng-Jun Mei, et al., *Th1 Shift in CIDP Versus Th2 Shift in Vasculitic Neuropathy in CSF*, 228 J. of the Neurological Sci. 75 (2005), filed as "Pet. Ex. 46 Ref No. 8."

[77] Jesus F. Bermejo-Martin, et al., *Th1 and Th17 Hypercytokinemia as Early Host Response Signature in Severe Pandemic Influenza*, 13 Critical Care 1 (2009), filed as "Pet. Ex. 48 Ref No. 20."

[78] Yinyao Lin, et al., *Th17 Cytokines and Vaccine-Induced Immunity*, 32 Seminars in Immunopathology 79 (2010), filed as "Pet. Ex. 48 Ref No. 21."

[79] F. Jadidi-Niaragh & A. Mirshafiey, *Th17 Cell, the New Player of Neuroinflammatory Process in Multiple Sclerosis*, 74 Scandinavian J. of Immunology 1 (2011), filed as "Pet. Ex. 48 Ref No. 22."

[80] Arya, et al., *supra*, note 47.

[81] Sharon K. 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 e67185 (2013), filed as "Resp. Ex. B-19."

[82] Sharon K. Greene, et al., *Near Real-Time Surveillance for Influenza Vaccine Safety: Proof-of-Concept in the Vaccine Safety Datalink Project*, 171 Am. J. of Epidemiology 177 (2010), filed as "Resp. Ex. B-22."

[83] Dr. Akbari addressed these studies and noted that they sourced their data from the Vaccine Safety Datalink. Dr. Akbari argued that the data from the VSD is flawed, as it underrepresents vaccinations administered in nontraditional settings, do not account for uninsured vaccine recipients, and coverage rates are higher in the VSD than in the general population. These and other factors make VSD data difficult to generalize. Pet. Ex. 46 at 7-8.

[84] Josef Finsterer, *Triggers of Guillain-Barré Syndrome:* Campylobacter jejuini *Predominates*, 23 Int'l J. of Molecular Sci. 14222 (2022), filed as "Resp. Ex. G-1."

[85] Marek Petráš, et al., *Is an Increased Risk of Developing Guillain-Barré Syndrome Associated with Seasonal Influenza Vaccination? A Systematic Review and Meta-Analysis*, 8 Vaccines 150 (2020), filed as "Resp. Ex. G-2."

elicits cytokines, drives inflammasome activation, elicits Th27 T cells, or that they could plausibly do so in fewer than three days. Resp. Ex. D-2 at 8; Resp. Ex. G at 8-9. He submitted that the studies relied upon by Dr. Akbari in support of his proposed mechanisms linking flu vaccine and GBS are insufficient. Some suggest that infection may be associated with autoimmune disease, but infection is different from a vaccination. Resp. Ex. G at 9. *See* Pet. Ex. 46 Ref No. 35;[86] Pet. Ex. 46 Ref No. 29;[87] Pet. Ex. 46 Ref No. 26.[88]

Dr. Tompkins asserted that the animal study Dr. Akbari relied upon, in which mice were seeded with millions of CD4+ and CD8+ T cells specific for the HA antigen present in the influenza vaccine which depleted the regulatory T cells and prevented "dampening of proinflammatory responses," is inapplicable to this case. Resp. Ex. G at 9. The vaccine used was a split-inactivated monovalent vaccine against the 2009 pandemic H1N1 virus and contained the adjuvant AS03, which is not currently used in any seasonal influenza vaccine or other human vaccine. The vaccine with the ASO3 adjuvant does significantly increase cytokine responses in vaccinated mice and was the only vaccine to elicit Th17 T cells. The authors specifically noted that nonadjuvanted influenza vaccines do not elicit Th17 T cells. *Id*. at 10; Resp. Ex. G-26.[89] Dr. Tompkins agreed that animal studies show increased cytokines and Th17 elicited with the use of ASO3 adjuvanted vaccines, but that has no bearing on this case. Resp. Ex. G at 9. While the studies presented may offer new insights for potential mechanisms for autoimmune disease, they fail to provide a clear mechanism connecting contemporary inactivate flu vaccination to GBS. *Id*. at 10.

Further, the studies relied upon as suggesting increased cytokine levels following inactivated or live attenuated flu vaccines did not look at onset of disease within two days of vaccination, or tested only women with sore arms, but found TNF-α, IFN- γ, IL-6, IL-8, and IL-10 were lower in all vaccinated groups. Resp. Ex. G at 8; Pet. Ex. 48 Ref No. 17 at 3, 4.[90] *See also* Pet. Ex. 48 Ref No. 1 at 11.[91]

Dr. Tompkins then examined Dr. Akbari's theory of molecular mimicry. Dr. Tompkins agreed with Dr. Akbari's definition of molecular mimicry. However, Dr. Tompkins argued that "there must be demonstrable evidence of the foreign antigen eliciting cross-reactive immune responses that cause tissue damage recapitulating clinical disease." Resp. Ex. B-1 at 5. In other words, Dr. Tompkins, citing the Institute of Medicine, would require a showing of the precise direct sequence homology or cross-reactive protein shared between the host and the vaccine for molecular mimicry to be sufficiently implicated. *Id*.; Resp. Ex. B-3.[92]

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[86] Jing Li, et al., *KIR^{+}CD8^{+} T Cells Suppress Pathogenic T Cells and Are Active in Autoimmune Diseases and COVID-19*, 376 Sci. eabi9591 (2022), filed as "Pet. Ex. 46 Ref No. 35."

[87] Jocelyn G. Labombarde, et al., *Induction of Broadly Reactive Influenza Antibodies Increases Susceptibility to Autoimmunity*, 38 Cell Reps. 110482 (2022), filed as "Pet. Ex. 46 Ref No. 29."

[88] Jon D. Laman, et al., *Guillain-Barré Syndrome: Expanding the Concept of Molecular Mimicry*, 43 Trends in Immunology 296 (2022), filed as "Pet. Ex. 46 Ref No. 26."

[89] Karen K. Yam, et al., *AS03-Adjuvanted, Very-Low-Dose Influenza Vaccines Induce Distinctive Immune Responses Compared to Unadjuvanted High-Dose Vaccines in BALB/c Mice*, 6 Frontiers in Immunology 1 (2015), filed as "Resp. Ex. G-26."

[90] Man & Kanneganti, *supra*, note 70.

[91] Salmon, et al., *supra*, note 42.

[92] Kathleen Stratton, et al., Institute of Medicine, *Adverse Effects of Vaccines: Evidence and Causality* 57 (2012)

The Vaccine Program however does not require scientific certainty to make a finding of entitlement. While the IOM may require a stringent identification of identical shared mimics to satisfy scientific rigor, here, "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program." *Knudsen*, 35 F.3d at 549. Indeed, while not dispositive or binding, other Special Masters have routinely found in favor of petitioners on Prong I in flu/GBS cases based in molecular mimicry. *See, e.g., Barone v. Sec'y of Health & Hum. Servs.*, No. 11-707V, 2014 WL 6834557, at *8 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (citing *Petronelli v. Sec'y of Health & Hum. Servs.*, No. 12-285V, 2013 WL 6054752, at *6 (Fed. Cl. Spec. Mstr. Oct. 25, 2013) (citing several Program flu/GBS cases in which entitlement was found for petitioners relying on molecular mimicry)). I have also previously noted that "[t]he association between flu vaccine and GBS is well-established in the Vaccine Program." *McCarter v. Sec'y of Health & Hum. Servs.*, No 20-1490V, 2025 WL 3458768, at *17 (Fed. Cl. Spec. Mstr. Nov. 7, 2025) (collecting cases). The Chief Special Master even favorably compared "the extensive science supporting causation for GBS after vaccination" to the relatively weaker evidence, in his opinion, for linking vaccination to chronic inflammatory demyelinating polyneuropathy, or CIDP, which he described as a distinct neuropathy. *Nieves v. Sec'y of Health & Hum. Servs.*, No. 18-1602V, 2023 WL 3580148, at *36 (Fed. Cl. Spec. Mstr. May 22, 2023).

Respondent ultimately argues that "there is no reliable scientific evidence – either epidemiologic or mechanistic – that a seasonal flu vaccine can cause GBS." ECF No. 130 at 50. Dr. Tompkins opined that an inactivated vaccine does not elicit cytokines, drive inflammasome activation, or elicit Th17 cells. Resp. Ex. G at 9. But if this was so, an inactivated flu vaccine without an adjuvant could not elicit the immune response necessary to develop adaptive immunity, which is required to be effective.

Petitioner has put forth a sound and reliable medical theory for how the flu vaccine can cause GBS and has supported that theory with expert opinions, including that of an immunologist, and reliable medical literature. I therefore find, based on the evidence and expert testimony, that the influenza vaccine can cause GBS. It is undisputed that petitioner received an influenza vaccine on October 21, 2016, and was diagnosed with GBS. Thus, Prong I, also known as the "can it cause?" inquiry, is satisfied.

B. *Althen* Prongs II and III: Logical Sequence of Cause and Effect and Proximate Temporal Relationship

The resolution of Prongs II and III in this case are intertwined and best addressed together.

1. Diagnosis and Medical History

The diagnosis of GBS is not in dispute. In dismissing petitioner's Table claim for flu-vaccine-related GBS, the Chief Special Master concluded, based on all the evidence, that the onset of petitioner's GBS occurred in fewer than the three days required by the Vaccine Injury Table. ECF No. 50 at 5. This was undisputed by petitioner who, in his brief in support of onset wrote, "'it appears the weakness in [his] hands 54-55 hours of [sic] vaccination may represent the first objective sign of GBS/AIDP alleged to be caused by vaccination.'" ECF No. 50 at 7 (quoting ECF

No. 47 at 2). Further, in his affidavits and reports to his medical providers, petitioner consistently reported receipt of the flu vaccine on Friday, October 21, 2016, between 4:00 and 5:00 PM, weakness in his hands on Sunday night, October 23, 2016, at around 10:00 PM, difficulty walking by Monday, October 24, 2016, and inability to walk on Tuesday, October 25, 2016. Pet. Ex. 4 at 5; Pet. Ex. 21 at 1-2; Pet. Ex. 11 at 199. Thus, petitioner's onset of GBS was between 54 to 58 hours post-vaccination.

The medical records support the following findings. Petitioner was born on June 26, 1967. He was 49 years old when he received the subject flu vaccination. Pet. Ex. 1 at 1. He had no known medical conditions before October of 2016. Pet. Ex. 7 at 21-26, 14-19, 20. His prior medical history was not questioned.

Petitioner presented on October 5, 2016, with body aches, chills, eye burning, throat pain, hoarse voice, pain on inspiration, chest and stomach pain, excessive thirst, decreased appetite, and fever of 102°. Pet. Ex. 7 at 1-2. Examination was normal, an influenza A/B test was negative, and the diagnosis was "flu-like" symptoms. He was prescribed Bentyl for stomach cramping. *Id*. at 1,4. Four days later, he presented to the ER with five to six days of abdominal pain, fever, chills, and diarrhea following binge drinking over the weekend. Pet. Ex. 15 at 14. He had upper abdominal tenderness upon examination but no fever. *Id*. at 17, 24. CT and ultrasound of the stomach revealed a fatty liver and gallstones without inflammation. *Id*. Stool sample revealed fecal leukocytes indicative of infection or inflammation but no campylobacter infection, parasites, or toxin. *Id*. at 120. His blood work was abnormal with elevated inflammatory markers, glucose, AIC, AST, and lipase. *Id*. at 116-119. He was diagnosed with diabetes for the first time and acute pancreatitis of unknown cause. *Id*. at 14. He was given Cipro for suspected C-diff but he tested negative with resolution of abdominal pain the following day. *Id*. at 119. The diagnosis on discharge was acute pancreatitis without necrosis or infection, viral intestinal infection not otherwise specified, and DM, for which Metformin and Glipizide were prescribed. Pet. Ex. 15 at 2-4, 13-14, 17, 97.

On October 15, 2016, petitioner presented with chest pain, back pain, and shortness of breath. Pet. Ex. 15 at 197, 201. Blood work showed elevated cardiac regulatory proteins and elevated WBC. *Id*. at 446, 181. Cardiac catheterization showed coronary artery disease, but the echocardiogram and stress test were normal. *Id*. at 295, 364, 370, 471. His CT was unchanged, and he was discharged with a diagnosis of acute pancreatitis. *Id*. at 181-82.

At a follow-up on October 21, 2016, petitioner had no complaints and had normal strength and intact sensory on examination. Pet. Ex. 17 at 3. He was taking medication for his recent diabetes diagnosis. *Id*. at 3-5. His blood work that day was abnormal with many highly elevated inflammatory markers, including but not limited to CRP, interleukin 6 and 8, and mildly elevated WBC. *Id*. at 101, 98-99. The subject flu vaccination was administered at about 4:00 to 5:00 PM. *Id*. at 4. Sunday night, October 23, 2016, at around 10:00 PM, petitioner noted weakness in his hands. The next morning, he walked slowly. By Tuesday, he could not walk at all. At that time, examination revealed weakness and diminished or absent reflexes. He was diagnosed with GBS. Pet. Ex. 11 at 199, 202, 203; Pet. Ex. 4 at 5. Petitioner's laboratory findings included but were not limited to strong positive GM1 Ab 180 and GD1b Ab, elevated lipase, and inflammatory markers. Pet. Ex. 11 at 69-70, 187, 64, 81. Lumbar puncture and EMG/NCS studies were consistent with GBS. *Id*. at 168, 155-156. The clinical summary included GBS following vaccination. *Id*. at 2.

2. Expert Reports

Petitioner's experts opined that the flu vaccine triggered petitioner's GBS, either directly or in combination with inflammatory processes caused by the acute pancreatitis and cholelithiasis, or gallstones, he suffered in the weeks prior to his receipt of the flu vaccine. Pet. Ex. 45 at 6; Pet. Ex. 45 Ref No. 6.[93] On the day of his flu vaccination, petitioner had a normal examination and no complaints, but his blood work showed elevated inflammatory markers including CRP, interleukin 6, and interleukin 8, with mildly elevated white blood cells and elevated platelets. Pet. Ex. 17 at 3-5, 101, 98-99. Petitioner's experts opined that the flu vaccine can cause GBS on its own and was a substantial factor in his development of GBS. And given petitioner's already systemic inflammatory state, receipt of the flu vaccination resulted in an earlier-than-typical onset of GBS within 54 to 58 hours, less time than the three days provided for by the Vaccine Table.

Conversely, respondent's experts argued that petitioner suffered from respiratory and gastrointestinal viral infections two to three weeks prior to the onset of GBS, as well as diabetes, recent surgery, and alcoholism. All of these are known causes of GBS and occurred within the risk interval for the onset of GBS. Therefore, the flu vaccination played no role in his GBS. *See generally* Resp. Exs. A-2, B-1, C, D-2, F, and G.

Petitioner's experts also responded to the alternative causes of alcoholic neuropathy, diabetes mellitus, and surgery raised by respondent's experts. However, respondent later disclaimed these as potential alternative causes. ECF No. 130 at 60-61 ("Petitioner also argues at length that petitioner's alcoholic neuropathy and diabetic neuropathy did not cause his GBS. Respondent does not assert that these conditions caused petitioner's GBS."). Thus, petitioner's arguments rebutting these potential alternative causes will not be addressed in full. Instead, respondent argues that petitioner suffered from a respiratory infection and/or a gastrointestinal infection, and that one of those more likely than not was the cause of petitioner's GBS.

i. Petitioner's Experts

Dr. Kinsbourne explained that petitioner's "flu-like" and gastrointestinal symptoms between October 5 and October 9, 2016, were manifestations of the inflammation caused by acute pancreatitis. They were not symptoms of flu virus, gastroenteritis, or bacteriuria. Pet. Ex. 45 at 5-6; Pet. Ex. 45 Ref No. 3;[94] Pet. Ex. 47 at 3-4, 5-6; Pet. Ex. 47 Ref No. 11;[95] Pet. Ex. 49 at 1-2; Pet. Ex. 53 Ref No. 1;[96] Pet. Ex. 53 Ref No. 2.[97] He added that bacteriuria is a complication of

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[93] Sekimoto, et al., *JPN Guidelines for the Management of Acute Pancreatitis: Epidemiology, Etiology, Natural History, and Outcome Predictors in Acute Pancreatitis*, 13 J. of Hepato-Biliary-Pancreatic Surgery 10 (2006), filed as "Pet. Ex. 45 Ref No. 6."

[94] A. Mora, et al., *Activation of Cellular Immune Response in Acute Pancreatitis*, 40 Gut 794 (1997), filed as "Pet. Ex. 45 Ref No. 3."

[95] Theo J. Verheij, et al., *Clinical Presentation, Microbiological Aetiology and Disease Course in Patients with Flu-Like Illness: A Post Hoc Analysis of Randomised Controlled Trial Data*, 72 British J. of Gen. Prac. e217 (2022), filed as "Pet. Ex. 47 Ref No. 11."

[96] Robert A. Balk, *Systemic Inflammatory Response Syndrome (SIRS): Where Did It Come from and Is It Still Relevant Today?*, 5 Virulence 20 (2014), filed as "Pet. Ex. 53 Ref No. 1."

[97] J. Toouli, et al., *Guidelines for the Management of Acute Pancreatitis*, 17 J. of Gastroenterology and Hepatology S15 (2002), filed as "Pet. Ex. 53 Ref No. 2."

pancreatitis, not a cause of it. He also noted that petitioner was never diagnosed with gastroenteritis. Pet. Ex. 47 at 4; Pet. Ex. 48 at 1-2; Pet. Ex. 7 at 1-5. Petitioner demonstrated all four of the hallmark signs of acute pancreatitis: raised temperature, elevated pulse rate, low blood pressure, and low oxygenation. Pet. Ex. 49 at 3; Pet. Ex. 49 Ref No. 3.[98] Further, acute pancreatitis has not been shown to be a trigger of GBS. A few cases of GBS have been reported in those with severe long-term pancreatitis, but no mechanism has been plausibly posited, suggesting that there were other causes for GBS in those patients. Pet. Ex. 45 at 6, 8; Pet. Ex. 45 Ref No. 6;[99] Pet. Ex. 45 Ref No. 1;[100] Pet. Ex. 45 Ref No. 2.[101]

Dr. Kinsbourne also noted that petitioner had a negative influenza A/B antigen test on October 4, 2016, but with symptoms referred to as "flu like" despite not having a cough, running nose, or shortness of breath. Pet. Ex. 45 at 5. He referred to *Ryu, et al.*, which showed that the flu test petitioner underwent had 94.5% sensitivity level for influenza A and 91.7% for influenza B. Pet. Ex. 47 at 10; Pet. Ex. 47 Ref No. 6.[102] He disagreed that petitioner had an influenza infection and that the influenza testing was a false negative. Pet. Ex. 47 at 10.

Subsequently, on October 9, 2016, petitioner's illness "declared itself plainly as a gastrointestinal issue, with nausea, vomiting, cramps and diarrhea, as well as continued fever" which was ultimately diagnosed as acute pancreatitis with abdominal symptoms resolving by October 11, 2016. *Id*. at 5-6.

Dr. Kinsbourne explained that acute pancreatitis is most frequently caused by alcoholism and cholelithiasis, or gallstones. Pet. Ex. 45 at 6; Pet. Ex. 45 Ref No. 6 at 1.[103] While *Gross, et al.* reported four cases of GBS following pancreatitis, the patients involved were seriously ill with severe long-lasting pancreatitis, not acute pancreatitis that resolved quickly as in petitioner. Pet. Ex. 45 at 6. *See generally* Pet. Ex. 45 Ref No. 1.[104] Further, in those cases, the GBS appeared months after treatment was initiated, prompting the authors to suggest alternative triggers for the patients' GBS. Pet. Ex. 45 at 6; Pet. Ex. 45 Ref No. 1 at 1.[105] *Harikumar, et al.* also reported polyneuritis in a patient with severe pancreatitis who later died. Pet. Ex. 45 at 6. *See generally* Pet. Ex. 45 Ref No. 2.[106] The patients in these studies were distinguishable from petitioner due to the uncertainty surrounding their diagnoses with no attempt by the authors to explain the mechanism of the polyneuropathy they suffered. Therefore, these cases of GBS following pancreatitis fall short of providing an alternative cause for petitioner's GBS. Pet. Ex. 45 at 6.

Dr. Kinsbourne added that the discovery of petitioner's "severe biliary tract disorder," or

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[98] Per-Olof Nyström, *The Systemic Inflammatory Response Syndrome: Definitions and Etiology*, 41 J. of Antimicrobial Chemotherapy 1 (1998), filed as "Pet. Ex. 49 Ref No. 3."
[99] Sekimoto, et al., *supra*, note 93.
[100] Gross, et al., *supra*, note 29.
[101] Harikumar, et al., *supra*, note 30.
[102] Sook Won Ryu, et al., *Comparison of Three Rapid Influenza Diagnostic Tests with Digital Readout Systems and One Conventional Rapid Influenza Diagnostic Test*, 32 J. of Clinical Lab Analysis e22234 (2018), filed as "Pet. Ex. 47 Ref No. 6."
[103] Sekimoto, et al., *supra*, note 93.
[104] Gross, et al., *supra*, note 29.
[105] *Id*.
[106] Harikumar, et al., *supra*, note 30.

cholecystitis, in late December during surgery could explain his acute pancreatis. Pet. Ex. 45 at 7-8. He explained how the cystic duct joins with the pancreatic duct to form the common bile duct. An obstruction of the bile duct by a gallstone is the most common cause of acute pancreatitis. The obstruction often leads to reflux of bile acids into the pancreatic duct, causing inflammation in the pancreatic acinar cells, which are the functional unit of the exocrine pancreas that synthesizes, stores, and secretes digestive enzymes. Activation of digestive enzymes before they get to the duodenum leads to acute pancreatitis. Pet. Ex. 45 at 7; Pet. Ex. 45 Ref No. 7 at 1-2.[107] If the gallstone dislodges, then the acute pancreatitis would resolve. Pet. Ex. 45 at 7. The diarrhea that accompanied petitioner's onset of acute pancreatitis "appears to have been part of the same disease process" or perhaps secondary to the duodenum involvement with a widespread chronic inflammatory process revealed during surgery. *Id*. at 8. No respiratory or gastrointestinal viral infections were found and there was no evidence of any additional event other than severe biliary system inflammation that contributed to petitioner's acute pancreatitis. *Id*. *Mora, et al.* showed inflammatory mediators that have been implicated as a component of acute pancreatitis onset. *Id*.; Pet. Ex. 45 Ref No. 3.[108] According to Dr. Kinsbourne, the immune activation caused by acute pancreatitis could have lingered to precipitate an earlier onset of GBS. Pet. Ex. 45 at 8.

Dr. Kinsbourne detailed the "extensive medical literature [that] demonstrates that an acute systemic inflammatory response is integral to the onset of acute pancreatitis." Pet. Ex. 47 at 6-7; Pet. Ex. 47 Ref No. 1;[109] Pet. Ex. 47 Ref No. 2;[110] Pet. Ex. 47 Ref No. 4;[111] Pet. Ex. 47 Ref No. 14.[112] *See also* Pet. Ex. 53 Ref No. 1.[113] The symptoms petitioner reported – abdominal pain, nausea, vomiting, and fever – were all due to inflammation mediated by inflammatory cytokines attendant to the onset of acute pancreatitis but often mistaken for flu-like symptoms. Pet. Ex. 47 at 4-6; Pet. Ex. 45 Ref No. 3;[114] Pet. Ex. 53 Ref No. 1 at 2[115] (referring to "SIRS," systemic inflammatory response syndrome, a concept used to describe the complex pathophysiologic response to an insult which includes pancreatitis). He added that the concept of SIRS "describes the complex, physiological response to an insult such as infection, trauma, burns, pancreatitis or a variety of other injuries. These clinical parameters are not unique to the septic patient and may be present in a diverse group of clinical disorders that result in a proinflammatory response." Pet. Ex. 53 at 2. *Frossard* explained that "'significant evidence has accumulated that synthesis and release of pro-inflammatory cytokines and chemokines is also responsible for local injury and systemic dispersion of the inflammatory mediators. Thus, inflammatory mediators produced within the gland increase pancreatic injury and radiate to distant organs.'" Pet. Ex. 47 at 7 (quoting Pet. Ex.

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[107] Guo-Jun Wang, et al., *Acute Pancreatitis: Etiology and Common Pathogenesis*, 15 World J. of Gastroenterology 1427 (2009), filed as "Pet. Ex. 45 Ref No. 7."
[108] Mora, et al., *supra*, note 94.
[109] Zilvinas Dambrauskas, et al., *Different Profiles of Cytokine Expression During Mild and Severe Acute Pancreatitis*, 16 World J. of Gastroenterology 1845 (2010), filed as "Pet. Ex. 47 Ref No. 1."
[110] Jean-Louis Frossard & Catherine M. Pastor, *Experimental Acute Pancreatitis: New Insights into the Pathophysiology*, 7 Frontiers in Bioscience d275 (2002), filed as "Pet. Ex. 47 Ref No. 2."
[111] J. Mayer, et al., *Inflammatory Mediators in Human Acute Pancreatitis: Clinical and Pathophysiological Implications*, 47 Gut 546 (2000), filed as "Pet. Ex. 47 Ref No. 4."
[112] Simon P. Young & Jonathan P. Thompson, *Severe Acute Pancreatitis*, 8 Continuing Educ. In Anaesthesia, Critical Case & Pain 125 (2008), filed as "Pet. Ex. 47 Ref No. 14."
[113] Balk, *supra*, note 96.
[114] Mora, et al., *supra*, note 94.
[115] Balk, *supra*, note 96.

47 Ref No. 2 at 2[116]). Petitioner's inflammatory response was sterile, "as is the case in acute pancreatitis." *Id*.

According to Dr. Kinsbourne, petitioner's GBS "was not caused by 'the triggering effect of respiratory and gastrointestinal infections,' as Dr. Jamieson insists," but by "part of the process of acute pancreatitis itself. . . . The relatively brief interval between the vaccination and the onset of the Guillain-Barre syndrome remains medically reasonable." Pet Ex. 47 at 8; Pet. Ex. 49 at 2. *Cruz-Santamaria, et al.* explained the "onrush of symptoms" that is part of acute pancreatitis. Pet. Ex. 49 at 2; Pet. Ex. 49 Ref No. 1.[117] *Nyström, et al.* referred to the diverse inflammatory manifestations of acute pancreatitis as evidence of SIRS. SIRS is symptomatically like sepsis and includes a reaction by endogenous mediators to non-infectious systemic inflammation resulting from trauma, burns, or acute pancreatitis. Pet. Ex. 49 at 2-3; Pet. Ex. 49 Ref No. 3.[118] *Singh, et al*. showed that acute pancreatitis is diagnosed by two or more of the following: raised temperature, elevated pulse rate, low blood pressure, and low oxygenation. Here, petitioner had all four. Pet. Ex. 49 at 3; Pet. Ex. 49 Ref No. 4.[119] *Kamisawa, et al.* reported that 64% of acute pancreatitis patients have lacrimal gland dysfunction including burning, painful, watery, itchy eyes, or dry mouth. Pet. Ex. 49 at 3; Pet. Ex. 49 Ref No. 2.[120] *Wu* reported that patients with acute pancreatitis have an onset of sudden severe epigastric pain that may radiate to their back, frequently with nausea, vomiting, fever and/or sweating. Pet. Ex. 49 at 6; Pet. Ex. 47 Ref No. 13.[121] The synchrony of the disease onset and rapid remission confirms that petitioner's symptoms were concomitants of petitioner's acute pancreatitis. *Vlachou, et al.* showed that the extra-pancreatic manifestations of acute pancreatitis derive from other organs such as the bile ducts, gallbladder, kidneys, retroperitoneum, mesentery, thyroid, lacrimal glands and orbits, salivary glands, lymph nodes, lungs, gastrointestinal tract, and blood vessels. Pet. Ex. 49 at 3; Pet. Ex. 49 Ref No. 5. [122]

Even if petitioner suffered from an upper respiratory or gastrointestinal virus, not every viral infection is associated with GBS. *Finsterer* listed 13 viruses and five vaccines, including the flu vaccine, associated with GBS out of hundreds of different respiratory and gastrointestinal viruses with no known connection to GBS. *Id*.; Resp. Ex. G-1.[123] It is known that the flu vaccine can rarely trigger GBS, and there is no evidence submitted to the contrary. Pet. Ex. 54 at 2; Resp. Ex. G-1;[124] Pet. Ex. 54 Ref No. 3.[125] See also Pet. Ex. 54 Ref No. 1[126] for a meta-analysis of the

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[116] Frossard & Pastor, *supra*, note 110.
[117] Dulce M. Cruz-Santamaría, et al., *Update on Pathogenesis and Clinical Management of Acute Pancreatitis*, 3 World J. of Gastrointestinal Pathophysiology 60 (2012), filed as "Pet. Ex. 49 Ref No. 1."
[118] Nyström, *supra*, note 98.
[119] Vikesh K. Singh, et al., *Early Systemic Inflammatory Response Syndrome is Associated with Severe Acute Pancreatitis*, 7 Clinical Gastroenterology and Hepatology 1247 (2009), filed as "Pet. Ex. 49 Ref No. 4."
[120] Terumi Kamisawa, et al., *Lacrimal Gland Function in Autoimmune Pancreatitis*, 48. Internal Med. 939 (2009), filed as "Pet. Ex. 49 Ref No. 2."
[121] Bechien U. Wu & Peter A. Banks, *Clinical Management of Patients with Acute Pancreatitis*, 144 Gastroenterology 1272 (2013), filed as "Pet. Ex. 47 Ref No. 13."
[122] Paraskevi A. Vlachou, et al., *IgG4-Related Sclerosing Disease: Autoimmune Pancreatitis and Extrapancreatic Manifestations*, 31 RadioGraphics 1379 (2011), filed as "Pet. Ex. 49 Ref No. 5."
[123] Finsterer, *supra*, note 84.
[124] Finsterer, *supra*, note 84.
[125] Lotte Sahin Levison, *Guillain-Barré Syndrome Following Influenza Vaccination: A 15-Year Nationwide Population-Based Case-Control Study*, 29. Eur. J. of Neurology 3389 (2022), filed as "Pet. Ex. 54 Ref No. 3."
[126] Arias, et al., *supra*, note 43.

risk of seasonal GBS between 1981 and 2014, which affirms an association between influenza vaccines and GBS. In this case, petitioner's medical records do not support an influenza infection, ruling it out with a negative Sofia test. Pet. Ex. 54 at 1. No gastrointestinal virus was identified either. Petitioner's abdominal pain and diarrhea were linked to his acute pancreatitis with both resolving when the pancreatitis resolved. Petitioner's pancreatitis was not caused by a virus but rather chronic inflammation of the gallbladder and cystic duct. Pet. Ex. 54 at 4.

Dr. Kinsbourne disagreed that petitioner's GBS was caused by viral or bacterial infections in the one to three weeks before onset. Pet. Ex. 47 at 2-3; Pet. Ex. 48 at 1. He added that Dr. Jamieson's "robust" list of triggers for GBS excluded flu vaccination and failed to explain how any of those triggers could be the cause of GBS in this case. "Certainly, [petitioner's] treating physicians did not trot out any of these allegedly robust 'known triggers'; they attributed the GBS to the closely antecedent influenza vaccination." Pet. Ex. 53 at 2.

Petitioner tested negative for influenza A/B and did not have upper respiratory symptoms of cough, problems breathing, or a gastrointestinal infection. His elevated inflammatory markers were due to acute pancreatitis which included diarrhea as part of the inflammatory process. Pet. Ex. 47 at 3; Pet. Ex. 7 at 22, 2; Pet. Ex. 48 at 2. Dr. Kinsbourne described a "flu-like illness" as an "illness that could clinically be mistaken for influenza." Pet. Ex. 47 at 4. *Verheij, et al*. defined flu-like symptoms as the "sudden onset of self-reported fever, with at least one respiratory symptom (cough, sore throat, or running or congested nose) and one systemic symptom (headache, muscle ache, sweats or chills, or tiredness) with a symptom duration of ≤72 hours during a seasonal flu epidemic." *Id*.; Pet. Ex. 47 Ref No. 11;[127] Pet. Ex. 48 at 2. Here, the Texas Medical Clinic diagnosis of "flu like" illness was "patently false" with no record of any such symptoms around the time of onset of petitioner's acute pancreatitis. Pet. Ex. 47 at 4; Pet. Ex. 48 at 1. Dr. Kinsbourne argued that petitioner's fever of 102°, chills, and night sweats were manifestations of the inflammatory factors associated with his acute pancreatitis that "spilled to extra-pancreatic locations" and resolved when the acute pancreatitis resolved two days later. Pet. Ex. 47 at 4; Pet. Ex. 7 at 12. "Contrary to notations in the medical records, [petitioner] did not have a flu-like illness at that time." Pet. Ex. 47 at 4.

Dr. Kinsbourne disagreed with Dr. Tompkins' opinions regarding the immunology but deferred to Dr. Akbari. But given that Dr. Tompkins did not deny that studies showed an increased rate of GBS following the 1976 U.S. flu vaccination campaign, Dr. Kinsbourne questioned what changed about the vaccine or the studies to make Dr. Tompkins prefer more contemporaneous studies. Pet. Ex. 47 at 8-9; Pet. Ex. 54 at 1; Pet. Ex. 48 at 4. Dr. Kinsbourne disagreed that epidemiologic studies are sufficiently powered to detect rare events. Pet. Ex. 47 at 8-9; Pet. Ex. 48 at 4. He added that Dr. Tompkins seemed to discount that the VICP included GBS as a known consequence of the flu vaccine and a Table injury. Pet. Ex. 48 at 4. He disagreed that the accuracy of the Influenza A/B tests was questionable citing to *Ryu, et al.*, which published a 94.5% sensitivity level for influenza A and 91.7% for influenza B on testing. Pet. Ex. 47 at 10; Pet. Ex. 47 Ref No. 6.[128] He disagreed that petitioner actually suffered from a flu infection and that the testing could have had a false negative result. *Id*.

[127] Verheij, et al., *supra*, note 95.
[128] Ryu, et al., *supra*, note 102.

Dr. Kinsbourne relied on *Schonberger, et al*. to show that, while the highest incidence of GBS was within the second and third week following flu vaccination, there were cases with onset between zero and one day, with the greater number between two and three days. Pet. Ex. 45 at 5. *See generally* Pet. Ex. 45 Ref No. 5.[129] Dr. Kinsbourne cited *Park, et al.* which showed that, of 48 GBS patients studied, more than half had onset within two days post-vaccination. Pet. Ex. 45 at 5; Pet. Ex. 45 Ref No. 4 at 5.[130] Petitioner's onset was 2.5 days after flu vaccination. Pet. Ex. 45 at 4.

In sum, Dr. Kinsbourne referred to respondent's proposed alternative causation theories as based upon a fictional and unspecified viral infection cobbled together from a report of non-specific inflammatory symptoms with no evidence in the medical record. This unspecified infection is then identified by respondent as ushering in petitioner's pancreatitis which in turn caused his GBS. Pet. Ex. 47 at 11; Pet. Ex. 53 at 1; Pet. Ex. 54 at 1. There was no evidence of infection in the risk period for petitioner's GBS and no evidence of alternative cause for his GBS. The flu vaccine was the only viable cause of the GBS in evidence. Pet. Ex. 47 at 11; Pet. Ex. 48 at 5. Petitioner's treating physicians diagnosed him with GBS "following vaccination" with no other diagnoses offered. Pet. Ex. 53 at 3; Pet. Ex. 11 at 2, 12, 14, 24, 150, 172, 175, 181, 192, 200. "There was no expression of uncertainty about vaccination as being the trigger. There was no search for any other trigger." Pet. Ex. 53 at 2.

Dr. Kinsbourne concluded that petitioner developed GBS 2.5 days after receipt of a flu vaccination consistent with "empirical evidence in the medical literature on GBS." Pet. Ex. 45 at 8. Acute pancreatitis has not been shown to be a trigger for GBS and there was no evidence of viral infection in this case that could have done so. *Id*. Flu vaccination is an accepted cause or trigger of GBS. There is no viable alternative in the evidence but the flu vaccine as the cause of petitioner's GBS. *Id*.

Next, Dr. Akbari described how the immune system functions in maintaining homeostasis while mounting an immunological memory response and how that same process can promote autoimmunity by aberrant response to a challenge, whether vaccination, infection, or another environmental trigger. Pet. Ex. 43 at 20. Dr. Akbari explained that, upon receipt of a flu vaccine, complex molecules called inflammasomes activate the production of cytokines, including those in the interleukin family which are potent stimulators for adaptive immune response. Pet. Ex. 43 at 5; Pet. Ex. 46 at 1-5; Pet. Ex. 46 Ref No. 1;[131] Pet. Ex. 46 Ref No. 2;[132] Pet. Ex. 46 Ref No. 3;[133] Pet. Ex. 46 Ref No. 4;[134] Pet. Ex. 46 at 5-7; Pet. Ex. 48 at 12-14; Pet. Ex. 48 Ref No. 14;[135] Pet. Ex. 48 Ref No. 15;[136] Pet. Ex. 48 Ref No. 16;[137] Pet. Ex. 48 Ref No. 17;[138] Pet. Ex. 48 Ref No. 18.[139] The activation of cytokines, particularly ILs, can occur quickly, within hours to days of

[129] Schonberger, et al., *supra*, note 26.
[130] Park, et al., *supra*, note 27.
[131] Crooke, et al., *supra*, note 63.
[132] Wan, et al., *supra*, note 64.
[133] Yang, et al., *supra*, note 65.
[134] Hartenian & Broz, *supra*, note 66.
[135] Tweedell, et al., *supra*, note 67.
[136] Christian, et al., *supra*, note 68.
[137] Tang, et al., *supra*, note 69.
[138] Man & Kanneganti, *supra*, note 70.
[139] Won, et al., *supra*, note 71.

vaccination. Cytokines work in combination with the tumor necrosis factor, or TNF-a, to experimentally induce demyelination, fever, and increased lassitude providing strong evidence for the role these cytokines play in the production of Th17 cells. Pet. Ex. 46 at 13-14, 24-25; Pet. Ex. 48 at 20-21, 23; Pet. Ex. 52 at 1-2. Both flu infection and flu vaccination can produce demyelinating inflammasomes and cytokines. Pet. Ex. 46 at 6; Pet. Ex. 48 at 14. There are often notably increased levels of Th17 cells in patients with peripheral and central demyelinating disease. Pet. Ex. 46 at 6; Pet. Ex. 46 Ref No. 8;[140] Pet. Ex. 48 at 14-15.

According to Dr. Akbari, highly activated T cells can enter the nervous system within hours after being in the periphery, exerting effector function on the nervous system by secreting proinflammatory cytokines and interleukins. This process likely resulted in the symptoms petitioner described in the days following his flu vaccination. Pet. Ex. 46 at 25. Petitioner already had activated ILs from pancreatitis and diarrhea prior to his receipt of the flu vaccination. The addition of the flu vaccination to a system already in an inflammatory state resulted in a quicker onset and was a substantial factor in petitioner's development of GBS. Pet. Ex. 46 at 13-14, 24-25; Pet. Ex. 48 at 20-21, 23; Pet. Ex. 52 at 1-2. Dr. Akbari noted that no scientific data exists to support a correlation between pancreatitis and GBS. Pet. Ex. 46 at 14. But studies have reported GBS within two to three days of immunization. Pet. Ex. 46 at 14; Pet. Ex. 48 at 1-6 (detailing each article showing onset of two to three days for GBS); Pet. Ex. 43 Ref No. 12;[141] Pet. Ex. 43 Ref No. 23;[142] Pet. Ex. 43 Ref No. 24;[143] Pet. Ex. 43 Ref No. 25.[144]

Further, *Schonberger* reported several cases in which the onset of GBS was within two days of vaccination, blurring the distinction between three and two days as an acceptable timeframe for onset. Dr. Akbari added that Dr. Jamieson dismissed the occurrence of GBS in 54.2% of patients in the *Park* report within 48 hours yet provided no robust evidence to refute these findings. Pet. Ex. 52 at 3. "Taken together all these data indicate that a recall response to H1N1 influenza and/or Influenza A antigens that cross-reacted with myelin can occur within a time interval of 54 hours or earlier from the flu immunization." Pet. Ex. 43 at 19-20; Pet. Ex. 46 at 25. Dr. Akbari agreed that the onset of symptoms "56-58 hours post-vaccination[] is atypical but consistent with established literature particularly regarding fast acting immune cells, GBS and flu vaccine." Pet. Ex. 52 at 19. Therefore, "[s]cience cannot definitively rule out that the flu vaccine administered to [petitioner] was not a substantial causal factor in the induction of demyelinating disease." Pet. Ex. 48 at 21. Within a reasonable degree of medical probability, had petitioner not received the flu vaccine, he would not have developed GBS. Pet. Ex. 43 at 20.

And in *Park,* GBS occurred within 48 hours in 54.2% of the patients studied, a study which Dr. Jamieson dismissed. Pet. Ex. 52 at 3; Pet. Ex. 48 Ref No. 3 at 1, 3.[145] According to Dr. Akbari, when all the data is taken together recall response to H1N1 influenza and/or influenza A antigens that cross react with myelin can occur within a time interval of 54 hours or earlier following flu vaccination. Pet. Ex. 43 at 19-20; Pet Ex. 46 at 25. Agreeing that onset of symptoms in 56-58 hours

[140] Mei, et al., *supra*, note 76.
[141] José Francisco Zambrano-Zaragoza, *Th17 Cells in Autoimmune and Infectious Diseases*, 2014 Int'l J. of Inflammation (2014), filed as "Pet. Ex. 43 Ref No. 12."
[142] Park, et al., *supra*, note 27.
[143] Takeda, et al., *supra*, note 73.
[144] Khoruts, et al., *supra*, note 72.
[145] Park, et al., *supra*, note 27.

post-vaccination is atypical, Dr. Akbari argued that it is consistent with established literature, particularly regarding fast-acting immune cells, GBS, and the flu vaccine. Pet. Ex. 52 at 19. He added that immunology is complex. Because "nothing in science is ever proven," the assertion that the flu vaccine caused petitioner's GBS by a preponderance of the evidence meets scientific rigor. Pet. Ex. 48 at 22. "Science cannot definitively rule out that the flu vaccine administered to [petitioner] was not a substantial causal factor in the induction of demyelinating disease." *Id*. at 21.

ii. Respondent's Experts

Dr. Tompkins summarized petitioner's medical record and concluded that he suffered from respiratory and gastrointestinal infections, both known to cause GBS, in the two to three weeks prior to the onset of GBS. Therefore, the flu vaccine was not a factor in the development of petitioner's GBS. Resp. Ex. B-1 at 10.

Dr. Tompkins then addressed each study on which petitioner relied in support of onset in fewer than three days, claiming that once the confounders were removed, so was the risk. Resp. Ex. B-1 at 4. *Schonberger* studied the 1976 swine flu vaccination but failed to consider prior infection, despite the peak attack rate for GBS occurring in December 1967. This study established the risk window for GBS as seven to 21 days following the antecedent event. Resp. Ex. B-1 at 4; Pet. Ex. 45 Ref No. 5;[146] Pet. Ex. 43 at 4, 11. *Polakowski* noted a slight increase in risk, with the greatest risk observed eight to 21 days after vaccination but did not account for infections within six weeks prior to GBS onset. Resp. Ex. B-1 at 4; Pet. Ex. 43 Ref No. 20 at 8-9.[147] *Perez-Vilar* did not detect an increase in GBS in the one-to-42- or the eight-to-21-day risk windows.[148] Resp. Ex. B-1 at 4-5; Pet. Ex. 43 Ref No. 21 at 1.[149] *Salmon* noted a slight insignificant risk of GBS following H1N1 vaccine with confounding effects of respiratory infection prior to onset. Resp. Ex. B-1 at 5; Pet. Ex. 43 Ref No. 22 at 5, 6.[150] Therefore, the publications relied upon by petitioner actually provided strong evidence for a risk window starting one week after the antecedent event. Any observations of onset sooner had confounding factors in the studies that weakened any conclusions regarding the risk from vaccination or early onset. Resp. Ex. B-1 at 5; Resp. Ex. D-2 at 7-13.

Dr. Tompkins later agreed that *Salmon* and *Polakowski* reported two to three cases of GBS within two days of receipt of the flu vaccine. However, "[b]eyond the absence of any statistical support for these cases to be significant, the authors and Dr. Akbari cannot demonstrate that these individual cases were causally associated with influenza vaccination. Rather, they were diagnosed with GBS following receipt of the 2009 H1N1 vaccine." Resp. Ex. G at 4-5; Resp. Ex. G-22;[151] Pet. Ex. 43 Ref No. 20.[152] Dr. Tompkins further criticized *Park* as having several problems

[146] Schonberger, et al., *supra*, note 26.
[147] Laura L. Polakowski, *Chart-Confirmed Guillain-Barré Syndrome After 2009 H1N1 Influenza Vaccination Among the Medicare Population, 2009-2010*, 178 Am. J. Epidemiology 962 (2013), filed as "Pet. Ex. 43 Ref No. 20."
[148] Dr. Akbari incorrectly attributed these findings to *Park.*
[149] Silvia Perez-Vilar, et al., *Guillain-Barré Syndrome After High-Dose Influenza Vaccine Administration in the United States, 2018-2019 Season*, 223 J. of Infectious Diseases 416 (2021), filed as "Pet. Ex. 43 Ref No. 21."
[150] Salmon, et al., *supra*, note 42.
[151] Daniel A. Salmon, et al., *Guillain-Barré Syndrome Following Influenza Vaccines Affords Opportunity to Improve Vaccine Confidence*, 223 J. of Infectious Diseases 355 (2021), filed as "Resp. Ex. G-22."
[152] Polakowski, et al., *supra*, note 147.

undermining the impact of the findings, including selection and data biases. *Park* used only individuals who received compensation for vaccine-related injuries, with more than a third of the participants not meeting the Brighton Criteria for GBS, and with onset of less than three days in children compared to a much longer period in adults with no explanation for the differences. "[T]he fact that older individuals had a much greater interval between vaccination and symptom onset reduces the suggested importance of this observation." Resp. Ex. G at 5; Pet. Ex. 48 Ref No. 3.[153] Further, studies by *Wise, Kwong*, and *Marks* showed onset within the first week following vaccination using data collected from the 2009 H1N1 pandemic vaccination campaign, the 1976 H1N1 vaccination campaign, or the Korea National Vaccine Injury Compensation Program from the 2009 pandemic, providing little reliable evidence for the two-day onset in this case. Resp. Ex. G at 5; Pet. Ex. 48 Ref No. 5;[154] Pet. Ex. 48 Ref No. 6;[155] Pet. Ex. 48 Ref No. 7.[156]

Dr. Tompkins concluded that while the studies presented by petitioner may offer new insights for potential mechanisms for autoimmune disease, they fail to provide a clear mechanism connecting the current inactivated flu vaccination with GBS. Resp. Ex. G at 5. Further, the studies relied upon as suggesting increased cytokine levels following inactivated or live attenuated flu vaccines either did not look at onset within two days of vaccination or tested only women with sore arms and found TNF-α, IFN- γ, IL-6, IL-8, and IL-10 to be lower in all vaccinated groups. *Id*. at 8; Pet. Ex. 48 Ref No. 17 at 3, 4.[157] *See also* Pet. Ex. 48 Ref No. 1 at 11.[158]

Finsterer discussed triggers for GBS including six bacteria, 13 viruses, and five vaccines that range from highly prevalent to rarely associated with GBS. *Id*.; Resp. Ex. G-1.[159] *Finsterer* noted that while GBS has been causally related to several vaccinations, other studies do not confirm this. Resp. Ex. G at 2. Further, 30% of GI and respiratory infections become asymptomatic prior to the onset of GBS. Dr. Tompkins concluded that infection is the most common trigger for GBS and that infection alone was sufficient to trigger the majority of GBS cases. *Id*.

Dr. Tompkins maintained that the repeated viral infections described in petitioner's medical records "are not only the most likely triggers for [petitioner's] GBS, they are also individually sufficient to trigger GBS." Resp. Ex. G at 3.

Dr. Jamieson also opined that in the weeks prior to the onset of his GBS, petitioner suffered from "multiple systemic viral and bacterial infections, including a flu-like illness, gastroenteritis, pancreatitis, bacteriuria, and cholecystitis" which were more likely the triggering event for his GBS than the flu vaccination. Resp. Ex. A-2 at 12; Pet. Ex. 7 at 1-5. She referenced petitioner's

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[153] Park, et al., *supra*, note 27.
[154] Matthew E. Wise, et al., *Guillain-Barré Syndrome During the 2009-2010 H1N1 Influenza Vaccination Campaign: Population-Based Surveillance Among 45 Million Americans*, 175 Prac. of Epidemiology 1110 (2012), filed as "Pet. Ex. 48 Ref No. 5."
[155] Jeffrey C. Kwong, et al., *Risk of Guillain-Barré Syndrome After Seasonal Influenza Vaccination and Influenza Health-Care Encounters: A Self-Controlled Study*, 13 Lancet Infectious Diseases 769 (2013), filed as "Pet. Ex. 48 Ref No. 6."
[156] James S. Marks & Thomas J. Halpin, *Guillain-Barré Syndrome in Recipients of A/New Jersey Influenza Vaccine*, 243 JAMA 2490 (1980), filed as "Pet. Ex. 48 Ref No. 7."
[157] Man & Kanneganti, *supra*, note 70.
[158] Salmon, et al., *supra*, note 42.
[159] Finsterer, *supra*, note 84.

medical records which documented "flu-like illness," acute pancreatitis possibly related to viral infection, and resolved gastroenteritis. Resp. Ex. A-2 at 12; Pet. Ex. 7 at 1-5; Pet. Ex. 20 at 197-199; Pet. Ex. 20 at 2-3. She added that, two months later, petitioner was treated with antibiotics for acute and chronic acalculous hemorrhagic cholecystitis. Resp. Ex. A-2 at 12; Pet. Ex. 4 at 178. Further, she argued that petitioner had AMAN particularly associated with gastrointestinal infections as the triggers. "His multiple preceding infections, including gastrointestinal infections, are a more usual and accepted trigger for AMAN, than is vaccination." Resp. Ex. A-2 at 12; Resp. Ex. C at 3-4.

Dr. Jamieson added that viral infections are diagnosed clinically without specific viral pathogen "so the absence of a specific identified virus by a an [sic] assay or culture is not dispositive." *Id*. at 3. Moreover, the presence of inflammation did not preclude viral infection since inflammation is often triggered by infection with multiple types of pathogens. *Id*. Further, since only two-thirds of patients with GBS report an antecedent event, leaving one-third developing GBS without any antecedent event, "[p]utting aside the copious evidence of [petitioner's] antecedent infections, Dr. Kinsbourne's argument is belied by the accepted truth about infections preceding GBS in only approximately two thirds of cases of GBS, as well as the shortened period of time of perceived risk." *Id*. at 4.

Dr. Jamieson next asserted that Dr. Akbari's discussion on onset did not align with his references in support of a zero-to-two-day onset after the flu vaccination. Resp. Ex. C at 5-6; Resp. Ex. A-18.[160] *See also* Resp. Ex. A-19[161] (finding no increase of GBS in the first six days post-vaccination); Pet. Ex. 43 Ref No. 20[162] (analyzing data from Medicare for the 2009 monovalent H1N1 influenza vaccine of those admitted to the hospital with GBS in the 126-day period post-vaccination, but not providing information about early risk after vaccination with seasonal vaccination); Resp. Ex. G-21[163] (performing a claims-based self-controlled risk interval analysis and finding no statistically significant increased risk for GBS in the one-to-42-day risk windows). She added that studies cited by petitioner were inapposite. The *Schonberger* study studied swine flu, while the *Park, et al*. study did not have normative data for comparison and could not be replicated by other papers including *Schonberger*. Resp. Ex. A-2 at 12; Resp. Ex. A-17;[164] Resp. Ex. A-18.[165] She argued that simply because ranges of onset include outliers occurring on either end of a bell-shaped curve, this does not suggest that inclusion of every time point on either end of the range automatically validates causation at all timeframes. Resp. Ex. C at 3. Dr. Jamieson concluded that none of these studies prove that the flu vaccine can cause GBS in under three days. Resp. Ex. C at 6; Resp. Ex. F at 3.

Dr. Jamieson did not find Dr. Akbari's conclusions about immune system stimulation following vaccination a persuasive and credible medical theory causally linking flu vaccination with the development of peripheral neuropathy and GBS in petitioner. The short timeframe between petitioner's vaccination and the multitude of other, more plausible triggers for his

[160] Park, et al., *supra*, note 27.
[161] Salmon, et al., *supra*, note 42.
[162] Polakowski, *supra*, note 147.
[163] Silvia Perez-Vilar, et al., *Surveillance for Guillain-Barré Syndrome After Influenza Vaccination Among U.S. Medicare Beneficiaries During the 2017-2018 Season*, 37 Vaccine 3856 (2019), filed as "Resp. Ex. G-21."
[164] *Id*.
[165] Park, et al., *supra*, note 27.

GBS/AMAN preclude finding that causal link.

3. Discussion

Petitioner's medical records provide no indication that he suffered from any health problems or from alcohol abuse prior to October 2016. However, his records during the month of October 2016 reflect several weeks of unfortunate medical events beginning on October 4, 2016. Petitioner experienced a host of symptoms ultimately determined to be acute pancreatitis, the onset of diabetes requiring medication, the need for cardiac catheterization, and ultimately gallstones with a blockage requiring surgery in December. Pet. Ex. 45 at 1-2; Pet. Ex. 53 at 2; Pet. Ex. 20 at 15; Pet. Ex. 2 at 45-46; Pet. Ex. 20 at 448-49; Pet. Ex. 4 at 5-6. On the day he received the flu vaccination, petitioner was finally feeling well, had no complaints, and had normal strength with sensory intact on examination. However, his blood work on that date revealed elevated ILs and other inflammatory markers at the time he received the subject flu vaccination meant to activate the immune system.

Case law is clear that a petitioner sustains their burden by demonstrating that the vaccination was a "substantial factor" and a "but for" cause of the injury, even if the vaccination was not the sole cause. *Pafford*, 451 F.3d at 1355; *Shyface*, 165 F.3d at 1352. In *Shyface,* petitioner Cheyenne Shyface was vaccinated with whole-cell DPT at the time he was developing an E. coli infection. Both the vaccine and the E. coli infection could and did cause a fever, which rose to 110°, resulting in his death four days later. *Shyface*, 165 F.3d. at 1345. Respondent defended the case arguing that the E. coli infection was the cause of Cheyenne's fever and death. Cheyenne's treating physician testified that both the vaccine and the infection were equally responsible for his fever and death. The Federal Circuit held that each of the two triggers, the vaccine and the infection, were substantial factors in causing Cheyenne's very high fever and death. But for the vaccination, Cheyenne would not have had the high fever and would not have died. Because it was not possible to determine whether the vaccine or the infection was the predominant factor, the Federal Circuit found that both were substantial factors in bringing about Cheyenne's death. *Shyface*, 165 F.3d at 1352-53.

Special masters have since found that where there is a concurrent infection with vaccination, both are substantial factors in bringing about the injury alleged. *Herkert v. Sec'y of Health & Human Servs.*, No. 97-518V, 2000 WL 141263, at *12 (Fed. Cl. Spec. Mstr. Jan. 19, 2000) (finding petitioner proved the DTaP vaccine and cytomegalovirus infection were both substantial factors in causing his son's transverse myelitis); *Nash v. Sec'y of Health & Human Servs.,* No. 00-149V, 2002 WL 1906501, at *19 (Fed. Cl. Spec. Mstr. June 27, 2002) (finding both meningitis infection and DPT vaccination were substantial factors in causing the child's condition). I have found that concurrent factors of infection and vaccination are both substantial factors in the resulting injuries or death. *See, e.g., Lehrman v. Sec'y of Health & Human Servs.,* No. 13–901, 2018 WL 1788477, at *19 (Fed. Cl. Spec. Mstr. Mar. 19, 2018); *Matten v. Sec'y of Health & Human Servs.,* No. 12-155V, 2021 WL 5768148, at *39 (Fed. Cl. Spec. Mstr. Nov. 2, 2021). In *Lehrman*, I also found that petitioner's GBS onset was in fewer than three days post-vaccination because of the already inflammatory state of petitioner's immune system at the time he received the flu vaccine. Respondent's expert in *Lehrman* agreed that under those conditions a shorter onset period was feasible. 2018 WL 1788477 at *18.

Here, petitioner's inflammatory markers were elevated resulting from acute pancreatitis and other conditions in the days and weeks prior to the subject vaccination and on the day of the vaccination. Therefore, the flu vaccination was administered to a petitioner whose immune system was already in an inflammatory state. This would account for the onset of GBS within 54 to 58 hours post-vaccination, rather than the typically expected three days. Based on the foregoing, I find that petitioner's flu vaccination was a substantial factor in the development of his GBS. I also find that petitioner's systemic inflammatory condition at the time he received the flu vaccine is the reason he suffered an onset of GBS within 54 to 58 hours of vaccination. Accordingly, I find that petitioner has provided preponderant evidence to satisfy Prongs II and III.

C. Burden Shifting: Respondent Must Show an Alternative Cause of Injury.

A petitioner who satisfies all three prongs of the Althen test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Human Servs.*, 98 Fed. Cl. 719, 726 (2011). Consequently, the burden now shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *de Bazan*, 539 F.3d at 1354; see also *Hammitt,* 98 Fed. Cl. at 726 (explaining that respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "does not include any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness, or condition.'" § 13(a)(2). *See also Doe/11 v. Sec'y of Health & Human Servs*., 83 Fed. Cl. 157 (2008) (holding that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

As detailed above, petitioner was ultimately diagnosed with acute pancreatitis and diabetes and underwent cardiac catheterization in the three weeks prior to his receipt of the flu vaccine. Dr. Kinsbourne showed that the symptoms with which petitioner presented on October 4 and 9, 2016, were symptoms of acute pancreatitis resulting in the elevation of various inflammatory markers on petitioner's blood work. Respondent's experts unpersuasively argued that petitioner suffered from upper respiratory and gastrointestinal infections and that those infections were the cause of his GBS. They did not dispute that petitioner's blood work on the day of the flu vaccination showed continued elevated inflammation. However, they maintained that petitioner's medical issues two to three weeks prior to his receipt of the vaccination were within a medically appropriate timeframe for the development of GBS and were the sole cause of his GBS. Regardless, as detailed above, I have found that petitioner's receipt of the flu vaccine, when combined with his already inflammatory state, as evidenced by the blood work performed that day, resulting from the inflammatory medical conditions from which he suffered in the two to three weeks prior to his vaccination, was a substantial factor in bringing about his GBS. Thus, respondent cannot sustain his burden in proving that any of petitioner's prior medical conditions were the sole substantial factor in causing his GBS.

VI. Conclusion

Petitioner has put forth preponderant evidence that the influenza vaccine that he received on October 21, 2016, contributed to and was a substantial factor in the development of his Guillain-Barré Syndrome. Therefore, petitioner has demonstrated entitlement to compensation. This case

shall proceed to the damages phase.

IT IS SO ORDERED.

s/ Mindy Michaels Roth
Mindy Michaels Roth
Special Master