In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

E-Filed: July 8, 2011

EARL L. STEWART, Petitioner, v. SECRETARY OF THE DEPARTMENT OF HEALTH AND HUMAN SERVICES, Respondent.	TO BE PUBLISHED **Case No. 06-777** Pre-Existing Neuropathy; Influenza Vaccine; Intercurrent Infection; Guillain-Barré Syndrome

Ronald C. Homer, Boston, MA, for petitioner.

Julia W. McInerny, Washington, DC, for respondent.

PUBLISHED RULING ON ENTITLEMENT[1]

Campbell-Smith, Chief Special Master

On November 16, 2006, petitioner, Earl L. Stewart, filed a petition seeking compensation under the National Vaccine Injury Compensation Program[2] (the Vaccine

[1] Because this decision contains a reasoned explanation for the undersigned's action in this case, the undersigned intends to post this decision on the United States Court of Federal Claims' website, in accordance with the E-Government Act of 2002, Pub. L. No. 107-347, 116 Stat. 2899, 2913 (Dec. 17, 2002). As provided by Vaccine Rule 18(b), each party has 14 days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the entire decision will be available to the public. Id.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3758, codified as amended, 42 U.S.C. § 300aa-10 through § 300aa-34 (2006) (Vaccine Act or

Program or the Act). Petitioner claims that he suffered Guillain-Barré Syndrome (GBS) as a consequence of a November 20, 2003 trivalent influenza vaccination. Pet. for Vaccine Comp. 1-2, Nov. 16, 2006.

Complicating petitioner's claim were three factual issues. First, petitioner's prior medical history was notable for his diabetic neuropathy in the same limbs later affected by his GBS. Pet. Ex. 4 at 14.[3] Second, at the time of petitioner's hospitalization for weakness in his extremities, he reported having had a "cough" two weeks after his receipt of the flu vaccine and two weeks prior to his hospital admission. Id. Third, petitioner tested positive for pneumonia shortly after his hospital admission. Id. The significance of these factual issues was a source of considerable contention between the parties.

In support of their respective positions on petitioner's claim for vaccine compensation, the parties presented medical testimony. Paul Willis, M.D., petitioner's treating neurologist during his hospitalization, testified on petitioner's behalf. Derek Smith, M.D., a neuroimmunologist, also testified for petitioner. Thomas Leist, M.D., a neuroimmunologist, testified for respondent.

Having carefully considered the record as a whole, the undersigned concludes that petitioner is entitled to compensation.

I. BACKGROUND

A. Procedural Development of this Claim

After the filing of petitioner's claim in November 2006, petitioner continued to develop the factual record in this case and began settlement discussions with respondent. The death of petitioner's counsel in 2009 halted discussions for several months. Upon the substitution of current counsel as petitioner's counsel of record, settlement negotiations resumed. Petitioner continued to develop the factual record of the case as well. The parties also filed expert reports. Settlement discussions ultimately proved unsuccessful, and an entitlement hearing was scheduled.

In anticipation of the entitlement hearing, petitioner filed a motion to narrow the issues to be addressed at hearing. See Pet'r's Mot. to Narrow, Apr. 27, 2010. Asserting that the doctrine of collateral estoppel has applicability in Vaccine Program proceedings, petitioner urged the court to hear evidence pertaining only to prong two of the standard

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the Act). All citations in this decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

[3] The page numbering in the citations to Petitioner's Exhibits 1-4 reflects the continuous pagination contained in those exhibits.

articulated in Althen v. Sec'y of Health and Human Servs., 418 F.3d 1274, 1278 (Fed. Cir. 2005). Prong two examines whether the vaccine claim involves a logical sequence of cause and effect. Id. This analytical prong of Althen has also been referred to as the "reason for the injury" test. Pafford v. Sec'y of Health and Human Servs., 451 F.3d 1352, 1356 (Fed. Cir. 2006). In moving to limit the issues at hearing to the second prong of Althen, petitioner asserted that prong one of the Althen inquiry--which focuses on whether a biologically plausible medical theory exists that causally connects the received vaccination to the sustained injury--had been established in other compensated vaccine cases. Pet'r's Mot. to Narrow, 1-2. Petitioner further asserted that prong three--which considers whether the timing between the received vaccination and the onset of injury is medically appropriate--had been established as a matter of fact in this case. Id.

Respondent opposed the motion to narrow the issues. Respondent argued that: (1) the Secretary "has never conceded the proposition that the flu vaccine can cause GBS," the prong one Althen inquiry; (2) petitioner does not satisfy the Althen prong three timing requirement by establishing a date of onset, without more; and (3) in the case of United States v. Mendoza, 464 U.S. 154, 162 (1984), the Supreme Court determined that nonmutual offensive collateral estoppel "'does not apply against the government.'" Resp't's Pre-Hearing Mem. and Resp. to Pet'r's Mot. to Narrow at 20-21, May 12, 2010.

By order dated June 15, 2011, the undersigned denied petitioner's motion to narrow the issues for hearing. See Ruling Denying Pet.'s Mot. to Narrow, June 15, 2011.

In May 2010, the undersigned heard testimony from petitioner, petitioner's treating neurologist, and the parties' respective experts. The parties submitted post-hearing briefing. Petitioner's claim is now ripe for a ruling.

B. Factual Background

Dr. Stewart is an accomplished composer, author and scholar of African-derived musical sounds. Tr. Vol. II, 58, May 19, 2010 (Tr. II). A former Fulbright Scholar,[4] he is an Associate Professor in the Department of Black Studies at the University of California, Santa Barbara. The Department of Black Studies, Univ. of Cal., Santa Barbara (June 27, 2011), http://www.blackstudies.ucsb.edu/people/bios/earl_stewart.html. He teaches a variety of music courses as well as music theory. Tr. II at 58-59.

Dr. Stewart's medical records show that prior to his receipt of the trivalent influenza vaccination in November 2003, he had a number of health problems, including

[4] As a Fulbright Scholar, he served as the composer and conductor in residence with the National Symphony Orchestra of Ghana in Accra, Ghana from 1992-1993. Tr. II at 59-60.

a history of obesity, uncontrolled hypertension, poorly controlled diabetes mellitus, hyperlipidemia (excess fat in the blood), coronary heart disease, basal ganglia infarct, and peripheral neuropathy. Pet. Ex. 4 at 14; Resp. Ex. D at 6466-70, 6489-92, 6527, 6606-08, 6610; see also Tr. II at 60.

On October 7, 2003, Dr. Stewart saw Rosanna Petronella, P.A., for insulin treatments. Resp. Ex. D at 6609-11. The examining physician's assistant noted that he continued to suffer from a "microvascular/neuropathy." Id. at 6609.

Nearly six weeks later, on November 20, 2003, Dr. Stewart received a trivalent influenza vaccination. Pet. Ex. 2 at 1.

On December 16, 2003, more than three weeks after he received the flu vaccination, Dr. Stewart awakened with "wobbly knees." Tr. II at 61. He also had a problem with his balance. Id. Concerned about how he was feeling, Dr. Stewart called a work colleague, who was also a close friend, to request a ride to the doctor's office. Id. Escorted by his friend to the doctor's office "close to 4:00" in the afternoon, Dr. Stewart was able to walk unassisted into the office. Tr. II at 61-62.

At the doctor's office, a physician's assistant evaluated Dr. Stewart. Pet. Ex. 3 at 2. On examination, Dr. Stewart's speech seemed slurred. Tr. II at 62. The physician's assistant noted:

> [Symptoms] [for] 1-2 days. 53 [year-old] [male] weak, difficulty walking, tingling hands, feet. [Complains of] 'off balance,' disequilibrium, 'tightness' [in] epigastric region, difficulty swallowing, disoriented. Paresthesias [in] hands [and] feet. . . . Ataxia, looks fatigued [and] disoriented, not alert to time of day. . . . Neuro - able to do finger to nose [and] rapid hand [movement], unable to stand for Romberg's, [abnormal] heel to toe. . . . [Assessment]: Altered mental status/disorientation. ? brain stem infarct vs. other. . . .

Pet. Ex. 3 at 2. At the insistence of the physician's assistant, Dr. Stewart was given a wheelchair and directed to go to the emergency room down the street. Tr. II at 62. His accompanying friend wheeled him from the doctor's office to the emergency room entrance. Id.

In the emergency room, Dr. Stewart was unable to climb onto the gurney. Id. Feeling very weak and concerned that he might be dying, Dr. Stewart directed his friend to go home. Id. Dr. Stewart was aware of his impaired speech. Id. He was losing his voice, and his hands were moving quickly. Tr. II at 63. He later learned from his attending neurologist, Dr. Paul Willis, that he had lost use of his tongue. Tr. II at 64. Dr. Stewart recalls being unable to move but being completely cognizant of everything around him. Id.

Dr. Stewart's hospital admission record contained a notation that he still had a cough from the upper respiratory infection (URI) he had ten days before his hospitalization. See Tr. II at 27-28. But he had no fever, had a normal white cell count, and showed no chest infiltrate on his x-ray, findings that indicated petitioner had no active infection when he initially presented to the emergency room. Tr. II at 29; see also Tr. II at 37-40.

An internal medicine record from the hospital indicated that Dr. Stewart had been admitted with complaints of "gait disturbance, leg weakness [and] slurred speech . . . not present the prior evening." Pet. Ex. 4 at 109-11. The initial assessment was a "Probable Acute [cerebrovascular accident]." Id. at 111.

On that same day of his hospital admission, December 16, 2003, Dr. Stewart was examined by Dr. Paul Willis. Dr. Willis became Dr. Stewart's attending neurologist. Dr. Willis noted the "gradual[] progress[ion]" of Dr. Stewart's symptoms—particularly, his slurred speech, difficulty swallowing, leg weakness and impaired coordination—over the day of his hospital admission. Pet. Ex. 4 at 9-11. Dr. Willis further noted "a recent viral illness about 10 days ago" that included "[t]hree days of [an] upper respiratory process" before medically clearing. Id. at 9. Dr. Willis observed that Dr. Stewart's initial presentation was suggestive of "a potential bulbar stroke," and his CT scan revealed evidence of the transient ischemic attack (stroke-like episode) Dr. Stewart had previously suffered. Id. The progression of Dr. Stewart's symptoms during hospitalization gave rise to a concern that Dr. Stewart may have had a progressive infarct (that is, an area of dying brain tissue)[5] in the basilar/bulbar region of his brain. Id. Dr. Stewart's MRI, however, "showed no evidence of an acute infarct in the basilar/bulbar region." Id.

Dr. Willis documented Dr. Stewart's deterioration during his clinical examination. "His ability to communicate [continued to] dissipate." Pet. Ex. 4 at 9. He exhibited "slight" drooping of his upper eyelids (a condition known as ptosis) and "[s]evere lower facial . . . weakness." Id. at 9-10. His "[g]ag [reflex] was diminished." Id. at 10. He "could . . . not protrude his tongue." Id. "Weakness evolved over [his] upper extremities with diminished grip and distal strength in the 1 to 2+ range and 2 to 3+ biceps flexion."[6]

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[5] See Dorland's Illustrated Medical Dictionary 948 (31st ed. 2007).

[6] Muscle strength is tested during a neurological examination by the patient's ability to move against the examiner's resistance. See Motor, Univ. of Fla. Coll. of Med. (June 27, 2011), http://medinfo.ufl.edu/year1/bcs/clist/neuro.html#Motor. The examiner compares the patient's muscle strength on one side to the other and then grades the patient's response on a scale from 1 to 5. Id. A description of various grades follows:

0/5 No muscle movement

Id. at 10. In addition, Dr. Stewart showed alarming signs of respiratory distress that led to his intubation.[7] Id.

Dr. Stewart's laboratory test results were also documented. On admission, he had a white cell count of 7800 and a low platelet count of 143,000. Pet. Ex. 4 at 10. His white cell count rose in context of his pneumonia and his platelets lowered to the 100,000 range.[8] Id. His electrolytes were essentially unremarkable. Id. His liver function test was normal. Id. His creatine phosphokinase (CPK) level was elevated at 361.[9] Id.

Dr. Willis recorded his impressions as follows:

> Progressive neuromuscular weakness with predominant bulbar and upper extremity findings without ocular motor paresis or significant impairment of cognitive function prior to sedation. . . . He presented initially with suggestive pattern of a bulbar infarct. This proves not to be the case. . . . Labs are notable for a slight elevation of CPK on Lipitor but without history of prior muscle pain. Concerns would be an atypical Guillain-Barr[é] Syndrome (no ocular involvement), myasthenic syndrome, botulism . . . or other post viral related

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1/5 Visible muscle movement, but no movement at the joint
2/5 Movement at the joint, but not against gravity
3/5 Movement against gravity, but not against added resistance
4/5 Movement against resistance, but less than normal
5/5 Normal strength

Id. On examination, Dr. Stewart's muscle strength was unquestionably diminished. Pet. Ex. 4 at 10.

[7] Intubation (endotracheal) is the procedure by which a tube is inserted through the mouth or nose and into the lungs. It can be used for the maintenance of an airway, for the aspiration of secretions, or for the ventilation of the lungs. Dorland's at 969, 2002.

[8] The normal white cell count for an adult is between 5,000-10,000. Mosby's Manual of Diagnostic & Lab. Tests 548, 552 (4th ed. 2010). An increase in white cell count readings is indicative of an infection. Id.

The normal platelet count for an adult falls within the range of 150,000-400,000. Id. at 416. A low platelet count is also indicative of an infection. Id. at 417.

[9] The normal range for the CPK test is within 55-170 units. Mosby's at 199. An elevated number is indicative of disease or injury affecting the heart muscle, the skeletal muscle, and the brain. Id. at 202.

neuromuscular process. He has critical issues related to autonomic instability, probable aspiration pneumonia, possible sepsis. . . . He is critically ill.

Pet. Ex. 4 at 9-11. Dr. Stewart's spinal fluid "showed lymphocytes, monocytes and a few neutrophils."[10] Pet. Ex. 4 at 28-30.

A culture taken from Dr. Stewart's endotracheal aspirate on December 18, 2003, two days after his hospitalization, tested positive for Haemophilus influenzae (H. influenzae) bacteria.[11] Tr. Vol. I, 28, May 18, 2010 (Tr. I.); Pet. Ex. 7 at 14513. The culture showed a heavy growth of bacteria, a result consistent with "a severe infection." Tr. I at 29.

On that same date, Dr. Stewart was evaluated for "[c]ritical care management of aspiration pneumonia." Pet. Ex. 4 at 14-16. The consulting physician noted that Dr. Stewart's change in functional status began approximately "one day" before his hospitalization, starting with a "loss of coordination" and "slurred speech." Id. at 14. The physician noted that Dr. Stewart "did have a flu shot." Id. At the time of the consulting physician's evaluation, Dr. Stewart was still intubuated, was on a ventilator machine, was unresponsive to "deep noxious stimulus," and was sedated. Id. at 15. The etiology of Dr. Stewart's neurological findings was "quite unclear." Id. at 16. Among the identified differential diagnoses were "stroke in evolution," "atypical Guillain-Barré syndrome," "myasthenia gravis," and "viral infection/encephalitis." Id.

Four days after his hospitalization, on December 20, 2003, Dr. Stewart began receiving plasmapheresis treatments for his possible GBS.[12] Pet. Ex. 4 at 133. He subsequently received intravenous immunoglobulin (IVIG) treatments and began to show improvement.[13] See Pet. Ex. 4 at 164.

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[10] The presence of lymphocytes, monocytes, and neutrophils in the spinal fluid are all indicative of a viral infection. Mosby's at 685.

[11] H. influenza is not a virus, but rather a type of bacteria that is a "normal inhabitant[] of the upper respiratory tract but may become [a] primary or secondary pathogen[]." It may also cause pneumonia in immunocompromised individuals. Dorland's at 827.

[12] Plasmapheresis involves the removal of serum from a patient's blood to filter out the immunologic substances suspected to be causing the injury. Tr. I at 12. Once filtered, the serum (plasma) is returned to the patient's blood. Id.; Dorland's at 1477.

[13] IVIG is another treatment intended to reduce the immunologic response suspected of causing the injury of concern. Tr. I at 12.

Dr. Stewart's two electroencephalograms[14] (EEGs) were abnormal[15] "due to the presence of . . . slowing" electrical activity in the brain that was suggestive of a mild encephalopathy.[16] See Pet. Ex. 4 at 18; Pet. Ex. 5 at 263; see also Mosby's at 573. His nerve conduction studies and electromyograph[17] (EMG) were indicative of "axonal involvement" and "demyelination," findings that were "consistent with a severe polyradiculoneuropathy"[18] and "not inconsistent with the suspected atypical Guillain Barré syndrome." Pet. Ex. 4 at 125; Pet. Ex. 5 at 272-73.

Dr. Stewart's acute hospitalization lasted three months. Tr. II at 64. His diagnosis at discharge was Guillain-Barré Syndrome. Pet. Ex. 4 at 28-30.

From the hospital, Dr. Stewart was transferred to a long-term care facility for more than a year. Tr. II at 64. Upon his discharge home, Dr. Stewart was confined to a wheel chair. Tr. II at 65. He received outpatient rehabilitative services, but remained in a wheelchair for more than two and a half years. Tr. II at 65-66.

Currently, he can walk with the assistance of a walker. Tr. II at 65. He has residual weakness on the left side of his body. Tr. II at 66. His ongoing inability to bend his hands or feet has compromised his ability to play the piano. Tr. II at 67, 69. His speech is not as clear as it was prior to his hospitalization in December of 2003, but he continues to work on the clarity of his speech through his class lectures. Tr. II at 68.

Nearly five and a half years after Dr. Stewart developed GBS, he suffered another stroke-like episode (transient ischemic attack). Tr. II at 70. As a result of this stroke event, he now has uncontrollable shaking of his left hand. Tr. II at 70-71.

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[14] An electroencephalogram measures the frequency, amplitude, and characteristics of brain waves. It is used to identify and evaluate seizures as well as to detect the presence of tumors and infections. Mosby's at 573.

[15] The first EEG was performed on December 19, 2003. Pet. Ex. 4 at 18. Another one was performed seven days later, on December 26, 2003. Pet. Ex. 5 at 263.

[16] An encephalopathy is defined as "any degenerative disease of the brain." Dorland's at 622.

[17] An electromyogram measures the electrical activity of skeletal muscle to detect muscular abnormalities caused either by muscular disorders or systemic diseases. Mosby's at 577.

[18] A polyradiculoneuropathy is defined as "any disease of the peripheral nerves and spinal nerve roots." Dorland's at 1515.

II. THE APPLICABLE LEGAL STANDARDS

To prevail on a non-Table vaccine claim such as petitioner has asserted here,[19] petitioner 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.'" Moberly v. Sec'y of Health & Human Servs., 592 F.3d 1315, 1321 (Fed. Cir. 2010) (quoting Shyface v. Sec'y of Health & Human Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)). Petitioner must prove his vaccine claim by a preponderance of the evidence. Althen, 418 F.3d at 1278.

The preponderant evidence standard under the Vaccine Act requires proof that a vaccine more likely than not caused the vaccinee's injury. Althen, 418 F.3d at 1279; see also In re Winship, 397 U.S. 358, 371–72 (1970) (Harlan, J., concurring) (quoting F. James, CIVIL PROCEDURE, 250–51 (1965)) (A preponderance of the evidence standard requires the trier of fact to "believe that the existence of a fact is more probable than its nonexistence before the [special master] may find in favor of the party who has the burden to persuade the [special master] of the fact's existence.") Mere conjecture or speculation will not establish a probability. See Snowbank Enter., Inc. v. United States, 6 Cl.Ct. 476, 486 (1984). This evidentiary standard "allows a finding of causation in a field bereft of complete and direct proof of how vaccines affect the human body." Capizzano v. Sec'y of Health & Human Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006).

Petitioner satisfies his burden of showing that the received vaccination brought about his injury by providing (1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a proximate temporal relationship between vaccination and injury. Althen, 418 F.3d at 1278.

Proof of vaccine causation must be supported by a sound and reliable "medical or scientific explanation that pertains specifically to the petitioner's case, although the explanation need only be 'legally probable, not medically or scientifically certain.'" Moberly, 592 F.3d at 1322 (quoting Knudsen v. Sec'y of Health & Human Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994)); see also Grant, 956 F.2d at 1148 (requiring that the medical theory must support actual cause). Mere temporal association is not sufficient to prove causation. Grant, 956 F.2d at 1148.

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[19] If petitioner alleges an injury listed on the Vaccine Injury Table (Table) that occurs within the correlative time frame set forth in the Table, petitioner's vaccine claim is deemed a Table claim, and a presumption of vaccine causation attaches. See §300aa-14; see also 42 CFR §100. If petitioner alleges an injury that is not listed on the Table (such as the GBS injury alleged in this case), the vaccine claim is deemed a non-Table case, and no presumption of causation attaches. Rather petitioner must satisfy his burden of proof. See §300aa-13(a)(1)(A).

A petitioner may use circumstantial evidence to prove his case, and "close calls" regarding causation must be resolved in favor of the petitioner. Althen, 418 F.3d at 1280. Causation can be supported by a treating physician's opinion that a vaccination was causally linked to the vaccinee's injury if the special master finds the opinion to be both reliable and persuasive. Moberly, 592 F.3d at 1324–25; see also Capizzano, 440 F.3d at 1326.

Should the petitioner succeed in establishing a prima facie case of causation, the burden then shifts to respondent to prove alternative causation by a preponderance of the evidence. Althen, 418 F.3d at 1278; see also de Bazan v. Sec'y of Health & Human Servs., 539 F.3d 1347, 1352 (Fed. Cir. 2008) ("So long as the petitioner has satisfied all three prongs of the Althen test, she bears no burden to rule out possible alternative causes."); Walther v. Sec'y of Health & Human Servs., 485 F.3d 1146, 1149-50 (Fed. Cir. 2007) ("[T]he Vaccine Act does not require the petitioner to bear the burden of eliminating alternative causes where the other evidence on causation is sufficient to establish a prima facie case.").

If petitioner fails to establish a prima facie case of causation, however, the burden does not shift. Doe 11 v. Sec'y of Health & Human Servs., 601 F.3d 1349, 1357-58 (Fed. Cir. 2010); see Cedillo v. Sec'y of Health & Human Servs., 617 F.3d 1328, 1335 (Fed. Cir. 2010) (citing Walther, 485 F.3d at 1151).

III. THE TESTIMONY OF THE MEDICAL WITNESSES

The parties presented witnesses offering medical opinions about whether Dr. Stewart's received flu vaccine led to the development of his GBS. The parties' medical witnesses did not dispute that petitioner suffered from Guillain-Barré Syndrome during his December 2003 hospitalization.[20] Rather, the witnesses' disagreement in this case

[20] The parties' medical witnesses agreed that Dr. Stewart suffered from GBS during his hospitalization in December 2003. See Tr. I at 12 (Dr. Willis); Tr. II at 103 (Dr. Smith), 156 (Dr. Leist). The witnesses expressed some uncertainty regarding whether petitioner suffered from the Miller-Fisher variant of GBS, a "very rare" variant characterized by eye movement disorders. See Tr. I at 15; Tr. II at 104.

While petitioner's expert neuroimmunologist, Dr. Smith, acknowledged that petitioner's documented eye drooping (ptosis) may have been consistent with the Miller-Fisher variant of GBS, he questioned whether Dr. Stewart's eye movement abnormality was sufficiently prominent to merit a Miller-Fisher diagnosis. Tr. II at 134. He concluded that Dr. Stewart's speech and swallowing difficulties were more consistent "with a standard demyelinating Guillain-Barré than Miller-Fisher." Tr. II at 135.

centered on what was the more likely cause of Dr. Stewart's GBS.

The parties' witnesses were qualified and knowledgeable. Having heard the testimony of the parties' witnesses and having considered the record as a whole, the undersigned concludes that petitioner has prevailed on his vaccine claim and thus, is entitled to Program compensation.

The opinions of the parties' medical witnesses were helpful and are set forth in further detail here.

A. Dr. Paul Willis, Petitioner's Treating Neurologist

As mentioned earlier, Dr. Willis was petitioner's attending neurologist during his acute hospitalization. See Pet. Ex. 4 at 9-11. He submitted an opinion of vaccine-related causation on Dr. Stewart's behalf and testified at hearing. See Pet. Ex. 33.

1. Dr. Willis's Qualifications

Dr. Willis attended Williams College in Massachusetts and medical school at the University of California Los Angeles (UCLA). Tr. I at 7-8. He completed his residency in neurology at UCLA and is a board certified neurologist. Tr. I at 8. He is a clinical neurologist practicing at Sansum Clinic, a multi-specialty clinic in Santa Barbara. Id. He is also an attending neurologist at Cottage Hospital in Santa Barbara. Id. He sees patients with a wide range of neurologic issues, including peripheral neuropathy and GBS, two neurologic conditions that are relevant in this case. Over his 30 year career, he has treated approximately 50 GBS patients. Dr. Willis testified as one of Dr. Stewart's treating physicians. See Tr. I at 9.

2. His Clinical Examination of Petitioner

Dr. Willis first examined Dr. Stewart on December 16, 2003 after his hospital admission. Tr. I at 9. As part of the medical history Dr. Stewart provided, he related to Dr. Willis that he had had an upper respiratory infection 10 days prior to his

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Respondent's expert neuroimmunologist, Dr. Leist, agreed that GBS can present clinically in different "soft forms," one of which is the Miller-Fisher variant. Tr. II at 155-56. He noted the possibility that petitioner's form of GBS was the Miller-Fisher type variant. But he declined to focus heavily on the type of GBS involved in petitioner's case. See id.

Because the type of GBS implicated in petitioner's case ultimately did not bear on the causation question presented here, the undersigned declines to resolve this factual issue.

hospitalization. Pet. Ex. 4 at 9; Tr. I at 28. Based on Dr. Stewart's history of diabetes and coronary artery disease and his clinical presentation with diffuse weakness in his extremities accompanied by difficulty speaking and swallowing, Dr. Willis initially suspected a stroke event. Pet. Ex. 4 at 9. He quickly ordered an MRI to determine whether Dr. Stewart had suffered a stroke. Id.

In the absence of evidence of a stroke, Dr. Willis began to consider other possible causes for Dr. Stewart's neuromuscular weakness. Among the considered possibilities were myasthenia gravis and poisoning by botulism or a diphtheria toxin. Tr. I at 11. Dr. Willis ordered a number of tests to assist in making a proper diagnosis. Id. The results of Dr. Stewart's lab work and a two to three day history of an "evolving pattern of weakness" were determined to be most consistent with a diagnosis of "some type" of GBS. Tr. I at 12. A course of plasmapheresis was initiated followed by a course of intravenous immunogobulin treatment. Id.

3. **His Opinion Regarding Causation**

Dr. Willis explained that GBS is an autoimmune disorder of the peripheral nerve system, first described more than 100 years ago. Tr. I at 13. The syndrome is believed to result from a stimulation of the immune system by "foreign invaders," including toxins such as vaccinations and infections, and the aberrant response of the body attacking its own tissues rather than the invading substance. Id. This mechanism of harm is known as molecular mimicry. Tr. I at 14.

Dr. Willis described classical GBS as involving an ascending, motor paralysis. Id. The progression of the weakness can occur "at varying rates" from an acute onset that occurs within 48 to 72 hours to an onset of over a week or two. Id. There are variants of GBS and, in Dr. Willis's view, Dr. Stewart suffered a particularly severe case of an atypical form of GBS. Tr. I at 15. The muscles controlling his speech and ability to swallow were affected. Id. In addition, he suffered damage to both the myelin sheathing protecting his nerves (demyelination) and well as damage to the nerve core (axonal degeneration). Id. The nature of Dr. Stewart's GBS injury was unusual but could occur in severe cases. Tr. I at 15-16. As residual damage from his GBS, Dr. Stewart continues to have hand and leg weakness. Tr. I at 18.

In Dr. Willis's view, Dr. Stewart "had two provocative issues preceding his catastrophic neurologic illness," specifically, a flu vaccine followed by an upper respiratory viral syndrome that provided an "additional immunological insult." Pet. Ex. 33 at 4. The neurologic effect of these factors was further complicated by the severe pneumonia infection Dr. Stewart suffered during his hospitalization. Id. at 3. As the treating neurologist during Dr. Stewart's hospitalization, Dr. Willis attributed the development of Dr. Stewart's GBS to the "synergistic effect" between his flu vaccine and his subsequent viral infection. Tr. I at 32. Dr. Willis noted that both events "up-

regulated" petitioner's immune system. Tr. I at 38.

Dr. Willis did not propose a mechanism for the posited synergy. But, he did address the timing of onset. Dr. Stewart presented with symptoms of GBS 25 days after he received the flu vaccine. Pet. Ex. 33 at 4. Dr. Willis initially opined that the 25-day period of onset was at the upper end of the time frame in which neurologic symptoms tend to arise. See Tr. I at 34. He later stated that an onset of 25 days was "well within" the six-week surveillance period used to monitor adverse effects after administration of the swine flu vaccine in 1976 and after administration of the H1N1 vaccine in 2009. See Pet. Ex. 33 at 4. In response to questioning at hearing about the timing of onset, Dr. Willis acknowledged that the appearance of neurologic symptoms within seven to 21 days after a precipitating event provided stronger evidence statistically of a causal relationship than the 25-day period of onset in this case. Tr. I at 34-35. He estimated that "60 to 70 percent [of cases] might occur in that time." Tr. I at 35. In an individual case such as this one, however, he did not know if the later timing of onset diminished the likelihood that the vaccine was causally related to petitioner's development of GBS. Id.

On questioning at hearing, Dr. Willis conceded that he had not described Dr. Stewart's GBS as post-vaccinal until he prepared his opinion letter of causation in 2010 for consideration in the filed vaccine claim. Tr. I at 32-33. Dr. Willis's notes in his treatment records refer to Dr. Stewart's GBS as post-infectious. Tr. I at 33.

Dr. Willis's view regarding the cause of Dr. Stewart's GBS was influenced strongly by his experience as a medical resident at UCLA in 1976 when he saw patients with GBS who had received vaccinations. Tr. I at 16. Dr. Willis testified that as he stood at Dr. Stewart's bedside with a group of medical residents, he observed the striking similarity between Dr. Stewart's condition and the dire condition of the swine flu vaccinated patients he had observed as a medical resident. Id. After making this observation to the residents, a pulmonary resident in the group drew Dr. Stewart's recent flu vaccination to Dr. Willis's attention. Id.

Dr. Willis opined that the flu vaccine that Dr. Stewart had received was a substantial contributing factor in the development of his GBS. Tr. I at 16-17. He testified that in the community of neurologists, vaccinations are one of the "well-recognized" precipitants of GBS. Tr. I at 21. He identified support in the medical literature, scientific acceptance of the described "immunologic mechanics," and the timing of Dr. Stewart's injury as the "major" factors informing his opinion of vaccine-related causation in this case. Id.

Dr. Willis conceded that he has no specialized training in immunology. Tr. I at 22. Nor is he a GBS specialist. Id. Rather, the basis for his opinion that a causal association exists between the flu vaccine and GBS is his experience as a clinician. See Tr. I at 25. He testified that in his clinical practice, he had seen two "relatively mild"

cases of post-vaccinal GBS in the previous year.[21] Tr. I at 26, 46. He diagnosed the two cases as post-vaccinal based primarily on the timing between vaccination and onset and the lack of any "other obvious antecedent cause." Tr. I at 26-27.

Dr. Willis's opinion that the onset of GBS can be triggered by a flu vaccine was unaffected by the conclusion reached by the Institute of Medicine (IOM) in its 2004 report. Tr. I at 24. The IOM stated in its 2004 report that the available epidemiologic evidence was inadequate to accept or reject a causal relationship between seasonal flu vaccines after 1976 and GBS. Id. Dr. Willis asserted that the IOM's conclusion neither proves nor disproves a causal relationship. Id. Instead, the report reveals the lack of determinative knowledge concerning whether a causal relationship exists. See id.

B. Dr. Derek Smith, Petitioner's Offered Expert

1. His Qualifications

Dr. Smith graduated from Rice University with one of his dual majors in biochemistry. Tr. II at 77. He attended medical school at the University of Texas Southwestern Medical School in Dallas, completed a residency in the Department of Neurology and Neuroscience at New York Hospital/Cornell University in New York, and completed a fellowship in neurology with the Multiple Sclerosis Study Group at Brigham and Women's Hospital in Boston. Pet. Ex. 30 at 1. Board certified in neurology, Dr. Smith has conducted "a fair amount of immunology research," has participated in clinical trials, and has received training in epidemiology. Tr. II at 78. He has published articles addressing the immunologic mechanism involved in treatments for multiple sclerosis patients. Tr. II at 78-79. Currently, he serves as the director of the Multiple Sclerosis Care Center in Connecticut. He also serves as a Clinical Assistant Professor at Harvard Medical School teaching residents and medical students. Tr. II at 78. In his 20 years of practice, he has diagnosed between 20 to 30 patients with GBS and has treated a number of GBS patients. Tr. II at 80, 144. The undersigned accepted Dr. Smith as an expert in neuroimmunology. Tr. II at 79.

2. His Opinion

Referencing petitioner's "complicated" pre-vaccination medical history, Dr. Smith posited that Dr. Stewart had "a number of diseases that may have already affected his

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[21] He testified that he has seen four or five cases of post-vaccinal GBS over his 30 years of practice. Tr. I at 45. Dr. Willis counted petitioner's case among these cases. Id. He did not count the swine flu cases he had observed in this number. Tr. I at 46. In contrast to petitioner's "severe" case of GBS, the other cases of post-vaccinal GBS observed by Dr. Willis were "relatively mild." See id.

peripheral nerves" and thus, predisposed him to injury in his peripheral nervous system.[22] Tr. II at 80. On November 20, 2003, he received a flu vaccine in his left shoulder muscle. See Pet. Ex. 2 at 2; Tr. II at 81. The medical records indicate that on or about December 6, 2003, he suffered a three-day course of an upper respiratory illness, and on December 16, 2003, he presented to the hospital with symptoms (specifically, weakness, difficulty walking, tingling in the hands and feet, and difficulty swallowing) that led to a diagnosis of GBS. Pet. Ex. 4 at 9-11; Tr. II at 82-85. Dr. Smith opined that a four-week time period for onset is medically appropriate for a "post-vaccinal immune-mediated disease," and the onset of Dr. Stewart's symptoms 26 days after receipt of his flu vaccine fell well within the four-week time period for the proposed mechanism of injury in this case.[23] See Tr. II at 82, 99. Dr. Smith testified that the likelihood that a vaccine-caused reaction has occurred is "highest" during the "peak" period of "two to three weeks" after vaccination. Tr. II at 130; see also Pet. Ex. 29[24] at 2; Pet. Ex. 29E[25] at 112; Pet. Ex. 29F[26] at 947-48. For injuries that occur outside of that peak time frame, vaccine-related causation becomes less likely. Tr. II at 99.

Dr. Smith addressed the mechanism by which GBS is thought to cause

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[22] However, Dr. Smith carefully distinguished any diabetic neuropathy that Dr. Stewart may have suffered from the impact on his peripheral nerves caused by his GBS. Dr. Smith reasoned that because the impairment of the peripheral nerves associated with diabetes does not present in the same demyelinating pattern discovered during Dr. Stewart's testing during his hospitalization, any effect on petitioner's peripheral nerves ascribed to his diabetes was distinguishable from that attributable to his GBS. Tr. II at 150.

[23] Dr. Smith added that an autoimmune complication that occurs within six weeks to three months following a vaccination reasonably can be considered causally connected to the vaccine. Tr. II at 96-97.

[24] Med. Expert Report of Dr. Derek Smith, June 26, 2007 (estimating that a suitable time frame for accepting an association between receipt of the influenza vaccine and GBS to be 0-4 weeks).

[25] Lawrence E. Schonberger, et al., Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program, United States, 1976-1977, 110 Am. Journal of Epidemiology 105, 112 (1979) ("The peak relative risks . . . occurred in weeks 2 and 3 after vaccination.")

[26] Thomas J. Safranek, et al., Reassessment of the Association Between Guillain-Barré Syndrome and Receipt of Swine Flu Influenza Vaccine in 1976-1977; Results of a Two-State Study, 133 Am. Journal of Epidemiology 940, 947-48 (1991) ("The attack rate of [GBS] in the vaccinated population peaked in the third week following vaccination.")

neurological injury. He explained that GBS "can be either primarily demyelinating (chiefly affecting the myelin sheathing that protects the nerves) or demyelinating and axonal (involving damage to both the nerve sheathing and the nerves)." Tr. II at 86. In Dr. Smith's view, the results of petitioner's first EMG, conducted two days after his hospitalization were supportive of a finding that Dr. Stewart's GBS was demyelinating. Tr. II at 88. That EMG showed "slowing motor conduction" that was noted in petitioner's medical records to be "suggest[ive]" of demyelination. Id. (citing Pet. Ex. 5 at 255). Dr. Smith observed that petitioner's normal lumbar puncture test results during the first week of his manifested symptoms of GBS could be explained as the result of an early effort to "indirect[ly] measure" the inflammation occurring in petitioner's peripheral nervous system by drawing spinal cord fluid from his central nervous system. See Tr. II at 87, 143. While aware that "there [could] be a delay from the time of an initial illness to the [appearance of] abnormalities in the spinal fluid," Dr. Smith could not speak, with any specificity, to the length of delay expected. Tr. II at 143-44.

Dr. Smith explained that some cases of GBS are considered "post-infectious" because they do not result from an active or persistent viral infection but rather from an "inflammatory illness" in the peripheral nerves. Tr. II at 89, 142. He stated that the "consensus hypothesis is that [GBS is caused by] an aberrant immune response . . . triggered by . . . acute infection that then caused the immune system to behave as though there might have been an infection in the nerves." Id. The aberrant immune response involves the immune system responding to a normal component of the body's nervous system in the manner in which it would respond to a viral or bacterial component associated with an infection. See Tr. II at 90. Such an immunological response--erroneously directed toward the body's own nervous system components rather than toward viral or bacterial components--is described as an autoimmune response. See id. The erroneously directed immune response is thought to occur because the immune system perceives a component of the body's nervous system to be a foreign invader. Tr. II at 91. This mechanism of injury—caused by a mistaken immunological attack on a component of the body's nervous system—is known as molecular mimicry, id., and by consensus of the neuroimmunological community, is the leading biological theory explaining how GBS occurs. Tr. II at 92. This theory finds support in "some laboratory-based research" mostly involving animals rather than humans. See Tr. II at 108.

Because GBS is a rare disorder, a very large epidemiological study of millions of subjects would be required to identify any factors causally associated with the disorder. See Tr. II at 95-96; see also Pet. Ex. 29C[27] at 606. Absent such a clarifying study, Dr. Smith testified that belief persists within the community of neuroimmunologists that flu

[27] Vittorio Govoni and Enrico Granieri, Epidemiology of Guillain-Barré Syndrome, 14 Current Op. in Neurology 605, 606 (2001) (estimating 1 to 2 cases of GBS per 100,000 persons).

vaccines can cause GBS. See Tr. II at 93-94. This belief is, in part, based on evidence that many patients develop neurological abnormalities after a prodromal syndrome. See Pet. Ex. 29B[28] at S25. Dr. Smith indicated that natural influenza virus has been implicated in the development of GBS, Tr. II at 98; see also Pet. Ex. 29A[29] at 2337, and he posited that the vaccine strain of influenza vaccine similarly could be implicated based on the theory that molecular mimicry exists between the components of the influenza vaccine and components of the body's myelin sheathing. Tr. II at 98; see also Pet. Ex. 36[30] at 230.

In support of the proposition that the vaccine strain of the flu has been causally associated with an increased incidence of GBS, Dr. Smith pointed to articles pertaining to the increased incidence of GBS following the 1976 swine flu vaccinations and to the more recent 1998 Lasky article in which investigators considered whether the influenza vaccines administered in 1992-1993 and in 1993-1994 contributed to a slight increase in the number of GBS cases beyond the estimated background rates of the disorder. See Tr. II at 107-10; see also Pet. Ex 29E at 109 (the 1979 Schonberger article); Pet. Ex. 29F at 948 (the 1991 Safranek article); Pet. Ex 29I[31] at 1560; Pet. Ex. 29D[32] at 1800.

With respect to Dr. Stewart's particular case, Dr. Smith speculated that after Dr. Stewart received the flu vaccine, he developed progressive weakness that first affected his bulbar muscles (those muscles responsible for speech and swallowing) and, in turn, led to the development of an upper respiratory infection prior to his hospitalization and to the development of pneumonia during his hospitalization.[33] See Tr. II at 100-01. Dr.

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[28] Dale E. McFarlin, Immunological Parameters in Guillain-Barré Syndrome, Supp. 27 Annals of Neurology S25, S25 (1990).

[29] P. E. Bosch and B. E. Smith, Disorders of Peripheral Nerves (Chapter 82), II Neurology in Clinical Practice: the Neurological Disorders 2299, 2337 (4th ed. 2004).

[30] Irving Nachamkin et al., Anti-Ganglioside Antibody Induction by Swine (A/NJ/1976/H1N1) and Other Influenza Vaccines: Insights into Vaccine-Associated Guillain-Barré Syndrome, 198 Journal Infectious Diseases 226, 230 (2008).

[31] Eugene S. Hurwitz, M.D., Guillain-Barré Syndrome and the 1978-1979 Influenza Vaccine, 304 New Eng. J. Med. 1557, 1560 (1981).

[32] Tamer Lasky, Ph.D., The Guillain-Barré Syndrome and the 1992-1993 and 1993-1994 Influenza Vaccines, 339 New Eng. J. Med. 1797, 1800 (1998).

[33] Dr. Smith allowed that a viral syndrome can occur several days after receipt of a flu vaccine, but that syndrome usually does not resemble an upper respiratory infection. Tr. II at 125. Instead, it presents as "mild," "low grade" viral-type symptoms. Id.

Smith characterized this proposed sequence of events as "certainly possible." See Tr. II at 101.

He also offered an alternative sequence of events that "very possibly" included both the influenza vaccine and the upper respiratory infection as contributing factors. Id. He posited that the vaccine and the infection "may have worked in concert" to activate "a specific immune response against the peripheral nerves." Tr. II at 101-02. He explained how the vaccine and infection might have worked together:

> [V]accines [are] frequently given with adjuvant. . . . [T]he purpose of the adjuvant is to create a more vigorous immune response. . . . [Also, a] specific protein or inactivated virus . . . is put in a vaccine . . . to create a specific immune response. . . . [B]oth specific and non-specific components [are added] to [potentiate] a vaccine. . . . [I]n Mr. Stewart's case, it's entirely plausible that the vaccine caused a specific immune response against one of the components of the peripheral nervous system, and . . . the respiratory infection then caused that [immune response] to become more vigorous.

Tr. II at 102; see also Tr. II at 136 (describing the "in concert" action as a boosted immune response effected by "one event [the vaccination] . . . providing the specific immune response, and then a second event [the upper respiratory infection] . . . providing essentially a non-specific boost to that specific immune response"). Dr. Smith stated that the specificity of an immune response is provided by antibodies and by T-cell receptors, and when considering immune-mediated neurological diseases, the "focus" is primarily on the T-cells. Tr. II at 118.

Dr. Smith discussed an experiment showing the similarity between a lipid (oil or fat) found in the infectious bacterial agent Campylobacter jejuni, a pathogen causally associated with the development of GBS, and a lipid found in the myelin sheathing located in the peripheral nervous system. Tr. II at 118. Dr. Smith also discussed evidence from an experimental study showing the similarity between a component of the flu vaccine, specifically a lipopolysaccharide,[34] and the myelin component of the

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Because Dr. Stewart's illness was described as an upper respiratory illness, Dr. Smith did not view that infection as a "direct consequence" of the flu vaccine. Id. He did express the view that the upper respiratory infection was possibly "part of the same process" immunologically triggered by the administered flu vaccine. See Tr. II at 128; but see Tr. II at 135 (expressing uncertainty regarding whether the upper respiratory event was a separate event by stating, "I don't know. It may have been; it may not have been.")

[34] Lipopolysaccharides are not proteins but are lipids or little fats bound to complex sugars. Tr. II at 217; see also Dorland's at 1079.

peripheral nervous system in mice and rabbits. See Tr. II at 120 (referencing Pet. Ex. 36 at 231-32). He asserted that these experiments provided supportive evidence for the molecular mimicry mechanism that he believed was responsible for petitioner's GBS. Tr. II at 119-20.

Dr. Smith acknowledged that of the events reported to have preceded the onset of GBS, the most commonly reported event is respiratory illness, followed by gastrointestinal illness. Tr. II at 127-28 (referencing Pet. Ex. 29A at 2337 (the 2004 Bosch neurology text) (finding that 58% of GBS cases were reported to have been preceded by a respiratory illness, 22% preceded by a gastrointestinal illness, and 3% preceded by vaccination). He observed, however, that most respiratory illnesses do not cause GBS, Tr. II at 128, and he asserted that the "evolution" of GBS is "thought to be similar" in patients who develop the condition whether precipitated by a received vaccination or an infection. Tr. II at 144. He maintained his opinion of vaccine-related causation in this case.

C. Dr. Leist, Respondent's Offered Expert

1. His Qualifications

Dr. Leist received a doctorate in biochemistry from the University of Zurich and a medical degree from the University of Miami. Tr. II at 152; Resp. Ex. K at 1. Like petitioner's expert, he trained as a neurologist in the Department of Neurology and Neuroscience at New York Hospital/Cornell University in New York, and he completed a fellowship with the National Institutes of Health in Bethesda, Maryland. Tr. II at 152; Resp. Ex. K at 1. Board certified in neurology, Dr. Leist currently serves as the director of the Multiple Sclerosis Center at Thomas Jefferson University in Philadelphia, Pennsylvania. Tr. II at 152-53. He also serves as an Associate Professor of Neurology and Clinical Neuroimmunology teaching the resident staff in his division. Tr. II at 153-54. In addition to teaching and maintaining an active clinical practice, Dr. Leist conducts research. See id. In his nearly 13 years of clinical practice, he has treated between 30 to 50 patients with GBS. Tr. II at 153-54. The undersigned accepted Dr. Leist as an expert in neuroimmunology. Tr. II at 155.

2. His Opinion

Dr. Leist agreed with the testimony from petitioner's medical witnesses that GBS is "a disease that leads to peripheral nerve injury . . . [and is] very often preceded by an infection." Tr. II at 155. He also agreed "that there is a relatively low incidence of GBS in the general population." Id.

He addressed whether certain risk factors existed for more severe cases of GBS, stating that "certain pathogens have been associated with forms of GBS that are much

more neuronopathic, [that is,] much more destructive of the neurons." Tr. II at 157. He explained that the neuronopathic forms of GBS cause "not just a demyelinating illness, but . . . actually leave[] the nerves injured or destroyed." Id. Such cases, which could be deemed "severe" cases of GBS, involve "very limited" patient recovery. Id.

Dr. Leist testified that "based on laboratory studies . . . [and] field observations . . . [from] the swine flu occurrences in the '70s . . . [and] the . . . Lasky [study in] . . . the early '90s," "a series of theoretical arguments" could be "put together for flu vaccine to cause GBS." Tr. II at 158. He pointed out, however, that subsequent studies suggested only a slight association between flu vaccine and GBS. See Tr. II at 159-60. Accordingly, he viewed the conclusion reached in the 2004 IOM Report (specifically, that the available data sets prior to 2003 did not support a finding either accepting or rejecting the likelihood of a causal association between the flu vaccine and GBS) as a "conservative statement" about the possibility of a causal relationship. See Tr. II at 160.

Dr. Leist noted that "in the last 10 to 20 years," doctors have recognized that there are lipopolysaccharides in the membranes of certain pathogens, including the bacterial agents Campylobacter jejuni and H. influenzae, that occur with structures similar to those found in the membranes of the human central nervous system. Tr. II at 163-64. In addition, he noted that "certain antibodies to certain subtype[s] of lipopolysaccharides" have been found in GBS patients. Tr. II at 164. Dr. Leist acceded that these findings arguably could support a causation theory of molecular mimicry for particular pathogens and GBS, but he was not aware of any corresponding evidence for the flu vaccine and GBS. Tr. II at 164-65. He added that "independent of vaccines, molecular mimicry in experimental models has been shown." Tr. II at 201.

Dr. Leist observed that there is evidence associating upper respiratory infections with the onset of GBS. Tr. II at 166 (citing the 2008 van Doorn article, filed as Resp. Ex. J3[35]). He agreed with petitioner's expert Dr. Smith that of the recognized antecedent events to GBS, upper respiratory infections are the "most common." Id. (referencing the same 2004 Bosch neurology text as did Dr. Smith). Dr. Leist indicated that he was not aware of any evidence that the severity of the antecedent upper respiratory infection was a factor in the development of GBS. Tr. II at 167.

Dr. Leist addressed Dr. Stewart's antecedent upper respiratory infection and noted that Dr. Stewart tested positively for H. influenzae bacteria during his hospitalization. See id. He further noted that H. influenzae bacteria can persist in a colonized state in the respiratory tracts of patients with diabetes, a condition that Dr. Stewart has. See Tr. II at 176-77.

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[35] Pieter A. van Doorn, et al., Clinical Features, Pathogens, and Treatment of Guillain-Barré Syndrome, 7 The Lancet: Neurology 939, 940 (2008).

Because H. influenzae is a bacterial pathogen that is "frequent[ly] . . . associated with . . . respiratory tract infections" and is one of the pathogens that has been associated with GBS, Dr. Leist opined that it was more likely than not that Dr. Stewart's GBS was caused by his antecedent upper respiratory infection. Tr. II at 168-69. This likelihood, in his view, outweighed the likelihood that the flu vaccine caused Dr. Stewart's GBS. Tr. II at 169. Dr. Leist conceded that petitioner's intubation during his hospitalization—which became necessary to address the respiratory distress that accompanied the progression of Dr. Stewart's GBS—was sufficient to have forced any diabetes-related colonized H. influenzae bacteria from Dr. Stewart's throat into his lungs to cause pneumonia. But, he maintained his opinion that Dr. Stewart's pneumonia was community-acquired rather than hospital-acquired.[36] Tr. II at 179-81.

Dr. Leist identified other factors that he believed weighed against the likelihood of vaccine-related causation. First, he pointed to the "marked axonal involvement" and the "significant atrophy of . . . multiple muscular areas" that led to an enduring functional impairment in Dr. Stewart's case that is thought to result from more than the demyelinating mechanism of molecular mimicry that petitioner has advanced in this case.[37] Tr. II at 170-71; see also Tr. II at 157-58 (asserting that "certain pathogens have been associated with forms of GBS that are much more . . . destructive of the neurons."). Second, he pointed to the 10-day time period between the upper respiratory infection and the onset of Dr. Stewart's GBS as stronger evidence of non-vaccine causation than the 26- day time period between the received flu vaccine and Dr. Stewart's presentation with GBS symptoms because the peak period for event-related causation is two weeks following the antecedent event. See Tr. II at 171-73 (citing the 1979 Schonberger article filed as Pet. Ex. 29E). Third, Dr. Leist pointed to the acute onset and rapid progression of Dr. Stewart's condition, as described in his medical records, as evidence weighing against Dr. Smith's proposition that the upper respiratory infection signaled the initial onset of Dr. Stewart's GBS following vaccination (rather than providing, as Dr. Leist believed, the triggering event for petitioner's GBS). Tr. II at 174-75; see also Tr. II at 190-91 (Dr. Leist asserting that the significantly axonal form of Dr. Stewart's GBS ("evidenced [by this] second EMG,") his antecedent respiratory tract infection, and his

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[36] Partially informing Dr. Leist's opinion was petitioner's high glucose reading on admission to the hospital. See Tr. II at 201. Dr. Leist explained that in his experience with multiple sclerosis patients that have the co-morbid condition of diabetes, he has learned that very high glucose levels could be indicative of "a nascent or developing . . . or presen[t] infection." Id.

[37] Dr. Leist clarified, later in his testimony, that the acute demyelinating form of GBS is the most common form of the disorder in North America. Tr. II at 182-83. While he concurred that secondary axonal damage can occur as a result of demyelination, he made clear that "severe" axonal damage does not occur by mere demyelination. Tr. II at 183.

later-detected H. influenzae infection—that could independently lead to a respiratory tract infection—presented a "not unreasonable explanation . . . [for] the whole symptom complex").

Contributing to Dr. Leist's doubt about the relation between Dr. Stewart's flu vaccine and his subsequent upper respiratory infection was the absence of any indication in Dr. Stewart's medical records or his own testimony that the two events were associated. Tr. II at 175. Dr. Leist added that the aspect of the theory presented by petitioner's witnesses that a subsequent infection could "augment" the immune response generated by a vaccination received more than 10 days earlier "is a relatively theoretical concept that has not really been borne out." Tr. II at 193. He explained that with respect to vaccinations, scientific consideration of the concept of synergy has focused primarily on differences in immune responses that occur when multiple vaccines are administered on the same day rather than spread out over time. See Tr. II at 194-95. He further explained that "our immune systems are specific enough to handle different tasks at different times." Tr. II at 195. In his view, the timing of 10 or more days between the received vaccine and the presentation of an upper respiratory infection "completely negate[d] even the theoretical consideration for an augmentation" of Dr. Stewart's immune response. Id.

Dr. Leist indicated that the natural flu virus has been associated with GBS but as a pathogen, the natural flu has not been associated with GBS as frequently as other pathogens. Tr. II at 204. He noted that the flu vaccine that Dr. Stewart received was "a non-li[v]e, non-attenuated[,] killed mixture of three viruses" and thus, bore no similarity to the natural live flu virus.[38] Tr. II at 165.

Dr. Leist indicated that some cases of GBS are idiopathic, that is, without any recognized antecedent event. Tr. II at 202. He also indicated that most recent epidemiological studies show no increased risk of GBS following vaccination. Tr. II at 210.

He discussed how he determines whether a case of GBS is causal or coincidental in the absence of epidemiological evidence. Tr. II at 211-12. Although he did not believe the flu vaccine was causally responsible for Dr. Stewart's GBS, he allowed that determining whether the onset of GBS following a vaccine was causally-related or merely coincidental was "a very difficult question." Tr. II at 162. The determination can be made in part by using the available tools in the clinical setting to rule out certain known causes first. Tr. II at 212-13.

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[38] The causation theory advanced in this case is one of molecular mimicry. It is not clear that the theory requires the mimicked pathogen to be a live one. However, it is clear that the theory requires the mimicked pathogen be recognized as a foreign invader to the body.

Dr. Leist acknowledged that when contemplating these matters, scientists "like to have certainty" and clinicians necessarily "live with uncertainty." Tr. II at 162. From his perspective as a clinician, he offered that if there is an identifiable antecedent event, he generally does consider the "possibility" that such antecedent event is causally-related to the later occurring event, rather than merely coincidental. Tr. II at 163.

Notwithstanding this admission as a clinician, Dr. Leist maintained his personal conviction that in general, the flu vaccine is not causally related to the onset of GBS. As an indication of the strength of his belief, he indicated that were he Dr. Stewart's treating doctor, he would recommend continued receipt of the flu vaccine.[39] Tr. II at 221-22.

IV. EVALUATING PETITIONER'S CLAIMS UNDER THE APPLICABLE LEGAL STANDARD

As described in the Legal Standards discussion above, the burden rests with petitioner to show, by preponderant evidence, that the vaccine he received brought about his injury. See Althen, 418 F.3d at 1278. To prevail on his vaccine claim for compensation, petitioner must present: (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 proximate temporal relationship between vaccination and injury. Id.

The undersigned turns now to consider whether petitioner has satisfied his burden under the Althen standard. Because the principal focus of the parties' disagreement is on

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[39] Of note, current guidance provided by the Centers for Disease Control (CDC) instructs persons who have had GBS to consult with a health care provider before receiving the flu vaccine. Inactivated Influenza Vaccine 2010-11: What You Need to Know, Centers for Disease Control & Prevention (July 6, 2011), http://www.cdc.gov/vaccines/pubs/vis/downloads/vis-flu.pdf. The guidance in place from CDC in November 2009, however, indicated that persons who have had GBS previously should not receive the flu vaccine. See 2009 H1N1 Influenza Vaccine and Pregnant Women: Information for Healthcare Providers, Centers for Disease Control & Prevention (July 6, 2011), http://www.cdc.gov/h1n1flu/vaccination/providers_qa.htm. See Ellis v. Int'l Playtex, Inc., 745 F.2d 292, 301 (4th Cir. 1984) (discussing the reliability of CDC reports and allowing consideration of such reports based on an inability to discern any "conceivable motive for carrying out . . . studies [or providing public health guidance] in any other manner than to inform the public fairly and accurately.") The CDC guidance clearly contemplates that whether a patient has had GBS should be considered when determining whether a flu vaccine should be given.

prong two, specifically whether the flu vaccine led to the development of petitioner's GBS, the undersigned addresses prongs one and three first.

A. Petitioner's Medical Theory

Petitioner's expert neuroimmunologist, Dr. Smith, and petitioner's treating neurologist, Dr. Willis, posited that the flu vaccine can cause GBS through the biological mechanism of molecular mimicry. See Tr. I at 23-24; Tr. II at 90-94, 117-21. In support of this theory of vaccine causation, petitioner's medical witnesses relied on the swine flu experience in the 1970s, the slight causal association between the flu vaccine and GBS detected after the 1992-1993 and 1993-1994 flu seasons, and the animal studies offering support for the theory, in particular, an experimental study identifying a flu vaccine component that bears similarity to a component of the myelin sheathing found in the peripheral nervous system of animals. See Tr. II at 117-22 (Dr. Smith discussing the process of molecular mimicry and the similarity between flu vaccine components and the myelin sheathing of the peripheral nervous system in mice and rabbits); Tr. II at 108-14 (Dr. Smith referencing various studies conducted over the identified time periods that showed an increase in GBS cases after administration of different influenza vaccines suggestive of a causal link between the administered vaccines and the resultant GBS).[40]

Respondent's expert neuroimmunologist, Dr. Leist, agreed that "a series of theoretical arguments" could be "put together for flu vaccine to cause GBS." Tr. II at 158. He did not challenge the scientific basis for the underpinnings of petitioner's theory. Rather he challenged the statistical significance of those studies reporting a detected association between the flu vaccine and GBS. See Tr. II at 159-60.

Because petitioner has offered a theory of vaccine causation that is supported by a sound scientific explanation, the undersigned finds that petitioner has satisfied his burden on prong one of the Althen test.

B. The Temporal Relationship between Petitioner's Vaccination and His Injury

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[40] See generally Pet. Ex. 29E at 120 (citing a 1976 study reporting "strong support" for an etiological link between the A/New Jersey Swine Flu vaccine and GBS); Pet. Ex. 29F at 949 (citing a 1991 review of the 1976 swine flu vaccine studies confirming the "statistically significant increased risk" of GBS following administration of the vaccine); Pet. Ex. 29D at 1801 (citing a 1992-93 and 1993-94 study finding a "small risk of GBS associated with the [seasonal] influenza vaccines"); Pet Ex. 36 at 230 (citing a 2008 article identifying the specific compounds found in various influenza vaccines likely to produce the antibodies which trigger the molecular mimicry suspected to play a role in the development of GBS).

The parties' medical expert witnesses agreed that the two-week period following the flu vaccine or other antecedent event was the peak period for the occurrence of adverse events. See Tr. II at 130 (Dr. Smith); Pet. Ex. 29 at 2 (Dr. Smith); Tr. II at 172 (Dr. Leist). Nonetheless, the parties' medical witnesses recognized the four-week time period as a medically appropriate time frame for the onset of GBS after a post-infectious process. See Tr. II at 88, 99 (Dr. Smith); Tr. at 223-24 (Dr. Leist). In this case, the parties agree that petitioner manifested symptoms of the onset of GBS within the medically acceptable time frame of four weeks. Accordingly, the undersigned finds that petitioner has satisfied his burden under prong three of the Althen standard.

C. The Sequence of Cause and Effect in Petitioner's Case

The parties diverge most significantly in their views regarding the cause of petitioner's GBS on the issue of the triggering antecedent event. Petitioner's attending neurologist and expert neuroimmunologist opined that petitioner's flu vaccine worked together with his subsequently acquired "infection"[41] to boost his immune system and thereby trigger the molecular mimicry that would lead to demyelination in petitioner's peripheral nervous system and cause petitioner to develop GBS. Pet. Ex. 33 at 4 (Expert Report of Dr. Willis); Tr. I at 29-32 (Dr. Willis); Pet. Ex. 29 at 2 (Expert Report of Dr. Smith); Tr. II at 89-92, 102 (Dr. Smith). Dr. Willis based his opinion of causation on the synergistic effect of the vaccine and subsequent infection. Pet. Ex. 33 at 4 (Expert Report of Dr. Willis); Tr. I at 29-32 (Dr. Willis). Dr. Smith shared that view. Pet. Ex. 29 at 2 (Expert Report of Dr. Smith); Tr. II at 89-92, 102 (Dr. Smith). Dr. Smith alternatively posited, as a possible but less likely causal sequence of events, that because petitioner's reported respiratory infection (following his flu vaccination) was marked by coughing, it was the first manifestation of the bulbar weakness that would eventually present as difficulty swallowing and slurred speech during the later development of petitioner's GBS. Tr. II at 100-01 (Dr. Smith).

Respondent's expert neuroimmunologist, Dr. Leist, had a different view. He regarded the 10-day period of time between Dr. Stewart's receipt of the flu vaccine and the presentation of his upper respiratory infection as too long a period of time between the two events to support "the relatively theoretical concept" of an augmented immune response. Tr. II at 193. Accordingly, he viewed the flu vaccination and the subsequent respiratory infection to comprise two separate and unrelated events, see Tr. II at 169, 175, and he devoted a good measure of his testimony to addressing why the respiratory infection (a factor unrelated to the vaccine) rather than the flu vaccine was the causal

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[41] The information was described variously in the medical records as a "viral illness," Pet. Ex. 4 at 9, an "upper respiratory process," Id., an "upper respiratory syndrome," Pet. Ex. 4 at 28, an "upper respiratory infection with a cough and sore throat," Pet. Ex. 33 at 1, and an "upper respiratory illness, [with] a mild cough" Pet. Ex. 21 at 2.

agent responsible for petitioner's GBS. Tr. II at 169-70, 172-73, 190-91.

In determining whether petitioner has satisfied his burden of proof, however, the undersigned does not focus on respondent's assertions concerning a factor unrelated.[42] Rather the undersigned considers first whether petitioner has satisfied his burden of showing a logical causal relationship between the vaccine he received and his sustained injury. The undersigned concludes that Dr. Stewart has done so.

Dr. Stewart has established that the flu vaccine could cause GBS under Althen prong one, and he has presented a causal sequence that logically implicates both the receipt of his flu vaccination and his subsequent respiratory infection in the development of his GBS. By incorporating his upper respiratory infection into the proposed causal sequence, petitioner rebuts respondent's claim that the infection was an independent and alternative causal factor. Dr. Stewart has offered opinions of causation from both his treating neurologist during his hospitalization and from an expert immunologist.

Making the closest of calls on the evidence presented, the undersigned finds that petitioner has satisfied, by a slight measure, his burden of proving that the vaccine he received more probably than not caused his injury. See Althen, 418 F.3d at 1280 (sanctioning the resolution of close calls in petitioner's favor). Dr. Stewart has presented a logical sequence of cause and effect supported by a plausible scientific explanation, and by satisfying the three elements of the Althen test, he has presented a prima facie claim for compensation.

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[42] By deferring the discussion of respondent's claim of alternative causation to a later discussion, the undersigned does not ignore the guidance from the Federal Circuit that respondent's position may be considered when evaluating petitioner's case in chief. See Doe 11 v. Sec'y of Health and Human Servs., 601 F.3d 1149, 1358 (Fed. Cir. 2010) (stating that "neither § 300aa-13 nor our cases limit what evidence the special master may consider in deciding whether a prima facie case has been established); de Bazan, 539 F.3d at 1353 ("The government, like any other defendant, is permitted to offer evidence to demonstrate the inadequacy of the petitioner's evidence on a requisite element of the petitioner's case in-chief.") But, structuring the discussion of the parties' respective positions differently would not have disturbed the undersigned's ruling. See also Torday v. Sec'y of Health & Human Servs., 2009 WL 51963 *5 (Fed. Cl. Spec. Mastr. Dec. 10, 2009) (in ruling for petitioner in a flu/GBS case, the deciding special master found that "[w]hether this case is analyzed under Walther and de Bazan as a factor unrelated or under Pafford[v. Sec'y of Health & Human Servs., 451 F.3d 1352 (Fed. Cir. 2006)] as petitioner's burden to eliminate other potential causes, . . . petitioner has established his right to recovery under the Vaccine Act"); Heinzelman v. Sec'y of Health & Human Servs., 2008 WL 5479123 *14 (Fed. Cl. Spec. Mstr. Dec. 4, 2008) (petitioner's case analyzed first before turning to consider respondent's arguments concerning alternative causation).

This showing, however, does not resolve the case. Further consideration of the vaccine claim is necessary because respondent attributes causation to a factor unrelated to the flu vaccine petitioner received. Accordingly, the burden shifts to respondent to prove alternative causation by preponderant evidence. See Althen, 418 F. 3d at 1280 (citing § 300aa-13(a)(1)(B)) (explaining that before a special master may grant compensation, he or she must find that there is "not a preponderance of evidence that the illness, disability, injury, condition or death described in the petition is due to factors unrelated to the administration of the vaccine . . . ."); de Bazan, 539 F.3d at 1352 ("Once the petitioner has established a prima facie case for entitlement to compensation and thus met her burden to prove causation-in-fact, the burden shifts to the government to prove '[by] a preponderance of the evidence that the [petitioner's injury] is due to factors unrelated to the administration of the vaccine described in the petition . . . .'"); Walther, 485 F.3d at 1149-50 ("A plain reading of the statutory text more naturally places the burden on the government to establish that there is an alternative cause by a preponderance of the evidence.").

The undersigned turns now to evaluate respondent's assertion that petitioner's upper respiratory infection was the causal factor responsible for his GBS.

D. Respondent's Claim Regarding a Factor Unrelated

The Vaccine Act authorizes compensation of a vaccine claim when petitioner has demonstrated by a preponderance of the evidence the matters that are statutorily required to be included in the petition, and "there is not a preponderance of evidence that the . . . condition . . . described in the petition is due to other factors unrelated to the administration of the vaccine described in the petition." § 300aa-13(a)(1)(A), (B) (emphasis added). The Federal Circuit has instructed that "when . . . multiple independent potential causes [of vaccine injury are alleged], the government has the burden to prove that the covered vaccine did not cause the harm." Walther, 485 F.3d at 1151. In proving alternative causation, the government must also satisfy the three prong Althen test. See Althen, 418 F.3d at 1278.

Respondent alleges an alternative cause for Dr. Stewart's injury. In particular, respondent's expert Dr. Leist challenged the causal sequence proposed by petitioner that involved both the received vaccine and the subsequent respiratory infection. Dr. Leist posited, as a more likely causal sequence, that petitioner's antecedent respiratory infection led to the development of his GBS. Tr. II at 169.

Pointing to evidence that an upper respiratory infection can cause GBS, Tr. II at

155, 204; Resp. Ex. J at 6-7; Resp. Ex. L1[43] at 53 (2004 IOM Report); Resp. Ex. J3 at 1-2 (van Doorn), respondent, through the testimony of Dr. Leist, has satisfied the first prong of Althen. Also pointing to evidence that a respiratory infection occurring within a four-week time frame prior to the onset of GBS may be causally associated, Tr. II at 223-24, respondent, again through the testimony of Dr. Leist has satisfied the third prong of the Althen test. But for the reasons more fully addressed below, respondent has failed to satisfy the second Althen prong.

Dr. Leist asserted that because upper respiratory infections are statistically the most common of the antecedent events associated with GBS and because petitioner had such an infection during the "peak" time frame of two to three weeks before the onset of his GBS, the infection alone—without any immunological boost from the received vaccine—was sufficient to trigger petitioner's GBS. Tr. II at 171-75. As further support for his view, Dr. Leist testified that as a diabetic patient, Dr. Stewart may have had colonized H. influenzae bacteria in his throat at the time he developed his respiratory infection. Tr. II at 188-99. Pointing out that H. influenzae is a pathogen known to be causally associated with the two conditions—pneumonia and GBS—that Dr. Stewart later developed and pointing out that Dr. Stewart tested positively for H. influenzae during his hospitalization, Dr. Leist speculated that Dr. Stewart's antecedent respiratory infection, in fact, may have involved the same H. influenzae bacteria later detected in Dr. Stewart's respiratory culture. Tr. II at 167-68. Dr. Leist further speculated that the H. influenzae bacteria may have been causally responsible for both his pneumonia and his GBS. Id.

Respondent's expert, in part, relies on statistical probability to diminish the likelihood of the causal sequence petitioner presented in this case and to enhance the likelihood of the causal sequence he has presented. However, in the view of the undersigned, the presented statistical evidence is not dispositive. Instead, such evidence is one of the factors that can be considered when evaluating a developed record "as a whole." See §300aa-13(a)(1) (providing that the "record as a whole" must be considered when making an entitlement determination). As discussed in more detail below, because the record as a whole does not support a finding that petitioner's antecedent respiratory illness was more likely than not the single, triggering agent for petitioner's GBS, respondent's claim of alternative causation does not prevail.

Dr. Leist speculated that petitioner's upper respiratory illness may have been caused by an H. influenzae bacterial infection. But this aspect of his causation theory cannot be confirmed by the medical records because petitioner did not seek treatment for the antecedent infection, and by the time petitioner did present to the hospital, he had no

[43] Immunization Safety Review: Influenza Vaccine and Neurological Complications, Institute of Medicine 1, 53 (2004).

clinical symptoms of a lingering infection.[44] Pet. Ex. 4 at 27 (Dr. Willis's initial medical history of Dr. Stewart on December 16, 2003, reflect that patient "reports a recent viral illness about 10 days ago. Three days of upper respiratory process . . . . [which] ha[s] cleared.") Moreover, the mere presence of a H. influenzae infection does not compel a finding that the bacterial agent is causally responsible for an ensuing case of GBS. As reported in an article filed by Dr. Leist, the bacterial agent H. influenzae "naturally inhabits the respiratory tract of the majority of humans," and thus cannot be considered to be the causal factor in GBS patients (particularly those with the Miller-Fisher variant) even when strains of the bacterium are cultured from the patients until "rigorous serological characterization" has occurred "to exclude the potential involvement of other pathogens." Resp. Ex. J4[45] at 8164 (emphasis added). Accordingly, a respiratory culture positive for H. influenzae bacteria, as in petitioner's case, points to only one factor—to be considered with other factors—when seeking to identify the causative agent in a GBS case.

In this case, petitioner's blood work after his admission to the hospital showed non-specific evidence of infection in his elevated white cell count, his low platelet count, and the detectible presence of lymphocytes, monocytes and neutrophils. Pet. Ex. 4 at 10. Dr. Stewart's glucose levels on hospitalization also were elevated, another indication of an emergent infection according to Dr. Leist. Id.; Tr. II at 201 ("The fact that these glucose levels were very high [upon presentation to the hospital] indicate[s] a nascent or a developing infection or presence of infection at that point in time.") In addition, petitioner's blood work revealed an elevated CPK level, that was suggestive of an injury affecting his skeletal muscle, as well as the heart and brain. Id. Whether these test results, when viewed together with petitioner's clinical picture, were more consistent with a 10-day old H. influenzae infection (as Dr. Leist asserted) than with an acute onset of GBS is not known.

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[44] Dr. Willis testified that petitioner did not exhibit "a severe infection" when he first entered the hospital. Tr. I at 48. He did not present with a fever. Tr. I at 29. His initial chest x-ray was normal. Tr. I at 29. Petitioner's initial hospital laboratory testing indicated a normal white blood cell count, demonstrating the absence of an active infection. Tr. I at 29; Tr. II at 177. Other test results were consistent, however, with an emergent infection.

[45] R. Scott Houliston, et al., A Haemophilus Influenzae Strain Associated with Fisher Syndrome Expresses a Novel Disialylated Ganglioside Mimic, 46 Biochemistry 8164, 8164 (2007) ("Because the organism naturally inhabits the respiratory tract in humans, strains cultured from patients with F[isher] S[yndrome] cannot be easily linked to the disease without rigorous serological characterization to exclude the potential involvement of other pathogens.")

Nor is it known whether the results of petitioner's respiratory culture (showing a heavy growth of H. influenza) two days after his hospitalization and petitioner's subsequent development of pneumonia are more likely indicators of an infection acquired prior to hospitalization (community-acquired) than an infection caused by the intubation necessitated by petitioner's worsening GBS condition during hospitalization. While Dr. Leist submitted medical literature with his expert report indicating that the onset of an infection less than 72 hours after hospitalization was deemed for purposes of the study to be a community-acquired infection, Resp. Ex. L4[46] at 78, he conceded that the very process of intubating petitioner could have forced colonized H. influenzae in petitioner's throat further into his respiratory tract and caused petitioner's pneumonia. Tr. II at 181.

Dr. Leist's hypothesis that the severity of Dr. Stewart's GBS and his limited recovery militated in favor of a finding that H. influenzae bacteria, and not petitioner's flu vaccine, was the more likely causal agent of petitioner's condition derived, at most, equivocal support from a letter to the editor about a case report. See Resp. Ex. L3.[47] As explained in the letter to the editor, the "most striking features" of the described patient with GBS subsequent to a H. influenzae infection were his severe symptoms and his poor recovery. Id. These features were notably "unlike those previously reported." Id. The authors' conclusion that "H. influenzae-related GBS does not always follow a benign clinical course" suggests to the undersigned that the severity of the clinical course of a patient's GBS is yet another factor to consider when evaluating whether a H. influenzae infection should be implicated as the causal agent. Id. But, it is not determinative.

Dr. Leist's opinion regarding alternative causation is premised upon a series of possibilities informed by current scientific understanding. The heft of the statistical probability to which Dr. Leist points can attach only if the series of presented possibilities are found persuasive. Even then, the statistical probability of a particular event occurring does not, in and of itself, defeat the legal probability that another event has occurred in an individual petitioner's case. The undersigned is mindful that legal probability and not medical certainty is the evidentiary guide for vaccine proceedings. Here, Dr. Leist urges the undersigned to accept the theory that petitioner's antecedent respiratory infection was caused by the later detected H. influenza infection. But petitioner's clinical presentation and test results at the time of his hospitalization infection were inconclusive regarding whether petitioner was suffering from a 10-day old infection. Moreover, Dr. Leist acknowledged that the act of intubation that occurred after petitioner's hospitalization was sufficient to have triggered petitioner's subsequent course of pneumonia.

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[46] D. H. Akbar, Bacterial Pneumonia: Comparison Between Diabetics and Non-Diabetics, 38 Acta Diabetol 77, 78 (2001).

[47] Shinji Tagami, et al., Fulminant Case of Guillain-Barré Syndrome with Poor Recovery and Depression Following Haemophilus Influenzae Infection (Letters to the Editor), 62 Psychiatry and Clinical Neurosciences 486, 486 (2008).

Having considered, on the whole, the evidence that supports respondent's position, the undersigned is not persuaded that respondent has met the shifted burden of proving alternative causation. The undersigned acknowledges that the evidence respondent has offered arguably could present a close call. But in the view of the undersigned, respondent's evidence does not defeat petitioner's claim. See Althen, 418 F.3d at 1280.

V. CONCLUSION

As established under prong one of the Althen analysis, petitioner has shown that the flu vaccine can lead to the onset of GBS. As established under prong three of Althen, petitioner has shown that within a medically appropriate time after receipt of the flu vaccine, he developed GBS. The complicating event in this case was petitioner's development of an unidentified respiratory illness after receipt of the flu vaccine and before the manifestation of the symptoms that led to petitioner's GBS diagnosis. Petitioner has presented a theory of causation that implicates both the flu vaccine and the subsequent respiratory infection and satisfactorily has established a logical causal sequence between receipt of his flu vaccine and the onset of his GBS as required under prong two of Althen. Respondent's claim of alternative causation is unavailing.

Having found that petitioner is entitled to vaccine compensation, the undersigned directs the parties to turn to evaluating petitioner's damages. **On or before July 22, 2011,** the parties shall contact chambers to schedule a status conference to discuss the damages phase of this case.

IT IS SO ORDERED.

s/ Patricia E. Campbell-Smith
Patricia E. Campbell-Smith
Chief Special Master