In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: February 20, 2026

JAMES G. STROUSE,	*	
Petitioner,	*	No. 19-793V
v.	*	Special Master Gowen
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Nancy R. Meyers, Turning Point Litigation, Greensboro, NC, for petitioner.
James V. Lopez, U.S. Department of Justice, Washington, D.C., for respondent.

RULING ON ENTITLMENT[1]

On May 30, 2019, James G. Strouse ("petitioner") filed his petition in the National Injury Vaccine Program. Petition[2] (ECF No. 1). Petitioner alleged that the hepatitis B vaccine he received on August 9, 2018 was the cause in fact of him developing Guillain-Barré syndrome ("GBS"). *Id.* After a review of the record, I find that petitioner has established by preponderant evidence that he is entitled to compensation.

[1] Pursuant to the E-Government Act of 2002, *see* 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services **because this opinion contains a reasoned explanation for the action in this case, I intend to post it on the website of the United States Court of Federal Claims.** The Court's website is at http://www.uscfc.uscourts.gov/aggregator/sources/7. Before the opinion is posted on the Court's website, each party has 14 days to file a motion requesting 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). An objecting party must provide the Court with a proposed redacted version of the opinion. *Id.* **If neither party files a motion for redaction within 14 days, the opinion will be posted on the Court's website without any changes.** *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. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to 34 (2012) (hereinafter "Vaccine Act" or "the Act"). Hereinafter, individual section references will be to 42 U.S.C. § 300aa of the Act.

I. Procedural history

Petitioner filed his claim for compensation on May 30, 2019. ECF No. 1. His claim was accompanied by medical records. *See* Petitioner's ("Pet'r") Exhibits ("Exs.") 1-10. On June 9, 2020, respondent filed a status report stating that he was not amenable to informal resolution of petitioner's claim and raised the issue of whether petitioner had GBS or chronic inflammatory demyelinating polyneuropathy ("CIDP"). Respondent ("Resp.") Status Report ("Rept.") ECF No. 21. I directed petitioner to file an expert report addressing the issue of petitioner's diagnosis.

Petitioner filed three expert reports of Lawrence Steinman, M.D.[3] Pet'r Exs. 13, 35, & 40. Respondent filed expert reports from Brian C. Callaghan, M.D.[4] and S. Mark Tompkins, PhD.[5] Resp. Exs. A, C, E, F. (ECF Nos. 32, 33, 38). On May 19, 2022, respondent filed the Rule 4(c) report, recommending against compensation, arguing that petitioner did not have GBS, and he cannot demonstrate by preponderant evidence that he had GBS and that petitioner's expert's theory of vaccine causation is unreliable, and that he cannot establish that the hepatitis B vaccine can cause GBS. Resp. Rept. 8-11.

[3] Dr. Lawrence Steinman is a neurologist and immunologist and is an Incumbent of GA Zimmerman Chair as Professor of Neurological Sciences, Neurology, and Pediatrics at Standford University. Pet'r. Ex. 34. He received his medical degree from Harvard University in 1973 and completed residency in Pediatrics and Adult Neurology at Standford University Hospital. *Id.* at 1. Dr. Steinman was elected to the National Academy of Sciences in 2015. *Id.* at 2. He is board certified in psychiatry and neurology. *Id.* Dr. Steinman is the Chairman of the Research Advisory Committee, Gulf War Illness for the U.S. Dept. of Veterans Affairs. *Id.* Dr. Steinman has been the lead investigator and author in numerous studies relating to multiple sclerosis and immunology. *Id.* at 6-49. Additionally, Dr. Steinman has been an editor to multiple journals relating to immunology and neurology. *Id.* at 5. Dr. Steinman has testified in the Vaccine Program before and admitted as an expert in immunology and neurology. Accordingly, Dr. Steinman is accepted as an expert in the field of immunology and neurology for purposes of deciding this matter.

[4] Dr. Brian Callaghan is a neurologist and Associate Professor of Neurology at the University of Michigan Health System. Resp't. Ex. B at 1. Dr. Callaghan received his medical degree from the University of Pennsylvania Medical Center and completed his residency at the University of Pennsylvania Medical Center. *Id.* He completed fellowships at the University of Michigan Health System. *Id.* Dr. Callaghan is board certified in psychiatry and neurology and electrodiagnostic medicine. *Id.* He is licensed to practice law in the state of Michigan. *Id.* Dr. Callaghan has been teaching neurology at the University of Michigan since 2009. *Id.* He is also a staff physician in the Neurology Department at the VA Ann Arbor Health System hospital. *Id.* at 2. Dr. Callaghan is a review for multiple medical journals, including JAMA Neurology and Neurology. *Id.* at 5. He has been authored or co-authored numerous medical articles in the field of neurology. *Id.* at 12-18. Dr. Callaghan has testified before the Vaccine Court on numerous occasions and has been admitted as an expert in the field of neurology. As such, Dr. Callaghan was accepted as an expert in the field of neurology for this matter.

[5] Dr. Stephen Mark Tompkins is the Assistant Department Head and Curriculum Coordinator for the Department of Infectious Diseases at the College of Veterinary Medicine at the University of Georgia. Resp't. Ex. D at 1-2. He received his undergraduate degree from the University of Illinois in 1990 and received his PhD from Emory University in 1997. *Id.* at 2. Afterwards, Dr. Tompkins became a research associate for the Department of Microbiology-Immunology at Northwestern University Medical School in Chicago, Illinois, and then became an Assistant Professor of Infectious Diseases at the College of Veterinary Medicine at the University of Georgia from 2005-2010. *Id.* Dr. Tompkins has remained at the College of Veterinary Medicine at the University of Georgia since 2005, and he became a tenured professor in 2016. *Id.* at 2. Dr. Tompkins' research is focused on infectious diseases and transmission, including influenza, and developing therapeutic approaches to viruses and infectious diseases. *Id.* at 16. He has authored or co-authored nearly 100 articles in the field of immunology. *Id.* at 29-39. Dr. Tompkins' opinion was accepted as an expert in the field of immunology.

On July 20, 2022, after reviewing the expert reports filed by both parties and the Rule 4(c) report, I held a Rule 5 Status Conference and recommended that the parties engage in litigative risk settlement negotiations. Rule 5 Order (ECF No. 42). Petitioner transmitted a demand to respondent and respondent filed a status report stating he wanted to "continue to defend." Resp. Status Rept. (ECF No. 44). After failed litigative risk negotiations, I ordered the case to ADR, where again the parties failed to resolve the matter. *See* Order Concluding ADR Proceedings (ECF No. 46).

On March 30, 2023, I held another status conference and ordered the parties to file any additional evidence to be considered and directed petitioner to file a motion for a ruling on the record. Scheduling Order (ECF No. 48). Petitioner filed additional medical records on July 17, 2023. Pet'r Exs. 41-44 (ECF No. 50).

On July 19, 2023, petitioner filed a motion for a ruling on the record. Pet'r Mot. (ECF No. 51). Respondent filed a response on January 16, 2024. Resp. Response to Pet'r Mot. (ECF No. 55). Petitioner filed a reply to respondent's response on March 21, 2024. Pet'r Reply (ECF No. 57). The undersigned granted respondent's motion to file a sur-reply, which was filed on June 25, 2024. Resp. Sur-Reply (ECF No. 61). Petitioner filed a response to respondent's sur-reply on September 5, 2024. Pet. Response Sur-Reply (ECF No. 64).

This matter is now ripe for adjudication.

II. Evidence submitted

a. Petitioner's medical records

On August 9, 2018, petitioner, a forty-three-year-old man, received a hepatitis B vaccination at the HMC Occupational Health Services. Pet'r. Ex. 1 at 1. He had received a hepatitis B vaccine one year earlier, on June 2, 2017. *Id.* Four days later, on August 15, 2018, petitioner had an appointment with Dr. James J. Martin, his primary care physician for high blood pressure, mild intermittent dizziness. Pet'r. Ex. 2 at 21. The note from this appointment indicates that petitioner's hypertension was stable on Losartan 100 mg/daily. *Id.* Petitioner was positive for palpitations. *Id.* at 22. He was diagnosed with "accelerated hypertension," and it was recommended that he switch to Losartan HCTZ 100 mg/25 daily and that he continue to monitor his blood pressure. *Id.* at 21.

Three days later, on August 18, 2018, petitioner went to the St. Luke's Hospital-Anderson Campus emergency department for a rapid heart rate, mild shortness of breath ("SOB") and left sided lower chest pain and tingling in his left arm that "lasted seconds." Pet'r. Ex. 3 at 8. Petitioner reported chest pain and shortness of breath. *Id.* Petitioner had elevated blood pressure but was discharged and advised to return if his symptoms worsened. *Id.* at 21.

On August 22, 2018, petitioner went to HMC Employee Health and reporting feeling "fatigued, elevated blood pressure." Pet'r. Ex. 10 at 84. He reported "weakness" "dizziness," and "bilaterally hand numbness." *Id.* He was rehydrated. *Id.* Petitioner's primary discharge diagnosis was "weakness." *Id.* at 92.

Petitioner returned to Dr. Martin on August 28, 2018 for a follow-up appointment after his two visits to the emergency room for elevated blood pressure. Pet'r. Ex. 2 at 24. Petitioner reported numbness and tingling in both feet and hands "with sensation of hand and lower extremity weakness." *Id.* Dr. Martin noted that petitioner had received the hepatitis B vaccine on August 9, 2018. *Id.* Under "neurological" petitioner reported that he was having difficulty finding words. *Id.* at 25. Petitioner's heart rate was elevated to 100 bpm. Petitioner's reflexes were all recorded at 1+ bilaterally. *Id.* at 25. Dr. Martin wrote that petitioner had "no ankle clonus," and that petitioner had normal strength in his upper and lower extremities. *Id.* Dr. Martin noted that petitioner was scheduled to see employee health the following day and he recommended that petitioner have a neurology consult "to rule out Guillain-Barré syndrome either at his place of employment or with St. Luke's neurology." *Id.* at 24.

On August 30, 2018, petitioner went to neurologist, Dr. Dipakkumar Pandya at MidJersey Orthopaedics for a consultation as recommended by Dr. Martin. Pet'r. Ex. 10 at 66; Pet'r. Ex 4 at 2. Dr. Pandya recorded that petitioner had received the hepatitis B vaccine on August 9th and that he had "tingling in legs and arms, as well as muscle weakness, fatigue." Pet'r. Ex. 4 at 2. The physical exam revealed that petitioner had "absent" deep tendon reflexes bilaterally, a wide based gait. *Id.* at 4. Under "Assessment and Plan," Dr. Pandya wrote, "Guillain-Barré syndrome following vaccination; Impression: Difficult call; started after vaccines; wide based gait; no DTRs; numbness; spreading numbness; neurologically-considering so many symptoms-he should be further evaluated. *Id.*

Petitioner returned to HMC Employee Health emergency department on August 30, 2018, to rule out Guillain-Barré syndrome. Pet'r. Ex. 10 at 71. Under the triage notes it stated, "pt sent by Dr. Pandya for eval r/o GB pt to be admitted and have lumbar tap, and IVIG, pt has 3 weeks of tingling in legs and arms on and off unsteady gait, no difficulty breathing." *Id.* Petitioner's strength was recorded as 4/5 in the lower extremities and "minimally reduced muscle strength but very close to 5/5 bilaterally in the upper extremities." Pet'r. Ex 5 at 7. Petitioner also had an "unsteady gait." *Id.* Petitioner was admitted for further evaluation and treatment with IVIG. *Id.*

Neurologist Dr. Pandya examined the petitioner on August 31, 2018. Pet'r. Ex. 5 at 10. He performed a physical examination and again noted that petitioner had absent deep tendon reflexes in both lower legs and that he was walking with a wide base. *Id.* at 13. Dr. Pandya wrote that petitioner had "probably normal" strength. *Id.* Dr. Pandya also noted that petitioner had some numbness in the upper hand and distal leg. *Id.* at 14. The "Impression and Discussion," was as follows:

> A 43-year-old gentleman who complains of a new onset of bilateral lower extremity numbness and weakness. He also reports that he did have a vaccination several weeks before. Clinical diagnosis would be difficult, presumably, the way we look at the clinical findings, this gentleman has a new onset of numbness in both lower legs; he had absent deep tendon reflexes in both lower legs; and at the same time he had a wide-based gait. He also has some numbness in the upper hand and distal leg. There could be multiple etiologies of his current symptoms. *Considering the symptoms started in such a way sub-*

> *acutely associated with this finding with the recent vaccination, the clinical diagnosis may be presumably Guillain-Barre syndrome.*

Id. at 14 (emphasis added). Dr. Pandya also opined that there may be some compounding issues, like hyperglycemia, but still concluded that "the subacute onset of symptoms were more concerning and that was the reason of having him evaluated with the current medical status." *Id.* Dr. Pandya initiated IVIG for five days. *Id.* A lumbar puncture showed that petitioner's CSF protein level was 41, with the normal range being 15-45 mg/dL. *Id.* at 60.

Petitioner was discharged from the hospital on September 4, 2018 and his discharge diagnosis was, "1) Suspected Guillain-Barré syndrome; 2) recent hepatitis B vaccine implicated in above." Pet'r Ex. 5 at 18. Petitioner had improved with IVIG and was able to ambulate without issue, according to the discharge summary. *Id.* Additionally, his hemoglobin A1c was 6.5 and controlled with diet. Petitioner reported that it had improved "from a few months ago." *Id.*

Petitioner had a physical therapy evaluation on September 11, 2018. Pet'r Ex. 6 at 2. Petitioner's primary diagnosis was, "Guillain-Barré syndrome following vaccination," and he was referred to physical therapy by neurologist, Dr. Pandya. *Id.* The assessment was that petitioner had "bilateral lower extremity and upper extremity weakness," and "gait slow speed, but otherwise normal." *Id.* at 3. It was recommended that petitioner return for two-three times a week.

On October 2, 2018, petitioner presented to MidJersey Orthopaedics for an EMG/NCS. Pet'r Ex. 4 at 43. The "Reason for Referral" stated, "A 43-year-old male who presented with hands and legs/feel numbness, trouble walking, absent DTRs on exam who was treated with 5 days of IVIG at HMC for GBS symptoms in leg started after Hep B vaccine. He had a spinal tap which was traumatic with normal CSF protein." *Id.* The results of the "motor nerve conduction study" showed slower conduction velocity in the bilateral peroneal and bilateral tibial nerves and the "sensory nerve action potential (SNAP) of the bilateral sural, bilateral medial and lateral planter and bilateral superficial peroneal nerves were absent." *Id.* at 44. The impression was of the EMG/NCS by Dr. Manish Viradia was, "an abnormal study suggestive of the mixed sensory/motor demyelinating polyneuropathy. This study is consistent with recent clinical diagnosis of Guillain-Barré syndrome." *Id.*

After the EMG/NCS, petitioner met with Dr. Viradia and petitioner reported that he was positive for gait disturbances and paresthesia. *Id.* at 12. Dr. Viradia noted that petitioner's gait was unsteady and he was having balance issues. *Id.* Petitioner's deep tendon reflexes were also absent bilaterally. *Id.* at 13. Dr. Viradia recommended that petitioner receive IVIG every two weeks and "no future hepatitis B vaccinations." *Id.* Additionally, an MRI of petitioner's spine was ordered. *Id.* at 14.

Petitioner returned to MidJersey Orthopaedics on November 16, 2018 and had an appointment with Dr. Pandya. *Id.* at 16. Petitioner reported that since re-starting IVIG he was doing "much better," and that his fatigue had improved. *Id.* However, he continued to have tingling and muscle spasms in his lower extremities. The MRI of petitioner's thoracic spine

showed diffuse degenerative joint disease at multiple levels and only mild degenerative disc disease in his cervical spine. *Id.* Petitioner stated that he was able to start driving short distances again and is occasionally using a cane. *Id.* Petitioner still had absent deep tendon reflexes bilaterally and a mild wide based gait. *Id.* at 18. Although petitioner's numbness was reported as "much better" Dr. Pandya ordered petitioner to complete six cycles of IVIG and to keep him updated on progress. *Id.* Additionally, petitioner was authorized to return to work part-time. *Id.*

On December 14, 2018, petitioner had a follow-up appointment with Dr. Pandya and he reported that his muscle weakness in legs had returned and the symptoms returned and continue to worsen. Pet'r Ex. 4 at 21. Petitioner still had absent deep tendon reflexes, and his gait was noted as "wide based." *Id.* at 22. Dr. Pandya ordered petitioner to complete six more cycles of IVIG and also recommended that petitioner seek treatment from his primary care physician for his high glucose number. *Id.* at 23. Additionally, Dr. Pandya noted that petitioner's "gait abnormality" was "multifactorial," and that petitioner should use a cane if necessary and continue to do physical therapy home exercises. *Id.*

Petitioner returned to Dr. Pandya on January 25, 2019, and petitioner reported that he had "plateaued with progress with PT in the past four weeks," however, he still experienced ongoing fatigue, along with having increased pain in the legs and forearms. Pet'r. Ex. 4 at 26. Petitioner also reported continued tingling in the lower legs and feet, with fatigue in his fingers. *Id.* Dr. Pandya noted that petitioner was to receive another dose of IVIG. *Id.* Petitioner's physical examination was remarkable for absent deep tendon reflexes and he was diagnosed with "sequelae of Guillain-Barre syndrome: continue IVIG as planned; continue physical therapy as you are." *Id.* at 28.

Petitioner had an appointment with his primary care physician, Dr. Martin, on February 21, 2019. Pet'r. Ex. 41 at 12. Dr. Martin diagnosed petitioner with Type 2 diabetes and elevated blood pressure. Dr. Martin noted that petitioner was experiencing "persistence fatigue since his diagnosis of GBS." Dr. Martin's physical examination included reflex scores, which showed petitioner had reflexes in all areas of 1+ bilaterally and in his upper and lower extremities. *Id.* at 15.

Petitioner returned to Dr. Pandya again on February 27, 2019 and then again on March 29, 2019. Pet'r. Ex. 4 at 31-38. At the March 29, 2019 appointment, petitioner reported "some tingling below the knee and scattered tingling," along with fatigue, dizziness, and leg soreness that was disturbing his sleep. *Id.* at 36. Petitioner again demonstrated absent deep tendon reflexes bilaterally (although it was not indicated if this was present in petitioner's upper or lower extremities). *Id.* at 37. Petitioner's diagnosis remained, "sequelae of Guillain-Barre syndrome," and it was recommended that petitioner continue his home exercise program. *Id.*

At a follow-up appointment with Dr. Pandya on May 13, 2019, petitioner reported "doing terrible," as a result of being recently diagnosed with pneumonia, which had increased the tingling sensation in his legs. Pet'r. Ex. 9 at 15. Dr. Pandya observed petitioner standing without support and noted that petitioner would use a cane occasionally. *Id.* The diagnosis of sequelae of GBS remained. *Id.* Dr. Pandya cleared petitioner for full-time work but recommend breaks for optimum functioning. *Id.* at 19. Petitioner reported 75% improvement of muscle

fatigue and weakness on June 21, 2019 to Dr. Pandya and stated that he was doing "much better." *Id.* at 21. Petitioner still had residual tingling in his hands and feet and he was working four days a week. *Id.*

At his next follow-up appointment on October 10, 2019, petitioner reported developing pain in his arms and legs, ranging from a 4/10-7/10, mostly at night. Pet'r. Ex. 9 at 26. Further, petitioner's tingling in his hands had worsened for four weeks and he had increased muscle fatigue and weakness, such that he began to re-use his cane. *Id.* The onset of these symptoms was reported as "towards the end of August." *Id.* Dr. Pandya ordered another EMG/NCS due to petitioner's age and sensory symptoms. *Id.* at 27.[6] Petitioner had a follow-up appointment on November 21, 2019, and petitioner was told that the "EMG-NCS was okay," although the record did not further elaborate. *Id.* at 3.

After an appointment on March 2, 2020, where petitioner described continued tingling in the legs and hands, Dr. Pandya prescribed petitioner 300 mg of Gabapentin. Pet'r. Ex. 11 at 10. This record does include some inconsistencies, including noting that petitioner's GBS diagnosis occurred after the flu vaccine. *Id.* at 9. The gabapentin appeared to improve petitioner's symptoms and at a June 2, 2020 appointment, petitioner reported doing better and having less pain. Pet'r. Ex. 43 at 7. Petitioner reported that the tingling had decreased and he felt almost near normal. *Id.* Dr. Pandya recommended that petitioner continue his current medication plan and also consider weight loss. *Id.* at 9. At an appointment on March 1, 2021, petitioner reported that his pain and tingling symptoms had decreased with the Gabapentin regime. *Id.* at 18. Dr. Pandya began to titrate petitioner down from 300 mg of Gabapentin to 100 mg over the course of two months. *Id.* at 21. However, by July 2021, when petitioner had stopped the Gabpentin, his symptoms returned. *Id.* at 24. Dr. Pandya returned petitioner to the 300 mg of Gabpentin. *Id.* at 27.

Over the course of the next year, petitioner continued to have appointments with Dr. Pandya. *See e.g.* Pet'r. Ex. 44. Petitioner continued with Gabapentin through 2022. At his appointment with Dr. Pandya on December 13, 2022, petitioner's symptoms were stable and his motor strength was recorded as "normal and symmetrical," but his reflexes were still absent. Pet'r. Ex. 44 at 9. Petitioner remained on 300 mg of Gabapentin until June 13, 2023, when Dr. Pandya suggested reducing the dosage again. *Id.* at 5. This was the last medical appointment relevant to petitioner's claim filed in this matter.

b. Petitioner's Expert's Opinion: Dr. Lawrence Steinman

Petitioner submitted three expert reports by Dr. Lawrence Steinman, neurologist. Pet'r. Exs. 13, 35, 40. Dr. Steinman opined that the hepatitis B vaccine petitioner received on August 8, 2018, was the cause of him developing GBS. Pet'r. Ex. 13 at 7.

1. Opinion Regarding Petitioner's Diagnosis

[6] Respondent correctly observed that the results of the EMG/NCS from November 2019 were not filed in this matter. *See* Resp. Response at n. 7.

First, Dr. Steinman addressed petitioner diagnosis, stating, "[t]he diagnosis here is GBS." Pet'r. Ex. 13 at 8. Dr. Steinman referred to the National Institute of Neurological Disorders and Stroke ("NINDS") Fact Sheet on GBS, describes symptoms of GBS as the following:

> Unexplained sensations often occur first, such as tingling in the feet or hands, or even pain (especially in children), often starting in the legs or back. Children will also show symptoms with difficulty walking and may refuse to walk. These sensations tend to disappear before the major, longer-term symptoms appear. Weakness on both sides of the body is a major symptom that prompts most people to seek medical attention. The weakness may first appear as difficult climbing stairs or with walking. Symptoms often affect the arms, breathing muscles, and even the face, reflecting more widespread nerve damage. Occasionally, symptoms start in the upper body and move down to the legs and feet.
>
> Most people reach the greatest stage of weakness within the first two weeks after symptoms appear, by the third week, 90 percent of affected individuals are at their weakest.

Pet'r. Ex. 16 at 2.[7] Further, NINDS explains that GBS is diagnosed by physician examinations and patients may have "deep tendon reflexes in the legs, such as knee jerks," are absent and reflexes in the arms may also be lost. *Id.* at 5. Key diagnostic findings of GBS are considered:

(1) Recent onset, within days to at most four weeks of symmetric weakness, usually starting in the legs;
(2) Abnormal sensations such as pain, numbness, and tingling in the feet that accompany or even occur before weakness;
(3) Absent or diminished deep tendon reflexes in weak limbs;
(4) Elevated CSF protein without elevated cell count;
(5) Abnormal nerve conduction velocity findings
(6) Sometimes, a recent viral infection or diarrhea.

Id. at 6. Relevant to this matter, the NINDS fact sheet also explains the long-term outlook for patients with GBS and explains that the point of greatest weakness occurs days to at most 4 weeks after the first symptom occurs. *Id.* at 7. Additionally, the Fact Sheet Provides:

> Symptoms usually stabilize at this level for a period of days, weeks, or sometimes even months. Recovery, however, can be slow or incomplete. The recovery period may be as little as a few weeks up to a few years. Some individuals still report ongoing improvement after 2 years. About 30 percent of those with GBS have residual weakness after 3 years.

Id. at 8. Finally, the Fact Sheet states, "Ongoing fatigue, pain, and other annoying sensations can sometimes be troublesome." *Id.*

[7] National Institute of Neurological Disorders and Stroke (NINDS): Guillain-Barre Syndrome Fact Sheet, Pub. June 2018. [Pet'r. Ex. 16].

In his second report, Dr. Steinman addresses respondent's expert, Dr. Callaghan's criticism that petitioner did not have GBS. *See generally* Resp't. Ex. A. Dr. Steinman stated that petitioner's treating neurologist, Dr. Pandya, is a board-certified neurologist who diagnosed petitioner with GBS. Pet'r. Ex. 35 at 2. Additionally, Dr. Steinman criticized Dr. Callaghan's reliance on the Brighton Collaboration diagnostic criteria for GBS for diagnosing patients in clinical practice, but that the GBS criterion developed by the Brighton Collaboration was for the use of case standardization for research for vaccine safety studies. Pet'r. Ex. 35 at 5-6; *see also* Pet'r. Ex. 36.[8]

Dr. Steinman referred to the Fokke et al. article which examined GBS patients' data and applying the Brighton criteria, found that only 61% of their patients would meet the level criterion. Pet'r. Ex. 36 at 8. Fokke explained that the "predominant cause" for not reaching the Brighton Level 1 classification was a normal protein level in the CSF. *Id.* at 9. Fokke discussed the clinical course of the GBS patients and stated that "all patients developed bilateral limb weakness, which generally involved both upper and lower extremities," and that "decreased reflexes in paretic arms or legs were found initially in 91% of patients and in all patients during follow-up." *Id.* at 1. Further, Fokke explained that 97% of GBS patients reached their nadir in four weeks and all patients in 6 weeks. *Id.* Finally, the authors noted that in a minority of GBS patients, they may "present with an extended progressive phase up to 6 weeks, weakness of the legs only and initial normal reflexes, and may show some clinical fluctuations later in the disease course." *Id.* at 11.

Quoting Fokke, Dr. Steinman wrote, "Without accurate biomarkers, the clinical features will remain the hallmark for the diagnosis of Guillain-Barre syndrome." Pet'r. Ex. 35 at 6; *see also* Pet'r. Ex. 36 at 11. He stated that he agreed with petitioner's neurologist, Dr. Pandya's decision to treat petitioner for GBS and that in clinical practice, it is impractical for a treating physician to wait for "all the boxes to be ticked." Pet'r. Ex. 35 at 6.

2. Opinion Regarding Vaccine Causation

With respect to vaccine causation, Dr. Steiman opined that through the mechanism of molecular mimicry, the hepatitis B vaccine can cause GBS. Pet'r. Ex. 13 at 11; Pet'r. Ex. 35 at 9-13. In his first report, Dr. Steinman cited the Fujinami et al. article, which provides that molecular mimicry is a mechanism that can initiate an autoimmune disease. Pet'r. Ex. 13 at 9; *see also* Pet'r. Ex. 19.[9]

Dr. Steinman stated that "[a]ll hepatitis B vaccines contain Hepatitis B surface antigen HepBsAg," and he focused his theory on an immune cross reaction between that antigen and Contacin-1, which is a protein in the peripheral nervous system, that has been found to be targeted by antibodies in GBS. Pet'r. Ex. 13 at 12-20. He performed a BLAST search between HepBsAg and contactin-1 to determine if there were homologies between the proteins and found

[8] Fokke, C. et al., *Diagnosis of Guillain-Barre Syndrome and Validation of Brighton Criteria,* 137 (Pt. 1) Brain 33-43 (2014). [Pet'r. Ex. 36].

[9] Fujinami, R. et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease,* 19 Clin. Microbio. Rev. 80-94 (2006). [Pet'r. Ex. 19].

that "several sequences…[have] at least five of 12 identical amino acids." *Id.* at 17. Using the sequences that have 6 out of 12 identical amino acids, he performed a search of the Immune Epitope Database ("IEDB") and reported that IEDB found, "The sequence SVFSKFIPLIPI has been described…in a human and the antigen is contactin-1," and "the sequence NILSPFLPLLPI has been seen [in a human] and is associated with HepBsAg." *Id.* at 19. Then applying the standards from the two Gautam papers, in which Dr. Steiman was a contributor, he asserted that "such a degree of homology would be sufficient to trigger clinically relevant neuroinflammation, since there are 6 out of 12 identical amino acids in the consecutive shared sequences between contactin-1 and HepBsAg." *Id.*

Dr. Steinman referred to the Devaux et al. and Miura et al. articles to support his theory that contactin-1 is targeted by autoantibodies in GBS. Pet'r Ex. 13 at 12. The Devaux researchers investigated the prevalence of antibodies against nodal adhesion molecules in patients with GBS or chronic inflammatory demyelinating polyneuropathy (CIDP). Pet'r. Ex. 25 at 1.[10] The authors explained that the "paranodal axoglial junctions composed of contactin-associated protein…form a barrier to the lateral diffusion of nodal channels." *Id.* at 2. Through their study, the authors found that in 43% of GBS patients had IgG targeting nodal and paranodal antigens and identified "NF186, gliomedin, and contactin as the immune targets of autoantibodies in many of these patients." *Id.* at 12. The authors concluded that, in conjunction with their work and other studies, that "the node is the locus of the immune attack in these neuropathies and suggest that several antigens located at nodes and paranodes in human nerves are targets of IgG antibodies." *Id.* at 14. Miura et al. also examined the role that autoantibodies to the nodes of Ranvier play in CIDP and found that "neurofascin-186, gliomedin and contractin-1 as the targets of autoantibodies in CIDP." Pet'r. Ex. 26 at 1.[11] The authors stated, "[I]n the current study we identified CNTN1 as a target for the IgG autoantibodies…We also showed that the IgG4 antibodies specifically recognize the paranodes on teased nerve fibers and human CNTN1 by ELISA." *Id.* at 6. Miura found "IgG autoantibodies against CNTN1 were identified in 16 sera from patients with CIDP and five with GBS." *Id.* at 4.

Additionally, Dr. Steinman referenced the Food and Drug Administration ("FDA") approved package insert for the hepatitis B vaccine petitioner received, and observed that it included a warning that serious adverse events post-vaccination include nervous-system disorders such as, "encephalitis, encephalopathy, migraine, multiple sclerosis, neuritis, neuropathy including hypoesthesia, paresthesia, *Guillain-Barré Syndrome,* and Bell's palsy, optic neuritis…." Pet'r. Ex. 13 at 12; *see also* Resp't. Ex. C, Tab 6.[12] Dr. Steinman acknowledged that the package insert includes a caveat that explains the reports of adverse events post-vaccination "are reported voluntarily from a population of unknown size, it is not always possible to reliably estimate their frequency or establish a causal relationship to the vaccine." Pet'r. Ex. 13 at 12.

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[10] Devaux, J. et al., *Nodal Proteins are Target Antigens in Guillain-Barré Syndrome,* 17 J. Peripheral Nervous Sys. 62-71 (2012). [Pet'r. Ex. 25].

[11] Miura, Y. et al., *Contactin-1 IgG4 Associates to Chronic Inflammatory Demyelinating Polyneuropathy with Sensory Ataxia,* 138 Brain 1484-1491 (2015). [Pet'r. Ex. 26].

[12] Engerix-B, Package Insert. [Resp't Ex. C, Tab 6].

In his supplemental expert report, Dr. Steinman addressed Dr. Callaghan's criticism that there were no epidemiological studies associating hepatitis B to GBS. Pet'r. Ex. 35 at 7. Dr. Steinman wrote that the Baxter et al. study referred to by Dr. Callaghan "found that patients receiving the hepatitis B vaccine had lower odds of developing GBS than those not receiving this vaccine." *Id.* Baxter et al. examined the possible risk of GBS following vaccination within a six-week period and ultimately concluded that there was no increased risk of GBS following any vaccination, including the influenza vaccination. Resp't. Ex. A, Tab 5.[13] Baxter identified 25 cases of GBS within six week of vaccination, documenting one case of GBS thirty-five days after a hepatitis B vaccine. *Id.* at 5. Importantly, Baxter concluded:

> Although we had limited power to fully assess the risk of GBS following vaccination due to the rarity of the outcome, the low numbers of GBS cases that were temporally associated with vaccination, coupled with our results, provide reassurance that the risk of GBS following any vaccine, including influenza vaccines, is extremely low.

Id. at 7.

Finally, Dr. Steinman addressed the onset of petitioner's GBS, opining that if the onset was two-days post-vaccination, is still an appropriate timeframe for a recall response sufficient for petitioner to develop symptoms related to GBS. Pet'r. Ex. 13 at 20; Pet'r. Ex. 35 at 13-15. Dr. Steinman observed that petitioner's August 9, 2018 hepatitis B vaccine was the second in the series of vaccinations, and petitioner would likely have circulating memory B and T cells from the prior vaccination. Pet'r. Ex. 13 at 20. Dr. Steinman referenced the Chapter in Vaccine by Siegrist, which discusses vaccine immunology, and wrote that following a second exposure to a vaccine there is a rapid onset of a secondary response. Pet'r. Ex. 13 at 21; *see also* Pet'r. Ex. 32 at 10.[14] The Siegrist chapter states, "booster exposure to antigen reactivates immune memory and results in a rapid (<7 days) increase of IgG antibody titer." Pet'r. Ex. 32 at 12. Siegrist explains that booster immunization or exposure to the antigen activates memory B cells, resulting in rapid proliferation and differentiation into plasma cells that produce very large amounts of higher affinity antibodies. *Id.* Dr. Steinman also referred to the Schonberger article, which examined the association between GBS and the 1976 swine flu vaccine, and stated that the article showed "an increased incidence in 0 to 1 days and 1 to 3 days." Pet'r. Ex. 13 at 22; *See also* Pet'r. Ex. 33 at 9.[15] Dr. Steinman asserted that both Schonberger and Siegrist demonstrate that the onset of GBS symptoms 48-hours post-vaccination is an appropriate timeframe for a recall response. Pet'r. Ex. 13 at 24; *see also* Pet'r. Ex. 35 at 14.

c. Respondent's Experts' Opinions

Respondent submitted expert reports from neurologist, Dr. Brian Callaghan and immunologist, Dr. Mark Tompkins. Resp't. Exs. A, C, E, & F. Dr. Brian Callaghan focused on

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[13] Baxter, R. et al., *Lack of Association of Guillain-Barre Syndrome with Vaccination,* 57(2) Clin. Infect. Disease 197-204 (2013). [Resp't Ex. A, Tab 5].

[14] Siegrist, C., *Chapter 2: Vaccine Immunology* (2013). [Pet'r Ex. 32].

[15] Schonberger, L. et al., *Guillain-Barré Syndrome Following Vaccination in the National Influenza Immunization Program,* 100 Am. J. of Epidem. 105-123 (1979). [Pet'r. Ex. 33].

petitioner's diagnosis, arguing that petitioner did not have GBS, but he also opined that the hepatitis B vaccine was not the cause of petitioner's condition. Resp't. Ex. A at 5. Dr. Tomkins focused his reports on whether the hepatitis B vaccine could cause GBS or other neurological conditions. Resp't Ex. C & F.

1. Opinion of Dr. Callaghan

Respondent submitted two reports by Dr. Callaghan, with his focus being on petitioner's diagnosis. Resp't. Ex. A & E.

Dr. Callaghan stated that "the cause of petitioner's symptoms is not GBS," mostly because "petitioner does not meet the Brighton Collaboration diagnostic criteria for GBS." Resp't. Ex. A at 4. After listing the Brighton Collaboration diagnostic criteria for GBS, Dr. Callaghan stated that petitioner did not meet any of criterion, except he acknowledged that petitioner had reduced reflexes in his legs. Resp't. Ex. A at 4.

With respect to the Brighton Collaboration criteria 2-4, Dr. Callaghan stated that petitioner's illness was not monophasic, failing criteria 2. Resp't. Ex. A at 4. He stated that petitioner's medical records "clearly demonstrated" "marked improvement in September 2018, followed by his subsequent decline, noted on October 2, 2018." *Id.* Dr. Callaghan also stated that petitioner's neurologist noted petitioner had disease progression on 12/14/2018, 3/29/2018, 5/13/2019, and 10/10/2019, thus being inconsistent with criteria 3 and 4. *Id.*

Further, Dr. Callaghan argued that petitioner's EMG/NCS was inconsistent with GBS, as it did not demonstrate a 20% reduction of conduction velocities in two or more nerves and/or 25% prolongation of distal motor latencies. *Id.*; Resp't Ex. E at 1. Dr. Callaghan referred to the Asbury et al. article, which in addition to providing a proposed electrodiagnostic criteria for demyelination of the peripheral nerve in GBS, also provides "Features Required for Diagnosis," which includes:

A. Progressive motor weakness of more than one limb. The degree ranges from minimal weakness of the legs, with or without mild ataxia, to total paralysis of the muscles of all four extremities and the trunk, bulbar and facial paralysis, and external ophthalmoplegia.
B. Areflexia (loss of tendon jerks). Universal areflexia is the rule, though distal areflexia with definite hyporeflexia of the biceps and knee jerks will suffice if other features are consistent.

Resp't. Ex. A, Tab 2 at 1.[16] Asbury also states mild sensory symptoms or signs and certain autonomic dysfunction such as tachycardia, postural hypotension, hypertension, and vasomotor symptoms, are "features strongly supportive of the diagnosis." *Id.* at 1. However, Dr. Callaghan wrote that petitioner's symptoms and findings, "including not feeling well, elevated blood

[16] Asbury, A. & Cornblath, D., *Assessment of Current Diagnostic Criteria for Guillain-Barre Syndrome*, 27 Ann. Neurol. S21-S24 (1990). [Resp't. Ex. A, Tab 2].

pressure, dizziness, fast heart rate, chest pain, and shortness of breath," are all quite atypical for GBS. Resp't. Ex. A at 4.

Dr. Callaghan also argued that petitioner had a normal protein in his CSF, which "makes a GBS diagnosis incredibly unlikely." Resp't. Ex. A at 4; Resp't. Ex. E at 2. Dr. Callaghan stated, "an elevated protein on a lumbar puncture approaches 100% after the first two weeks after symptom onset and the petitioner's lumbar puncture was performed 3-4 weeks after onset." Resp't. Ex. A at 4. To Dr. Callaghan, the normal lumbar puncture was further evidence that petitioner did not have GBS.

Dr. Callaghan also opined that hepatitis B was not the likely cause of GBS, as "there is no convincing evidence supporting a likely association between hepatitis B vaccination and GBS." Resp't. Ex. A at 5. He acknowledged that there has been temporal association reported between the onset of hepatitis B and GBS, but argued that case reports are not reliable evidence of causation. *Id.* He referred to the McMahon et al. article, which examined adverse reactions to the hepatitis B vaccine, and asserted that this article finds that "the incidence of GBS is not increased in those receiving the hepatitis B vaccine" compared to the incidences of GBS in the general population. Resp't. Ex. A at 5. Dr. Callaghan also referenced the Baxter et al. study, a more recent study than the McMahon study, and posited that Baxter also found that "patients receiving the hepatitis B vaccine had a lower odd of developing GBS than those not receiving the vaccine." Resp't. Ex. A at 5. Baxter et al. examined incidences of GBS six-weeks after vaccination and identified 25 patients with confirmed GBS diagnoses. Resp't. Ex. A, Tab 5 at 5.[17] Baxter acknowledged, "Cases of GBS have been noted following receipt of multiple vaccines, including tetanus, rabies, polio, and hepatitis B, but to date there has been insufficient evidence to show that these cases were causally related to the immunization." *Id.* Further, Baxter stated that they "are unable to exclude any possible association between vaccines and GBS" despite the review of a very large captured population. *Id.* Baxter concluded, "…the low number of GBS cases that were temporally associated with vaccination, coupled with our results, provide reassurances that the risk of GBS following any vaccine, including influenza vaccines, *is extremely low*." *Id.* at 7 (emphasis added).

Dr. Callaghan's ultimate conclusion was that petitioner did not have GBS and that "there is no reliable support for the hypothesis that hepatitis B (vaccine) is a likely cause of GBS." Resp't. Ex. A at 5; Resp't. Ex. E at 2.

2. Opinion of Dr. S. Mark Tompkins

Respondent submitted two expert reports from immunologist, Dr. Mark Tompkins, who opined that the hepatitis B vaccine cannot cause GBS and Dr. Steinman's theory of molecular mimicry is unreliable. Resp't. Ex. C at 11; Resp't. Ex. F at 4.

Dr. Tompkins first discussed the safety of the hepatitis B vaccine, as evidenced by the World Health Organization's 2017 and the Advisory Committee on Immunization Practices 2018 reports recommendations on the hepatitis B vaccine. Resp't Ex. C at 4. Focusing on the AICP

[17] Baxter, R. et al., *Lack of Association of Guillain-Barre Syndrome with Vaccinations*, 57(2) Clin. Infect. Disease 197-204 (2013). [Resp't. Ex. A, Tab 5].

Report, Dr. Tompkins states that a review of the Vaccine Safety Datalink, found that there was no evidence of association between the hepatitis B vaccine and GBS. *Id.*; *see also* Resp't. Ex. C, Tab 5.[18]

He then opined that there is not sufficient evidence to support Dr. Steinman's theory of molecular mimicry. Resp't. Ex. C at 6. Dr. Tompkins acknowledges that Dr. Steinman's "description of the limited amino acid identity required to elicit neuroinflammation" is accurate, but that these experiments were run on mice using an "extremely potent, inflammatory adjuvant," and two injections of the pertussis toxin to induce neuroinflammation, which is very different than the hepatitis B vaccine. *Id.* at 6.

Dr. Tompkins then argues that Dr. Steinman's identification of 50% homology is not indicative of molecular mimicry. Resp't. Ex. C at 6. He provided information from the BLAST website regarding E-values, which explains that "Expect-value is a parameter that describes the number of hits one can "expect" to see by chance when searching a database of a particular size," and that, "The lower the E-value, or the closer it is to zero, the more "significant" the match is. However, keep in mind that virtually identical short alignments have relatively high e-values. This is because the calculation of the E values takes into account the length of the query sequence. This high e-values make sense because shorter sequences have a higher probability of occurring in the database purely by chance." *Id.* at 6-7. Dr. Tompkins stated that Dr. Steinman's search for GGIYTCFAENNR, with five out of 12 matched amino acids has an expect value of 6.6, thus given the high e-value, there is a "reasonable likelihood of having six or less amino acids with homology between 1007 and 226 amino acids due to chance alone." *Id.* at 7. Dr. Tompkins stated that he replicated Dr. Steinman's BLAST search, only reducing the e-values of peptides with four amino acids similarity and found "no significant similarity found." *Id.*

Dr. Tompkins acknowledged that Dr. Steinman's search of the Immune Epitope Database ("IEDB") resulted in identifying peptides that have partial homology with Contactin-1 or HepBsAg may be isolated from HLA proteins expressed on a tumor cell line or stimulate cytokine production in a culture assay," but stated that this finding does not show evidence "that these or any other peptides derived from the hepatitis B vaccine are molecular mimics or elicit neuroinflammation." *Id.* at 7.

Dr. Tompkins also argued that identification of similar amino acid sequence homology is insufficient alone to prove molecular mimicry. Quoting the Institute of Medicine's "Adverse Effects of Vaccines: Evidence and Causality," Dr. Tompkins wrote:

> Linear amino acid sequence homology of even similar conformational structure between an exogenous agent and a self-antigen alone are not sufficient to prove that molecular mimicry is the pathogenic mechanism for a disease. Many such homologies exist, and the vast majority of these are not associated with biologically relevant autoimmune phenomena or actual human disease.

[18] Centers for Disease Control and Prevention, *Prevention of Hepatitis B Virus Infection in the United States: Recommendations of the Advisory Committee on Immunization Practices*, 67 Morb. & Morality Weekly Report (2018). [Resp't. Ex. C, Tab 5].

Id. at 8; *see also* Resp't. Ex. C, Tab 7.[19] The IOM also provides, "While molecular mimicry is a well-established mechanism in selected animal models, its relevance to human autoimmune disease remains in most cases to be convincingly proven," and identifies "experimental evidence that suggests or implicates [molecular mimicry] in certain human autoimmune diseases." Resp't. Ex. C, Tab 7 at 100. One such molecular mimic identified by the IOM is "demyelinating diseases and hepatitis B," providing:

> Amino acid homology between myelin basic protein (MBP) and hepatitis B virus polymerase has been reported. In addition, injection of a HBPV immunologic epitope shared with MBP into rabbits resulted in an EAE-like disease, antibodies against MBP, and T-cell reactivity. However, infection with hepatitis B is not associated with the development of demyelinating disease. Furthermore, the recombinant vaccines contain hepatitis B surface antigen, not hepatitis B virus polymerase.

Id.

Dr. Tompkins additionally cites to the Ricco article, which explore the amino acid sequence of the hepatitis B virus and human proteins, and stated that the authors identified "more than 65 peptides having at least five consecutive amino acids with complete identity" and the demonstration of homology of five to nine amino acids is "unremarkable." Resp't. Ex. C at 8. The authors of this study, however, stated that their study "provid[ed] clear-cut analysis of the identity platform linking [hepatitis B virus] and homo sapiens proteomes," and "[b]ased on the need for five or six amino acids to induce a monoclonal antibody response, the 65 heptapeptide overlaps might clearly induce autoimmune reactions." Resp't. Ex. C, Tab 9 at 2.[20] The authors also wrote that, "the nature of the overlapping is also of interest since a number of viral motifs occur in human proteins that are crucially involved in the neuronal structure and functions." *Id.*

In his supplemental report, Dr. Tompkins argued that the Lanz et al. article which Dr. Steinman contributed to, does not support the theory of molecular mimicry as the mechanism for the hepatitis B vaccine to cause GBS. Resp't. Ex. F at 2. Dr. Tompkins stated that Dr. Steinman and his colleagues "used extensive empirical analysis to identify a proposed molecular mimic," and did not rely on a BLAST search, followed by an IEDB search to identify the human proteome epitope to which the EBV would bind to, to cause multiple sclerosis. *Id.* at 3. Dr. Tompkins stated that "it makes it impossible to predict possible cross-reactive epitopes based on linear sequence identity," because "key contact amino acids have long spans of amino acids between them or are located on different polypeptides." *Id.* at 2.

With respect to the onset of petitioner's GBS, Dr. Tompkins argues that the onset of GBS symptoms two days after petitioner received the second hepatitis B vaccine is "improbable." Resp't. Ex. C at 10. Dr. Tompkins asserts that the medical literature Dr. Steinman relied upon to support his opinion that a two-day onset after vaccination is medically plausible for a recall

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[19] Institute of Medicine ("IOM"), *Adverse Effects of Vaccines: Evidence and Causality* (2012). [Resp't. Ex. C, Tab 7].

[20] Ricco, R. et al., *Hepatitis B virus and Homo sapiens Proteome-wide Analysis: A Profusion of Viral Peptide Overlaps in Neuron-specific Human Proteins*, 4 Biologics: Targets & Therapy 75-81 (2010). [Resp't. Ex. C, Tab 9].

response, is unsupportive because the articles either reference a different type of vaccine or discuss a different type of immune response, such as a B-cell, instead of a T-cell mediated response. *Id.*

Specifically, Dr. Tompkins states that the chapter by Siegrist found in Vaccines, depicts a rapid B-cell response after secondary exposure to a vaccine, and Dr. Steinman's theory of molecular mimicry "does not involve a B cell epitope mimic, [but] rather, he proposes a cross-reactive T-cell response between an epitope in the HBV vaccine and Contactin-1." Resp't. Ex. C at 9 (citing Pet'r. Ex. 13). Dr. Tompkins also argues that the Schonberger article is equally inapplicable to this case, because "97% of the GBS cases associated with the A/New Jersey vaccine were in persons ≥20 years of age (median age of 46), so all the affected individuals had pre-existing immunity to the influenza virus from repeated exposures to whole virus vaccines and (more likely) infection. Thus, the GBS cases associated with the 1976 A/New Jersey vaccine could be related to prior influenza virus infection, repeated priming and boosting, expanding memory cell populations, thus enabling a more rapid immune response following vaccination." *Id.* Dr. Tompkins wrote, "In contrast, [petitioner] had a single prior exposure to 20ug HBsAg from the initial Energix-B vaccine, which would not expand the memory immune cell population to the extend that the repeated influenza exposures would have done for the 1976 influenza vaccine." *Id.* Dr. Tompkins also states a potential association between the swine flu vaccine and GBS does not provide "substantial support for a recombinant HBsAg vaccine…being associated with GBS, as these are two different vaccines." *Id.*

Dr. Tompkins also argued that the Degrassi article does not demonstrate a "significant proliferative response" of antibody or humoral response. Resp't. Ex. C at 10.[21] Dr. Tompkins referred to the Leroux-Roels study, which studied the humoral and cellular immune response to the hepatitis B vaccines and stated that the authors "did not see significant proliferative response at six days following booster," and that "less than one third of vaccinated subjects seroconverted by day six-post boost." *Id.* Leroux-Roels studied the cellular and humoral antibody responses in fifty vaccine recipients, and found it was difficult to induce or detect hepatitis B antigen specific lymphoproliferation in cultures and explained, "Antigen specific proliferation could only be revealed after a second round of *in vitro* stimulation with HBsAg. Comparable results were obtained by Celius et al. who found that HBsAg specific response in only one out of ten vaccinees if fresh PBMC were used and in three out of these ten subjects when PBMC were first stimulated *in vitro* with HBsAg maintained in IL-2 and rechallenged with HBsAg." Resp't. Ex. C, Tab 14.[22] The authors, however, did observe that some vaccinees were called "fast responders" and others were "slow or non-responders," and that there was variability of the humoral immune response in vaccine recipients, raising the question of "why HBsAg is recognized less efficiently and induces less vigorous T-cell responses in some individuals and how the selective deficiency can be overcome." *Id.* at 6. Dr. Tompkins characterized the Leroux-Roels article standing for the proposition that "there was no measurable T-cell response to the Energix-B vaccine," however, the study did not actually make this conclusion, but instead

[21] Degrassi, A. et al., *Cellular Response and Anti-HBs Synthesis in vitro After Vaccination with Yeast-Derived Recombinant Hepatitis Vaccine* 10 Vaccine 617-622 (1992). [Resp't. Ex. C, Tab 13].
[22] Leroux-Roels, G. et al., *Correlation between In Vivo Humoral and In Vitro Cellular Immune Responses Following Immunization with Hepatitis B Surface Antigen (HBsAg) Vaccines*, 12 Vaccine 812-818 (1994). [Resp't. Ex. C, Tab 14].

showed that 24 patients were classified as "fast responders" with a anti-HB response ≥ 10 IU six days after vaccination. Resp't. Ex. C, Tab 14 at 3.

Dr. Tompkins concluded in both reports that "there is no evidence of association between HBV vaccination and GBS," and that Dr. Steinman's theory of molecular mimicry between the HBsAG and Contactin-1 protein, is unreliable because there "no evidence that these epitopes are elicited by vaccination or found in GBS patients." Resp't. Ex. C at 10; Resp't. Ex. F at 4. Further, Dr. Tompkins concluded that the timing between vaccination and the onset of petitioner's symptoms is not supported by the medical literature. Resp't. Ex. F at 4.

III. Legal Standard

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

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

A. Nature of Injury

The Federal Circuit established the test for actual causation of an off-Table injury in *Althen v. Sec'y of Health & Hum. Servs.*, 418 F.3d 1274, 1278. In that case: "There was no dispute as to whether the petitioner, Margaret Althen, actually suffered from a central nervous system demyelinating disorder. Therefore, the Federal Circuit was not presented with a case in which the diagnosis itself was questioned, but one in which causation of the injury by the vaccine was the issue in dispute." *Doe v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 597, 611 (2010) (citing *Althen*, 418 F.3d at 1282), *aff'd, Lombardi v. Sec'y of Health & Hum. Servs.*, 656 F.3d 1343 (Fed. Cir. 2011).

Special masters are generally not tasked with diagnosing injuries. *See Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 549 (Fed. Cir. 1994). In *Contreras*, the Federal Circuit explained: "The general rule is that a special master should not conduct a differential diagnosis, at the outset of the causation analysis, to choose one diagnosis over another, or over a

combination of diagnoses." *Contreras v. Sec'y of Heath & Hum. Servs.*, 107 Fed. Cl. 280, 293 (2012) (citing *Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 149–50 (Fed. Cir. 2011); *Lombardi*, 656 F.3d at 1351; *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1345 (Fed. Cir. 2010); *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1382 (Fed. Cir. 2009); *Althen*, 418 F.3d at 1277, 1280–81).

However, the Federal Circuit has recognized two exceptions that permit special masters to opine on a petitioner's diagnosis. *See Contreras*, 107 Fed. Cl. at 293. In *Broekelschen*, the Federal Circuit determined that "in an atypical case, where 'the question of causation turns on which injury [the petitioner] suffered,' the special master is permitted to choose between two competing diagnoses of dissimilar diseases as a first step in the causation analysis." 618 F.3d at 1346. There, the Federal Circuit noted that the case was atypical because "1) the injury itself was in dispute; 2) the proposed injuries differed significantly in their pathology; and 3) the question of causation turns on which injury petitioner suffered." *Id.* at 1343–44. Importantly, the *Broekelschen* court distinguished cases where differing diagnoses of the alleged vaccine injury were along a continuum of related conditions, observing that "in those cases, a precise diagnosis would be unnecessary for the causation analysis." *Id.* at 1346 (distinguishing *Andreu*, 569 F.3d at 1378, 1381 and *Kelley v. Sec'y of Health & Hum. Servs.*, 68 Fed. Cl. 84, 100–01 (2005)).

In *Lombardi*, the Federal Circuit recognized a second exception to the general rule that special masters should not diagnose alleged vaccine injuries. 656 F.3d at 1353; *Contreras,* 107 Fed. Cl. at 294–95. There, petitioner's experts disagreed as to petitioner's injury: one concluding that she suffered from transverse myelitis while the other expert testified that petitioner suffered from either chronic fatigue syndrome or systemic lupus erythematosus. *Lombardi,* 656 F.3d at 1346, 1352.

The Secretary's expert disputed those diagnoses and proposed five alternative possible diagnoses. *Id.* at 1352. In holding that it was appropriate for the special master to initially determine "what injury, if any, was supported by the evidence presented in the record before applying the Althen test to determine causation," the Federal Circuit reasoned that "[i]n the absence of a showing of the very existence of *any* specific injury of which the petitioner complains, the question of causation is not reached." *Id.* at 1352–53. Therefore, the Federal Circuit held that a special master is permitted under *Lombardi* "to attempt to diagnose the petitioner's illness in an unusual case where: (1) the petitioner presents conflicting diagnoses of her alleged vaccine injury; (2) the experts have 'extreme disagreement' as to the malady suffered; and, (3) the diagnoses are not along a continuum of similar conditions." *Id.*; *Contreras*, 107 Fed. Cl. at 294.

In contrast, in *Contreras*, the Court of Federal Claims held that the special master erred by diagnosing the petitioner's illness – as TM and not Guillain-Barré syndrome ("GBS") – before evaluating the *Althen* prongs. 107 Fed. Cl. 280, 292-93. The Court reasoned that the case contained "ample evidence that TM and GBS are similar diseases with similar pathologies" and the parties' "unified position [was] that an exact diagnosis of [the petitioner's illness] was not required to rule on causation." *Id.* at 293. The Court of Federal Claims articulated that "the general rule is that the special master should not conduct a differential diagnosis, at the outset of the causation analysis, to choose one diagnosis over another, or over a combination of

diagnoses." *Id.*, *aff'd* 844 F.3d 1363; *see also Andreu*, 569 F.3d 1367, 1378 (holding that the special master need not determine whether an initial seizure was febrile or afebrile for purposes of assessing vaccine causation).

Relevant to this inquiry, the Vaccine Act provides that a special master must consider the record as a whole including any medical diagnosis contained therein. Section 300aa-13(b)(1). However, no diagnosis in the medical records is "binding on the special master." *Id.* Rather, "[i]n evaluating the weight to be afforded to any such diagnosis… the special master… shall consider the entire record and the course of the injury, disability, illness, or condition until the date of the judgment of the special master." *Id.* The special master shall also consider any expert opinions and additional medical scientific evidence in the record. *Id.*

B. Causation

To receive compensation through the Program, petitioner must prove either (1) that she suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that she received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); *Capizzano v. Sec'y of Health & Hum. Servs.,* 440 F.3d 1317, 1319-20 (Fed. Cir. 2006). When, as here, petitioners allege that they suffered an injury that was actually caused by the vaccination, petitioners must establish, by preponderant evidence: (1) a medical theory causally connecting the vaccine and her injury ("*Althen* Prong One"); (2) a logical sequence of cause and effect showing that the vaccine was the reason for her injury ("*Althen* Prong Two"); and (3) a showing of a proximate temporal relationship between the vaccine and her injury ("*Althen* Prong Three"). § 13(a)(1); *Althen*, 418 F.3d at 1278. There must be preponderant evidence for each *Althen* prong. *Caves v. Sec'y of Health & Human Servs.*, 100 Fed. Cl. 119, 132 (2011), *aff. per curiam,* 463 Fed. Appx. 932 (Fed. Cir. 2012).

The causation theory must relate to the injury alleged. The petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." *Knudsen v. Sec'y of Health & Hum. Servs*., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Recently, in *Kottenstette,* the Federal Circuit reiterated that proof of causation does not "require identification and proof of specific biological mechanisms[.]" *Kottenstette v. Sec'y of Health & Hum. Servs.,* -- Fed.Appx.—(Fed. Cir. June 15, 2021) (citing *Knudsen v. Sec'y of Health & Hum. Servs.,* 35 F.3d 543, 549 (Fed. Cir. 1994). Causation "can be found in vaccine cases….without detailed medical and scientific exposition of the biological mechanisms." *Knudsen,* 35 F.3d 543, 548-49 (Fed. Cir. 1994). It is not necessary for a petitioner to point to conclusive evidence in the medical literature linking a vaccine to the petitioner's injury, as long as the petitioner can show by a preponderance of evidence that there is a causal relationship between the vaccine and the injury, whatever the details of the mechanism may be. *Moberly v. Sec'y of Health & Hum. Servs.,* 592 F.3d 1315, 1325 (Fed. Cir. 2010). The petitioner need not make a specific type of evidentiary showing such as "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." *Capizzano,* 440 F.3d at 1325. Instead, petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. *Id.* at 1325-26.

Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in petitioner's favor when the evidence weighs in his favor. *See Moberly*, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); *Althen*, 418 F.3d at 1280 (noting that "close calls" are resolved in petitioner's favor).

In Vaccine Act cases, expert testimony may be evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594-96 (1993); *see also Cedillo*, 617 F.3d at 1339 (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). In Vaccine Program cases, the *Daubert* analysis has been used in the weighing of the scientific evidence actually proffered and heard rather than as a tool for the pre-trial exclusion of expert testimony. *Davis v. Sec'y of Health & Hum. Servs.*, 94 Fed. Cl. 53, 66–67 (Fed. Cl. 2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted"), *aff'd*, 420 F. App'x 923 (Fed. Cir. 2011). The flexible use of the *Daubert* factors to determine the persuasiveness and/or reliability of expert testimony in Vaccine Program cases has routinely been upheld. *See, e.g.*, *Snyder v. Sec'y of Health & Hum. Servs.*, 88 Fed. Cl. 706, 742–45 (2009).

When both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs.*, 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe v. Sec'y of Health & Hum. Servs.*, 219 F.3d 1357, 1362 (Fed. Cir. 2000)). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 ("[a]ssessments as to the reliability of expert testimony often turn on credibility determinations"); *see also Porter v. Sec'y of Health & Hum. Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) ("this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act").

If the petitioner makes a *prima facie* case supporting vaccine causation-in-fact, the burden shifts to respondent to show by a preponderance of the evidence that the injury is instead due to factors unrelated to the administration of the vaccine. *Deribeaux v. Sec'y of Health & Human Servs.*, 717 F.3d 1363, 1367 (Fed. Cir. 2013) (citing Section 13(a)(1)(B)). Respondent

has the burden of demonstrating that: "[A] factor unrelated to the vaccination is the more likely or principal cause of injury alleged. Such a showing establishes that the factor unrelated, not the vaccination, was 'principally responsible' for the injury. If the evidence or alternative cause is seen in equipoise, then the government has failed in its burden of persuasion and compensation must be awarded." *Knudsen,* 35 F.3d at 551. Close calls regarding causation must be resolved in favor of the petitioner. *Althen,* 418 F.3d at 1280 (holding that Congress created a system in which "close calls regarding causation are resolved in favor of injured claimants").

IV. Analysis

a. Petitioner's Diagnosis

As a threshold matter, a petitioner must establish that he suffered from the condition for which he seeks compensation. *Broekelschen v. Sec'y of Health & Humn. Servs.,* 618 F.3d 1339, 1346 (Fed. Cir. 2010). The Federal Circuit has made clear "the statute places the burden on petitioner to make a showing of at least one defined and recognized injury." *Lombardi v. Sec'y of Health & Hum. Servs.,* 656 F.3d 1343, 1353 (Fed. Cir. 2011) (affirming a special master's decision to dismiss a petition when the petitioner could not establish that she had any of the three diagnoses alleged."). "The function of the special master is not to 'diagnose' vaccine-related injuries, but instead to determine based on the record evidence as a whole and the totality of the case, whether it has been shown by a preponderance of evidence that a vaccine caused [petitioner's] injury." *Id.* at 1352-53 (internal citations omitted). Thus, where "the existence and nature of the injury itself in dispute, it is the special master's duty to *first determine* which injury is best supported" by the evidence before applying the *Althen* test to determine causation. *Id.* at 1352 (citing *Broekelschen,* 618 F.3d at 1345) (emphasis added).

The parties dispute petitioner's proper diagnosis in this matter. Petitioner alleges that he suffered Guillain-Barré syndrome ("GBS"), as alleged in his petition. Petition (ECF No. 1). Respondent argues that petitioner did not have GBS for a variety of reasons, including that petitioner did not meet the Brighton criteria for GBS; his clinical course was not monophasic; and his EMG/NCS was inconsistent with a study demonstrative of GBS. Resp't. Br. at 44-53; Resp't. Sur-Reply at 11. Respondent concedes in the sur-reply "it was reasonable for petitioner's treating providers to suspect GBS initially," but that "petitioner's subsequent clinical history demonstrates this initial diagnosis was incorrect." Resp't. Sur-Reply at 11.

1. Petitioner has Preponderantly Established a Diagnosis of GBS

I have concluded that there is preponderant evidence to support the diagnosis of GBS following petitioner's hepatitis B vaccination on August 9, 2015. This finding is based on petitioner's clinical presentation, diagnostic testing, the diagnosis of his treating neurologist, the opinion of petitioner's expert, Dr. Steinman, and the medical literature.

Petitioner received his second hepatitis B vaccine on August 9, 2015. On August 18, 2018, petitioner went to the emergency room of St. Luke's hospital complaining of a rapid heart rate and tingling in his left arm. Pet'r. Ex. 3 at 8. Ten days later, on August 28, 2018, petitioner went to his primary care physician reporting numbness, tingling, and weakness in both his feet

and hands. Pet'r. Ex. 2 at 24. While petitioner's physical exam was normal, his treating physician recommended he seek treatment from a neurologist. *Id.* At his appointment with neurologist, Dr. Pandya on August 30, 2018, petitioner had absent deep tendon reflexes bilaterally, petitioner was unable to walk with a tandem gait, and petitioner continued to report tingling in his legs and arms. Pet'r. Ex. 4 at 2. When petitioner got to the emergency room at Hunterdon Medical Center the same day, the HPI states:

> The patient reports 2-3 weeks of symptoms that began with weakness and tingling in his feet bilaterally, and symptoms rising up his legs and now have gotten past his knees and he has started feeling symptoms in his hands and arms as well.

Pet'r. Ex. 5 at 4. His motor strength was recorded as 4/5 bilaterally in his lower extremities and an "unsteady gait noted." *Id.* at 7. The following day, on August 31, 2018, petitioner had a neurology consult with Dr. Pandya, and he wrote that petitioner had "absent deep tendon reflexes in both lower legs," and "walks with a wide base." *Id.* at 13. Petitioner underwent a lumbar puncture and his CSF protein level was 41 mg/dl, with the normal range being 15-45 mg/dl. *Id.* at 60. Dr. Pandya suspected that petitioner had GBS and ordered five rounds of IVIG treatment. *Id.* at 18.

Petitioner had a follow-up appointment with Dr. Pandya on September 18, 2018, and petitioner reported residual distal numbness, but his gait had improved and petitioner was using a cane for walking distances. Pet'r. Ex. 4 at 7. His physical exam was positive for absent deep tendon reflexes. *Id.* at 8. Dr. Pandya ordered an EMG/NCS for petitioner and diagnosed petitioner with Guillain-Barre syndrome following vaccination. *Id.* at 9. Dr. Pandya did not prescribe petitioner gabapentin or other medication at this appointment. *Id.*

The findings of the EMG/NCS performed on October 2, 2018 were "an abnormal study suggestive of the mixed sensory/motor demyelinating polyneuropathy," and that the study was "consistent with recent clinical diagnosis of GBS." Pet'r. Ex. 4 at 44. The motor nerve conduction study of petitioner's bilateral peroneal and bilateral tibial nerves showed "slower conduction velocity," and "[t]he sensory nerve action (SNAP) of the bilateral sural, bilateral medial and lateral plantar and bilateral superficial peroneal nerves were absent." *Id.*

At petitioner's October 2, 2018 appointment with neurologist Dr. Viradia, petitioner endorsed more sensory symptoms, including "tingling" in his bilateral toes, feet, and legs, and a "cold sensation from toes into legs with prolonged sitting or standing." Pet'r. Ex. 4 at 12. Petitioner also reported a sensation of tingling in his left arm and 5th finger and left leg and 5th toe. *Id.* Petitioner's neurological exam was positive for "stocking hypoesthesia" and an "unsteady gait" with "[deep tendon reflexes] absent bilaterally." *Id.* at 13. Dr. Viradia prescribed petitioner IVIG every two weeks at this appointment. *Id.* Petitioner then received a total of 12 additional IVIG infusions, from October 16, 2018 through March 25, 2019. *See* Pet'r. Ex. 7 at 2-7; Pet'r. Ex. 8 at 7-14.

From October 2, 2018 through March 29, 2019, petitioner reported ongoing complaints of numbness and tingling in his lower legs and feet, fatigue, and muscle weakness. *See* Pet'r. Ex. 4 at 12 ("Symptoms are tingling b/l legs, feet, toes, arm, and hands…Walking more than 2

minutes causes weakness in legs."); *Id.* at 16 ("Continuing tingling and muscle spasms mostly in lower extremities."); *Id.* at 21 ("patient reports increased muscle weakness in his legs and fatigue has returned"); *Id.* at 26 ("Ongoing fatigue….Continued tingling in lower legs and with fatigue in fingers."); *Id.* at 36 (03/29/2029-"Last few days increasing fatigue, has some tingling below the knee, some scattered tingling.").

On October 10, 2019, approximately one year after the onset of his GBS, petitioner's residual symptoms included tingling in legs and hands, and he had continued muscle fatigue and weakness. Pet'r. Ex. 9 at 26. Dr. Pandya wrote that petitioner had "more sensory and pain related symptoms," as "Sequelae of Guillain-Barre Syndrome." *Id.* Petitioner reported improvement in his symptoms on November 21, 2019, stating that he had "less tingling, less pain, [and] less fatigue." Pet'r. Ex. 11 at 3. Petitioner had also stopped using his cane to walk. *Id.* Sixteen months later, on March 2, 2020, petitioner reported "doing very much the same since last visit," although he had continued pain along with paresthesias in his hands and feet. *Id.* at 10. He was prescribed 300 mg per day of Gabapentin. *Id.* At his follow-up appointment on June 2, 2020, petitioner reported less tingling and feeling "near normal," and having a good response to the Gabapentin. Pet'r. Ex. 43 at 7.

According to the medical literature filed by both parties, progressive motor weakness in more than one limb, loss of tendon reflexes, and sensory symptoms such as numbness and tingling are signs supportive of GBS. Resp't. Ex. A, Tab 2 at 1 ("Progressive motor weakness of more than one limb."); Resp't. Ex. C, Tab 2 ("Unexplained sensations often occur first, such as tingling in the feet or hands…"); Resp't. Ex. A, Tab 1 ("The weakness is associated with decreased or absent deep tendon reflexes, and tend to be relatively symmetric."). Here, petitioner endorsed numbness and tingling in his feet and hands on August 28, 2018 and he had documented bilateral absent deep tendon reflexes in his lower extremities documented on August 30, 2018. His bilateral leg weakness, along with the numbness and tingling, began to ascend, and he developed ataxia, which is also consistent with the progression of GBS. *See* Pet'r. Ex. 4

Dr. Callaghan argues that petitioner's later treatment with IVIG shows that his GBS progressed past 4 weeks, which inconsistent with the diagnosis of GBS. Resp't. Ex. A at 4; Resp't. Ex. E at 1-2. However, Fokke explained that "the expected clinical course with a successive progressive, plateau and recovery phase is remarkably variable." Pet'r. Ex. 36 at 10. "The duration of the plateau phase was equally variable: most patients start recovering within less than a week after reaching nadir, but 6 months follow-up without clear signs of recovery is still compatible with the diagnosis of Guillain-Barre syndrome." *Id.* Additionally, "secondary deteriorations during the follow-up period were seen in 15% of patients. Two-thirds of these patients (10%) had a typical treatment related fluctuation." *Id.*

Petitioner's medical records show that after he was initially hospitalized for GBS and treated with IVIG, he reported feeling better but still had "distal numbness" and absent deep tendon reflexes in his lower extremities. Pet'r. Ex. 4 at 7. After petitioner had the EMG/NCS on October 2, 2018, it was recommended that he restart IVIG treatment to address petitioner's ongoing numbness and tingling in his feet, legs, and hands. *Id.* at 12. These sensory symptoms petitioner reported are consistent with GBS, likely a result of nerve damage that was apparent on his EMG/NCS. From there, it appears that petitioner began to experience treatment related

symptom fluctuation, when he would report improvement of certain symptoms such as fatigue or tingling in his upper extremities, but then a rebound of those symptoms. *See e.g.* Pet'r. Ex. 4 at 18 (11/16/2018-he feels much better; numbness improved); *Id.* at 26 (1/25/2019-Continued tingling in lower legs/feet and with fatigue in fingers); *see also* Pet'r. Ex. 6 at 102 (1/3/2019- "Patient continues to make steady improvements with upper extremity and lower extremity strength; however, fatigue persists, which is typical of Guillain Barre Syndrome."). It appears that the continued treatment with IVIG was for his ongoing residual symptoms and caused treatment related fluctuations in his clinical course, and not that petitioner's receipt of IVIG treatment was due to worsening symptoms or disease progression, as suggested by Dr. Callaghan.

Dr. Callaghan also argues that petitioner's CSF protein level and EMG/NCS study were inconsistent with the diagnosis of GBS, as well as petitioner's EMG/NCS. Resp't. Ex. A at 4. To meet the Level 1 Brighton criteria, a patient must have an elevated protein in CSF to meet the Level 1 Brighton criteria diagnosis for GBS *and* an EMG/NCS consistent with GBS. Resp't. Ex. A, Tab 1 at 6. Importantly, the Brighton GBS diagnostic criterion was established to create uniform case definitions and guidelines for data collection, and analysis for epidemiological studies. Resp't. Ex. A, Tab 1 at 1. Further, Sejvar provides that, "The case definitions are structured in three GBS and three FS levels of diagnostic certainty each as described in the overview paper. *It should be stressed that, although potentially applicable in a clinical setting, the level of diagnostic certainty is primarily intended for epidemiologic purposes and not as a criterion for treatment. Id.* at 5 (emphasis added). Petitioners asserting Table GBS claims in the Vaccine Program do not have to provide evidence of an elevated CSF protein or EMG findings consistent with GBS, although that evidence is supportive of the diagnosis. *See* 42 C.F.R. § 100.3(c)(15)(iv).

Fokke also explains that CSF protein levels may be normal in GBS cases, particularly if obtained early in the patient's clinical course. Pet'r. Ex. 36 at 9; Resp't. Ex. A, Tab 1 at 4. While Brighton requires the protein count to be above 50 cells/ul, Fokke stated that they found 15% of their patients with a CSF protein count between 5 and 50, indicating a "mild pleocytosis is compatible with a diagnosis of GBS." Pet'r. Ex. 36 at 10. Therefore, even with petitioner's normal CSF protein level, petitioner's treating physicians made the diagnosis of GBS based on his clinical presentation, including his absent deep tendon reflexes bilaterally and his ascending weakness, along with numbness and tingling. *See* Pet'r. Ex. 5 at 14.

Petitioner's EMG/NCS demonstrated that he had absent responses in his bilateral sural, bilateral, medial and lateral plantar and bilateral superficial peroneal nerves on the NCS. Pet. Ex. 4 at 44. The impression was: This is an abnormal study, suggestive of a mixed sensory/motor demyelinating polyneuropathy. This study is consistent with recent clinical diagnosis of GBS." *Id.* Fokke also discusses the reliance on EMG/NCS by the Brighton criteria and stated that only 48% of their patients fulfilled the specific diagnostic criteria for demyelinating polyneuropathy and that "there is no definite agreed-upon diagnostic electrophysiological criteria for the diagnosis of GBS." Pet'r. Ex. 36 at 11. Given the interpretation of petitioner's EMG/NCS by petitioner's treating neurologist and the findings suggestive of mixed sensory and motor demyelinating polyneuropathy, petitioner's EMG/NCS is consistent with the diagnosis of GBS.

Overall, based on petitioner's clinical course and his signs and symptoms, his diagnosis of GBs is consistent with the medical literature filed by both parties, and is also consistent with opinions of petitioner's treating neurologists. I find that petitioner has preponderantly established he developed GBS after receiving the hepatitis B vaccine on August 9, 2018.

b. *Althen* Analysis

1. *Althen* prong one

Under *Althen* prong one, petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1375 (Fed. Cir. 2009); *Pafford*, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. *Boatmon*, 941 F.3d at 1359; *see also Knudsen*, 35 F.3d at 548; *Veryzer v. Sec'y of Health & Hum. Servs.*, 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable."). If petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. *See Broekelschen*, 618 F.1339 at 1347 ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); *Perreira v. Sec'y of Health & Hum. Servs.*, 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing *Fehrs v. United States*, 620 F.2d 255, 265 (Ct. Cl. 1980)).

I find that petitioner has provided preponderant evidence that molecular mimicry is a sound and reliable theory to explain how the hepatitis A vaccine can cause GBS. This finding is based on petitioner's expert opinion and supporting medical literature.

Dr. Steinman proposed a theory of molecular mimicry between the hepatitis B vaccine components and proteins in the peripheral nerves to explain how the vaccine can cause GBS. In general, molecular mimicry, as explained in the Fujinami paper, is when there is a "shared immunologic epitope with a microbe and the host," and antibodies to the microbe can cross-react with self-antigens, resulting in an autoimmune condition. Pet'r. Ex. 19 at 1.[23] Molecular mimicry can involve the development of B or T cells that cross-react with self-antigens. Fujinami wrote:

> We had previously shown that the hepatitis B virus polymerase shared an immunologic epitope with myelin basic protein ("MBP"). When the viral peptide was injected into rabbits, some of the animals developed experimental autoimmune encephalomyelitis (EAE)-like disease, had T cell reactivity, and developed antibodies to MBP.

Id. at 3. Dr. Steinman opined that the hepatitis B vaccine contained "molecular mimics to a

[23] Fujinami, R. et al., *Molecular Mimicry, Bystander Activation, or Viral Persistence: Infections and Autoimmune Disease*, 19 Clin. Micro. Rev. 80-94 (2006). [Pet'r. Ex. 19].

protein known as Contactin-1 that has been associated with GBS," and that "antibodies to contactin-1 are found in patients with CIDP and GBS." Pet'r. Ex. 13 at 12. Dr. Steinman identified homology between the HepBsAg component of the hepatitis B vaccine and contactin-1, stating that the proteins share 6 out of 12 identical amino acids, which is sufficient to trigger neuroinflammation. *Id.* at 19.

Respondent's experts argue that there is no association between the hepatitis B vaccine and GBS. *See* Resp't. Ex. A at 5; Resp't. Ex. C at 5; Resp't. Ex. F at 2. Dr. Tompkins argues that the hepatitis B vaccine is safe and the Advisory Committee on Immunization Practices ("ACIP") has found no association between the hepatitis B vaccine and GBS. Resp't. Ex. C at 5; Resp't. Ex. F at 2. Dr. Tompkins does not appear to dispute molecular mimicry as a mechanism for inducing autoimmune conditions, instead, he argues that the steps Dr. Steinman has taken to develop his theory are unreliable. Resp't. Ex. C at 6-8. Dr. Tompkins acknowledges that some of the literature Dr. Steinman refers to demonstrates that "immunization with a peptide with 4 of 11 amino acids sharing identity with a MBP peptide could elicit neuroinflammation," he asserts that simply demonstrating sequence homology is insufficient to demonstrate that molecular mimicry is a pathogenic mechanism for GBS. Resp't. Ex. C at 7.

Molecular mimicry has been accepted as a sound and reliable theory in many Vaccine Program cases dealing with demyelinating conditions, including GBS, forming the basis for petitioners to be compensated. *See e.g. Musick v. Sec'y of Health & Hum. Servs.,* No. 18-451V, 2025 WL 2452232, at *35-39 (Fed. Cl. Spec. Mstr. July 8, 2025) (accepting molecular mimicry as the causal mechanism for how the Prevnar-13 vaccine can cause GBS); *J.G. v. Sec'y of Health & Hum. Servs.*, No. 20-664V, 2023 WL 2752634, at *30 (Fed. Cl. Spec. Mstr. Feb. 13, 2023) (accepting molecular mimicry as the causal mechanism for how the hepatitis A vaccine can cause GBS); *Quirino v. Sec'y of Health & Hum. Servs.,* No. 17-989V, 2023 WL 9229145, at *21 (Fed. Cl. Spec. Mstr. Dec. 18, 2023) (finding that molecular mimicry between the hepatitis B vaccine and the peripheral nerves can result in small fiber neuropathy); *Conte v. Sec'y of Health & Hum. Servs.*, No. 17-403V, 2020 WL 5743696, at *23 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program.").

Additionally, the Hepatitis B-Neurological Demyelinating Omnibus Proceeding determined that a causal association existed between the hepatitis B vaccine and several demyelinating diseases, including GBS. *See Stevens v. Sec'y of Health & Hum. Servs.,* 2006 WL 659525; *Peugh v. Sec'y of Health & Hum. Servs.,* No. 99-638V, 2007 WL 1531666 (Fed. Cl. Spec. Mstr. May 8, 2007) (finding that the hepatitis B vaccine can cause GBS). More recently, former Chief Special Master Dorsey found that molecular mimicry was a sound and reliable theory to explain how the hepatitis B vaccination can cause GBS. *See Osso v. Sec'y of Health & Hum. Servs.*, No. 18-575V, 2023 WL 5016473, at *22-24 (Fed. Cl. Spec. Mstr. July 13, 2023).

Petitioner has also submitted preponderant evidence in this case to establish that the hepatitis B vaccine can cause GBS through molecular mimicry. First, Dr. Steinman identified a sequence of 6 of 12 amino acids that share consecutive sequences between the HepBsAg and contactin-1 protein. Pet'r. Ex. 13 at 19. He then explained how viral peptides with homology of 5 amino acids to a self-peptide can stimulate T cells to induce EAE. Pet'r. Ex. 13 at 13. The

1998 Gautam paper, which Dr. Steinman co-authored, explored whether a viral peptide could cross-react with myelin basic protein and cause EAE. Pet'r. Ex. 29 at 6. The authors found that a microbial peptide with limited similarity with myelin basic protein could stimulate MBP-reactive T-cells, causing neuroinflammation. *Id.* Gautam explained that molecular mimics between an infectious agent and self can result in the development of either autoreactive T or B cells. *Id.* Gautam explains how the cross-reaction leads to autoimmune disease:

> One possibility is that self-reactive T-cells that have escaped the thymic deletion encounter cross-reactive microbial peptides bound to MHC molecules. This may result in the low-level stimulation of self-reactive T cells, migration into a site, and the causing of some tissue damage. Once the tissue destruction has begun, the release of self-antigens from the target tissue may perpetuate the immune response against its own antigens, even after the microbial pathogen has cleared.

Id.

Next, Dr. Steinman credibly explained that antibodies to contactin-1 are found in patients with GBS. Pet'r. Ex. 13 at 12. Miura explains that contactin-1 is a "key axonal adhesion molecule" and "is essential for the formation of the paranodal septate-like junction." Pet'r. Ex. 26 at 3. While Miura was examining patients with CIDP for autoantibodies to contactin-1, the researchers identified four patients with GBS that had anti-contactin-1 antibodies. *Id.* at 7. Devaux also explored the immune attacks to the nodal and paranodal structures of the peripheral nerves and found that in AIDP, the common form of GBS in North America, autoantibodies were found at the nodes of Ranvier and its paranodes. Pet'r. Ex. 25 at 4. Devaux identified auto-antibodies specific to contactin-1 in GBS patients and believed that these immune attacks to the nodes and paranodes were pathogenic in GBS, not related to a secondary immune reaction against demyelinated or damaged myelinated fibers. *Id.* at 16.

Respondent, through Dr. Tompkins, criticizes how Dr. Steinman developed his theory, including the use of the BLAST and IEDB searches, to identify molecular mimics between the HepBsAg and contactin-1. Resp't. Ex. C at 6-7; Resp't. Ex. F at 2-3. Dr. Tompkins argued that the Lanz article, which found an association between the EBV and MS, "used extensive empirical analysis" to find the molecular mimic, and did not just rely on a BLAST search. Resp't. Ex. F at 3. However, the exacting science Dr. Tompkins is demanding to demonstrate molecular mimicry in this case is not required and would be totally impractical in Vaccine Program cases. *See Knudsen v. Sec'y of Health & Hum. Sevs.,* 35 F.3d 543, 549 (1994) ("to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program."). Requiring testable evidence or insisting on direct evidence, such as a study similar to Lanz, impermissibly heightens petitioners' burden of proof. *See also Brayboy v. Sec'y of Health & Hum. Servs.,* No. 15-183V, 2021 WL 4453146, at *19 (Fed. Cl. Spec. Mstr. Aug. 30, 2021) ("Petitioners identified cross-reaction between components of the vaccine and proteins in the body that are directly responsible for the health and productivity of the organ at issue. Respondent is requiring an additional step and insisting on direct testable evidence of pathology."). Here, Dr. Steinman performed a BLAST search to identify homology between proteins in the hepatitis B vaccine and proteins in the body which are targeted in GBS by immune attacks. Requiring the type of massive testing and

experimental work that was performed in the Lanz paper to demonstrate the role of molecular mimicry between the Epstein Barr virus and multiple sclerosis is not required.

Respondent also argues that epidemiological evidence does not demonstrate an association between the hepatitis B vaccine and GBS. Resp't. Ex. C at 5; *see also* Resp't. Sur-Reply at 6-8. Respondent submitted the McMahon and Baxter studies as epidemiological evidence that the hepatitis B vaccine is not associated with GBS. A lack of epidemiological evidence is not dispositive. Where such evidence is submitted, "the special master can consider it in reaching an informed judgment as to whether a particular vaccination likely caused a particular injury." *Andreu,* 569 F.3d 1367,1379 (2009).

The Baxter study examined 415 cases of GBS in the Kaiser Permanent Northern California ("KPNC") patient population to compare "the odds of vaccination in the 6 and 10 weeks prior to the onset of GBS to the odds of vaccination during the same time intervals in all vaccinated individuals in the entire KPNC population." Resp't. Ex. A, Tab 5. The study only identified 25 cases of vaccination in the six weeks prior to the onset of GBS, with one occurring after administration of the hepatitis B vaccine. *Id.* at 4. The authors found "no increased risk of GBS following vaccination," including the influenza vaccine. *Id.* But despite this conclusion, the authors stated, "we are unable to exclude any possible association between vaccines and GBS," and noted that their study can provide "reassurance that the risk of GBS following any vaccine, including influenza vaccines, is extremely low." *Id.* The McMahon study is also not persuasive in discrediting Dr. Steinman's theory. The McMahon study examined rates of GBS following vaccination in Alaskan natives and found no evidence that the hepatitis B vaccine caused an increase in the incidence of GBS among the population. Resp't. Ex. A, Tab 4 at 3. However, the authors noted that "the number of vaccinees was considerably smaller in this study than in the CDC study and therefore the power to detect an increase in the incidence of GBS may not be sufficient." *Id.* at 3. The McMahon authors noted that in a post-marketing surveillance study of the hepatitis B vaccine, the CDC found nine cases of GBS in 850,000 persons who received a hepatitis B vaccination, which was higher than the annual background incidence rate of GBS. *Id.*

I have considered these two studies presented by respondent and find that they do not undermine the viability of Dr. Steinman's theory. Both studies explain that their findings suggest that the vaccines studied do not *increase* the risk of GBS post-vaccination, not that vaccines are not the *cause* of GBS in rare instances. "While epidemiological studies can establish an increased incidence, and this increased incidence can support an inference of causation, epidemiological studies cannot absolutely refute a causal connection. Epidemiological studies cannot prove a negative. *Harris v. Sec'y of Health & Hum. Servs.,* 2014 WL 3159377, at *11 (Fed. Cl. Spec. Mstr. June 10, 2014), *mot. for review dismissed,* 2015 WL 2129036. A vaccine injury is a rare event that cannot be disproved because a vaccinee did not experience a response consistent with that of the general population. *Walls v. Sec'y of Health & Hum. Servs.* No. 16-557V, 2020 WL 13801342, at *16 (Fed. Cl. Spec. Mstr. June 23, 2020). Finally, petitioner does not need to provide epidemiological evidence to show that a vaccine can cause an injury. *See Andreu,* 569 F.3d at 1378 (quoting *Capizzano,* 440 F.3d at 1325-26); *see also Althen,* 418 F.3d at 1280 (noting that "close calls" are resolved in a petitioner's favor).

After considering the evidence as a whole, including the expert opinions and medical literature submitted by both parties, I find that petitioner has proved a sound and reliable medical theory by preponderant evidence, satisfying *Althen* prong one.

2. *Althen* prong three

Althen prong three requires petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." *Id.* Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan,* 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under *Althen* prong one). *Id.*; *Koehn v. Sec'y of Health & Hum. Servs.,* 773 F.3d 1239, 1243 (Fed. Cir. 2014); *Shapiro,* 101 Fed. Cl. at 542; *see Pafford*, 451 3d at 1358. A temporal relationship between a vaccine and an injury, standing alone, does not constitute preponderant evidence of vaccine causation. *See e.g. Veryzer,* 100 Fed. Cl. at 356 (explaining that "a temporal relationship alone will not demonstrate the requisite causal link and that petitioner must posit a medical theory casually connecting the vaccine and injury").

Petitioner states that the onset of his GBS began approximately 2 days after he received the hepatitis B vaccine, based on the medical records. Pet'r. Mot. at 23; *see also* Pet'r. Ex. 5 at 10 ("These symptoms started 2 days after he received the hepatitis B vaccination."). The medical records are somewhat unclear as to the exact onset of petitioner's symptoms, with some records stating 2 days post-vaccination, and others suggesting 3 days post-vaccination. For example, on August 29, 2018, petitioner reported that two days after receiving the second hepatitis B vaccine he "started to feel unwell and noted increase and fluctuating [blood pressure]…he was also complaining of generalized weakness, dizziness, and nausea." Pet'r. Ex. 10 at 100. But two days later, on August 30, 2018, under "Reason for Admission" the record states, "received hep b 2nd vaccine at [occupational] health on 8/9/2018. 3 days later tachy, weakness of legs and numbness and tingling of extremities. Onset 2 ½ weeks." Pet'r. Ex. 5 at 146. Dr. Spall also noted an onset of three days post-vaccination when he examined the petitioner upon his admission to the hospital. The "History of Present Illness" taken by Dr. Spall states, "The patient reports 2-3 weeks of symptoms that began with weakness and tingling in his feet bilaterally, and symptoms rising up his legs and now have gotten past his knees and he has started felling symptoms in his hands as well. He reports 3 days prior to initiation of these symptoms he had the second dose of his hepatitis B vaccination." *Id.* at 4-5.

Dr. Steinman opines that a two-day onset of GBS symptoms after receipt of the second hepatitis B vaccine is consistent with a recall response, triggering the pre-existing immune cells to react to both the vaccine components and self-tissue, resulting in symptom onset. Pet'r. Ex. 13 at 19-22. Respondent's experts argue that the onset of petitioner's GBS is too soon to be associated with the hepatitis B vaccine. Resp't. Ex. C at 10.

To support his opinion, Dr. Steinman referenced the Siegrist chapter from the text "Vaccine Immunology", which indicates that "secondary immune responses, booster exposure to

antigen reactivates immune memory and results in a rapid (<7 days) increase of IgG antibody titer[s]." Pet'r. Ex. 32 at 10. Dr. Steinman also referred to the Schonberg article, which examined cases of GBS following the 1979 Swine Flu vaccinations and noted that Schonberg identified sixteen cases of GBS 1-3 days following vaccination. Pet'r. Ex. 33 at 9; *see also* Pet'r. Ex. 13 at 23.

Respondent relied upon the Leroux and Degrassi articles to rebut Dr. Steinman's opinion that a two-day onset is appropriate, arguing that these articles demonstrate that the "hepatitis B booster vaccinations trigger weak and slow T cell and B cell responses," and thus, symptoms of GBS would not appear two days post-vaccination. Resp't. Resp. at 25-26; *see also* Resp't. Ex. F at 4. In their briefs, the parties disagree about the relevance of those articles in determining whether a rapid immune response to the hepatitis B vaccine occurs. *See* Pet'r Sur-Reply at 2 ("blood samples in Degrassi and Lerous-Roels were not drawn with sufficient frequency to shed light on the rapidity of the subjects' immune response."); Resp't. Sur-Reply at 3 ("Petitioner erroneously claims that two studies referenced by Dr. Tompkins (Degrassi and Leroux-Roels) do not address what would be medically reasonable time between vaccination and symptom onset to infer vaccine causation under *Althen* prong three.).

Degrassi drew blood samples four weeks apart between the first and second dose, and then 20 weeks after the third dose. Resp't. Ex. C, Tab 13 at 2. The authors noted that two out of 12 vaccinated patients developed antibodies to HepBsAg after the first dose and after the second dose, all vaccinated patients developed antibodies, however, when they tested the samples against a purified HepBsAg, they found no proliferative response. *Id.* at 3. However, the relevance of Degrassi is somewhat undermined by the Leroux-Roles study, which was published two years later. Leroux-Roles took serum samples six days after administration of the second and third doses of hepatitis B vaccines and found a group of vaccinees that rapidly developed antibodies to hepatitis B, designated "fast responders." Resp't. Ex. C, Tab 14. Leroux-Roles simultaneously monitored humoral responses in vaccinees and in vitro cellular immune responses in 50 subjects and found that the two responses are closely correlated. *Id.* at 5. The authors concluded:

> We have clearly demonstrated that the variability of humoral immune responses to the HepBsAg in vaccine recipients is reflected by a concurrent variability of *in vivo* cellular immune responses to this antigen. The intriguing question now are why HBsAg is recognized less efficiently and induces less vigorous T-cell responses in some individuals and how this selective deficiency can be overcome.

Id. at 6. Ultimately, Leroux-Roles demonstrated that there is high variability in both the humoral and cellular immune response to the hepatitis B vaccine, with some people demonstrating rapid antibody responses, and others having a relatively slow response. Neither of these studies dispositively demonstrate that a rapid immune response to the hepatitis B booster is "improbable" as opined by Dr. Tompkins and argued by respondent.

Additionally, a two-day onset of a neurological disease has been found to be temporally acceptable in several Vaccine Program cases. In *Quirino,* the special master accepted a two-day onset of small fiber neuropathy as medically acceptable after the petitioner received both a

hepatitis B and Tdap vaccine. *Quirino,* 2023 WL 9229145, at 23-24. In *Le,* the petitioner developed symptoms of transverse myelitis between two and three days after the flu vaccine, and the special master found this timeframe to be appropriate given the mechanism of molecular mimicry. *Le v. Sec'y of Health & Hum. Servs.,* No. 16-1078V, 2023 WL 3049203, at *34-35 (Fed. Cl. Spec. Mstr. Mar. 30, 2023). Finally, in *Augustynski*, a case consolidated in the Hepatitis B Omnibus proceedings, the petitioner developed neurological symptoms of a demyelinating condition (later diagnosed with multiple sclerosis) one day after he received his hepatitis B booster vaccination. *Augustynski v. Sec'y of Health & Hum. Servs.,* No. 99-611V, 2007 WL 3033614, at *8-9 (Fed. Cl. Spec. Mstr. Sept. 28, 2007). The special master explained that the expert credibly explained that the "first hepatitis B vaccination primed [the] immune T-cells. With the second hepatitis B vaccination, the T-cells had some memory of the antigen and the subsequent response happened very quickly, within 24-hours….The second hepatitis B vaccine was a rechallenge of petitioner's immune system." *Id.* at 4-5.

I find that Dr. Steinman's opinion is more persuasive than respondent's experts' opinions on this matter. The medical literature, particularly the Leroux-Roles study, demonstrates that the immune response to the hepatitis B vaccine is highly variable and some individuals demonstrate a rapid response, while others may lag. Further, the onset of neurological symptoms two to three days post-vaccination is consistent with a recall response, where an individual's immune system can more quickly recognize an antigen it previously encountered. Finally, petitioner's medical records support that he was experiencing a response to the second hepatitis B vaccine, reporting "not feeling well over the last several days." *See* Pet'r. Ex. 2 at 21.

Given the above, I find that petitioner has preponderantly demonstrate that the onset of his symptoms, whether two or three days after his second hepatitis B vaccination on August 9, 2018, is a medically appropriate timeframe, consistent with the theory of molecular mimicry. Therefore, petitioner has demonstrated *Althen* prong three by preponderant evidence.

3. *Althen* prong two

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

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. *Andreu,* 569 F3d at 1367; *Capizzano,* 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.' " (quoting *Althen,* 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. *Cucuras,* 993 F. 2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court.").

As explained above, petitioner has proffered a sound and reliable mechanism of vaccine causation under *Althen* prong one and the onset of petitioner's symptoms was approximately two or three days after vaccination, an appropriate timeframe in which a recall immune response could cause a cross reaction secondary to molecular mimicry and cause GBS. "Evidence demonstrating petitioner's injury occurred within a medically acceptable timeframe bolsters a link between the injury alleged and the vaccination at issue under the "but-for" prong of the causation analysis." *Capizzano,* 440 F. 3d at 1326 (finding medical opinions that explain how a vaccine can cause the injury alleged coupled with evidence demonstrating a close temporal relationship "are quite probative" in proving actual causation.") *Pafford,* 451 F.3d at 1358; *see also Contreras,* 107 Fed. Cl. at 295 (finding that there is a "logical overlap between three *Althen* prongs, and that evidence that goes to one prong may also be probative for another prong"). However, it is not only the finding of *Althen* prongs one and three for which the undersigned finds preponderant evidence to support *Althen* prong two.

Petitioner's medical records support a clinical course consistent with the mechanism of molecular mimicry. Petitioner received his second hepatitis B vaccine on August 8, 2019 and seven days later went to his primary care physician's office, complaining of "not feeling well over the last several days," with increased blood pressure. Pet'r. Ex. 2 at 21. Three days later, petitioner went to the emergency department, reporting shortness of breath, chest pain, and tingling in his left arm. Pet'r. Ex. 3 at 8. On August 28, 2018, petitioner's primary care physician suspected petitioner had GBS, when petitioner explained he was experiencing numbness and tingling in both feet and hands, along with lower extremity weakness, and recommended petitioner consult with a neurologist. Pet'r. Ex. 2 at 24. Neurologist Dr. Pandya assessed petitioner on August 30, 2018, found petitioner had absent deep tendon reflexes bilaterally in both lower extremities, and diagnosed him with "Guillain-Barre syndrome following vaccination." Pet'r. Ex. 4 at 4. Petitioner was hospitalized with ascending numbness and tingling, absent deep tendon reflexes, diagnosed with GBS and treated with IVIG. *See* Pet'r. Ex. 5 at 13-18. The EMG/NCS performed on October 2, 2018 was "suggestive of the mixed sensory/motor demyelinating polyneuropathy," and petitioner's study was found to be "consistent with recent clinical diagnosis of [GBS]." Pet'r. Ex. 4 at 43-44.

Petitioner's treating physicians also made statements associating petitioner's hepatitis B vaccine as the cause of his GBS. When Dr. Pandya first assessed petitioner on August 28, 2018, he diagnosed petitioner with "Guillain-Barre syndrome following vaccination." Pet'r. Ex. 4 at 4. When Dr. Pandya assessed petitioner in the hospital on August 31, 2018, he wrote, "43-year old gentleman who complains of new onset of bilateral lower extremity numbness and weakness…this gentleman has a new onset of numbness in both lower legs; he had absent deep tendon reflexes in both lower legs; and at the same time he had a wide-based gait. He also has some numbness in the upper hand and distal leg. There could be multiple etiologies of the current symptoms. *Considering the symptoms started in such a way subacutely associated with this finding with the recent vaccination.*" Pet'r. Ex. 5 at 12-13 (emphasis added). Petitioner's discharge diagnosis was, "Suspected Guillain-Barré syndrome; recent hepatitis B vaccine implicated in above." *Id.* at 17. Petitioner's primary care physician, Dr. Martin, documented that petitioner was diagnosed with "Guillain-Barré 08/2018 after receiving a hepatitis B vaccine booster on 08/9/2018." Pet'r. Ex. 41 at 14.

Further, there is nothing in the medical records to suggest an alternative cause. Prior to the vaccination on August 9, 2018, the most recent medical appointments were from May 2018, when petitioner was complaining about a chronic cough, and he was diagnosed with an asthma exacerbation and seasonal allergies. Pet'r. Ex. 2 at 13. He also reported having no recent illnesses when admitted to the hospital on August 30, 2018. Pet'r. Ex. 5 at 4. Finally, respondent's experts do not identify any potential alternative causes for petitioner's condition, nor did they identify any possible alternative diagnoses. *See* Resp't. Ex. A at 5; Resp't. Ex. C at 11.

Consistent with the finding above that petitioner's diagnosis was GBS, I find petitioner demonstrated a logical sequence of cause and effect by preponderant evidence, as he developed his symptoms two to three days after receiving the second hepatitis B vaccine on August 9, 2018, his treating physicians associated his condition to the hepatitis B vaccine, and there is nothing in the record to demonstrate an alternative cause for petitioner's GBS. Accordingly, petitioner has satisfied *Althen* prong two.

V. Conclusion

For the reasons discussed above, the undersigned finds that petitioner has established by preponderant evidence that the immune response to the second hepatitis B vaccine he received on August 9, 2018 was the cause-in-fact of his GBS. Therefore, petitioner is entitled to compensation. A separate damages order will be issued.

IT IS SO ORDERED.

s/Thomas L. Gowen
Thomas L. Gowen
Special Master