In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

No. 21-317V

SUZANNE ZACHARSKI, *on behalf of* DIANE LEMANSKI, *decedent*, Petitioner, v. SECRETARY OF HEALTH AND HUMAN SERVICES, Respondent.	*	Chief Special Master Corcoran Filed: March 26, 2025

Joseph Alexander Vuckovich, Mctlaw, Washington, DC, for Petitioner.

Felicia Langel, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On January 8, 2021, Suzanne Zacharski ("Petitioner"), on behalf of Diane Lemanski, filed a petition for compensation under the National Childhood Vaccine Injury Act. Petition (ECF No. 1). Petitioner alleges that the Hepatitis A vaccine administered to Ms. Lemanski on February 2, 2018, caused or significantly aggravated her "chronic motor axonal polyneuropathy" ("CMAP"), and ultimately led to her death. Petition at 1-2.

A trial was held in this matter on September 23-24, 2024. Now, based upon my review of the record and consideration of the hearing testimony, including expert input, I deny entitlement. Petitioner succeeded in establishing that Ms. Lemanski's injury was something other than

[1] Under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction "of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy." Vaccine Rule 18(b). Otherwise, the whole Ruling will be available to the public in its present form. *Id.*

amyotrophic lateral sclerosis ("ALS")[2], as contended by Respondent's experts. But Petitioner has not preponderantly shown that the Hepatitis A vaccine can cause a chronic, immune-mediated neuropathy, or did so to Ms. Lemanski.

I. Fact Summary

Pre-Vaccination History

Ms. Lemanski was born on August 25, 1959, and was 58 years old at the time of the relevant vaccination. Ex. 6.01 at 78; Ex. 1 at 1. Her medical history was relevant for asthma, hyperlipidemia, hypertension, hypothyroidism, insomnia, anxiety, and migraines. Ex. 2 at 6-7. She had also experienced foot, knee, and leg problems well before the vaccination at issue. Ex. 6.01 at 103-06.

In the spring of 2015, for example, Ms. Lemanski visited her family practice complaining of left knee pain and stiffness that had progressed to right knee "pinching" pain over the course of several months. Ex. 6.01 at 103-05. A right knee x-ray was unremarkable. *Id.* at 104. On August 11, 2016, Ms. Lemanski returned to her family practice with a complaint of foot pain "for weeks," and an x-ray showed mild degenerative changes. *Id.* at 78.

On December 2, 2016, Ms. Lemanski visited her primary care physician ("PCP") complaining of a dull ache on the top of her left foot for three months. Ex. 6.01 at 75. Her podiatrist had injected her foot a few months before, and ever since her foot had been "burning and tingling intermittently." *Id.* Soon thereafter, on December 8, 2016, Ms. Lemanski visited a neurophysiologist for numbness and tingling in her left leg for about three months. *Id.* at 74. An EMG[3] of her left leg yielded normal results. Ex. 6.02 at 145-46. At an October 2017 visit with Dr. Steven Kotsonis, her PCP, she complained of persistent foot pain and was diagnosed with plantar fasciitis. Ex. 6.01 at 62.

Vaccination and Initial Symptoms – February-July 2018

On February 2, 2018, Ms. Lemanski received a Hepatitis A vaccine through Beaumont Hospital Royal Oak's employee vaccination program. Ex. 1 at 1. Three days later, on February 5,

[2] ALS is a fatal type of motor neuron disease that involves progressive degeneration of nerve cells in the spinal cord and brain. E. Tiryaki & H. Horak, *ALS and other Motor Neuron Diseases*, 20 Continuum 1185, 1185 (2014), filed as Ex. 34 (ECF No. 26-18).

[3] An Electromyography (EMG) test "measures muscle response or electrical activity in response to a nerve's stimulation of the muscle. The test is used to help detect neuromuscular abnormalities." *Electromyography (EMG)*, John Hopkins Medicine, https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/electromyography-emg#:~:text=Electromyography%20(EMG)%20measures%20muscle%20response,the%20skin%20into%20the%20muscle (last visited Mar. 7, 2025).

2018, she visited her PCP complaining of aching and twitching in both legs that she reported began *three weeks earlier*:

> "The patient is a 58-year-old female who presents with leg pain . . . The patient describes the pain as aching. *Onset was 3 week(s) ago*. Note for "Leg pain": Pt states that she is having bilateral twitching in both knee areas x 3 weeks. She denies any recent trauma."

Ex. 6.01 at 58 (emphasis added).

Ms. Lemanksi repeated claims of a pre-vaccination onset a few days later, on February 9, 2018, when she returned to her PCP to complain of aching and twitching in both legs:

> "The patient is a 58-year-old female who presents for a Recheck of Leg pain. Symptoms include leg pain. The pain is located symmetrically. There is no radiation. The patient describes the pain as aching. *Onset was 4 week(s) ago*. Note for "Leg pain": Pt states she's here for recheck on her blood work. Pt states both legs are still twitching."

Ex. 6.01 at 56 (emphasis added).[4]

Ms. Lemanski's neurologic exam was normal at both visits. Ex. 6.01 at 57, 59. An EMG subsequently performed on March 1, 2018, showed "[d]iffuse, ongoing denervation at multiple nerve root segments, L4-S1 bilaterally [but] . . . [n]o evidence of neuropathy or myopathy." Ex. 6.02 at 83-85. Otherwise, the EMG results were read to be consistent with "conditions such as lumbar polyradiculopathy from spinal compressive etiologies." *Id.* at 84.

Ms. Lemanski returned to her PCP on March 7, 2019, with the same complaints. Ex. 6.01 at 54. Her treater now ordered MRIs of her brain and lumbar spine – and although the results of the brain MRI were normal, the lumbar MRI showed multilevel neural foraminal narrowing.[5] *Id.* at 54; Ex. 6.02 at 78, 80-81. He referred Ms. Lemanski to physical therapy ("PT") and to a neurologist. Ex. 6.01 at 55. At her PT evaluation on March 12, 2018, Ms. Lemanski reported that she had been experiencing weakness in her left foot/ankle and muscle twitches throughout her body (but primarily her legs) for the past 5-6 weeks (which again would potentially predate

[4] As noted below, Petitioner contends these claims of a pre-vaccination onset are inaccurate.

[5] "Foraminal Stenosis" occurs when narrowing in parts of a person's spine causes compression of their spinal nerves. *Foraminal Stenosis*, Cleveland Clinic, https://my.clevelandclinic.org/health/diseases/24856-foraminal-stenosis (last visited Feb. 24, 2025).

vaccination). Ex. 6.02 at 86. Ms. Lemanski also reported that her right lower back and hip began hurting the previous day, and she had a history of sciatica. *Id.*

On March 28, 2018, Ms. Lemanski returned to her PCP and reported continued aching and tingling of her legs, but noted that PT was helping. Ex. 6.01 at 52. The notes from her PT reevaluation on April 16, 2018, however, stated that there were "no significant changes with the left leg weakness and calf tightness," that her left ankle was weak, and that she had fallen twice. Ex. 6.02 at 73, 75.

Ms. Lemanski subsequently visited Dr. Sunitha Santhakumar, a neurologist, on April 18, 2018, for treatment of weakness in both legs. Ex. 2 at 5, 7. Ms. Lemanski was tearful, and she reported that "a little after" her vaccination in February, she had developed random, spontaneous muscle twitching, left calf soreness and stiffness, and an unsteady gait. *Id.* at 7. She also reported tripping and falling. *Id.* On exam, Ms. Lemanski displayed a slight left foot drop, difficulty walking, and a few muscle fasciculations of her left calf. *Id.* Dr. Santhakumar questioned whether Ms. Lemanski's neurological symptoms were due to a vaccine reaction. *Id.* Noting that the existing EMG results were consistent with a spinal cord compression, Dr. Santhakumar diagnosed Ms. Lemanski with lumbar polyradiculopathy.[6] *Id.* Dr. Santhakumar ordered an autoimmune/inflammatory workup and a repeat EMG, and she prescribed a Medrol Dosepak and continued PT. *Id.*

Ms. Lemanski's bloodwork from May 10, 2018, was normal. But the next EMG (performed on May 18, 2018) showed "left sciatic neuropathy proximal to the short head of the biceps femoris with functional continuity documented to all muscles sampled . . . [and] pathology in the right S1 nerve root distribution," and although "[t]he abnormalities noted are not consistent with a diagnosis of diffuse pathology of the motor neurons and/or their axons," the results were overall deemed abnormal. Ex. 12 at 57-63; *see also* Ex. 5 at 25-26.

Ms. Lemanski thereafter attended 17 PT sessions from March 2018 to June 2018, and her progress over the course of the sessions was variable. *See generally* Ex. 4. During this time, Ms. Lemanski was fitted for an ankle foot orthosis for her left foot drop, and she walked with a cane. *Id.* at 17. At her PT discharge on June 12, 2018, the therapist wrote that Ms. Lemanski "has not reported a fall in over 3 weeks, but reports having good days and bad days with variable strength." *Id.*

On June 19, 2018, Ms. Lemanski returned to Dr. Santhakumar and complained of muscle spasms particularly in the left leg, a cold sensation in the left leg, and a "vibration" sensation in

[6] "Lumbar polyradiculopathy" occurs when nerve roots in a person's lower back become pinched or damaged. *Radiculopathy*, Johns Hopkins Medicine, https://www.hopkinsmedicine.org/health/conditions-and-diseases/radiculopathy (last visited Feb. 24, 2025).

the bottom of her left foot. Ex. 2 at 9, 11. Dr. Santhakumar interpreted the EMG of May 18, 2018, as evidence of left sciatic neuropathy.[7] *Id.* at 11. On exam, Ms. Lemanski displayed intact sensation but mild edema, and a cold sensation of the left ankle. *Id.* Dr. Santhakumar diagnosed Ms. Lemanski with a generalized immune process or plexitis[8] and treated her with a second Medrol Dosepak. *Id.* Dr. Santhakumar also prescribed Flexeril for muscle spasms and recommended an IVIg[9] trial. *Id.*

On June 30, 2018, Ms. Lemanski received stitches for a finger laceration after she fell in the shower. Ex. 3 at 21. On July 16, 2018, Ms. Lemanski went to her family practice to get her stitches removed and reported gait instability. Ex. 6.01 at 47. At this time, she expressed the view that her neurological symptoms were due to an autoimmune reaction to the Hepatitis A vaccine. *Id.* at 46.

Throughout July and August 2018, Ms. Lemanski received several rounds of steroid infusions and attended ten more PT sessions. Ex. 6.02 at 57; Ex. 5 at 40-57. During this period, she initially felt stronger, but after the steroid infusions stopped, her condition declined. Ex. 5 at 40-47. Ms. Lemanski complained of waxing/waning strength, fatigue, twitching in her upper body, edema in her left leg, and foot drop now on her right side. *Id.* at 47. Her PT discharge summary on August 3, 2018, concluded that Ms. Lemanski had "made little to no progress during her course of therapy." *Id.* at 56.

Hospitalization and Inpatient Rehabilitation – August 2018

Due to her worsening condition and the appearance of her right foot drop, Dr. Santhakumar recommended that Ms. Lemanski go to the hospital. Ex. 6.02 at 57. Upon arrival on August 6, 2018, Ms. Lemanski was admitted with a diagnosis of "unclear." Ex. 17.05 at 70. One of Ms. Lemanski's treating physicians noted that "[s]he reports that in Feb[ruary], she had a hepatitis A vaccine and began to have muscle twitching a few hours later, over the course of the next few months, this progressed to include fatigue and weakness of the [left] foot." *Id.* at 103. Laboratory

[7] Sciatic neuropathy, also known as "sciatica," is "a syndrome characterized by pain radiating from the back into the buttock and along the posterior or lateral aspect of the lower limb; it is most often caused by protrusion of a low lumbar intervertebral disk." *Sciatica*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=44946 (last visited Feb. 24, 2025).

[8] "Plexitis" is inflammation of intermingled nerves or nerve fiber bundles. *Plexitis*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=39634&searchterm=plexitis (last visited Feb. 24, 2025).

[9] "Intravenous Immunoglobulin (IVIg)" is defined as "[a] therap[y] prepared from a pool of immunoglobulins (antibodies) from the plasma of thousands of healthy donors. Immunoglobulins are made by the immune system of healthy people for the purpose of fighting infections…IVIG/SCIG work in different ways to prevent the body from attacking itself and to decrease several types of inflammation in the body." IVIG, https://rheumatology.org/patients/intravenous-immunoglobulin-ivig (last visited Feb. 25, 2025).

workup was unremarkable for autoimmune/inflammatory markers, but remarkable for extremely elevated Epstein-Barr Virus ("EBV") IgG[10] antibodies. *Id.* at 192.

On August 8, 2018, Dr. Iuliana Niculescu evaluated Ms. Lemanski for inpatient rehabilitation, and wrote that her "workup thus far has not revealed a specific etiology to her symptoms [and her EMGs] showed ongoing denervation at L4-S1 bilaterally but did not show AIDP [acute inflammatory demyelinating polyneuropathy]/CIDP [chronic inflammatory demyelinating polyradiculoneuropathy] or motor neuron disease." Ex. 17.05 at 159. On August 10, 2018, midway through a five-day IVIg treatment regimen, a neurology consult noted that Ms. Lemanski was able to use a walker, move the toes of her right foot, and her leg tingling had improved with Lyrica. *Id.* at 131. The neurologist noted that "[i]nitially when she had the hepatitis vaccine, she had chills and nights sweats," but listed the etiology as "not clear." *Id.* at 133.

Another physician in the hospital ordered a "CT chest and abdomen for possible paraneoplastic syndrome due to the atypical time course after the vaccine." Ex. 6.02 at 36. During her pulmonology consult, Ms. Lemanski reported chills and night sweats that began within 48 hours of receiving the Hepatitis A vaccine (a contention *not* reflected in the contemporaneous medical records, it should be noted), which had progressed to muscle aches and twitching in her left leg a few days later. *Id.* at 31. A chest CT scan performed on August 8, 2018, showed non-neoplastic nodules (non-cancerous abnormal growths) and a pulmonary embolus (blood clot) in her right lung. Ex. 7 at 39. A lower extremity venous doppler on August 9, 2018, showed an acute deep vein thrombosis ("DVT")[11] of Ms. Lemanski's left leg. Ex. 17.05 at 132.

Ms. Lemanski's discharge summary on August 10, 2018, deemed the etiology of her condition to be unclear. Ex. 17.05 at 53. That same day, Ms. Lemanski was transferred to inpatient rehabilitation ("IPR"), where she received five plasmapheresis treatments over the course of two weeks. Ex. 6.02 at 5; Ex. 17.04 at 623. She experienced chest pain associated with the injection port and was assessed for cardiac disease. Ex. 17.04 at 624. The DVT in her left leg was noted, and medication adjustments were proposed. *Id.* at 623.

Another EMG (performed on August 22, 2018) now showed a worsening of Ms. Lemanski's neurological condition. Ex. 6.01 at 373-76. Julie Ferris, M.D., a rehabilitative

[10] IgG antibodies, which are present in all body fluids, are proteins that the immune system makes to fight bacteria and viruses. The body keeps a "blueprint" of all the IgG antibodies that have been created so if a person is exposed to the same germs again, the immune system can quickly make more antibodies. *Immunoglobulins Blood Test*, Medline Plus, https://medlineplus.gov/lab-tests/immunoglobulins-blood-test/ (last visited Mar. 12, 2025). In effect, evidence of an IgG antibody is deemed to reflect a resolved infection rather than a new/active infection. *See Knorr v. Sec'y of Health & Hum. Servs.*, No. 15-1169V, 2018 WL 6991548, at *4 n.7 (Fed. Cl. Spec. Mstr. Dec. 7, 2018).

[11] "Deep Vein Thrombosis" is a blood clot in a vein located deep within a person's body, usually in their leg. *Deep Vein Thrombosis (DVT)*, Cleveland Clinic, https://my.clevelandclinic.org/health/diseases/16911-deep-vein-thrombosis-dvt (last visited Feb. 25, 2025).

medicine specialist, noted that the findings "showed diffuse denervation in upper and lower extremities including paraspinals with cranial nerve sparing consistent with diffused axonal peripheral neuropathy." Ex. 17.04 at 623. At Ms. Lemanski's discharge from IPR on September 7, 2018, Dr. Ferris wrote in the discharge summary, "[p]rogressive axonal neuropathy of unclear etiology." *Id.* Dr. Ferris referred Ms. Lemanski to outpatient PT and occupational therapy ("OT"). Ex. 8 at 7.

Outpatient Treatment and Tentative Polyneuropathy Diagnosis

Ms. Lemanski thereafter attended 10 PT/OT sessions but made minimal progress. *See generally* Ex. 8. At the initial assessment on September 10, 2018, Ms. Lemanski arrived in a wheelchair. Ex. 8 at 9. On exam, she displayed significant weakness in both legs, decreased tone, intact light touch sensation, and decreased vibration sensation. *Id.* at 10. Ms. Lemanski's PT/OT sessions were interrupted when she fell in her bedroom and fractured her left tibia and fibula on September 11, 2018. Ex. 6.01 at 321-27.

On September 20, 2018, Ms. Lemanski visited Rebecca Grysiewicz, a neurologist, and complained of tingling in her arms and on her head, and twitching of her upper body, hands, and lips. Ex. 10 at 41. Dr. Grysiewicz wrote that

> "the most likely initial diagnosis was acute axonal motor neuropathy, which is a variant or subtype of GBS ["Guillain-Barré syndrome"]. While this is much more rare, there are case reports associated with vaccinations. Symptoms have been present for more than a few weeks and may be consistent with a chronic axonal motor neuropathy (CAMN); findings are not consistent with neurophysiological CIDP."

Id. at 45.

By September 26, 2018, Ms. Lemanski's upper body symptoms had worsened. Ex. 10 at 35. Ms. Lemanski thereafter received a second round of five plasmapheresis treatments in October 2018. Ex. 6.01 at 235-62. On October 19, 2018, Ms. Lemanski reported that the treatments had helped, although she was still experiencing leg weakness, twitching, and burning pain in her feet at night. Ex. 10 at 30. Dr. Grysiewicz planned to repeat the plasmapheresis again in a month. *Id.* at 34. During her third round of plasmapheresis treatment, Ms. Lemanski saw Dr. Grysiewicz again, on November 16, 2018, and complained of fatigue, headaches, arm weakness, foot swelling, and unusual pain sensations. *Id.* at 26. Dr. Grysiewicz noted a slight improvement in Ms. Lemanski's leg strength. *Id.* at 28.

On December 13, 2018, Ms. Lemanski visited Dr. Grysiewicz and reported that she was experiencing more twitching, trembling, and weakness in her arms and core. Ex. 10 at 20. Dr. Grysiewicz ordered a fourth round of plasmapheresis and wrote, "[f]indings presumed to be secondary to an immune mediated response due to Hepatitis A vaccination; however, she is experiencing progression in to [sic] her [upper extremities] and core with increasing fasciculations. The progression of symptoms is atypical for an immune mediated peripheral neuropathy." *Id.* at 24.

A new EMG (performed on December 13, 2018) showed that Ms. Lemanski's denervation had spread. Ex. 10 at 13; Ex. 12 at 35-42. There was "evidence of diffuse active denervation and neurogenic recruitment patterns with limited if any features of re-innervation" that was worse in her legs. Ex. 10 at 13. The report concluded that "findings in this study are indicative of diffuse primarily axonal motor neuropathy versus neuronopathy. Possible causes may include inflammatory axonal motor neuropathies including those due to paraneoplastic causes; however, motor neuron disease should be considered in the differential diagnosis." *Id.*

At a visit with Dr. Grysiewicz on December 20, 2018, and during her fourth round of plasmapheresis, Ms. Lemanski again reported improvement, except for weakness in her arms, neck, and shoulders. Ex. 10 at 15. Ms. Lemanski insisted that she never had extremity twitches or weakness prior to the Hepatitis A vaccine. *Id.* On exam, Ms. Lemanski had no fasciculations, positive deep tendon reflexes, positive sensation, and positive, although diminished, motor strength. *Id.* at 17. Dr. Grysiewicz noted "definitive improvement with steroids and plasmapheresis [, . . . and] a strong association temporal with the administration of the vaccination." *Id.* at 18.

Cardiac Arrest and Death

On January 7, 2019, Ms. Lemanski visited Dr. Grysiewicz and reported improvement from the plasmapheresis, but noted that she was depressed and anxious about her condition. Ex. 10 at 9. On exam, Dr. Grysiewicz observed occasional tremors in her hands and weakness in her upper body. *Id.* at 13. Dr. Grysiewicz also noted that Ms. Lemanski had tachycardia and was panting. *Id.* at 9. On January 9, 2019, Dr. Ferris noted that Ms. Lemanski's most recent EMG "was consistent with a severe axonal neuropathy although [motor neuron disease could not] fully be ruled out." Ex. 7 at 6. Dr. Ferris also noted that Ms. Lemanski was seeing a pulmonologist for positional breathing difficulties. *Id.*

On January 16, 2019, sometime between 7:30 a.m. and 8:15 a.m., Ms. Lemanski went into cardiac arrest at her home. Ex. 11 at 9. Petitioner initiated CPR, with assistance from emergency responders who arrived not long after. *Id.* Ms. Lemanski was transported to Henry Ford Macomb Hospital but could not be resuscitated. *Id.* at 10. She was pronounced dead at 9:30 a.m. *Id.* Ms. Lemanski's death certificate was signed by Dr. Kotsonis, PCP, on January 22, 2019, and it listed

the cause of death as "Demyelinating Polyneuropathy." Ex. 13. Ms. Lemanski's autopsy of March 9, 2019, however, was inconclusive as to her cause of death. Ex. 11 at 33-37. That autopsy identified no evidence of myocardial infarction, pulmonary embolism, or histopathological changes of her brain. *Id.* at 33.

II. Fact Witnesses

A. *Suzanne Zacharski*

Petitioner (Ms. Lemanski's daughter) testified at the hearing. *See generally* Tr. 5-27. She maintained that Ms. Lemanski had been very healthy prior to receiving the vaccine. *Id.* at 7-8. In fact, the two of them regularly ran half-marathons together. *Id.* She also explained that she had lived with Ms. Lemanski for more than 30 years, and that they talked about everything. *Id.* at 6, 9. Petitioner testified that Ms. Lemanski never complained about muscle twitching, fatigue, or weakness prior to receiving the vaccine. *Id.* at 8.

Petitioner also testified that she was "100 percent" certain that her mother's symptoms began after the vaccine. Tr. at 9. When asked why the doctor at Ms. Lemanski's February 5, 2018 appointment listed her twitching symptoms as "ongoing for three weeks," Petitioner surmised that a provider likely clicked the wrong button when she took Ms. Lemanski's history. *Id.* at 10. Petitioner theorized that the error carried over into the next appointment on February 9, 2018, which is why the doctor's second notes stated that Ms. Lemanski's symptoms had been ongoing for one additional week beyond the prior record. *Id.*

Recalling the onset and course of Ms. Lemanski's symptoms, Petitioner testified that Ms. Lemanski first experienced muscle twitching and then fasciculations, followed by left leg and then right leg weakness. Tr. at 11. By the end of her illness course, she had difficulty moving her arms as well. *Id.* Referencing a physical therapy note from April 2018 that described Ms. Lemanski having fallen twice, Petitioner explained that the weakness in her left leg caused her to fall, and she clarified that Ms. Lemanski did not have a tendency to fall prior to vaccination. *Id.* at 12. Petitioner also recounted Ms. Lemanski's fall during their trip to Disney World in June 2018. *Id.* She explained that while in the shower, Ms. Lemanski lost her balance and fell, lacerating her finger on the glass doorjamb. *Id.* at 13.

In July 2018, Ms. Lemanski received five doses of intravenous steroid treatment. Tr. at 13. Petitioner testified that Ms. Lemanski felt that the steroid treatments always gave her more energy, and were more helpful than the physical therapy sessions. *Id.* at 13-14. During Ms. Lemanski's August 2018 hospitalization, she received a five-day course of IVIg treatment. *Id.* at 14. Petitioner explained that while Ms. Lemanski was very excited about the treatment, it resulted in only slight improvement in her leg function for a short while. *Id.* At the end of August 2018, Ms. Lemanski

began a course of plasmapheresis. *Id.* at 14-15. Petitioner testified that this treatment was deemed worthwhile by Ms. Lemanski's treaters because "they felt that her symptoms were from the vaccine." *Id.* at 15. Petitioner explained that the plasmapheresis seemed to work well, as reflected in medical records in which Ms. Lemanski so informed treaters. *Id.* at 15, 16.

Petitioner recalled that Ms. Lemanski suffered yet another fall in September 2018, when her legs crumbled underneath her after Petitioner attempted to transfer her out of her wheelchair. Tr. at 17. Around this time, Ms. Lemanski began seeing Dr. Grysiewicz, who believed that her illness was caused by the vaccine. *Id.* at 17-18. Because the plasmapheresis seemed to help Ms. Lemanski, Dr. Grysiewicz ordered two more rounds in October and November 2018. *Id.* at 18. Petitioner testified that the plasmapheresis was "extremely difficult" for her mother, and she often suffered several side effects. *Id.* at 18-19. Still, she always felt better post-treatment, so she continued with multiple rounds. *Id.* at 19. Petitioner testified that while the treatment certainly helped, Ms. Lemanski's symptoms continued to progress, and by the end of 2018, Ms. Lemanski was wheelchair-bound. *Id.*

Petitioner then recalled the morning of Ms. Lemanski's death. Tr. at 21. She noted that they had an appointment that day to see a specialist about getting fitted for noninvasive ventilation because, by this point, Ms. Lemanski had trouble breathing due to her weakened chest muscles. *Id.* When Petitioner tried to wake Ms. Lemanski up that morning, she discovered she was not breathing. *Id.*

Petitioner concluded her testimony by clarifying that Ms. Lemanski had been concerned that she might have had ALS. Tr. at 26. At almost every appointment she went to, she would ask Dr. Grysiewicz about ALS. *Id.* Dr. Grysiewicz always checked for cranial nerve involvement and upper motor neuron nerve involvement, and finding nothing, reassured Ms. Lemanski that she did not likely have ALS. *Id.*

B. *Daniel Lemanski*

Daniel Lemanski (Ms. Lemanski's husband) also testified briefly. *See generally* Tr. 28-35. He noted that he and Ms. Lemanski had been married for over 40 years by the time she passed. *Id.* at 29. He explained that they often communicated about important things, and maintained that Ms. Lemanski would have spoken to him if she had serious health concerns. *Id.* at 30. He then testified that Ms. Lemanski was in very good health, and did not verbalize any health concerns in the months leading up to the vaccine. *Id.* at 31-32. As far as he could recall, her symptoms began post-vaccination. *Id.* at 33.

III. Expert Witnesses

A. *Petitioner's Experts*

1. Dr. Vincent Lau – Dr. Lau, a neurologist, prepared two written reports and testified on behalf of Petitioner. *See generally* Tr. at 35-127; Report, dated June 26, 2022 (ECF No. 26-2) ("First Lau Rep."); Report, dated Jan. 9, 2023 (ECF No. 32-2) ("Second Lau Rep."). Dr. Lau opined that the Hepatitis A vaccine was a substantial factor in Ms. Lemanski's development of CMAP, which ultimately led to her death. Tr. at 117.

Dr. Lau is the Director of Clinical Research at Alnylam Pharmaceuticals in Cambridge, MA. CV, dated June 20, 2024, filed as Ex. 123 (ECF No. 49-2) ("Lau CV"). He received his medical degree from Icahn School of Medicine in Mount Sinai before completing his residency in neurology at Yale New Haven Hospital and his fellowship in neuromuscular medicine at Harvard Medical School. Lau CV at 1; Tr. at 36-37. He is board certified in neurology and neuromuscular medicine. Tr. at 39. Earlier in his career, Dr. Lau was an Assistant Professor of Neurology at Boston University and the Program Director of the Neuromuscular Medicine Fellowship at Boston University. Lau CV at 1; Tr. at 37. In his current role as Director of Clinical Research, Dr. Lau designs clinical trials on diseases that include peripheral neuropathy. Tr. at 37. He has also lectured and published articles on various topics concerning neuropathies and EMG cases/simulations. *Id.* at 38; Lau CV at 3-4.

Dr. Lau began his testimony by reviewing Ms. Lemanski's medical history. Tr. at 42. He noted that she had a history of foot and knee pain and had been diagnosed with plantar fasciitis. *Id.* He also acknowledged that she had experienced numbness and tingling in her lower extremities in December 2016, but noted that the EMG from that visit was normal and did not indicate the existence of any serious underlying neuromuscular disorder at that time. *Id.* at 43-44; Ex. 6.01 at 74-75; Ex. 6.02 at 528.

Turning to Ms. Lemanski's post-vaccination medical records, Dr. Lau noted that Ms. Lemanski presented to her PCP on February 5, 2018, complaining of symmetrical aching leg pain and bilateral twitching in both knees. Tr. at 45; Ex. 6.01 at 58. Dr. Lau admitted that the medical records listed the onset of these symptoms as "three weeks ago." Tr. at 45; Ex. 6.01 at 58. On February 9, 2019, Ms. Lemanski returned to her PCP with the same complaints. Tr. at 45; Ex. 6.01 at 56. Her neurologic exam at both visits was normal. Tr. at 45-46; Ex. 6.01 at 56-58.

Dr. Lau then reviewed the results of Ms. Lemanski's four EMGs, in an effort to illustrate the progression of her symptoms (and also to rebut Respondent's contentions that some of the EMGs' findings were consistent with ALS). In his view, the first EMG – from March 1, 2018 – revealed that she was at that time experiencing some acute denervation. Tr. at 47; Ex. 6.02 at 104.

The results of this EMG showed decreased recruitment, which occurs when nerve fibers are unable to access (i.e., "recruit") the correct amount of muscle fibers. Tr. at 48. These denervation changes suggested that Ms. Lemanski was undergoing a neuropathic injury at this time – although Dr. Lau maintained that it was not yet chronic. *Id.* at 50-51. While an increase in amplitude (one of the markers for chronic denervation) was also present, the results of the EMG *as a whole* did not offer convincing evidence of chronic denervation. *Id.* at 50. Dr. Lau testified that it typically takes up to two to three months after the onset of an ongoing neuromuscular process for chronic denervation changes to appear on an EMG (thereby suggesting that the onset of Ms. Lemanski's neuropathy could not have been earlier than February 2018). *Id.*; First Lau Rep. at 10.

Dr. Lau then discussed Ms. Lemanski's second EMG, which was conducted in May 2018. Tr. at 56; Ex. 17.05 at 465. In his view, the results of this EMG established the presence of the same kind of early, ongoing denervation that was apparent in the March EMG – but with no evidence of increased amplitude (and hence *still* not evidencing a chronic process). Tr. at 56. Indeed, Dr. Lau specifically acknowledged that the absence of any abnormalities on amplitude, duration, and polyphagia (the three markers for chronic denervation) meant that there was nothing to suggest chronic neuropathy occurring at this time, and he characterized the evidence of increased amplitude from the first EMG (performed two months before) as likely "overcall or something transient that wasn't the signal of something." *Id.* at 57.

Dr. Lau also maintained, however, that the impressions section of the May 2018 EMG deemed the observed abnormalities to be inconsistent with anything like a motor neuron disease, such as ALS. Tr. at 57; Ex. 17.05 at 463. Responding to Dr. Robbins's assertion that the finding of an absent left superficial fibrillary sensory nerve response might be a technical artifact, Dr. Lau noted that Ms. Lemanski's measured reflex was very robust on the right side (which underscored the extent to which the reflex on the left side was truly absent). Tr. at 58-59. Dr. Lau also pointed out that Dr. Daniel Menkes – a recognized medical leader and the "gold standard that people should aspire to in terms of performing EMGs" – had been responsible for conducting this second EMG. *Id.* at 60.

Dr. Lau moved on to the results of Ms. Lemanski's August 2018 EMG. Tr. at 68; Ex. 17.04 at 227. Its results showed increased duration, amplitude, and phases in multiple muscles, which now indicated the presence of chronic denervation changes. Tr. at 68; Ex. 17.04 at 227. Because chronic denervation typically appears two to three months after the onset of a neuropathy, these results suggested to Dr. Lau that Ms. Lemanski's neuropathy could not have begun before February 2018 (although they also allowed for the possibility of an onset *several months later*, since two to three months before August would fall in the May-June period).[12] Tr. at 69-70. Dr.

[12] During Petitioner's rebuttal case, Dr. Lau was asked on cross examination about this issue. He now attempted to stretch the relevant period, arguing that even if chronic denervation typically becomes evident *at least* two to three months after onset, it could take longer. Tr. at 380. He thus deemed evidence of chronicity *any time* between May and

Lau also pointed out that the August 2018 EMG revealed evidence of diffuse, severe, primarily axonal motor neuropathy without cranial nerve involvement, which (again) made a diagnosis of motor neuron disease less likely. *Id.* at 71; Ex. 17.04 at 228.

Finally, Dr. Lau evaluated Ms. Lemanski's fourth EMG from December 2018, which he noted showed evidence of ongoing acute denervation. Tr. at 82; Ex. 12 at 36-37. The results also revealed more evidence of increased duration, amplitude, and polyphagia, confirming that the chronic denervation changes first detected in August had progressed. Tr. at 83; Ex. 12 at 37.

Turning to Ms. Lemanski's symptoms development, Dr. Lau contended that her overall presentation was most consistent with an ongoing chronic neuropathy rather than motor neuron disease. Tr. at 52; First Lau Rep. at 11. For example, Ms. Lemanski had reported head "twitching" at a March 28, 2018 visit with her PCP – beginning the month before but with some improvement. Tr. at 52; Ex. 6.01 at 52. Dr. Lau deemed this significant, maintaining that a patient's twitching would not improve if they were experiencing motor neuron disease. Tr. at 52. He also highlighted the records from Ms. Lemanski's April 18, 2018 appointment. *Id.* at 52-53; Ex. 2 at 5. At this time, Ms. Lemanski noted a "left foot drop," which Dr. Lau testified was consistent with an ongoing neuropathic process. Tr. at 53; Ex. 2 at 7. Dr. Lau also pointed out that the assessment section of this report noted the question of etiology, and listed the Hepatitis A vaccine as a possible trigger/explanation. Tr. at 54; Ex. 2 at 7.

Regarding the specific nature of Ms. Lemanski's neuropathy, Dr. Lau testified that Ms. Lemanski's EMGs showed more of an axonal than demyelinating neuropathy. Tr. at 67. Indeed, because Ms. Lemanski experienced severe motor symptoms but without accompanying sensory issues, her neuropathy was best classified as predominantly (or solely) motor. *Id.* at 100. He further supported his diagnostic theory by referencing Dr. Grysiewicz's assessment from September 11, 2018, in which she wrote that Ms. Lemanski's initial presentation was most in line with a diagnosis of acute axonal motor neuropathy – but because her symptoms had lasted for more than four weeks, the diagnosis was likely chronic axonal motor neuropathy. *Id.* at 74-75; Ex. 10 at 45.

Dr. Lau then elaborated on the differences between demyelinating and axonal polyneuropathies, referencing literature offered in support. Tr. at 97; K. Doppler et al., *Destruction of Paranodal Architecture in Inflammatory Neuropathy with Anti-Contactin-1 Autoantibodies*, 86 J. of Neurol., Neurosurg. and Psych. 720, 724-25 (2015), filed as Ex. 59 (ECF No. 36-12) ("Doppler"). Doppler's authors considered a cohort of 53 patients with CIPD, plus 21 with GBS, and compared their blood with controls (some healthy, some with different autoimmune diseases). Doppler at 720. Four of the CIDP patients were found to possess evidence of IgG anti-contactin-1

August 2018 to be reasonable, even if onset of Ms. Lemanski's neuropathic injury had occurred in February. *Id.* at 381.

antibodies, with the authors focusing on this antibody because contactin-1 is a "paranodal protein[13] that seems to induce an immune response." *Id.* at 720, 722. Clinical data for this subset of CIDP patients (plus histopathology testing) revealed that they all had experienced an acute form of CIDP onset (sufficient for treaters to initially mistake it as GBS), plus "severe motor and moderate sensory symptoms and relapsing-remitting course of disease." *Id.* at 725. A sural nerve biopsy performed on three of the patients showed axonal loss but only few thinly myelinated fibers, resulting in a diagnosis of axonal (rather than demyelinating) neuropathy. *Id.* at 724. Doppler's authors concluded that an anti-contactin-1-driven neuropathy might constitute "another form of paranodopathy," featuring "disruption of paranodal architecture, subsequent axonal loss and slower nerve condition," but without the common features of demyelination usually found with CIDP. *Id.* at 727.

Doppler, Dr. Lau claimed, thus established that anti-contactin-1 autoantibodies are one of the multiple immune antibodies that can cause chronic, axon-specific neuropathies. Tr. at 98. Based on the nerve biopsies conducted in Doppler, anti-contactin-1 neuropathies are more likely to be associated with an axonal neuropathy, like the one Ms. Lemanski had, as opposed to a demyelinating neuropathy. *Id.* at 99; Doppler at 724. Doppler does not, however, comment on *how* this class of autoantibodies would come into existence.

To further support his contention that anti-contactin-1 antibodies are associated with axonal neuropathies, Dr. Lau referenced a review article that discussed antibodies studied in the context of CIDP cases. Tr. at 101; L. Querol et al., *Autoantibodies in Chronic Inflammatory Neuropathies: Diagnostic and Therapeutic Implications*, 13 Nature Rev. Neurol. 533-47 (2017), filed as Ex. 77 (ECF No. 36-30) ("Querol II").[14] The authors of Querol II referenced a prior study of three CIDP patients who experienced an "aggressive disease phenotype" featuring acute onset, predominantly motor symptoms, older age onset, evidence of denervation at the first EMG, and a poor response to IVIg treatment. Querol II at 535 (*citing* L. Querol et al., *Antibodies to Contactin-1 in Chronic Inflammatory Demyelinating Polyneuropathy*, 73 Ann. Neurol. 370-80 (2013), filed as Ex. 78 (ECF No. 36-31) ("Querol I")). Dr. Lau testified that this clinical description fit Ms. Lemanski's presentation – she had "100 percent" of the listed features. Tr. at 102. (As noted below, however, the evidence on the record is equivocal as to the effectiveness of IVIg treatment in Ms. Lemanski's case.) Anti-contactin-1 antibodies were detected in this group of subjects. Querol II at 535. Thus,

[13] Specifically, contactin-1 is a cell surface protein that promotes the stability of sodium ion channel clusters located at the nodes of Ranvier along a nerve (with these nodes responsible for facilitating conduction of the nerve impulse down the axon). First Berger Rep. at 9; A. Uncini et al., *Nodo-Paranodopathy: Beyond the Demyelinating and Axonal Classification in Anti-Ganglioside Antibody-Mediated Neuropathy*, 124 Clin. Neurophysiology 1928, 1931 (2013), filed as Ex. 84 (ECF No. 36-37).

[14] Petitioner has offered two items of literature with the same primary author (Dr. Luis Querol) – but because Querol II was published later in time, it receives the short-form title I have given it, even though it is the *first time* in this Decision that I have referred to either article.

even though the umbrella of "CIDP" predominantly consists of demyelinating neuropathies, Dr. Lau deemed an anti-contactin-1 neuropathy to be a "more axonal picture" falling within that umbrella. Tr. at 103. In his view, anti-contactin-1 neuropathy should be suspected when there is prominent motor involvement and signs of axonal damage at onset. *Id.* at 104.

Addressing the "motor" classification of Ms. Lemanski's illness, Dr. Lau took note of an item of literature offered by Respondent. S. Oh et al., *Chronic Inflammatory Axonal Polyneuropathy*, 91 J. of Neurol., Neurosurg. and Psych. 1175-80 (2020), filed as Ex. A-17 (ECF No. 30-18) ("Oh"). Oh examined 33 patients with immunotherapy-responding chronic axonal polyneuropathy. Oh at 1175. The authors of Oh acknowledged that axonal CIDP has not yet been proven as a proper diagnostic classification, but contended that this form of polyneuropathy should be viewed as a distinct kind of immunotherapy-responsive subset of CIDP, referring to it as "CIAP" (chronic inflammatory axonal polyneuropathy). *Id.* at 1175, 1178. CIAP and CIDP could be distinguished by test results establishing the existence of inflammation, plus EMG/NCS[15] results suggesting an axonal focus over demyelination. *Id.* at 1179-80. As Dr. Lau pointed out, three of the 33 patients with CIAP considered in Oh exhibited motor-only symptoms. *Id.* at 1176; Tr. at 113. Looking at the overall clinical presentation of these three patients, Dr. Lau deemed Oh supportive of the conclusion that a motor-oriented chronic axonal neuropathy was rare – but nevertheless possible. Tr. at 112-13.

Dr. Lau then discussed Ms. Lemanski's demonstrated improvement in response to steroids and immunotherapies (specifically plasmapheresis), and how this was consistent with an immune-mediated disease. Tr. at 72. Physician's notes from August 26, 2018, established that Ms. Lemanski felt better after plasmapheresis, allowing her to ambulate more freely. *Id.*; Ex. 7 at 80. These short-term improvements, Dr. Lau explained, would be expected in someone suffering from a form of an autoimmune neuropathy. Tr. at 73. Ms. Lemanski also reported improvement after starting her fourth course of plasmapheresis in December 2018. *Id.* at 83. According to the notes from that day, she was able to pet the top of her dog's head and give him a treat, which she was unable to do the week prior. *Id.* at 84; Ex. 10 at 15. Dr. Lau pointed out that this is a very clear change in function and improved strength. Tr. at 84. In addition, Dr. Grysiewicz's notes from the December 2018 visit noted that Ms. Lemanski was experiencing "definitive improvement with steroids and plasmapheresis," leading her to order another round of the treatment. Ex. 10 at 18; *see also Id.* at 9. Dr. Lau acknowledged that Dr. Grysiewicz documented that the progression of Ms. Lemanski's symptoms was "atypical" for immune-mediated peripheral neuropathy. Tr. at 85; Ex. 10 at 24. But he deemed this to mean that the disease was more aggressive than usual. Tr. at 85.

[15] A Nerve Conduction Study (NCS) test "measures how fast an electrical impulse moves through your nerve." During the test, a person's nerve is stimulated with electrode patches placed on their skin. The test is used to identify nerve damage. *Nerve Conduction Studies*, John Hopkins Medicine, https://www.hopkinsmedicine.org/health/treatment-tests-and-therapies/nerve-conduction-studies (last visited Mar. 7, 2025).

Ms. Lemanski also experienced improvements as a result of her IVIg treatments. Tr. at 65, 97; Ex. 17.05 at 131; Ex. 6.01 at 379. But this form of treatment was not without side effects. Tr. at 64. According to Ms. Lemanski's assessment and plan from August 2018, she had a pulmonary embolism (blood clot in the lung). Ex. 7 at 39. Dr. Lau explained that an individual with a pulmonary embolism in the past would be at greater risk for developing a pulmonary embolism with IVIg. Tr. at 64. But Ms. Lemanski had continued with IVIg treatments despite such risks because she felt she was improving. *Id.* at 65, 97.

Of course, the medical record suggested that Ms. Lemanski had enjoyed no *long-term* improvement from these immunotherapies. But Dr. Lau maintained that improvement secondary to IVIg in a chronic axonal neuropathy was never guaranteed, with regression possible if the treatment was interrupted or discontinued. Tr. at 72, 107; J. Godil et al., *Refractory CIDP: Clinical Characteristics, Antibodies, and Response to Alternative Treatment*, 418 J. of Neurological Sci. 1-3 (2020), filed as Ex. 23 (ECF No. 26-7) ("Godil"). Although Godil established that approximately three-quarters of CIDP patients responded well to immunotherapies (Godil at 2), such treatments did not guarantee success. Tr. at 108. Thus, the possibility that Ms. Lemanski had experienced an immune-mediated neuropathy was not wholly rebutted by the treatment's ultimate ineffectiveness. *Id.* at 108-09.[16]

Dr. Lau concluded his testimony with the argument that Ms. Lemanski's condition was not ALS. He began with a reference to Ms. Lemanski's autopsy, noting that it indicated no significant histopathological (brain) changes – inconsistent with what would be seen for ALS. Tr. at 87-89; Ex. 11 at 33. In support, he cited to an article offered by Respondent regarding brain activity in ALS patients. *See* G. Coan & C. Mitchell, *An Assessment of Possible Neuropathology and Clinical Relationships in 46 Sporadic Amyotrophic Lateral Sclerosis Patient Autopsies*, 15 Neurodegen. Diseases 301-12 (2015), filed as Exhibit A-6 (ECF No. 30-7) ("Coan"). Tr. at 88. Coan's authors studied 46 autopsies of patients who had ALS and had passed away. Coan at 303. The majority of the changes that were discovered were observed in the brain. *Id.* at 306.

Dr. Lau went on to further distinguish Ms. Lemanski's condition from ALS by citing an article that discussed the pathophysiology of ALS. Tr. at 89; L. Foster & M. Salajegheh, *Motor Neuron Diseases: Pathophysiology, Diagnosis, and Management*, 132 Am. J. of Med. 32-37 (2019), filed as Ex. 22 (ECF No. 26-6) ("Foster"). Foster noted that hyperreflexia is a key feature of ALS. Foster at 33. Ms. Lemanski, by contrast, had experienced *decreased* reflexes, which Dr. Lau explained is "much more consistent with neuropathy versus a motor neuron disease, which is

[16] Dr. Lau also addressed arguments from Respondent's experts that some of Ms. Lemanski's reported immunotherapy improvement might simply be due to a placebo effect. *See, e.g.*, Tr. at 80-82. But because I am finding that Ms. Lemanski likely experienced some form of axonal-oriented chronic neuropathy, this dispute need not be delved into in greater detail (although as noted below, Ms. Lemanski's fluctuating treatment course and her responsiveness to different kinds of treatments does bear on causation in other regards).

kind of notoriously, famously [sic] associated with increased reflexes." Tr. at 92. Furthermore, motor neuron disease is "famously progressive," meaning that a patient's treatment course should be marked by a consistent worsening of symptoms. *Id.* at 90-91. But Ms. Lemanski's condition tended to fluctuate. *Id.* at 91. And although there are medications that slow the progression of ALS, Ms. Lemanski was never treated with any of them, nor was she referred to any clinical trials for ALS. *Id.* at 93. It could thus be inferred that Ms. Lemanski's treaters largely did not suspect ALS as a possible explanation for her presentation.

On cross, Dr. Lau reiterated his opinion that Ms. Lemanski had suffered from a purely axonal motor neuropathy. Tr. at 120. He agreed that articles he offered, like Doppler or Querol I and II, all involved patients with CIDP (which did not properly fit Ms. Lemanski's clinical symptoms and testing results), but deemed them relevant because they all allowed for the possibility of subset of CIDP-like conditions that did not primarily involve demyelination. *Id.* at 121. Thus, they provided diagnostic guidance relevant to the facts of this case as well. *Id.*

Dr. Lau further clarified on cross-examination that although it could not be shown on this record that Ms. Lemanski had in fact ever possessed the anti-contactin-1 antibody – or even that she likely *did* suffer from this specific form of neuropathy – her phenotypic presentation remained consistent with the form of neuropathy discussed for the small minority of CIDP patients in the Doppler and Querol articles (adding – although this was not part of his expert opinion – that anti-contactin-1 was "the most adjacent thing," in terms of homology, to the Hepatitis A capsid). Tr. at 122-23. Her also maintained that although the record established that Ms. Lemanski was ultimately diagnosed with CMAN (referred to by Dr. Grysiewicz as "CAMN" (Ex. 10 at 45)), such a diagnosis was functionally equivalent to CMAP. *Id.* at 124. At the same time, however, Dr. Lau admitted that Ms. Lemanski did not have anti-ganglioside antibodies, which would be associated with CMAN, but argued that this alone did not defeat the diagnosis. *Id.* And while Ms. Lemanski's autopsy did not show evidence of CMAP, Dr. Lau noted that the nerves were not examined. *Id.* at 124.

2. Dr. Mel Burger – Dr. Burger prepared three written reports and testified on behalf of Petitioner. See generally Tr. at 130-189; Report, dated July 31, 2023 (ECF No. 35-2) ("First Berger Rep."); Report, dated Feb. 9, 2024 (ECF No. 42-2) ("Second Berger Rep."); Report, dated June 11, 2024 (ECF. No. 47-8) ("Third Berger Rep."). Dr. Berger opined that the Hepatitis A vaccine caused Ms. Lemanksi to suffer an aberrant immune response, resulting in the development of CMAP. First Burger Rep. at 12; Tr. at 172.

Dr. Berger is an independent consultant and subject matter expert in the areas of immunoglobulins, immunologic diseases, biopharmaceutical drug development, and vaccine adverse effects. CV, dated July 31, 2023, filed as Ex. 48 (ECF No. 35-3) ("Berger CV). He received

his medical degree and Ph.D. in biochemistry at Case Western Reserve University. Berger CV at 1; Tr. at 131. He went on to complete a residency in pediatrics at Boston Children's Hospital and a fellowship in allergy and immunology at the National Institute of Health. Tr. at 131. From 1984-2001 and 2006-2008, he served as the Chief of the Allergy-Immunology-Rheumatology Division at Rainbow Babies and Children's Hospital. *Id.* at 131-32; Berger CV at 2. In this role, he saw patients with immune deficiencies and autoimmune diseases, taught students and residents, and conducted research in both the clinic and laboratory. Tr. at 132. He was also an Associate Professor and Professor of Pathology at Case Western from 1985-2008. *Id.*; Berger CV at 2. Dr. Berger is board-certified in pediatrics and allergy-immunology. Tr. at 135; Berger CV at 4. He has completed numerous clinical research studies and has published over 100 articles. Berger CV at 6-18.

Key to Dr. Berger's opinion was the purported role of the human contactin-1 protein as a target for an autoimmune attack in conditions resembling Ms. Lemanski's overall presentation. Tr. at 142. Contactin-1, Dr. Berger explained, is a protein found on the surface of the nerve cells which facilities attachment of the myelin fingers onto the axon. *Id*. at 155, 157. This protein "also serves to organize different domains within the axon which have different aggregations of ion channels that are necessary for propagation of the electrical impulse along the nerve." *Id*. Contactin-1 is specifically found at the node of Ranvier – the locations along the nerve that allow for ion currents to propagate an electrical impulse. *Id.* at 156.

An autoantibody attack on contactin-1, Dr. Berger contended, could block the node of Ranvier, resulting in harm to the junction that holds the myelin and axon together, as well as to the axon itself. Tr. at 146. And this attack could lead an individual to experience short circuits in nerve electrical impulses, which might be perceived as twitching or fasciculations. *Id*. at 157. According to Dr. Berger's first report, there has been increased recognition among immunologists and neuroimmunologists that proteins in and/or adjacent to the nodes of Ranvier can be targets of immunologic attack in conditions that resemble GBS or CIDP (although these conditions may have clinically distinctive features – perhaps more indicative of axonal damage than the demyelination common to GBS and CIDP). First Berger Rep. at 9. And there is potential, biologically-significant homology between components of contactin-1 and the Hepatitis A capsid, which means there are regions/epitopes within both proteins that could bind to the same antibody or Major Histocompatibility Complex ("MHC")[17] molecule and T-cell receptor, resulting in production of

[17] MHC molecules (also known as "Human Leukocyte Antigens") are a "group of genes that code for proteins found on the surfaces of cells that help the immune system recognize foreign substances." *Major Histocompatibility Complex*, Britannica Online, https://www.britannica.com/science/major-histocompatibility-complex (last visited Mar. 7, 2025). As noted in *Blackburn v. Sec'y of Health & Hum. Servs*., No. 10-410V, 2015 WL 425935, at *8-9 (Fed. Cl. Spec. Mstr. Jan. 9, 2015), MHC molecules have sometimes been compared by Program experts to a "'catcher's mitt' . . . holding a peptide from the vaccine which would then be presented to a host T cell. . . . [the] peptide would "fit" into the catcher's mitt due to its structural homology with the host protein, . . . and "would thereupon give instructions to the T cell," resulting in an autoimmune attack. But "although T cells and B cells recognize similar antigens, they

antibodies capable of mistaken, autoimmune self-attack. Tr. at 142. Dr. Berger opined that this likely occurred in Ms. Lemanski's case. First Berger Rep. at 13.

There are, Dr. Berger admitted, limitations to the applicability of molecular mimicry in establishing how an autoimmune disease likely occurs. For example, mere sequence homology is not sufficient for a molecular mimicry reaction to take place. Tr. at 144. In order to trigger a reaction, the complexes of the vaccine antigen (epitope) must bind to the groove of the MHC II molecule, which in turn, stimulates T-helper cells. *Id.* at 145, 148. Once the T-helper cells detect epitopes on the MHC II molecule, they activate B-cells that also recognize the epitope and produce antibodies against it. *Id.* at 148; Second Berger Rep. at 5. These B-cells then differentiate into long-lived plasma cells, which produce and secrete antibodies against vaccine antigens and, in some cases, against cross-reactive host antigens. Second Berger Rep. at 5.

Another issue with homology's limited applicability in this context is whether a short sequence of amino acids in a protein sub-component is sufficient to spark a cross-reaction – and why, if so, autoimmune disease is not more rampant in the population. Tr. at 152. Dr. Berger allowed that autoimmune reactions are very rare, but attributed this to the fact that an aberrant immune reaction is subject to a number of compensatory/regulating mechanisms that usually prevent its occurrence. Second Berger Rep. at 8. In addition, an individual with an autoimmune disease must possess an exact combination of MHC genes that allow for a specific cross-reactive response – and there is extraordinary diversity of MHC genes (meaning most individuals do not likely possess the necessary extra factors). Tr. at 152; Second Berger Rep. at 7. So, and for reasons that remain poorly understood, mechanisms of immune tolerance (which ordinarily would prevent an autoimmune attack) break down only in a small number of unfortunate (but predisposed) individuals, like Ms. Lemanski. Second Burger Rep. at 8.

Nevertheless, Dr. Berger contended, a five amino acid homologic sequence would in most cases be sufficient to spark a cross-reaction between the Hepatitis A VP1 component and contactin-1. Tr. at 143-44; Second Berger Rep. at 4. He also explained that the antigen and host epitopes do not have to be *identical* to trigger a molecular mimicry reaction. Tr. at 144. In support, he offered some literature. M.W. Cunningham et al., *Human and Murine Antibodies Cross-Reactive with Streptococcal M Protein and Myosin Recognize the Sequence GLN-LYS-SER-LYS-*

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play different roles in the adaptive immune system; T cells can only recognize structures that are bound to the [MHC], while antibodies are not so constrained." *Blackburn*, 2015 WL 425935, at *9.

MHC I molecules are expressed on almost all cells, while MHC II molecules are primarily expressed on antigen-presenting cells. Second MacGinnitie Rep. at 2. But while MHC I molecules interact with primary T cells (those more directly responsible for attacking foreign pathogens), MHC II molecules react with T-helper cells, which assist the process by which B cells encourage the production of antibodies. *Agarwal v. Sec'y of Health & Hum. Servs.*, No. 16-191V, 2020 WL 5651683, at *26 (Fed. Cl. Spec. Mstr. Aug. 31, 2020). Accordingly, and in either case, *MHC molecules do not themselves cross-react, nor are they situses for additional homology with a foreign antigen*. Rather, they assist in the process by which molecular mimicry occurs.

GLN in M Protein, 143 J. of Immuno. 2677-83 (1989), filed as Ex. 99 (ECF No. 43-10) ("Cunningham"). *Id.* at 143. Cunningham is a more than 40-year-old paper describing the mechanism by which a five amino acid sequence can cross react with a streptococcal surface protein and major heart muscle protein, leading to rheumatic fever. Cunningham at 2677. (Notably, however, rheumatic fever – unlike CMAP – is well understood by medical science to be propagated by an autoimmune cross-reaction – and Cunningham's findings are thus to that context, saying nothing about what degree of homology would be required for any *other* autoimmune diseases). Dr. Berger still deemed Cunningham relevant. Tr. at 143.

In response to the question of whether and how an autoimmune condition triggered by vaccination can become chronic, Dr. Berger explained that plasma cells (generated by a B cell-initiated process, and responsible for production of antibodies) live for as long as the patient is alive, and do not require additional antigenic stimulation to keep producing autoantibodies. Tr. at 149; *see also* F. Hiepe et al., *Long-lived Autoreactive Plasma Cells Drive Persistent Autoimmune Inflammation*, 7 Nat'l Rev. of Rheumatology 170-78 (2011), filed as Ex. 65 (ECF No. 36-18) ("Hiepe"). Hiepe is a review article that discusses chronic autoimmunity, noting that "long-lived plasma cells can support chronic inflammatory processes in autoimmune diseases by continuously secreting pathogenic antibodies, and they can contribute to flares of symptoms." Hiepe at 170. Hiepe goes on to state that "[plasma cells] reside immobilized in specific survival niches in the bone marrow and inflamed tissues, where they secrete antibodies for months, years, or a lifetime, independent of antigenic stimulation." *Id*. at 171.

Hiepe does not, however, say anything about whether and/or how any foreign antigenic stimulation (from a vaccine or wild infection) would cause this chronicity. In fact, it seems to suggest that (a) not all plasma cells necessarily will become long-lived, and (b) their chronicity is dependent on whether the cells find a "survival niche" – in bone marrow or inflamed tissues. Hiepe at 171-72.

Turning to Ms. Lemanski's specific presentation, Dr. Berger cited Querol II to support his argument that Ms. Lemanski likely suffered from an axonal-oriented polyneuropathy. Tr. at 158. Querol II's authors noted that Querol I had attempted to subclassify a large group of patients characterized as having CIDP according to the actual antibodies involved in their disease, identifying a subset of patients with anti-contactin-1 autoantibodies. Querol II at 535. These patients tended to be older, have an aggressive course, predominantly motor symptoms, and characteristically axonal features. *Id*. Because Ms. Lemanski's disease presentation resembled these patients, it could be inferred that contactin-1 autoantibodies were likely a cause of her symptoms as well. Tr. at 159, 373-74; *see also* Querol I at 372 (four out of 46 CIDP patients had antibodies that reacted with contactin-1). Dr. Berger also cited Doppler in support of this theory. Tr. at 164; Doppler at 720. When Doppler's authors conducted nerve biopsies on the relevant patients exhibiting the contactin-1 antibodies, it was discovered that the neural nodes were

elongated, but with no evidence of demyelination (again reinforcing that this kind of neuropathy was an axonal subset of CIDP cases (which are typically demyelinating)). Doppler at 724.

As further proof of the disease potential of the anti-contactin-1 antibody, Dr. Berger referenced an animal study involving a "passive transfer" of the proposed pathogenic autoantibody itself. *See* C. Manso, *Contactin-1 IgG4 Antibodies Cause Paranode Dismantling and Conduction Defects*, 139 Brain 1700-12 (2016), filed as Ex. 69 (ECF No. 36-22) ("Manso"). In Manso, researchers sought to evaluate whether human contactin-1 autoantibodies derived from two patients with CIDP would be pathogenic in animal subjects (rats). Manso at 1701-02. It was determined that one antibody form was able to "access paranodal regions, [and] induce the loss of paranodal specialization, motor conduction deficits, and ataxia," leading Manso's authors to characterize the antibody as "pathogenic and reliable biomarkers for CIDP." *Id.* at 1708. However, Manso does not specify whether the CIDP patients from whom the antibodies were derived had clinical presentations consistent with what Ms. Lemanski (or other individuals with the purported axonal-oriented form of chronic neuropathy) experienced. *Id.* at 1701, 1708.[18] And – as with other literature regarding this autoantibody – the article says nothing about what *causes* the antibodies to come into existence in the first place.

Dr. Berger then discussed whether Ms. Lemanksi's limited response to treatment shed any light on the likely nature of her condition or its etiology. Tr. at 167. In slight contrast to Dr. Lau, Dr. Berger deemed Ms. Lemanski's response to immunotherapy to be "poor and "transient," analogizing it to the limited response to IVIg observed in in Querol II. *Id.* Querol II's authors had clarified that a "poor response" to IVIg treatments should be construed as "a reduced response rate or suboptimal level of response relative to that obtained in patients with typical seronegative CIDP – and not as a complete absence of response in all patients." Querol II at 534. Like the patients in Querol II, Ms. Lemanski had a suboptimal response to IVIg – while still good enough for treaters to continuously prescribe additional rounds. *Id.* at 168, 170. IVIg and plasmapheresis, he maintained, are difficult treatments that can trigger a variety of side effects. *Id.* at 169. Ms. Lemanski's treatment team likely continued with these treatments, despite the fact that they did not obviously help her in the long run, because they likely intuited that her disease was an immunologically-mediated process, and that the benefits of immunotherapy outweighed the risks. *Id*. at 170. But Dr. Berger admitted that these types of treatments are not ultimately effective in treating chronic axonal neuropathy; they can only reduce the number of autoantibodies in a patient's blood at any given time but cannot stop their production on an ongoing basis. *Id*. at 187.

In concluding his testimony, Dr. Berger briefly addressed the timing of Ms. Lemanski's disease onset and whether it was medically acceptable when measured from the date of

[18] All Manso says about the patients, in fact, was that they had CIDP, possessed high titers of the antibodies the researchers wished to evaluate, and had "similar clinical phenotypes" – but it does not specify what they were, making it impossible to ascertain the degree to which they mirrored the kinds of symptoms Ms. Lemanski experienced. Manso at 1701.

vaccination. Tr. at 170. He contended that Ms. Lemanski's disease onset could have begun as early as a week after vaccination, noting that there was independent support for that timeframe for the generation of antibodies after an immune stimulus. *Id*.; Hiepe at 171 ("[I]t was recently shown that after primary immunization with a T-cell dependent antigen, germinal-center-derived memory B cells and plasma cells can appear early after one week in the peripheral blood").[19] And even if Ms. Lemanski's neuropathy began prior to vaccination, as Respondent contended (and as medical records suggest), the vaccination still likely played a role in encouraging the aberrant and chronic subsequent process. Tr. at 172.

On cross-examination, Dr. Berger was asked about the four criteria often relied upon by scientists when attempting to weigh the explanatory power of molecular mimicry as a possible mechanism for an autoimmune disease. *See* A. Jasti, *Guillain-Barre Syndrome: Causes, Immunopathogenic Mechanisms and Treatment*, 12 Expert Rev. of Clinical Immunology 1175-89 (2016), filed as Ex. 26 (ECF No. 26-10) ("Jasti"). These criteria require (a) evidence of significant homology between the vaccination and human proteins; (b) demonstration of a cross-reactive immune response; (c) an animal model of disease after infection; and (d) proof of an epidemiologic association between the vaccination and disease. Jasti at 1176.

Dr. Berger proposed the first two Jasti criteria had clearly been met. Tr. at 174-77. As to the fourth, he referenced case reports. *Id*. at 179; S. Huber et al., *Combined Acute Disseminated Encephalomyelitis and Acute Motor Axonal Neuropathy After Vaccination for Hepatitis A and Infection with Campylobacter jejuni*, 246 J. of Neuro. 1204-06 (1999), filed as Ex. 25 (ECF No. 26-9) ("Huber"). But the patient in Huber had been experiencing a GI infection for three weeks prior to receiving the Hepatitis A vaccine. Huber at 1204. The patient also complained of immediate malaise-like symptoms three days post-vaccination, and was eventually diagnosed with AMAN, which Petitioner does not claim Ms. Lemanski had. *Id.* Another such report also involved AMAN and a wild Hepatitis A infection as opposed to the vaccine. A. Samadi et al., *A 30-Year Old Man with Acute Motor Axonal Neuropathy Subtype of Guillain-Barre Syndrome Having Hepatitis A Virus Infection*, 11 Middle Eastern J. of Digestive Diseases 110-15 (2019), filed as Ex. 81 (ECF No. 36-34) ("Samadi") (A 30-year old man who had been experiencing flu-like symptoms for ~10 days prior to hospital admission, tested positive for Hepatitis A IgM. On his second day of admission, he began to experience neurological symptoms of muscle paralysis and was subsequently diagnosed with AMAN).

Still more case reports involved GBS, which it is agreed Ms. Lemanski did not have. Tr. at 180; X. Roux et al., *Guillain-Barre Syndrome and Anti-Hepatitis A Vaccination*, 40 Medecine et Maladies Infectieuses 490-92 (2010), filed as Ex. 29 (ECF No. 26-13) ("Roux"); S. Ono et al.,

[19] I note, however, that "immunization" of a test subject with a suspected disease-causing antigen, as described by this citation from Hiepe, is not at all comparable to receipt of a vaccine (although the idea that a vaccine can within a week's time begin to cause production of antibodies is certainly more than plausible).

Guillain-Barre Syndrome Following Fulminant Viral Hepatitis A, 33 Internal Med. 799-801 (1994), filed as Ex. 74 (ECF No. 36-37) ("Ono"). And another article relied on data derived from VAERS[20] reports. N. Souayah et al., *Analysis of Data From the CDC/FDA Vaccine Adverse Event Reporting System (1990-2009) on Guillain-Barre Syndrome After Hepatitis Vaccination in the USA*, 19 J. of Clin. Neurosci. 1089-1092 (2012), filed as Ex. 31 (ECF No. 26-15) ("Souayah"). Souayah's authors used data from VAERS to identify patients who developed GBS secondary to Hepatitis immunization. Souayah at 1089. They found that 70% of vaccinated patients who developed GBS did so within six weeks of vaccine administration. *Id.* at 1090. The authors concluded that this unbalanced distribution suggests that the Hepatitis vaccine may potentially trigger GBS. *Id.* at 1092.

After reviewing these articles, Dr. Berger conceded that they were not sufficient to meet the "epidemiological evidence criteria" of molecular mimicry as framed in Jasti. Tr. at 181-82. And he acknowledged that he was unaware of the existence of animal models in which the Hepatitis A *vaccine* caused neurologic damage (even though Manso seemed to possibly suggest the contactin-1 antibody could do so). *Id*. at 182.

3. Dr. Steven Bradfute – Dr. Bradfute, a viral immunologist, prepared two written reports and testified on behalf of Petitioner. *See generally* Tr. at 189-218; Report, dated Feb. 9, 2024 (ECF No. 42-3) ("First Bradfute Rep."); Report, dated June 11, 2024 (ECF No. 47-3) ("Second Bradfute Rep."). Dr. Bradfute opined that Ms. Lemanski's neuropathy was caused, in part, by a molecular mimicry reaction triggered by the Hepatitis A vaccine. Tr. at 195.

Dr. Bradfute is an Associate Professor in the Department of Internal Medicine at the University of New Mexico. CV, dated Feb. 9, 2024, filed as Ex. 90 (ECF No. 42-4) ("Bradfute CV"). He received his Ph.D. in immunology at Baylor College of Medicine in Houston, TX. Bradfute CV at 1; Tr. at 190. From 2005-2011, Dr. Bradfute completed a postdoctoral fellowship at the United States Army Medical Research Institute of Infectious Diseases, where he researched vaccine development, therapeutic development, and host pathogen interactions for a wide range of viruses. *Id.* From 2011-2013, he completed a second postdoctoral fellowship at the University of New Mexico on the topic of autophagy, which is essentially "the garbage disposal of the cell." Tr. at 191. Dr. Bradfute has served as a section editor for the Journal of Vaccines and as a reviewer for dozens of scientific journals on topics involving immunology, virology, vaccine development, and therapeutics against viral pathogens. *Id.* at 193. He also been published extensively. Bradfute CV at 15-21.

[20] VAERS is the Vaccine Adverse Event Reporting System, a database maintained by the Centers for Disease Control. VAERS collects information about adverse events that occur after the administration of licensed vaccines in the U.S. *See About VAERS*, Vaccine Adverse Event Reporting System (VAERS), http://vaers.hhs.gov/about/index (last visited Mar. 20, 2025).

In opining for Petitioner, Dr. Bradfute discussed the scientific theory of molecular mimicry, and the process by which biologically-relevant amino acid sequence similarities between components of the contactin-1 human protein (a cell surface protein found on axons) and the VP1 (one of the major capsid proteins of Hepatitis A included in the vaccine) could result in an autoimmune, cross-reacting attack on the nerve axons (at the contactin-1 location). Tr. at 197; First Bradfute Rep. at 1. Molecular mimicry, he contended, is one of the principal mechanisms driving autoimmune disease. Tr. at 196. But it occurs only if there is biologically-significant sequence homology between peptide components of the foreign antigen and the host antigen. *Id.* When significant homology exists, the antibodies produced in response to the foreign antigen may begin attacking the human protein, due to the homologic similarity. *Id.* Nevertheless, Dr. Bradfute admitted that sequence homology alone is insufficient to cause a cross-reaction. *Id.* at 199. Rather, additional stimulants are necessary to cause an autoimmune reaction via molecular mimicry, including activation of cells through inflammation, cell-to-cell contact, and a breakdown in immune regulatory function, along with other factors. *Id.* at 200.

Dr. Bradfute attempted to identify homology for this case. To do so, he used the European Bioinformatics Institute protein alignment algorithm "Clustal Omega" to identify homologic similarity.[21] Tr. at 197; First Bradfute Rep. at 1-2. He found stretches of peptide sequences with similar biochemical properties between the VP1 and contactin-1. Tr. at 198.; First Bradfute Rep. at 2. In his view, this demonstrated "there could be, within two antigens, the building blocks to generate a cross-reactive immune response due to the similarity of their sequence." *Id.* at 199.

Dr. Bradfute also utilized the Immune Epitope Database ("IEDB") as an alternative means of demonstrating the likelihood of a cross-reaction. After running a few searches, Dr. Bradfute found that peptides from *both* contactin-1 and the Hepatitis A capsid antigen are predicted to bind to several of the *same* MHC II alleles. Tr. at 200-01; First Bradfute Rep. at 2. This is significant because a T-helper cell (a master regulator of the body's immune response) could generate an immune response against both antigens, since they are similar enough to bind to the same MHC allele. Tr. at 200. Dr. Bradfute then referenced a table that he included in his first expert report. *Id.* at 201 (citing First Bradfute Rep. at 2). According to the table (which listed the results of his IEDB analysis), eight different MHC II alleles are predicted to bind to both VP1 and anti-contactin-1 antigens. First Bradfute Rep. at 2. Respondent's immunologic expert, Dr. Andrew MacGinnitie, had contended that the MHC II analysis only showed that these two antigens *could* in theory bind to the MHC II molecule – it did not show that they *do* bind. Second MacGinnitie Rep. at 3. But

[21] In response to Dr. MacGinnitie's contention that the Clustal Omega program can only demonstrate alignment via three *or more* peptide sequences, Dr. Bradfute maintained that Clustal Omega has recently changed, but was still acceptable for aligning two or more sequences at the time he performed the analysis for this action. Tr. at 197-98; First Bradfute Rep. at 2. He offered some literature to support this contention. F. Sievers et al., *Fast, Scalable Generation of High-Quality Protein Multiple Sequence Alignments Using Clustal Omega, 7 Molecular Systems Biology*, 1 (2011), filed as Ex. 111 (ECF No. 43-22) (Clustal Omega "can align virtually any number of protein sequences").

Dr. Bradfute explained in response that IEDB is 85% accurate in predicting binding. Tr. at 203. He also testified that this concept of anchor amino acids in MHC II binding is widely accepted among immunologists. *Id.* at 205.

Dr. Bradfute then addressed other criticisms offered by Respondent's experts. He explained his rationale for considering the possible stimulative potential of T-helper cells, even though Dr. Berger had focused on the role of anticontactin-1 antibodies produced by B-cells (and of course vaccines are *primarily aimed* at encouraging production of antibodies). Tr. at 206; Second Bradfute Rep. at 1-2. An autoimmune disease mediated by long-lived antibodies targeting host proteins, he argued, requires the engagement of these T-helper cells, and this occurs via the presentation of peptides on MHC II present on the surface of B cells to T-helper cell receptors. Second Bradfute Rep. at 1; *see also* Y. Huang, et al., *T Peripheral Helper Cells in Autoimmune Diseases: What do We Know?*, 14 Frontiers in Immuno. 1, 1 (2023), filed as Exhibit 117 (ECF No. 47-7) ("[T]he pathogenesis and development of autoimmune diseases is largely dependent on immune responses, which are mediated by interactions between T and B cells"). These T-helper cells are integral to the production of long-lived antibody-producing B cells (plasma cells). Tr. at 207-08; Second Bradfute Rep. at 1. Accordingly, the existence of similar peptide sequences between the Hepatitis A virus capsid protein in the vaccine and human contactin-1 also suggests that the generation of cross-reactive T-helper cells can occur after vaccination, further aiding in the generation of antibodies that target contactin-1. *Id.* In addition, although Dr. MacGinnitie argued that different peptides bound to the same MHC molecule will, in general, lead to activation of different T cells (Tr. at 211), in Dr. Bradfute's view it is just as likely that two different peptides will bind to the same receptor, leading to a cross reactive response. *Id.*

Dr. Bradfute also testified about immune tolerance – the mechanisms that protect against autoimmune reactions generally. Tr. at 211. He differentiated between central tolerance (which eradicates B and T cells before they mature), and peripheral tolerance (which stops the autoreactivity of cells that have survived central tolerance). *Id.* at 212. Such immune safeguards are not routinely successful for every individual. *Id.* In patients like Ms. Lemanski, tolerance mechanisms are likely bypassed, and therefore unable to end the cross-reactive response. *Id.* at 213. In fact, autoimmune processes like molecular mimicry may actively undermine tolerance on their own. *See* M. Rojas et al., *Molecular Mimicry and Autoimmunity in the Time of COVID-19*, 139 J. of Autoimmunity 1, 1 (2023), filed as Ex. 115 (ECF No. 47-5) ("[E]pidemiological data and animal studies on multiple autoimmune diseases suggest that molecular mimicry is one of the likely mechanisms for loss of peripheral tolerance and the development of clinical disease").

On cross examination, Dr. Bradfute reiterated his opinion that BLAST[22] searches were not useful to compare to dissimilar proteins. Tr. at 215. He also reiterated his belief that co-stimulation is necessary in order to activate T-helper cells following binding with an antigen-presenting cell. *Id.* at 216. But he admitted that he was unable to find evidence of co-stimulation in Ms. Lemanski's case, because the necessary tests had never been performed. *Id.* He further admitted that he could not determine that the MHC II molecules that could potentially bind the Hepatitis A capsid antigen, VP1, and contactin-1 were present in Ms. Lemanski – again, however, because the relevant analyses were not conducted. *Id.* at 217.

B. *Respondent's Experts*

1. Dr. Nathaniel M. Robbins – Dr. Robbins prepared two written reports and testified on behalf of Respondent. *See generally* Tr. at 219–322; Report, dated Nov. 30, 2022 (ECF No. 30-1) ("First Robbins Rep."); Report, dated July 20, 203 (ECF No. 34-1) ("Second Robbins Rep."). Dr. Robbins opined that Ms. Lemanski's proper diagnosis was ALS. First Robbins Rep. at 8; Tr. at 262.

Dr. Robbins is an Assistant Professor of Neurology at Dartmouth Geisel School of Medicine. CV, dated Dec. 7, 2022, filed as Ex. A-1 (ECF No. 30-2) ("Robbins CV"). He is also the Department of Neurology Director of Clinical Operations and the Medical Director of the Neurology APP Fellowship. Robbins CV at 1. Dr. Robbins received his medical degree from Albert Einstein College of Medicine, and then went on to complete a residency in neurology at the University of California San Francisco and a fellowship in clinical neurophysiology at Dartmouth. *Id.*; Tr. at 219. Dr. Robbins is board-certified in clinical neurophysiology and electrodiagnostic medicine, psychiatry, and neurology. Robbins CV at 2. In his current role, Dr. Robbins teaches residents, medical students, fellows, and junior faculty on subjects such as peripheral nerve disorders, neuromuscular disorders, and central autonomic disorders. Tr. at 220. About 60% of Dr. Robbins's current practice involves treating clinical patients with a variety of illnesses, including peripheral neuropathies and ALS. *Id.* He is currently studying the diagnosis and treatment of peripheral nerve disorders. *Id.* at 221. Dr. Robbins is a reviewer for several medical journals and has published 30-40 journal articles. *Id.*; Robbins CV at 2-4.

Dr. Robbins first addressed Petitioner's proposed diagnosis of CMAP. He noted that "chronic motor axonal polyneuropathy" is not a recognized, official medical diagnosis, pointing out its absences in diagnostic reference materials like PubMed.[23] Tr. at 224. And Ms. Lemanski

[22] BLAST, or Basic Local Alignment Search Tool, is a program that "finds regions of similarity between biological sequences." *Blast Local Alignment Tool Search*, National Library of Medicine, blast.ncbi.nlm.nih.gov/Blast.cgi (last visited Mar. 20, 2025).

[23] PubMed is a free online database compromised of more than 38 million citations for biomedical literature from MEDLINE, life science journals, and online books. *See* https://pubmed.ncbi.nlm.nih.gov/ (last visited Mar. 20, 2025).

was never formally diagnosed with any other chronic neuropathies, including CIAP, CMAN, or CIDP. *Id.* at 225-28. (Of course, Dr. Grysiewicz had proposed something like CMAN as a possible diagnosis in September 2018. Ex. 10 at 45). CMAP, Dr. Robbins contended, is not simply a generalized congruent term for CIAP. *Id.* at 229.

Other important diagnostic criteria for immune-mediated neuropathies were also absent from this record, in Dr. Robbins's view. He outlined three important criteria: (i) the disease must adhere to a pattern consistent with a known clinical syndrome of autoimmune neuropathy; (ii) evidence of autoimmunity must be present; and (iii) alternative causes must be excluded. Tr. at 240; First Robbins Rep. at 8-9. But (as noted in the review of CIAP criteria), Ms. Lemanski's symptoms did not fit many of the symptoms requirements. Tr. at 241. For example, the patients in Oh all displayed areflexia, which Ms. Lemanski did not have. *Id.* at 229-30; Oh at 1176; *see also* A. Uncini et al., *Chronic Progressive Steroid Responsive Axonal Polyneuropathy*, 19 Muscle & Nerve 365, 368 (1996), filed as Ex. A-21 (ECF No. 30-22) (describing areflexia/hyporeflexia in patients with an axonal-oriented neuropathy). Rather, Ms. Lemanski had displayed pathologically brisk reflexes, which means her reflexes were "too jumpy" and spread to adjacent areas. Tr. at 234; Ex. 2 at 7; Ex. 17; Ex. 10 at 13. The records from August 2018 in fact revealed that Ms. Lemanski's reflexes were normal, even though she was unable to move voluntarily. Ex. 6.02 at 440. And the records from January 2019 note that Ms. Lemanski was still experiencing knee jerks and had normal upper extremity reflexes. Ex. 10 at 13.

In addition, most CIAP patients experienced *sensorimotor* symptoms. Tr. at 239; First Robbins Rep. at 9. Yet Ms. Lemanski's presentation involved *pure motor* symptoms – atypical for an immune-mediated disease. Tr. at 230, 239. And the few patients that did experience motor-only symptoms did so on a symmetric basis. Tr. at 238; Oh at 1176. Ms. Lemanski's weakness, however, was strikingly asymmetric. Tr. at 238-39; First Robbins Rep. at 9. Finally, the vast majority of those patients tested positive for elevated protein, and Ms. Lemanski did not. First Robbins Rep. at 10; Oh at 1176. All of the foregoing suggested to Dr. Robbins that a CIAP-like condition was not the best diagnostic classification for what Ms. Lemanski had experienced. Tr. at 230.

On cross-examination, Dr. Robbins was confronted with evidence suggesting that a presentation of purely motor symptoms was not in every case inconsistent with existing chronic neuropathy diagnostic classifications. Tr. at 295. One article filed by Respondent seemed to at least allow for a category of "atypical" chronic polyneuropathies that would encompass Ms. Lemanski's condition. *Id.* at 295-96; P. Van den Bergh et al., *European Federation of Neurological Societies/Peripheral Nerve Society Guideline on Management of Chronic Inflammatory Demyelinating Polyradiculoneuropathy: Report of a Joint Taks Force of the European Federation of Neurological Societies and the Peripheral Nerve Society—First Revision*, 17 Eur. J. of Neuro. 356, 360 (2010), filed as Ex. 42 (ECF No. 32-8) ("Van den Bergh") (the clinical diagnostic criteria

for atypical CIDP can include pure motor symptoms). Dr. Robbins agreed that atypical forms of polyneuropathy can be purely motor. Tr. at 296.

There was also a lack of record evidence corroborating the existence of an ongoing autoimmune process. Thus, Ms. Lemanski was never shown to possess certain antibody biomarkers, like anti-ganglioside antibodies. Tr. at 241. In Dr. Robbins's understanding, most patients with acute motor axonal polyneuropathies would display anti-ganglioside antibodies as biomarkers, but Ms. Lemanski did not test positive for them in May 2018. *Id.* at 296. But at least one article filed by Petitioner found that anti-ganglioside antibodies decline significantly six to seven weeks after disease induction – and Ms. Lemanski's May 2018 test occurred in a greater post-vaccination period. *Id.* at 297; H. Willison et al., *Guillain-Barre Syndrome*, 388 Lancet 717, 718 filed as Ex. 85 (ECF. No. 36-38) ("Willison"). Dr. Robbins pointed out on redirect, however, that Willison involved GBS, which is monophasic. Tr. at 311-12. Because Ms. Lemanski's condition was chronic, it less likely that anti-ganglioside antibodies would have declined at this point. *Id.* at 312. Dr. Robbins also maintained (as further evidence that she was not experiencing an immune-driven neuropathy) that Ms. Lemanski never tested positive for elevated proteins in her cerebrospinal fluid – although he later admitted that it is possible, while uncommon, for a patient with immune-mediated polyneuropathy to have normal protein levels. *Id.* at 296.

Dr. Robbins further highlighted the fact that it did not appear that Ms. Lemanski had overall experienced sufficient improvement from her immunomodulatory therapies, like IVIg or plasmapheresis. In so contending, he acknowledged that Ms. Lemanski had reported brief periods of feeling better in response to treatment, but noted that "a little variation from visit to visit is not enough of an objective response." Tr. at 242. Even if Ms. Lemanski *felt* like she was getting better, the objective record did not demonstrate true improvement. *Id.* at 244. And referencing the reliability of the INCAT scale,[24] Dr. Robbins noted that many patients can feel like they are improving when they are actually getting worse. *Id.* at 246. Dr. Robbins favored his interpretation of Ms. Lemanski's reported improvement as proof of a placebo effect over medical records that suggested the improvements were objective. *Id.* at 288-90; Ex. 10 at 18, 34.

Ms. Lemanski's EMG results were additional evidence against the conclusion that she had experienced an immune-mediated neuropathic illness. Tr. at 247. For one thing, such testing could not be assumed to be accurate. While EMGs and NCSs are good tests of nerve injury, they come with a relatively high rate of interrater reliability issues. *Id.* at 247-48; P. Narayanaswami et al., *Critically Re-evaluating a Common Technique: Accuracy, Reliability, and Confirmation Bias of EMG*, 86 Neurology 218, 221 (2016), filed as Ex. A-16 (ECF No. 30-17) ("[T]here was only 60.4% agreement [among reviewers] for the presence of reinnervation"). Thus, it was not necessarily the case that each EMG's findings were wholly reliable.

[24] The Inflammatory Neuropathy Cause and Treatment (INCAT) disability scale assesses the functional ability of the arms and legs in patients with CIDP. *See* https://vyvgarthcp.com/vyvgarthytrulo-cidp/about/cidp.

More significantly, however, the EMGs themselves did not track with a chronic neuropathic disease attributable to an autoimmune process. The initial March 2018 EMG results, for example, revealed increased amplitude motor units, suggesting the existence of chronic reinnervation *at that time*. Tr. at 249-50; Ex. 7 at 147. This was consistent with Ms. Lemanski's overall history. At this point in her medical course, Ms. Lemanski had experienced ongoing injury in multiple muscles in both of her legs, with the left leg predominantly affected. Tr. at 249. But a patient must suffer injury to a significant proportion of their nerve and muscle before they develop clinical weakness. *Id.* at 251. So, Dr. Robbins did not deem it unusual that Ms. Lemanski was not also complaining of right leg weakness at this time. *Id.* at 252.

Dr. Robbins thus disagreed with Dr. Lau's contention that chronicity of Ms. Lemanski's injury did not exist as of this first EMG. Tr. at 252. Ms. Lemanski could not have had these large amplitude motor units by March 2018 if her condition had only begun in February, because these kinds of readings more typically reflect the existence of a chronic nerve injury of at least several months duration. Second Robbins Rep. at 4. In so contending, Dr. Robbins emphasized Dr. Lau's acknowledgement that evidence of chronic changes on an EMG are consistent with a damaging process that began two to three months before. Tr. at 253. Because the March 2018 EMG showed damage to multiple nerve roots, Ms. Lemanski's disease onset had likely begun a few months *before* this March 2018 EMG. *Id.*

Next, Dr. Robbins analyzed Ms. Lemanski's May 2018 EMG. Tr. at 254; Ex. 12 at 67. The motor amplitudes were beginning to decrease at this point, suggesting more widespread nerve injury. Tr. at 254; Ex. 12 at 67. Furthermore, this EMG showed no evidence of demyelination. Tr. at 255. Dr. Robbins admitted that there was no evidence of chronic reinnervation in this EMG, but argued that the results were likely incorrect. *Id.* at 257. The inaccuracies could be attributed to the fact that Ms. Lemanski's limbs were inadequately warmed prior to the test; nerves must be a certain temperature in order to produce accurate results. *Id.* at 255.

Dr. Robbins then turned to Ms. Lemanski's August 2018 EMG, which now showed active denervation and chronic reinnervation in many of the muscles tested. Tr. at 258-59; Ex. 6.01 at 373. The EMG also showed ongoing nerve injury up in the cervical paraspinal muscles, which is highly suggestive of motor neuron disease or ALS. Tr. at 259; Ex. 6.01 at 373. But he later admitted that this EMG showed no evidence of cranial nerve involvement – a hallmark of ALS (although he opined in response that it was unlikely at this point in her disease progression that Ms. Lemanski's ALS had progressed to her brainstem – and had not even at the time of her death). Tr. at 304; Ex. 7 at 97. Finally, Dr. Robbins examined the results of Ms. Lemanski's final EMG from December 2018. Tr. at 260; Ex. 12 at 35. He noted that the sensory amplitudes in this EMG were normal, but the motor nerve findings were very abnormal. *Id.* Once again, the EMG showed no

evidence of demyelination, but revealed widespread ongoing nerve injury in almost every muscle tested. Tr. at 260.

Ms. Lemanski's autopsy results were also, Dr. Robbins contended, unsupportive of a "demyelinating neuropathy" as her diagnosis (or even the cause of her death). Tr. at 267. This cause of death was only certified by Ms. Lemanski's PCP, Dr. Kotsonis, but was not shown to be the result of the autopsy. *Id.* Indeed, the examiners did not study the nerves or the spinal cord, "which seems to have been where the problem was." *Id.* And although the autopsy did not find histopathological changes (a necessary criteria for ALS), Dr. Robbins theorized that the examiners conducting the autopsy may not have been specialized enough, and may not have performed the requisite immuno-staining required for this sort of finding. *Id.* at 268. But he admitted the autopsy also lacked other corroboration for ALS, such as Purkinje cell[25] loss, localized neuronal loss, or neurofibrillary tangles. *Id.* at 306-07; Ex. 11 at 33.

Dr. Robbins's testimony included some discussion of what medical science knows about purported contactin-1 immune mediated neuropathies, such as CIDP, AMAN, and/or GBS. Tr. at 269, 276. He denied that articles like Doppler or Querol I applied to Ms. Lemanski's case, because the patients in these articles all had CIDP, which requires demyelination. *Id.* at 269-70. In response to Dr. Lau's contention that some of these CIDP cases did not involve demyelination, Dr. Robbins explained that an anti-contactin-1 neuropathy is, *by definition*, a demyelinating neuropathy that likely involves sensory motor neurons. *Id.* at 273. Because Ms. Lemanski only experienced motor nerve issues, she could not have had an anti-contactin-1 neuropathy. *Id.* at 274. And while the Oh paper reported three patients with purely motor symptoms, the rest of the studied subjects' symptoms did not mirror Ms. Lemanski's – they were completely areflexic, for example, or displayed elevated proteins and symmetric symptoms. *Id.* at 321; Oh at 1176.

ALS was a more evidentiarily-supported diagnosis for Ms. Lemanski's condition, in Dr. Robbins's view. Tr. at 262. He characterized ALS to be a disease in which the motor neurons die in the brain, the spinal cord, and in the periphery. *Id.* The etiology of ALS is unknown, although it is a relatively common condition. *Id.* at 262-63. It is distinguishable from other diseases of motor nerves because it is not a neuropathy that just affects the lower motor nerves in the periphery; the upper motor nerves die too. First Robbins Rep. at 11 (citing N. Garg et al., *Differentiating Lower Motor Neuron Syndromes*, 88 J. of Neurol., Neurosurg. and Psych. 474, 476 (2017), filed as Ex. A-10 (ECF No. 30-11) ("The combination of upper and LMN signs is the pathognomonic hallmark with this variant referred to as ALS")).

[25] Purkinje cells are "large neurons in the cerebellar cortex that have piriform cell bodies in the Purkinje layer ... and large branching dendrite trees going through the outer (molecular) layer towards the surface." *Purkinje cells*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=64371&searchterm=Purkinje+cells (last visited Mar. 20, 2025).

A clinical diagnosis of ALS is based on insidious progression of weakness, fasciculations, and cramps accompanied by upper motor neuron signs (most commonly hyperreflexia). Tr. at 264. The presence of reflexes in muscles that are otherwise profoundly weak is specific to ALS – no other disease features this. *Id.* Dr. Robbins noted that Ms. Lemanski exhibited upper and lower motor neuron signs. *Id.* at 265. She experienced pathologic hyperreflexia while experiencing weakness and denervation. *Id.* Ms. Lemanski's hyperreflexia, Dr. Robbins argued, cut strongly against a diagnosis of "chronic motor axonal polyneuropathy" because "*hyporeflexia* is the rule in both AMAN and CIDP," while exaggerated reflexes are common in ALS. Second Robbins Rep. at 7 (emphasis added).[26] Dr. Robbins further pointed out that Ms. Lemanski reported that her fasciculations began one month prior to vaccination, in January 2018. Tr. at 278. He opined that this likely marked the onset of Ms. Lemanski's disease, noting that it was highly implausible that these fasciculations were a separate and coincidental phenomenon related to "anxiety", as Dr. Lau suggested, or that they represented a nonspecific symptom that coincidentally began at the same time as her progressive motor nerve disease. First Robbins Rep. at 15.

Dr. Robbins acknowledged that Ms. Lemanski did not exhibit other common symptoms of ALS, like muscular atrophy or pseudobulbar effect, but asserted that these criteria do not all have to be present in order to make the diagnosis. Tr. at 265. He emphasized that it is very difficult to reach a definitive diagnosis of ALS, since there are no laboratory tests that can confirm it. *Id.* at 316. Dr. Robbins also admitted that there exist two FDA-approved drugs that can slow the progression of ALS, but that Ms. Lemanski was prescribed neither. *Id.* at 265-66. Yet he deemed this not unusual, since patients with ALS are often diagnosed late in their disease progression – too late to receive these medications for treatment. *Id.* at 266. He also agreed that ALS patients usually have a post-diagnosis survival period of several years, in contrast to Ms. Lemanski's experience. *Id.* at 285, 301. And importantly, none of Ms. Lemanski's providers ever diagnosed her with ALS, even though they consistently considered it as part of the differential diagnosis. *Id.* at 308.

2. Dr. Andrew J. MacGinnitie – Dr. MacGinnitie, an allergist/immunologist, prepared two written reports and testified on behalf of Respondent. *See generally* Tr. 322-70; Report, dated Oct. 23, 2023 (ECF No. 37-1) ("First MacGinnitie Rep."); Report, dated Apr. 26, 2024 (ECF No. 45-1) ("Second MacGinnitie Rep."). Dr. MacGinnitie opined that Ms. Lemanski's illness was not caused by the Hepatitis A vaccine. First MacGinnitie Rep. at 14; Tr. at 328.

Dr. MacGinnitie is the Division Chief for the Division of Allergy, Asthma, and Immunology at Children's Wisconsin in Milwaukee and is a Professor of Pediatrics at the Medical

[26] Hyporeflexia is characterized by weakening of the reflexes. *Hyporeflexia*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24379&searchterm=hyporeflexia (last visited Mar. 7, 2025). Hyperreflexia is characterized by an exaggeration of the reflexes. *Hyperreflexia*, Dorland's Medical Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=23992&searchterm=hyperreflexia (last visited Mar. 7, 2025).

College of Wisconsin. Tr. at 323. He received his medical degree and Ph.D. in Pathology from the University of Chicago. CV, dated Oct. 24, 2023, filed as Ex. C-1 (ECF No. 37-2) ("MacGinnitie CV"). Thereafter, he completed a residency in pediatrics in the Boston Combined Residency Program, a joint venture of Boston Children's Hospital and Boston Medical Center, followed by a fellowship in allergy/immunology at Boston Children's Hospital. MacGinnitie CV at 1; Tr. at 323. Dr. Robbins is board-certified in both pediatrics and allergy and clinical immunology. MacGinnitie CV at 11; Tr. at 325. In his prior role as the Clinical Chief of the Division of Immunology at Boston Children's Hospital, he saw approximately 1,500 patients annually. MacGinnitie CV at 2; Tr. at 324. In his current role, he sees patients in the clinical setting two days per week. Tr. at 324. Most of his journal publications involve immunological topics. *Id.* at 326; CV at 12-16.

In addressing Petitioner's proposed autoimmune mechanism, Dr. MacGinnitie opined that it is "very improbable" that molecular mimicry between the VP1 component of the Hepatitis A vaccine and contactin-1 led Ms. Lemanski to develop her neurologic illness. Tr. at 328. In support, he referenced the four criteria for determining molecular mimicry set forth in Jasti, attempting to apply them to Ms. Lemanski's case. Tr. at 329-30; First MacGinnitie Rep. at 7-11. He acknowledged that requiring satisfaction of these criteria amounted to demanding a level of proof "pretty close to scientific certainty" (which is not equivalent to the Program's preponderance requirement), but maintained that Petitioner had not offered sufficient evidence to meet *any* of the four criteria. Tr. at 350.

First, Dr. MacGinnitie represented that Dr. Bradfute had not demonstrated a biologically significant degree of homology between VP1 and contactin-1. Tr. at 331. In order to test Dr. Bradfute's contention, he performed his own homology evaluation, running these two proteins through a BLAST search. *Id.* In so doing, Dr. MacGinnitie only found similarity between the proteins when he extended the search to a very permissive, non-stringent range. *Id.* And even then, the statistical tool in BLAST revealed that the degree of homology was not notable. *Id.*

Dr. Bradfute had questioned the reliability of a BLAST-reliant methodology to assess homology, but Dr. MacGinnitie maintained that in fact it was sound, citing literature as support. *See* David Wheeler & Medha Bhagwat, *9. BLAST QuickStart*, *in* Comparative Genomics (Nicholas H. Bergman, ed. 2007), filed as Ex. C-3 (ECF No. 37-4) ("Wheeler & Bhagwat"). Tr. at 332. Wheeler & Bhagwat does not state, as Dr. Bradfute argued, that the BLAST tool can only be used to compare similar proteins across species. Tr. at 332; Wheeler & Bhagwat at 1 ("The introduction of an intermediate layer in which nucleotide sequences are translated into their corresponding protein sequences according to a specified genetic code allows cross-comparisons between nucleotide and protein sequences"). Dr. MacGinnitie admitted that he is less familiar with Clustal Omega, the tool used by Dr. Bradfute, but he noted that Dr. Bradfute did not report any statistical significance between the sequences he found. *Id.* at 333.

Another reason to doubt the power of Dr. Bradfute's homology showing, Dr. MacGinnitie argued, arose from the fact that sequence similarity alone is insufficient to demonstrate causality.

First MacGinnitie Rep. at 10. On the contrary – medical science is well aware of how common sequential amino acid homology is in nature, yet autoimmune disease is not rampant. *See* D. Kanduc et al., *Massive Peptide Sharing between Viral and Human Proteomes*, 29 Peptides 1755-66 (2008), filed as Ex. C-7 (ECF No. 37-8) ("Kanduc"). Kanduc found that 90% of the viral, five amino acid peptides are repeatedly scattered throughout the human proteome. Kanduc at 1757. If molecular mimicry were really enough of a basis alone to trigger autoimmunity, then these sequence similarities would support a 100% rate of autoimmune disease development following vaccination or infection – which is clearly not observed. Tr. at 335.

Dr. MacGinnitie also discussed the breakdown of immune tolerance that is needed to trigger autoimmunity. Tr. at 337. While he acknowledged that it is not always understood what can cause such a breaking of tolerance, significant inflammation as a background condition is often required. *Id.* at 339. But his review of the medical records revealed no evidence that Ms. Lemanski had suffered from any inflammation in the wake of her vaccination, let alone a level sufficient to promote an aberrant immune reaction. *Id.* at 338. At bottom, all Dr. Bradfute established via his search was "some small area of homology" – a showing that, if accepted, could apply to virtually any vaccine received before onset of the disease in question. *Id.* at 350.

Turning to the second Jasti criterion, Dr. MacGinnitie maintained that there was an absence of reliable scientific evidence supporting the proposition that the Hepatitis A vaccine could likely lead to an autoimmune cross-reaction. Tr. at 339. For example, Dr. Berger had failed to identify evidence of a cross-reactive antibody or T-helper cell response between the VP1 protein of the Hepatitis A vaccine and contactin-1. *Id.*; First MacGinnitie Rep. at 7. The fact that T-helper cells promote antibody production did not necessarily also mean that they would encourage an antibody response to the same antigen or portion of antigen deemed to be a human peptide/protein mimic. Tr. at 345. Rather, it would need to have been shown that Ms. Lemanski's T-helper cells could specifically react to the VP1 component of the Hepatitis A vaccine sufficient to also produce the specific anti-contactin-1 antibodies deemed pathogenic under Petitioner's theory. *Id.* at 347. The chance that this could occur (along with the proposed immune tolerance break) all at once – and in an absence of evidence that Ms. Lemanski possessed anti-contactin-1 antibodies in the first place – was, in Dr. MacGinnitie's assessment, remote. *Id.*

Another evidentiary omission that Dr. MacGinnitie maintained undermined the application of molecular mimicry as explanatory in this case was Dr. Bradfute's contentions about MHC molecules. MHC molecules show great diversity, but there is no evidence that Ms. Lemanski's T-helper cells would lead to expression of the MHC II molecules identified by Dr. Bradfute as potentially binding Hepatitis A and contactin-1 peptides. Tr. at 336; Second MacGinnitie Rep. at 3. And even if two peptides were to bind to the same MHC II molecule, Dr. MacGinnitie explained, it was "extremely unlikely" that this would also stimulate the same T-helper cell. Tr. at 343.

Addressing the third Jasti criterion, Dr. MacGinnitie maintained that Dr. Berger had failed to identify any animal model that demonstrates the possibility of autoimmunity between VP1 and

contactin-1. Tr. at 347. Nor had Petitioner offered any reliable epidemiologic evidence. *Id.* at 348. Case reports were not helpful, and reliance on VAERS reports was problematic. *Id.* Anyone, Dr. MacGinnitie emphasized, can make out a VAERS report – there is no independent post-report investigation that confirms the patient actually had the relevant disease or received the relevant vaccine. *Id.* Thus, articles like Souayah, which relied on VAERS reports, were not convincing evidence (especially since Souayah involved GBS, a distinguishable condition). *Id.*

Other record evidence also undercut the conclusion that the Hepatitis A vaccine had caused Ms. Lemanski to experience an inflammatory reaction consistent with an autoimmune process. Thus, there was no evidence she possessed anti-ganglioside or anti-contactin-1 antibodies (although such testing was never done), and there was no objective evidence in the medical record that immunomodulatory treatments had improved her condition. Tr. at 352-53; First MacGinnitie Rep. at 12. While Ms. Lemanski claimed "continued improvement" from these treatments, Dr. Grysiewicz's exams in December 2018 and January 2019 did not reveal objective support for her beliefs. First MacGinnite Rep. at 12.

In conclusion, Dr. MacGinnitie discussed the timing of Ms. Lemanski's disease process. Tr. at 350. He proposed that it would take between three days to up to two months for someone to develop an autoimmune disease after vaccination, assuming the proposition that the vaccine instigated production of cross-reacting antibodies. *Id.* at 350-51. Thus, because Ms. Lemanski received the Hepatitis A vaccine on February 2, 2018, her symptom onset should have occurred between February 5th and early April. *Id.* at 351. But when Ms. Lemanski saw her doctor on February 5, 2018, she reported that her symptoms began *before* vaccination. *Id.*

IV. Procedural History

As noted, this case was initiated at the start of 2021. On April 14, 2022, Respondent filed his Rule 4(c) Report disputing entitlement. (ECF No. 23). Parties filed several rounds of experts reports (as reflected in the summary of trial testimony above) and briefed the matter before it went to trial in September 2024. The matter is now fully ripe for resolution.

V. Applicable Legal Standards

A. *Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed.

Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).[27] There is no Table claim for the injury of CMAP – or for any form of neuropathy in reaction to the Hepatitis A vaccine, for that matter.

For both Table and Non-Table claims, Vaccine Program petitioners bear a "preponderance of the evidence" burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the "trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact's existence." *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. V. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec'y of Health & Hum. Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec'y of Health & Hum. Servs*., 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen v. Sec'y of Health and Hum. Servs.*, 418 F.3d 1274, 1278 (Fed. Cir. 2005): "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury."

Each *Althen* prong requires a different showing. Under *Althen* prong one, petitioners must provide a "reputable medical theory," demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec'y of Health & Hum. Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing

[27] Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec'y of Health & Hum. Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff'd* 104 F. App'x. 712 (Fed. Cir. 2004); *see also Spooner v. Sec'y of Health & Hum. Servs.*, No. 13-159V, 2014 WL 504728, at *7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

Capizzano, 440 F.3d at 1325–26). Special masters, despite their expertise, are not empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed "not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act's preponderant evidence standard." *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245 ("[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one" (emphasis in original)).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing the proposed causal theory's scientific or medical *plausibility*. *See Kalajdzic v. Sec'y of Health & Hum. Servs.*, No. 2023-1321, 2024 WL 3064398, at *2 (Fed. Cir. June 20, 2024) (arguments "for a less than preponderance standard" deemed "plainly inconsistent with our precedent" (*citing Moberly*, 592 F.3d at 1322)); *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); *see also Howard v. Sec'y of Health & Hum. Servs*., 2023 WL 4117370, at *4 (Fed. Cl. May 18, 2023) ("[t]he standard has been preponderance for nearly four decades"), *aff'd*, 2024 WL 2873301 (Fed. Cir. June 7, 2024) (unpublished). And petitioners always have the ultimate burden of establishing their *overall* Vaccine Act claim with preponderant evidence. *W.C. v. Sec'y of Health & Hum. Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell v. United States*, 133 Fed. Cl. 782, 793 (2017) (noting that *Moberly* "addresses the petitioner's overall burden of proving causation-in-fact under the Vaccine Act" by a preponderance standard).

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine "did cause" injury, the opinions and views of the injured party's treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 ("medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury'") (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec'y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder v. Sec'y of Health & Hum. Servs*., 88 Fed. Cl. 706, 746 n.67 (2009) ("there is nothing . . . that mandates

that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec'y of Health & Hum. Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians' conclusions against each other), *aff'd*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec'y of Dept. of Health & Hum. Servs.*, No. 06-522V, 2011 WL 1935813, at *17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review den'd*, 100 Fed. Cl. 344, 356 (2011), *aff'd without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a "proximate temporal relationship" between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase "medically-acceptable temporal relationship." *Id.* A petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation." *De Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is37edicaly acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one's requirement). *Id.* at 1352; *Shapiro v. Sec'y of Health & Hum. Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. Den'd after remand*, 105 Fed. Cl. 353 (2012), *aff'd mem.*, 503 F. Appx. 952 (Fed. Cir. 2013); *Koehn v. Sec'y of Health & Hum. Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. den'd* (Fed. Cl. Dec. 3, 2013), *aff'd*, 773 F.3d 1239 (Fed. Cir. 2014).

B. *Legal Standards Governing Factual Determinations*

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11I(2). The special master is required to consider "all [ ] relevant medical and scientific evidence contained in the record," including "any diagnosis, conclusion, medical judgment, or autopsy or coroner's report which is contained in the record regarding the nature, causation, and aggravation of the petitioner's illness, disability, injury, condition, or death," as well as the "results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions." Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec'y of Health & Hum. Servs*., 3 F.3d 415, 417 (Fed. Cir. 1993) (determining that it is within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

As noted by the Federal Circuit, "[m]edical records, in general, warrant consideration as trustworthy evidence." *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Hum. Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd, Rickett v. Sec'y of Health & Hum. Servs*., 468 F. App'x 952 (Fed. Cir. 2011) (non-precedential opinion). A series of linked propositions explains why such records deserve some weight: (i) sick people visit medical professionals; (ii) sick people attempt to honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Hum. Servs.*, No. 11–685V, 2013 WL 1880825, at *2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013); *Cucuras*, 993 F.2d at 1525 ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

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

However, the Federal Circuit has also noted that there is no formal "presumption" that records are accurate or superior on their face to other forms of evidence. *Kirby v. Sec'y of Health & Hum. Servs.*, 997 F.3d 1378, 1383 (Fed. Cir. 2021). There are certainly situations in which compelling oral or written testimony (provided in the form of an affidavit or declaration) may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Hum. Servs*., 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at *19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Hum. Servs*., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be "consistent, clear, cogent, and compelling." *Sanchez*, 2013 WL 1880825, at *3 (citing *Blutstein v. Sec'y of Health & Hum. Servs*., No. 90–2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person's failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional's failure to document everything reported to her or him; (3) a person's faulty recollection of the events when presenting testimony; or (4) a person's purposeful recounting of symptoms that did not exist. *La Londe v. Sec'y of Health & Hum. Servs*., 110 Fed. Cl. 184, 203–04 (2013), *aff'd*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

C. *Analysis of Expert Testimony*

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec'y of Health & Hum. Servs*., 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharm., Inc.*, 509 U.S. 579, 594–96 (1993). *See Cedillo v. Sec'y of Health & Hum. Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec'y of Health & Hum. Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999). Under *Daubert*, the factors for analyzing the reliability of testimony are:

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

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

In the Vaccine Program the *Daubert* factors play a slightly different role than they do when applied in other federal judicial settings, like the district courts. Typically, *Daubert* factors are employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable or could confuse a jury. By contrast, in Vaccine Program cases these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec'y of Health & Hum. Servs*., 94 Fed. Cl. 53, 66–67 (2010) ("uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of

expert testimony already admitted"). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. *See, e.g.*, *Snyder*, 88 Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be "based on the credibility of the experts and the relative persuasiveness of their competing theories." *Broekelschen v. Sec'y of Health & Hum. Servs*., 618 F.3d 1339, 1347 (Fed. Cir. 2010) (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion "connected to existing data only by the *ipse dixit* of the expert," especially if "there is simply too great an analytical gap between the data and the opinion proffered." *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 146 (1997)); *see also Isaac v. Sec'y of Health & Hum. Servs*., No. 08–601V, 2012 WL 3609993, at *17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for review den'd*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. App'x. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particul'r 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").

D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not all such items factor into the outcome of this decision. While I have reviewed all the medical literature submitted, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Hum. Servs.*, No. 2015–5072, 2016 WL 1358616, at *5 (Fed. Cir. Apr. 6, 2016) ("[w]e generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.,* 527 F. App'x 875, 884 (Fed. Cir. 2013) ("[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered").

ANALYSIS

I. The Record Supports the Conclusion that Ms. Lemanski Suffered From Some Form of Chronic, Axonal-Oriented Neuropathy

As should be evident from my foregoing review of the expert testimony offered in this case, the parties vigorously dispute the nature of the diagnosis – with Petitioner very committed to establishing that Ms. Lemanski experienced some form of immune-mediated, chronic neuropathy.

This case ultimately turns more on Petitioner's success in meeting the *Althen* prongs. But nevertheless (and as Federal Circuit precedent establishes) in certain cases it is appropriate to determine the nature of the alleged injury (especially since acceptance of ALS as the best evidentiarily-supported diagnosis might render causation close to impossible, given the lack of association between it and vaccination).[28] *Broekelschen*, 618 F.3d at 1346. And because "each prong of the *Althen* test is decided relative to the injury[,]" determining facts relating to the claimed injury can aid overall in the claim's resolution. *Id.*

Chronic inflammatory neuropathies ("CIN") are rare, very disabling autoimmune disorders characterized by motor and sensory symptoms of diverse severity. Querol II at 533. Most CINs are diagnosed using clinical and electrophysiological criteria. *Id.* The existence of pathological and radiological evidence of inflammation in nerve and nerve roots, the pathogenic role of immune cells and, most importantly, the favorable response to immunotherapies support a diagnosis for CIN. *Id.* at 533-34. Although immune-mediated pathophysiology for CIN is often linked to the presence of demyelinating features on EMG, some CINs are primarily axonal and do not involve demyelination and its attendant clinical features, like paresthesia. *Id.* at 544.

ALS is a fatal type of motor neuron disease that involves progressive degeneration of nerve cells in the spinal cord and brain. E. Tiryaki & H. Horak, *ALS and other Motor Neuron Diseases*, 20 Continuum 1185, 1185 (2014), filed as Ex. 34 (ECF No. 26-18) ("Tiryaki & Horak"). While most motor neuron diseases affect only the lower motor neurons (those located in the spinal cord anterior horn and the brainstem), ALS can also affect upper motor neurons. Tiryaki & Horak at 1185. The clinical hallmarks of motor neuron diseases like ALS include atrophy, weakness, and fasciculations of the affected motor units. *Id.* at 1186. And while ALS shares some similarities with chronic, motor-oriented neuropathies, the two can be clearly distinguished. Thus, levels of cranial nerve involvement, response to immunotherapy, reflexes, and pain levels, to name just a few subjects, are different when the two are compared. *See generally* Van den Bergh; Tiryaki & Horak.

[28] *See e.g.*, *Bailey v. Sec'y of Health & Hum. Servs.*, No. 15-1417V, 2020 WL 10486107, at *16 (Fed. Cl. Spec. Mstr. Apr. 24, 2020) (concluding that Petitioner failed to establish vaccine causation after determining that Petitioner's correct diagnosis was ALS, rather than GBS), *mot. for review den'd*, 151 Fed. Cl. 396 (2020).

The medical record in this case preponderates against Respondent's proposed ALS diagnosis. Although some of Ms. Lemanski's symptoms may have been unusual or rare, and it was clearly difficult for her treaters to ever arrive at a common diagnosis, her overall clinical presentation was more consistent with *some kind* of chronic neuropathy. Ms. Lemanski's symptoms were in many ways consistent with a neuropathic disease akin to what Petitioner contends Ms. Lemanski experienced. Furthermore, Ms. Lemanski did not exhibit several clinical features that are commonly seen in ALS, such as cranial nerve involvement or histopathological changes. Ex. 11 at 33; Ex. 10 at 44. And Ms. Lemanski's clinical course was marked by periods of transient improvement. Thus, she experienced periods of improved strength and motor function, and improved fasciculations at various times throughout her disease course. By contrast, and as Dr. Lau testified, ALS is "famously progressive," meaning a patient's condition should show worsening on each exam. Tr. at 90-91.

In addition (and although it took several months for a diagnosis to be embraced), contemporaneous treaters eventually concluded that Ms. Lemanski was suffering from an immune-mediated neuropathy, offering treatments specifically targeted in that manner (although as discussed below these treatments were not ultimately effective). *See* Ex. 2 at 11 (Ms. Lemanski diagnosed with a "generalized immune process," and IVIg treatment recommended); Ex. 17.04 at 623 (Dr. Ferris diagnosed Ms. Lemanski with "progressive axonal neuropathy of unclear etiology" after finding no cranial nerve involvement in her August 2018 EMG); Ex. 10 at 45 (Dr. Grysiewicz noted that Ms. Lemanski's "[s]ymptoms have been present for more than a few weeks and may be consistent with a chronic axonal motor neuropathy"). It is true that the opinions of treating physicians are not binding on a special master, and should always be weighed against the evidence presented in the record. *Snyder*, 88 Fed. Cl. at 746 n.67 ("there is nothing ... that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted"). But here, the treaters' opinions are supported by the symptoms and EMG results reflected in the medical records.

Respondent points out that Ms. Lemanski's specific clinical presentation (*i.e*. axonal and purely motor) was inconsistent with certain well-known neuropathies, like CIDP. Certainly Ms. Lemanski never experienced demyelination, or the kinds of sensory, clinical symptoms that would manifest due to demyelination. Yet some of the literature Petitioner filed supports the existence of a rare subset of chronic neuropathies, with symptoms comparable to Ms. Lemanski's medical course. *See, e.g.*, Querol II (identifying three CIDP patients who experienced an aggressive phenotype with acute onset, predominantly motor symptoms, older age onset, and evidence of denervation at the first EMG). And Petitioner is *not* arguing that Ms. Lemanski should have been diagnosed with the most common form of CIDP. Instead, Ms. Lemanski may have had what the authors of Doppler describe as a "paranodopathy," characterized by axonal loss and severe motor symptoms. Doppler at 727. Oh also notes that a diagnosis of chronic axonal neuropathy is marked by symmetrical *or* asymmetrical polyneuropathy. Oh at 1175. Thus, the fact that Ms. Lemanski's

presentation was strikingly asymmetrical thus does not prevent the diagnosis from being reasonable in this case.

The absence of certain lab work corroborating the proposed diagnosis is similarly not fatal to a diagnostic finding in Petitioner's favor (although as noted below it is problematic for Petitioner's causation theory). Respondent argues, for example, that Ms. Lemanski never displayed anti-ganglioside antibodies or elevated protein in her cerebral spinal fluid – both of which are common in patients with immune-mediated neuropathies. But as Dr. Robbins admitted in his testimony, it is possible for patients with immune-mediated neuropathies to have normal protein levels. Tr. at 296. And even if *most* patients with acute motor axonal polyneuropathies have anti-ganglioside antibodies, their presence is not essential for a diagnosis (and in any event would be more consistent with a *demyelinating* neuropathy). *Id.* at 125.

The question of Ms. Lemanski's areflexia presents a more difficult issue. Respondent correctly points out that the patients in Oh (who experienced a form of chronic axonal neuropathy) all had areflexia (Oh at 1776), while Ms. Lemanski was hyper-reflexive. Tr. at 265. In fact, Ms. Lemanski was documented as having "brisk reflexes" in the middle of her disease course (Ex. 2 at 7; Ex. 6.02 at 71), but further progression of her disease resulted in reduced or absent reflexes. Thus, by September 2018, Ms. Lemanski was documented as having the following: "Reflexes 2+ upper extremities, +1 right knew, trace left knee, absent ankles." Ex. 10 at 44. Such findings of reduced reflexes, especially in her lower extremities, remained consistent through January 2019. Ex. 10 at 13, 17, 23, 27, 33, 38. Accordingly, there is some evidence to support the conclusion that over time, Ms. Lemanski developed the areflexia characteristic of a chronic, motor nerve-oriented neuropathy – although in ALS the lower extremities are more likely than the upper extremities to feature hyperreflexia. Ex. 33 at 2; T. Takeda et al., *Phenotypic Variability and Its Pathological Basis in Amyotrophic Lateral Sclerosis*, 40 Neuropathology 40, 41 (2020), filed as Ex. 33 (ECF No. 26-17). Ultimately, this factor does not strongly favor Petitioner's preferred diagnosis – but also does not preponderate the other way, or prevent a finding on this issue in Petitioner's favor.

Finally, Ms. Lemanski's autopsy results cut against the proposed ALS diagnosis. The examiners found no evidence of histopathological changes (a necessary criteria for ALS). Ex. 11 at 33. Histopathological findings commonly observed in ALS patients include Purkinje cell loss (85%), localized neuronal loss (83%), and neurofibrillary tangles (78%). Coan at 301. None of these were observed in Ms. Lemanski's autopsy. Ex. 11 at 33. While the autopsy may not have been exhaustive, it also does not help Respondent in proving his preferred diagnosis.

For these reasons, I cannot find that the medical records support a diagnosis of ALS. However, I note several caveats in making this determination.

First, I am unable based upon the evidence before me to embrace "CMAP" as the most evidentiarily-supported diagnostic classification for Ms. Lemanski's neuropathy. As Respondent established, no such diagnosis recognized by medical science even exists, and the overall concept of CMAP remains poorly-understood or researched. Tr. at 224; First Robbins Rep. at 8. All I can conclude from this record is that the neuropathy Ms. Lemanski likely experienced impacted her nerve axon more than it caused demyelination (i.e., damaging to the nerve myelin covering of the axon), that motor nerves rather than sensory nerves were likely implicated, and that it was a chronic process.

Second, in offering their interpretations of the medical records, the two competing diagnostic experts (Drs. Lau and Robbins) made several important points relevant to causation. For example, because Ms. Lemanski could not have been properly diagnosed with an acute form of neuropathy, evidence specific to AIDP/GBS is unhelpful, given the faster, self-limiting progression of those forms of neuropathy. The same is true of CIDP – which may be chronic but would also feature demyelination. This possible diagnosis (which Petitioner rejects – and which I agree the record does not support) is of greater relevance, since many of the items of literature offered in this case to demonstrate the potentially pathogenic character of contactin-1 are found in CIDP-specific studies. While many recorded treatment findings lean in favor of the general diagnosis proposed by Petitioner, some of those same findings are far less supportive of a vaccine-mediated injury. And the diagnostic experts' discussion of EMG results was also very illuminating.

II. Petitioner Has Not Carried Her *Althen* Burden of Proof[29]

A. *Althen* Prong Three

The timing of Ms. Lemanski's illness onset, as laid out in the medical records, does not support a finding of causation. First, unrebutted record evidence from close in time to the February 2018 vaccination suggests *a pre-vaccination onset*. The records from Ms. Lemanski's first doctor visit, which took place three days post-vaccination, identified an onset of "three weeks ago." Ex. 6.01 at 58. Then, when Ms. Lemanski returned to the doctor on February 9, 2018 (now one-week post-vaccination), her onset was listed as beginning four weeks earlier. *Id.* at 56. In either case, vaccine causation would not be possible. And this kind of timeframe fits the medical record evidence of Ms. Lemanski's history of foot and leg complaints; while some of her pre-vaccination history involved complaints too long before vaccination to be given much weight, she did report foot complaints in October 2017 (and the timeframe from then to February 2018 is not significantly different from the timeframe from vaccination to May and June 2018, which Petitioner's experts deemed medically acceptable for purposes of causation). Ex. 6.01 at 62.

[29] I address the *Althen* prongs in order of their significance to my determination.

Petitioner's attempt at hearing to gainsay these record onset citations as erroneous (*see* Tr. at 9-10) was uncompelling. It is well-established that oral testimony that conflicts with contemporaneous documents is entitled to little evidentiary weight, unless some reasoned grounds for varying the record is offered. *Cucuras*, 993 F.2d at 1528. My review of these records does not suggest to me that they likely included automatically-added fields, due to drop-down menus. They instead appear to be memorialized summaries of what Ms. Lemanski actually said at the time. Ex. 6.01 at 56, 58. And although a month after these initial appointments Ms. Lemanski began to consistently report a post-vaccination onset, I would give greater weight to evidence contemporaneous with the vaccination and first complaints of clinical symptoms.

Second, the overall course of Ms. Lemanski's illness makes it impossible for me to ascertain *when* a post-vaccination onset would have occurred – thereby preventing me from finding that the timeframe measured from the date of vaccination to onset was medically acceptable. Dr. Lau's efforts to show how the progression of EMG results was inconsistent with ALS only worked to cast doubt on Petitioner's prong three arguments.

The EMGs establish that Ms. Lemanski did not appear to be experiencing significant neuropathic symptoms as of March 2018, despite some evidence of denervation (which actually would be consistent with a preexisting process already underway). Ex. 6.02 at 83-85. The second EMG (from May 2018) was more expressly deemed "not consistent with a diagnosis of diffuse pathology of the motor neurons and/or their axons" (Ex. 5 at 25-26; Ex. 12 at 57-63), and Dr. Lau was ambivalent in pinpointing this time as when the chronic nature of her axonal injury was sufficiently robust to deem it confirmed. Tr. at 69-70; 380-81. Another treater felt these EMG results confirmed merely a sciatic neuropathy, as opposed to an autoimmune-oriented injury. Ex. 2 at 11. Only by the August 2018 EMG did Dr. Lau seem to believe the nature of Ms. Lemanski's injury was better substantiated – but this allows for an onset predating the EMG of anytime in the *prior two to three months* – meaning no sooner than two to three months *after* vaccination. Dr. Berger contended, however, that "several days to 6 weeks" would be the "expected interval [] within which an immunologic reaction to the vaccine would occur." Second Berger Rep. at 2. An onset after May 2018 falls outside of that timeframe.

Given the foregoing, this record does not allow me to preponderantly connect the February 2nd vaccination with what came later, or to firmly identify a precise onset of Ms. Lemanski's axonal-oriented chronic neuropathy. Ms. Lemanski's initial reports of aching and twitching suggest an onset of early January 2018, and thus pre-vaccination. And such an onset is somewhat consistent with her prior history of foot and leg complaints. In direct contrast, her EMG results support an onset of mid-June (two months before the August EMG, which confirmed chronicity). If so (and assuming all prior complaints were unrelated), Ms. Lemanski had a several-month quiescent period post-vaccination before her neuropathic symptoms truly manifested. That gap has not been adequately explained by Petitioner's experts.

Such factual variability in the record prevented Petitioner from preponderantly showing *when* Ms. Lemanski's illness likely began. And she cannot prevail merely by contending that virtually *any* onset within days to several months post-vaccination is medically acceptable – and I need not accept contentions from Petitioner's experts to that end either. I accordingly cannot find that the onset of Ms. Lemanski's neuropathy was temporally associated in a "medically acceptable manner" with the Hepatitis A vaccine.

B. *Althen* Prong Two

Even assuming that Petitioner had been able to show that the Hepatitis A vaccine could cause the production of the allegedly-harmful autoantibodies capable of initiating a chronic, axonal-oriented neuropathy, the record in this case does not preponderantly support the conclusion that the Hepatitis A vaccine "did cause" Ms. Lemanski's neuropathy.

First, certain evidence needed to corroborate the theory unfolding as proposed is absent from the medical record. Petitioner's theory requires the existence of anti-contactin-1 autoantibodies, but Ms. Lemanski was never shown to possess them. Tr. at 122-23. Because such testing would not likely be common in treating a person like Ms. Lemanski, I do not give this record omission great weight. And yet Petitioner's argument is that Ms. Lemanski's clinical presentation suggests she likely *did* possess this antibody. *See, e.g.*, Tr. at 386 (Dr. Berger contending that the fact of Ms. Lemanski's presentation is proof that "the sequence of low probability events" can occur in a single individual). This is the kind of conclusory reasoning I have often criticized when discussing a claimant's success in substantiating the second *Althen* prong. *See e.g., W.G. v. Sec'y of Health & Hum. Servs.*, No. 18-1735V, 2024 WL 2312366, at *18 n.10 (Fed. Cl. Spec. Mstr. Feb. 9, 2024) ("Though the idea of an immune trigger may be consistent with [the expert's] theory, his assertion of that trigger in this specific case, based on no clinical evidence apart from Petitioner's very manifestation of the allegedly vaccine-caused injury, amounts to a circular logic that has been rejected in prior cases").

Petitioner's experts similarly proposed that specific MHC II molecules might bind VP1 and contactin-1 peptides, but as Dr. Bradfute admitted on cross-examination there is no evidence that Ms. Lemanski's T-cells expressed any of the relevant MHC II molecules needed for this cross-reaction. Tr. at 217. When Dr. MacGinnitie used an online tool to examine the frequencies of the some of the relevant MHC II alleles, he discovered that the frequency of these alleles in the U.S. population is generally low (with most presenting in between 1-20% of people). Second MacGinnitie Rep. at 4. Thus, Petitioner's case *assumes* not only that Ms. Lemanski likely possessed them, but that she also happened to be one of the minority of individuals who did so. Again – Petitioner cannot persuasively prove the vaccine likely caused Ms. Lemanski's injury by always invoking the existence of the injury itself as proof that the vaccine caused it.

Second, there is no evidence in the record that Ms. Lemanski ever experienced an inflammatory reaction following the administration of the Hepatitis A vaccine. At Ms. Lemanski's first appointment following the vaccine, on February 5, 2018, she complained only of leg pain and twitching. Ex. 6.01 at 58. She made no mention of the Hepatitis A vaccine at this appointment, nor did she recount experiencing any sort of inflammatory reaction over the previous three days. According to my review of the medical records, it appears Mr. Lemanski did not link her condition to the Hepatitis A vaccine until two and a half months later, on April 18, 2018, when she informed a treater that she had developed random, spontaneous muscle twitching and soreness "a little after" her February vaccination. Ex. 2 at 7. Otherwise, no testing results for a month or more post-vaccination suggested that Ms. Lemanski was experiencing the kind of significant inflammation that might herald an aberrant vaccination-related event, and she never *contemporaneously* reported such a reaction (even if she did much later on).

Petitioner points to evidence of treater speculation of a vaccine association, yet in no such cases did any treaters provide a reasoned explanation for *how* the two were associated. *See* Ex. 2 at 7; Ex. 10 at 45. In September 2018, Dr. Grysiewicz (Ms. Lemanski's neurologist, who seemed to first identify the possibility that she was experiencing a chronic, axonal-oriented neuropathy) noted that "there are case reports [of acute axonal motor neuropathy] associated with vaccinations," but then went on to propose that because Ms. Lemanski's symptoms had lasted for over four weeks, her neuropathy was more likely chronic (thus suggesting that Dr. Grysiewicz only put confidence in a vaccine association with acute neuropathic responses). Ex. 10 at 45. Unlike acute/monophasic conditions, chronic illnesses relapse and remit over a long period of time. Because such a disease process is so different, evidence of vaccine association with acute conditions cannot be thoughtlessly applied to chronic conditions. For this reason, Dr. Grysiewicz's opinion that Ms. Lemanski's disease was chronic makes her preceding note about vaccine association far less probative of causation.

At the same time, other treaters remained wholly unconvinced of a vaccine connection, repeatedly listing the etiology of Ms. Lemanski's disease as unclear. *See* Ex. 17.05 at 133, 159; Ex. 17.04 at 623. And even if treaters had more compellingly explained a basis for their opinion of vaccine causation, Program law clearly does not obligate special masters to accept as sacrosanct the statements of contemporaneous treaters. *Snyder*, 88 Fed. Cl. at 746 n.67 ("However, there is nothing. . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted").

Ms. Lemanski's response to immunotherapy was also invoked at times by Petitioner as establishing that her neuropathy was immune-mediated. And indeed, the medical record provides instances in which Ms. Lemanski deemed her IVIg or plasmapheresis to be beneficial. *See* Ex. 7 at 80; Ex. 5 at 40-47. Treaters also thought the same at times. Ex. 10 at 17-18. But the contention that this positive reaction to immunotherapy "proves" the autoimmune nature of Ms. Lemanski's

injury is undercut significantly by a competing fact – that immunotherapies are *not* effective for treatment of a primarily-axonal chronic neuropathy. *See e.g.*, Van den Bergh at 246. Tragically, these treatments did not arrest Ms. Lemanski's health decline. And (as Petitioner's experts maintained) this is not a matter in which nerve demyelination characterized Ms. Lemanski's injury; were it otherwise, reports of the benefits of immune therapy would be stronger indirect proof of the nature of the injury. I therefore do not give significant weight to this evidence in ascertaining whether the Hepatitis A vaccine "did cause" Ms. Lemanski's injury.

C. *Althen* Prong One

The first *Althen* prong requires a claimant to provide a "persuasive medical theory" demonstrating that the vaccine at issue can cause or significantly aggravate the type of injury alleged. *Althen*, 418 F.3d at 1278. Petitioner contends that the Hepatitis A vaccine triggered (or significantly aggravated) Ms. Lemanski's chronic neuropathy, via molecular mimicry. But this theory lacks preponderant, reliable support (although the evidence on this matter presents a more difficult-to-resolve question).

There are few Program cases involving the Hepatitis A vaccine and *any* form of neuropathy. Based on a cursory review of existing decisions, it appears that in only two instances have special masters found a causal link between the Hepatitis A vaccine and some form of neuropathy, but neither of these cases involve the injury alleged herein. *See Johnson v. Sec'y of Health & Hum. Servs.*, No. 16-1630V, 2024 WL 5349150 (Fed. Cl. Spec. Mstr. Dec. 30, 2024); *J. G. v. Sec'y of Health & Hum. Servs.*, No. 20-664V, 2023 WL 2752634 (Fed. Cl. Spec. Mstr. Feb. 13, 2023).

In *Johnson*, a special master concluded that the Twinrix vaccine (Hepatitis A and Hepatitis B combination) can cause small fiber neuropathy via molecular mimicry. *Johnson*, 2024 WL 5349150, at *34. The finding relied on the conclusion that a small fiber neuropathy is an autoimmune condition akin to GBS (which has been shown to be causally linked to the Hepatitis B vaccine). *Id.* at *32-33. But the discussion focused entirely on the Hepatitis B vaccine, and included no review of evidence specific to the Hepatitis A vaccine. *J.G.* is somewhat more on point. There, a special master found that the Hepatitis A vaccine can cause GBS via molecular mimicry. *J.G.*, 2023 WL 2752634, at *30. But that decision was based in large part on a few case reports, and did not include a discussion of homology or cross-reactions. *Id.* at *30-31. Of course, neither determination is binding herein. *Hanlon v. Sec'y of Health & Hum. Servs.*, 40 Fed. Cl. 625, 630 (1998). But I otherwise find them to offer limited guidance value in resolving this case, which involves a wholly different kind of neuropathy.

In this case, the causation theory offered by Petitioner has several components. Her experts began by purporting to show homology between contactin-1 and VP1. She focused on contactin-

1 as a target self-antigen, because of the existence of reliable literature showing that this specific antibody is likely associated with an axonal-oriented chronic neuropathy akin to what Ms. Lemanski experienced. Tr. at 99; Doppler at 724.

I do find that Petitioner's experts persuasively established an association between this autoantibody and some kind of (as-yet-undefined) chronic, axonal-focused neuropathy. The best evidence offered for this point was Manso, which shows an instance in which direct introduction of the putative cross-reacting contactin-1 antibodies could cause chronic, axonal-oriented harm in animal subjects. Admittedly, the putative causal impact of these antibodies is derived from studies involving very small patient samples, such as Querol I and Doppler. It is also not fully clear that the specific antibodies are *only* so associated with a rare subset of axonal neuropathies, with some of the studies suggesting the antibodies were also associated with concurrent demyelination (which Ms. Lemanski did not appear to experience). *See* Querol I at 374. But on this issue, Petitioner's showing was successful.

Using Clustal Omega, Dr. Bradfute identified stretches of peptide sequences with similar biochemical properties between VP1 and contactin-1. Tr. at 198. But Dr. MacGinnite called the validity of this "homology" finding into question, based in part on his own counter-"research," albeit relying on a BLAST search. *Id.* at 331. According to Dr. MacGinnitie's own search, similarity between the two proteins was only found when he extended the search to a very permissive, non-stringent range. *Id.* And even then, the statistical tool in BLAST revealed that the homology between the two proteins was insignificant. *Id.*

The two opposing immunology experts disputed which chosen databases and methodologies for establishing homology constituted the superior approach,[30] but I do not decide who prevailed on this point. This is primarily, however, due to the fact that the case does not turn on which expert "better performed" their desktop homology prediction. Rather, the issue is whether a showing of homology *in general* deserves much probative weight.

I find that it does not. Homology *alone* does not establish causation. *See Sullivan v. Sec'y of Health & Hum. Servs.*, No. 10-398V, 2015 WL 1404957, at *17-18, n.30 (Fed. Cl. Spec. Mstr. Feb. 13, 2015) (while the law does not require Petitioner to "prove" homology in a Program case,

[30] Although Dr. Bradfute maintained that a BLAST search is somehow less reliable or accurate (Tr. at 215), it is extremely common in Program cases for experts acting for petitioners to rely *solely* on BLAST results. *See J.C. v. Sec'y of Health & Hum. Servs.*, No. 17-69V, 2024 WL 3412625, at *18 (Fed. Cl. Spec. Mstr. May 16, 2024) ("Here, the core element of Dr. Steinman's molecular mimicry theory is his purported demonstration via 'BLAST' search results of homology . . ."). If Dr. Bradfute is correct, his view should be shared immediately with other experts who often act on petitioners' behalf in other Program cases. At the same time (and somewhat ironically), Respondent often attacks the utility of BLAST searches – undermining the finding that a BLAST search is superior to Dr. Bradfute's methodology. *See, e.g., J.C.*, 2024 WL 3412625, at *18 ("Respondent's experts have presented detailed criticisms regarding [expert] reliance on BLAST searches as evidence of molecular mimicry and his specific methodology in producing his results").

mere assertion that HPV strain shares sequences with the human body such that molecular mimicry might occur resulting in injury was by itself insufficient to satisfy burden). Rather, as Dr. MacGinnitie demonstrated, medical science only *begins* with an evaluation of homology when considering whether molecular mimicry best explains a putative autoimmune injury process. Jasti at 1176.[31] And Dr. Bradfute's Clustal Omega search offers nothing more than a *possible* showing of homology in any event.

This evidence thus underscores the speculative quality of Petitioner's evidence, no matter how specific to the theory it may be. *See Tullio v. Sec'y of Health & Hum. Servs.*, No. 15-51V, 2019 WL 7580149, at *15 (Fed. Cl. Spec. Mstr. Dec. 19, 2019) ("…because all proteins, which have hundreds of amino acids, are built from the same 20 amino acids, it is inevitable that some sequences of amino acids will repeat…Thus, the finding of sequence homology does not necessarily mean the similarity has significance to the immune system"), *mot. for review den'd*, 149 Fed. Cl. 448 (2020). Indeed, as Dr. Lau admitted, Petitioner's very focus on the purported homologic similarities between a component of the vaccine and contactin-1 was a reverse-engineered effort, inspired by Ms. Lemanski's clinical presentation. *See* Tr. at 122-23 (". . . we're not saying she has anti-contactin neuropathy because that wasn't what was detected, but that is the most adjacent thing to the Hepatitis A capsid").

In order to bulwark this part of her showing, Petitioner attempted to offer other evidence of how different aspects of the immune system might contribute to cross-reactivity. Thus, Dr. Bradfute conducted an analysis using IEDB, which revealed that both contactin-1 and VP1 antigens are predicted to bind to the same MHC II allele, thereby triggering a cross-reactive response. Tr. at 200-01. But as Dr. MacGinnite pointed out, this analysis shows only that these two antigens are *capable* of binding to the same MHC allele – not that they *likely* do, or have been

[31] Petitioner correctly noted when cross-examining Respondent's experts that Jasti's four criteria for analyzing the utility of molecular mimicry as an autoimmune mechanism in a particular context are not congruent with the preponderant evidentiary standard applied in the Vaccine Program. Tr. at 350. I am fully aware that petitioners need *never* prove causation with certainty – and I would never find a petitioner had failed his evidentiary burden merely because one of Respondent's experts maintained that scientific certainty was not established.

But this does not mean that Jasti's criteria have *no* relevance in my weighing of the evidence. For these criteria do stand as the kinds of factors medical science takes into account when assessing if molecular mimicry has value as a putative mechanism for a specific autoimmune disease's pathogenesis. *See e.g., Cerrone v. Sec'y of Health & Hum. Servs.*, No. 17-1158V, 2023 WL 3816718, at *27 (Fed. Cl. Spec. Mstr. June 1, 2023) ("[T]he [four] factors reflect what knowledgeable members of the relevant scientific community would deem important when determining if molecular mimicry explains a likely disease process"), *mot. for review den'd*, 168 Fed. Cl. 745 (2023), *appeal docketed*, No. 24-1281 (Fed. Cir. Dec. 22, 2023). Thus, while I do not deem the Jasti factors to constitute a "mini-test" that Petitioner must satisfy to prevail, I can reasonably give weight to Respondent's experts' contentions about the best intellectual framework for "testing" whether molecular mimicry is a sound and reliable theory in the specific context before me. This approach is consistent with the fact that I can take into account *Daubert* criteria for testing medical theories, even if those criteria do not rise to the level of literal tests that a Vaccine Program claimant must satisfy. *See Terran*, 195 F.3d at 1316 (ruling that it is appropriate for special masters to utilize *Daubert*'s factors as a framework for evaluating the reliability of causation-in-fact theories presented in Program cases).

shown to by reliable scientific evidence. Second MacGinnitie Rep. at 3. In his second report, Dr. MacGinnite explained that "T cells recognize peptides bound to MHC molecules and the T-cell receptor recognizes both the peptide and the MHC molecule." *Id.*; K. Ashby & K. Hogquist, *A Guide to Thymic Selection of T Cells*, 24 Nature Rev. Immuno. 103, 104 (2024), filed as Ex. D-1 (ECF No. 45-2) ("[U]nlike B cell receptors that recognize antigen directly, T cell receptors (TCRs) recognize a combinatorial ligand composed of an MHC molecule 'presenting' a small peptide"). This makes it highly unlikely that different peptides bound to the same MHC molecule will lead to the activation of the same T-cell. Second MacGinnite Rep. at 3.

More significantly, Petitioner has failed to show that an autoimmune reaction *likely does occur* due to the demonstrated sequence homology, leading to a neuropathy of the sort Ms. Lemanski experienced. *See Yalacki v. Sec'y of Health & Hum. Servs.*, No. 14-278V, 2019 WL 1061429, at *34 (Fed. Cl. Spec. Mstr. Jan. 31, 2019), *mot. for review den'd*, 146 Fed. Cl. 80 (2019) (commenting on petitioner's theory that the Hepatitis B vaccine could trigger a pathogenic process resulting in an autoimmune attack, but finding that it was "not enough for a claimant to invoke the concept of molecular mimicry" as petitioner needed to "cite to evidence, circumstantial or otherwise, suggesting reason to find it plausible that the proposed autoimmune cross-reaction triggered by the relevant vaccine does occur"). Articles like Manso, Querol I, or Doppler may establish the *importance* of anti-contactin-1 antibodies in causing chronic axonal neuropathies, but they do not also stand for the proposition that these antibodies come into existence via molecular mimicry. Indeed, the literature offered provides no hint as to what might produce these antibodies (for example, via an association between them and some analogous wild virus).

Proof connecting the Hepatitis A vaccine and the production of anti-contactin-1 antibodies leading to the proposed neuropathy was also in short supply. Petitioner's experts were forthcoming in admitting they could not offer evidence directly implicating the Hepatitis A vaccine as even capable of causing the production of allegedly-pathogenic anti-contactin-1 antibodies. *See*, *e.g.*, Tr. at 388 (Dr. Berger admitting that "[t]here's not evidence [in the filed literature] that the VP1 protein was the trigger"). They therefore relied on different kinds of indirect proof (which of course Program claimants may do). But these items of evidence were not especially probative.

For example, Petitioner referenced a number of case reports – a kind of evidence not generally given significant weight when evaluating causation. *See Campbell v. Sec'y of Health & Hum. Servs.*, 97 Fed. Cl. 650, 668 (2011) ("Case reports do not purport to establish causation definitively, and this deficiency does indeed reduce their evidentiary value compared particularly to formal epidemiological studies"). Most of these reports do not even involve the Hepatitis A vaccine and/or chronic immune-mediated neuropathy. Roux and Ono, for example, involve GBS, while Samadi involves a wild Hepatis A infection. Petitioners must present evidence that links the specific vaccine at issue to the alleged injury. *Broekelschen*, 618 F.3d at 1345 ("a petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's

case"). And as Dr. MacGinnitie noted, none of these articles explain how the allegedly-causal anti-contactin-1 antibody even came to exist in the studied individuals. Tr. at 361.

Petitioner also cites Souayah, which relies on VAERS reporting (supplemented by data provided by the Center for Biologics Evaluation and Research). But as Dr. MacGinnite correctly pointed out during his testimony (and beyond the fact that Souayah involves GBS rather than chronic axonal neuropathies), reliance on VAERS reports is problematic. Tr. at 348. Such reports can be filed by anyone, there is no check on accuracy or validity, and the claimed adverse post-vaccination event itself is not confirmed. *Id.* The quantity and quality of information obtained in VAERS reports is often insufficient to make an informed decision regarding the causal link between a vaccine and an injury. *Ryman v. Sec'y of Health & Hum. Servs.*, 65 Fed. Cl. 35, 43 (2005). And the reports may be biased towards pre-existing notions of adverse events. *Ryman*, 65 Fed. Cl. at 43. The authors of Souayah acknowledged some of these limitations, stating that VAERS "is subject to underreporting, differential reporting, ascertainment bias, and variability in report quality and completeness." Souayah at 1091.[32] For these very reasons, special masters have properly not afforded substantial weight to VAERS reports.

In sum, the evidence offered by Petitioner amounts to a general showing of homology, coupled with an outline for a *potential* cross-reaction, and some non-specific case report-level evidence. This allows for a slightly *plausible* possibility that the vaccine *could* lead to production of the relevant autoantibodies – which in turn were preponderantly demonstrated to be capable of causing an axonal neuropathy *akin* to what Ms. Lemanski experienced (although it has not been shown she in fact possessed anti-contactin-1 antibodies). Plausibility, however, is not the evidentiary standard in Program cases. *See Oliver v. Sec'y of Health & Hum. Servs.*, 900 F.3d 1357, 1361 (Fed. Cir. 2018) ("To demonstrate causation, the petitioner's 'burden is to show by preponderant evidence' each of the requirements set forth in *Althen* . . .") (quoting *Althen*, 418 F.3d at 1278); *Moberly*, 592 F.3d at 1322. And this is properly the case. For if Program claims could succeed on such an easy-to-meet standard, it is hard to envision (absent strong direct proof refuting causation) circumstances in which any vaccine could not be found capable of plausibly causing an injury, especially if the injury is mediated by the immune system. *Myers v. Sec'y of Health & Hum. Servs.*, No. 21-1205V, 2025 WL 407049, at *20 (Fed. Cl. Spec. Mstr. Jan. 2, 2025).

The parties' dispute on the "can cause" *Althen* prong admittedly presents a thornier issue for resolution. Certainly, Petitioner's experts noted an association between contactin-1 antibodies and chronic axonal neuropathies facially comparable to Ms. Lemanski's clinical presentation. If

[32] In fact, Souayah's authors themselves seemed to recognize the dangers of simply accepting VAERS data at face value. Thus, instead of relying on the subject patients' self-diagnoses, the article's authors deferred to the conclusions of a board-certified neuromuscular specialist who reviewed the patients' reported test results in order to identify diagnostic criteria for GBS. Souayah at 1089. The authors also excluded all patients with incomplete reports, even if the limited information provided supported a diagnosis of GBS. *Id.* at 1091.

the homology showing were given more weight, and the putative association between the Hepatitis A vaccine and the development of this *specific* kind of neuropathy better substantiated, a favorable prong one finding could have been reached. This was not a case in which *no* evidence having substantive merit was offered by Petitioner. Many special masters – moved by the idea that any vaccine can cause virtually any kind of neuropathy via molecular mimicry – might well deem the showing in this case enough to meet the first *Althen* prong (although I have criticized the analytic strength of that kind of determination). *See Gamboa-Avila v. Sec'y of Health & Hum. Servs.*, No. 18-925V, 2023 WL 6536207, at *25 (Fed. Cl. Sept. 11, 2023), *mot. for review den'd*, 170 Fed. Cl. 441 (2024); *appeal docketed*, No. 24-1765 (Fed. Cir. May 1, 2024). But even if I am in error on this aspect of my Decision, I would still be compelled to deny entitlement – for the evidence against Petitioner on the other two *Althen* prongs is far greater. *Dobrydnev v. Sec'y of Health & Hum. Servs.*, 566 Fed. Appx. 976, 980 (Fed. Cir. 2014) (stressing that all three prongs must be satisfied).

III. Petitioner Did not Establish a Significant Aggravation Claim

At trial, most if not all of Petitioner's efforts were directed at proving that the Hepatitis A vaccine *caused* Ms. Lemanski's injury – not that it made a preexisting neuropathic injury worse. Indeed, as noted above, Petitioner affirmatively testified that Ms. Lemanski's symptoms began only *after* vaccination (Tr. at 9-10), and none of her testifying experts put any real effort at hearing into outlining how the Hepatitis A vaccine could have worsened her condition (although their reports may have glancingly addressed the possibility).

However, Petitioner's pre-hearing briefing devoted some time to maintaining an alternative theory of recovery, in which the vaccination did worsen an existing course of CMAP or something like it. Petitioner's Brief, dated June 14, 2024 (ECF No. 48) ("Br.") at 80-86. Respondent also included a refutation of such a claim in his brief. Respondent's Opposition, dated July 17, 2024 (ECF No. 50) ("Opp.") at 18-22. I will therefore briefly address whether this alternative version of an injury claim has preponderant record support.

A. Standard for Significant Aggravation Claim

Where a petitioner alleges significant aggravation of a preexisting condition, the *Althen* test is expanded, and the petitioner has additional evidentiary burdens to satisfy. *Loving v. Sec'y of Health & Hum. Servs.*, 86 Fed. Cl. 135, 144 (2009). In *Loving*, the Court of Federal Claims combined the *Althen* test with the test from *Whitecotton v. Sec'y of Health & Hum. Servs.*, 81 F.3d 1099, 1107 (Fed. Cir. 1996), which related to on-Table significant aggravation cases. The resultant "significant aggravation" test has six components, which require establishing:

> (1) the person's condition prior to administration of the vaccine, (2) the person's current condition (or the condition following the vaccination if that is also

> pertinent), (3) whether the person's current condition constitutes a 'significant aggravation' of the person's condition prior to vaccination, (4) a medical theory causally connecting such a significantly worsened condition to the vaccination, (5) a logical sequence of cause and effect showing that the vaccination was the reason for the significant aggravation, and (6) a showing of a proximate temporal relationship between the vaccination and the significant aggravation.

Loving, 86 Fed. Cl. at 144; *see also W.C.*, 704 F.3d at 1357 (holding that "the *Loving* case provides the correct framework for evaluating off-table significant aggravation claims"). In effect, the last three prongs of the *Loving* test correspond to the three *Althen* prongs.

In *Sharpe v. Sec'y of Health & Hum. Servs.*, 964 F.3d 1072 (Fed. Cir. 2020), the Federal Circuit further elaborated on the *Loving* framework. Under Prong (3) of the *Loving* test, a Petitioner need not demonstrate an expected outcome, but merely that her current-post vaccination condition was worse than pre-vaccination. *Sharpe*, 964 F.3d at 1081. And a claimant may make out a prima facie case of significant aggravation overall without eliminating a preexisting condition as the potential cause of her significantly aggravated injury (although the Circuit's recasting of the significant aggravation standard still permits Respondent to attempt to establish alternative cause, after the burden of proof has shifted to Respondent). *Id.* at 1083.

B. *Loving* Prongs One through Three

Assuming that Ms. Lemanski's chronic, axonal-oriented neuropathy pre-dated vaccination, Petitioner has provided sufficient evidence to establish the first three *Loving* prongs (an easy-to-meet burden in the wake of *Sharpe*).

As established in Petitioner's brief, Ms. Lemanski was in relatively good health prior to her receipt of the Hepatitis A vaccine in February 2018, even if her undiagnosed neuropathy already existed. Br. at 82. Thus, although she complained of knee and foot pain in the years leading up the vaccine, a neurological exam and EMG did not reveal abnormalities. *Id.* at 83; Ex. 6.01 at 61-62; Ex. 6.02 at 144-43. Medical records suggest that Ms. Lemanski began experiencing more aching pain and muscle twitching in both legs sometime in January 2018, about three weeks prior to receiving the vaccine. Ex. 6.01 at 56, 58.

Following her receipt of the vaccine on February 2, 2018, however, Ms. Lemanski's condition progressively deteriorated over time. Br. at 83. She experienced spasms, muscle twitching, and pain in both of her legs. *Id.* By March 2018, she had difficulty walking and completing daily activities. *Id.*; Ex. 6.02 at 86. And by April 2018, Ms. Lemanski had developed left foot drop and continued to experienced muscles aches and lower extremity weakness. Br. at 83; Ex. 2 at 7. Eventually treaters proposed she was likely experiencing some form of neuropathy.

Her disease continued to progress over the next several months, with temporary periods of improvement, and she ultimately passed away in January 2019. Br. at 83-84; Ex. 11 at 8-10.

C. *Loving* Prongs Four through Six

In addressing the final three *Loving* prongs, I hereby incorporate by reference my discussion of causation under the *Althen* prongs. Petitioner has failed to meet these final prongs, for largely the same reasons set forth in my discussion above.

In particular, Petitioner has failed to demonstrate a proximate temporal relationship between the vaccine and the significant aggravation of Ms. Lemanki's condition. As I noted earlier in my analysis, Petitioner could not clearly identify when Ms. Lemanski's condition began – not only with respect to the possibility that onset predated vaccination, but also *when* in the post-vaccination period the aggravating impact of vaccination was evident. Thus, Ms. Lemanski was complaining of symptoms within *three days* of vaccination –shorter than what Dr. Berger suggested would be the timeframe for a chronic process to "set up" (*see* Second Berger Rep. at 2 (proposing several days to six weeks as a medically acceptable interval) – but her testing and clinical exam results did not suggest that a heightened inflammatory process was underway. The first two 2018 EMGs were also inconsistent with the existence of a chronic process, as Dr. Berger maintained. But arguably *they should have been*, had her pre-vaccination injury been suddenly worsened by vaccination – for why then did the first EMG *not* establish the existence of a chronic neuropathy? Or the second for that matter? Petitioner was, therefore, equally unable to establish that the worsening of Ms. Lemanski's chronic axonal neuropathy occurred in a medically-acceptable timeframe in comparison to the date of vaccination (unless "worsening" is defined to mean her immediate post-vaccination symptoms, followed by the indeterminate four to six-month period of symptoms that followed).

In addition, Petitioner has failed to establish that the Hepatitis A vaccine could likely worsen an existing chronic axonal neuropathy, as required under the fourth *Loving* prong. In fact (and unlike with respect to Petitioner's showing on *Althen* prong one, as discussed above), Petitioner's showing on this prong was even less well-substantiated. Thus, although Petitioner may have been able to demonstrate an association between anti-contactin-1 antibodies and this kind of neuropathy, she did not also establish how this vaccine would interact with an existing autoimmune process mediated by these antibodies. At most, Dr. Lau maintained that there are reports in literature of underlying chronic immune-mediated neuropathies worsening after vaccination. Second Lau Rep. at 5. But the articles he cited were a few case reports involving the Covid-19 vaccine and the flu vaccine, not the Hepatitis A vaccine. *See e.g.*, A. Fotiadou et al., *Acute-onset Chronic Inflammatory Demyelinating Polyneuropathy Complicating SARS-CoV-2 Infection and Ad26.COV2.S Vaccination: Report of Two Cases*, 58 The Egyptian J. of Neuro., Psych. and Neurosurg. 116-19 (2022), filed as Ex. 37 (ECF No. 32-3). There is insufficient support for the

conclusion that a vaccine "would have amplified an autoimmune attack" already underway, as suggested by Dr. Berger. Second Berger Rep. at 13-14.

Otherwise, Petitioner contended (again, more as an afterthought than as a foundational contention in her case) that exacerbation would be possible via molecular mimicry. Br. at 84-85; Second Lau Rep. at 5; Second Burger Rep. at 13. But I have already noted the deficiencies of the generic molecular mimicry argument offered in this case, and I find it no more convincing when applied to a significant aggravation claim. It has not been shown that the Hepatitis A vaccine likely results in production of the anti-contactin-1 antibodies in the first place due to molecular mimicry, let alone that the vaccine would stimulate further the existing illness by causing even more of these antibodies to be produced.[33]

CONCLUSION

Petitioner and her family have suffered greatly due to Ms. Lemanski's illness and subsequent passing, and for that they have my deepest sympathy. But it has not been preponderantly shown that Ms. Lemanski's chronic neuropathy was likely caused or significantly aggravated by the Hepatitis A vaccine. As a result, I cannot find entitlement in Petitioner's favor.

In the absence of a motion for review filed pursuant to RCFC Appendix B, the Clerk of the Court **SHALL ENTER JUDGMENT** in accordance with the terms of this Decision.[34]

IT IS SO ORDERED.

s/Brian H. Corcoran
Brian H. Corcoran
Chief Special Master

[33] This also highlights *Loving* prong five problems with a significant aggravation claim – there is no evidence (beyond Petitioner's clinical presentation) that she possessed the anti-contactin-1 antibodies, and no evidence of a vaccine reaction beyond her reported three-day post-vaccination symptoms.

[34] Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment if (jointly or separately) they file notices renouncing their right to seek review.