In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: September 17, 2025

JOHN BUEN,	*	PUBLISHED
Petitioner,	*	No. 21-1314V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Dismissal; Influenza ("Flu") Vaccine; Sudden Sensorineural Hearing Loss ("SSNHL").
Respondent.	*	

Ronald Craig Homer, Conway, Homer, P.C., Boston, MA, for Petitioner.
Austin Josel Egan, U.S. Department of Justice, Washington, DC, for Respondent.

DECISION[1]

I. INTRODUCTION

On May 4, 2021, John Buen ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018).[2] Petitioner alleges that he suffered sudden sensorineural hearing loss ("SSNHL") as a result of an influenza ("flu") vaccine he received on September 15, 2019. Petition at Preamble (ECF No. 1). Respondent argued against compensation, stating that "this

[1] Because this Decision contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Decision will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Decision to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

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case is not appropriate for compensation under the terms of the Vaccine Act." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 29) (emphasis omitted).

After carefully analyzing and weighing the evidence presented in this case in accordance with the applicable legal standards, the undersigned finds that Petitioner failed to provide preponderant evidence that his flu vaccine caused his SSNHL. Thus, Petitioner has failed to satisfy his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is not entitled to compensation.

II. ISSUES TO BE DECIDED

The parties stipulate that Petitioner received a flu vaccination on September 15, 2019 in the United States. Joint Submission, filed Aug. 16, 2024, at 1 (ECF No. 74). The parties agree Petitioner was properly diagnosed with SSNHL. Id.

In dispute is causation, specifically all three Althen prongs: (1) whether the "flu vaccine can cause SSNHL," (2) whether "there is a logical sequence of cause and effect between [Petitioner's] September 15, 2019 flu vaccination and the development of his SSNHL," and (3) whether "the onset of [Petitioner's] SSNHL occurred within a medically appropriate timeframe with respect to his September 15, 2019 flu vaccination." Joint Submission at 2.

III. BACKGROUND

A. Procedural History

Petitioner filed his petition on May 4, 2021, followed by medical records[3] from June 2021 to October 2021. Petition; Petitioner's Exhibits ("Pet. Exs.") 1-16. The case was assigned to the undersigned on April 15, 2022. Notice of Reassignment dated Apr. 15, 2022 (ECF No. 22). Respondent filed his Rule 4(c) report, arguing against compensation, on September 2, 2022. Resp. Rept. at 1.

On February 2, 2023, Petitioner filed an expert report from Dr. Edwin Monsell. Pet. Ex. 19. Respondent filed an expert report from Dr. Jay T. Rubinstein on August 16, 2023. Resp. Ex. A. Petitioner filed a supplemental expert report from Dr. Monsell on November 14, 2023. Pet. Ex. 91. On January 22, 2024, Respondent filed a responsive expert report form Dr. Rubinstein. Resp. Ex. C.

Thereafter, at request of the parties, the undersigned held a Rule 5 conference on March 5, 2024. Rule 5 Order dated Mar. 5, 2024 (ECF No. 63). The undersigned was unable to provide preliminary findings as to Althen prong one. Id. at 2. She preliminarily agreed with the parties' experts that onset was five to six days post-vaccination. Id. Following the Rule 5 conference, Respondent indicated he was not amenable to settlement discussions, and a briefing schedule was set. Joint Status Rept., filed June 6, 2024 (ECF No. 71); Order dated June 7, 2024 (ECF No. 72); Order dated June 20, 2024 (ECF No. 73).

[3] Updated medical records were filed throughout litigation.

Petitioner filed his motion for a ruling on the record on August 19, 2024. Pet. Motion for an Entitlement Ruling on the Record ("Pet. Mot."), filed Aug. 19, 2024 (ECF No. 75). Respondent filed his response on October 25, 2024, and Petitioner filed a reply on November 19, 2024. Resp. Response to Pet. Mot. ("Resp. Response"), filed Oct. 25, 2024 (ECF No. 87); Pet. Reply to Resp. Response ("Pet. Reply"), filed Nov. 19, 2024 (ECF No. 88).

This matter is now ripe for adjudication.

B. Sudden Sensorineural Hearing Loss

Sensorineural hearing loss ("SNHL") is "[h]earing loss resulting from abnormal function of the cochlea, auditory nerve, or higher aspects of central auditory perception or processing." Pet. Ex. 37 at 4 tbl.1.[4] SNHL is "generally idiopathic," but some cases are "associated with infections, vasculitides, tumors, [] genetic conditions, and cardiovascular risk factors." Pet. Ex. 27 at 1;[5] see also Pet. Ex. 94 at 7 (noting "viral infection is speculated as one of the etiologies of SSNHL" with "several studies [] propos[ing] a possible association between viral infection and SSNHL");[6] Pet. Ex. 93 at 1 ("The etiology [of SSNHL] can be classified into various categories such as autoimmune diseases, infections, functional, metabolic disorders, vascular disorders, traumatic causes, due to toxins, and neurological disorders.").[7]

SSNHL is a subset of SNHL that "occurs within a 72-hour window" and meets specific audiometric criteria. Pet. Ex. 37 at 3. Idiopathic SSNHL has "no identifiable cause despite investigation" and accounts for "90% of patients with SSNHL." Id.; see also Pet. Ex. 94 at 1. Despite being "one of the most common" otolaryngology diseases, "[t]he etiology of SSNHL remains unknown." Pet. Ex. 94 at 9; see also Pet. Ex. 93 at 1 ("The etiology of the majority of [SSNHL] cases is unknown.").

[4] Sujana S. Chandrasekhar et al., Clinical Practice Guideline: Sudden Hearing Loss (Update), 161 Otolaryngol. Head & Neck Surg. S1 (2019).

[5] Roger Baxter et al., Sudden-Onset Sensorineural Hearing Loss After Immunization: A Case-Centered Analysis, 155 Otolaryngol. Head & Neck Surg. 81 (2016).

[6] Satoshi Yamada et al., Update on Findings About Sudden Sensorineural Hearing Loss and Insight into Its Pathogenesis, 11 J. Clin. Med. 1 (2022).

[7] Priyanshi Tripathi & Prasad Deshmukh, Sudden Sensorineural Hearing Loss: A Review, 14 Cureus 1 (2022).

C. Factual History

1. Relevant Medical History[8]

On September 15, 2019, Petitioner, age 50, received a flu vaccination[9] in his right arm. Pet. Ex. 1 at 1; Pet. Ex. 16 at 1.

Petitioner's pre-vaccination medical history included cardiac issues, obstructive sleep apnea, hypertension, hyperlipidemia, post-traumatic stress disorder, and orthopedic issues. Pet. Ex. 3 at 57, 131-32, 139.

Respondent noted tinnitus[10] in Petitioner's pre-vaccination history. Resp. Response at 2. The medical records Respondent cited regarding tinnitus identified it as a "Service Connection/Rated Disabilit[y]" at 10%. See, e.g., Pet. Ex. 3 at 93, 100. It is not clear from these records how "Service Connection/Rated Disabilities" are assessed by the U.S. Army, nor when this assessment was done. It is also not clear in which ear or ears Petitioner had tinnitus. At a physical examination on August 21, 2019, less than four weeks prior to vaccination, Petitioner "[did] not complain of . . . difficulty hearing, hearing loss, [or] tinnitus." Id. at 128-32. And on September 15, 2019, the date of vaccination, Petitioner underwent a hearing test as part of his bi-annual physical examination for the U.S. Army Reserve, and the audiogram was normal. See Pet. Ex. 18 at 1; Pet. Ex. 19 at 2 (Dr. Monsell opining Petitioner's "hearing test on Sept[ember] 15, 2019 was normal"); Resp. Ex. A at 2 (Dr. Rubinstein agreeing this hearing screening "revealed normal hearing in both ears"). In remarks dated September 18, 2019, tinnitus was noted. Pet. Ex. 18 at 1.

On September 24, 2019, nine days post-vaccination, Petitioner saw an audiologist for left-sided hearing difficulties, tinnitus, and vertigo[11] that began on September 22, 2019. Pet. Ex. 3 at 120. Petitioner expressed that he might have had "some ear infections prior," but no illness preceded the hearing loss, tinnitus, or vertigo. Id. An examination revealed no "significant abnormalities," however, the "ear canal appeared slightly irritated, but no significant redness was

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[8] This summary of medical records is largely taken from the parties' briefs, as the undersigned finds the parties provided an accurate representation of the records. See Pet. Mot. at 4-14; Resp. Response at 2-4. The undersigned has edited the summary.

[9] This was not Petitioner's first flu vaccination. See Pet. Ex. 16 at 1-3 (documenting flu vaccinations almost yearly).

[10] Tinnitus is "a noise in the ears, such as ringing, buzzing, roaring, or clicking" that "is usually subjective." Tinnitus, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=50114 (last visited Sept. 9, 2025).

[11] Vertigo is "an illusory sense that either the environment or one's own body is revolving; it may result from diseases of the internal ear or may be due to disturbances of the vestibular centers or pathways in the central nervous system." Vertigo, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=52968 (last visited Sept. 9, 2025).

noted." Id. "Comprehensive audiometry (air, bone[,] and speech tests) indicated normal hearing [in the] right ear, and moderately severe to profound SNHL in the left ear. Speech discrimination was nonexistent [in the] left ear. Stenger speech was negative."[12] Id. Petitioner was diagnosed with sudden hearing loss in his left ear, tinnitus in his left ear, and "reported vertigo, symptoms consistent with labyrinthitis."[13] Id. Petitioner was instructed to follow up with his primary care provider ("PCP") for medical therapy and for a magnetic resonance imaging ("MRI") of the brain/internal auditory canal to rule out retrocochlear pathology.[14] Id. at 121. Petitioner was also directed to see an ear, nose, and throat ("ENT") specialist for an evaluation. Id.

Later that day, Petitioner saw his PCP for his sudden hearing loss, tinnitus, and vertigo. Pet. Ex. 3 at 113. Petitioner's PCP noted Petitioner developed sudden tinnitus in his left ear on September 21, 2019 and left-sided hearing loss on September 22, 2019. Id. Petitioner's PCP diagnosed him with left-sided sudden idiopathic hearing loss, prescribed prednisone, and ordered an MRI without contrast of the internal auditory canal. Id. at 117.

On September 26, 2019, Petitioner underwent an internal auditory canal MRI without contrast. Pet. Ex. 3 at 56. The MRI was normal. Id. The seventh and eighth cranial nerve complex revealed normal caliber and did not show enhancement. Id. The study did not show any "cerebellar pontine angle region abnormality."[15] Id.

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[12] A Stenger test "(for simulated unilateral hearing loss) a signal is presented at an intensity less than the admitted threshold to the affected ear, and a less intense signal of the same frequency is presented simultaneously to the unaffected ear. If the subject is feigning a loss of hearing, the signal in the unaffected ear will not be heard." Stenger Test, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=112989 (last visited Sept. 9, 2025).

[13] Labyrinthitis is "inflammation of the internal ear" that "may be accompanied by hearing loss or vertigo." Labyrinthitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=27360 (last visited Sept. 9, 2025).

[14] Retrocochlear hearing loss is "[SNHL] in which the lesion is proximal to the cochlea, in the vestibulocochlear nerve or one of the auditory areas of the brain." Retrocochlear Hearing Loss, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=80206 (last visited Sept. 9, 2025).

[15] Cerebellopontine angle is the region between the cerebellum and the pons. Angulus Pontocerebellaris, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=56635 (last visited Sept. 9, 2025). A tumor in this region presents with tinnitus, unilateral hearing loss, and vertigo. See Michie Hamiter, Vestibular Schwannoma, Merck Manual, https://www.merckmanuals.com/professional/ear-nose-and-throat-disorders/inner-ear-disorders/vestibular-schwannoma (last reviewed May 2023); Table: Common Localizing Manifestations of Primary Brain Tumors, Merck Manual, https://www.msdmanuals.com/professional/multimedia/table/common-localizing-manifestations-of-primary-brain-tumors (last visited Sept. 9, 2025); Table: Cranial Nerves, Merck Manual, https://www.msdmanuals.com/professional/multimedia/table/cranial-nerves (last visited Sept. 9, 2025).

Petitioner saw a physical therapist for assistive device assessment and training later that day, on September 26, 2019, for benign paroxysmal vertigo of the left ear. Pet. Ex. 3 at 71-73, 109-11. Assessment was "[a]cute vestibular condition requiring increased gait [stability]." Id. at 74, 111. Petitioner was discharged from physical therapy with a cane, and the physical therapist remarked that Petitioner had good rehabilitation potential. Id.

On October 1, 2019, Petitioner saw a psychiatrist for a "primary care-mental health integration follow-up." Pet. Ex. 3 at 107. The psychiatrist noted that Petitioner "ha[d] been having physical problems due to potential [] reaction to a vaccine" and that Petitioner thought his "hearing loss, increased tinnitus, and vestibular imbalance [were] due to reaction to a flu vaccine." Id.

On October 3, 2019, Petitioner followed up with his audiologist for re-evaluation. Pet. Ex. 3 at 106. Petitioner had no abnormalities in his ear canals, no nystagmus or abnormalities shown with Hallpike,[16] normal hearing in his right ear, and "profound hearing loss" in the left ear. Id. No subjective vertigo was noted. Id. at 107. The audiologist documented Petitioner's left-sided hearing loss as "stable." Id.

On October 10, 2019, Petitioner saw an otorhinolaryngologist, Dr. Benjamin Westbrook, for an out-patient consultation at William Beaumont Army Medical Center. Pet. Ex. 2 at 25-27. Petitioner reported sudden-onset dizziness and hearing loss two weeks prior. Id. at 25. Dr. Westbrook noted that Petitioner had "machine-noise tinnitus," no prior episode of symptoms, and "[n]o preceding illness." Id. Petitioner stated that his vertigo symptoms resolved within twenty-four hours and the remaining disequilibrium seemed to be resolving. Id. His complaints including "[h]earing loss and earache," "[d]izziness, vertigo, and difficulty with balance." Id. Dr. Westbrook noted Petitioner's first audiogram showed "severe panfrequency SNHL" and his follow-up audiogram conducted after ten days of prednisone showed "profound panfrequency loss." Id. Physical examination of the ears was normal. Id. at 26. Assessment was SNHL in the left ear, "with unrestricted hearing on the contralateral side," and tinnitus in the left ear. Id. at 27. Dr. Westbrook indicated "[p]ossible vestibular neuronitis[17] as a cause" of Petitioner's SSNHL. Id. A left transtympanic steroid injection was administered. Id. Petitioner was to follow up in ten days for a new audiogram. Id.

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[16] Dix-Hallpike maneuver is "a test for benign positional vertigo." Dix-Hallpike Maneuver, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=87854 (last visited Sept. 9, 2025). "[T]he examiner turns the head of the seated patient to one side and pulls the patient backward into a supine position with the head hanging over the edge of the examining table; the patient then looks straight ahead and the examiner observes for positional nystagmus, which is indicative of benign positional vertigo." Id.

[17] Vestibular neuronitis is "a disturbance of vestibular function consisting of a single attack of severe vertigo, usually accompanied by nausea and vomiting but without auditory symptoms." Vestibular Neuronitis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=92648 (last visited Sept. 9, 2025).

Petitioner returned to William Beaumont Army Medical Center and saw otorhinolaryngologist Dr. Jacqueline Anne Anderson on October 23, 2019. Pet. Ex. 2 at 22. Dr. Anderson noted Petitioner reported "subjective improvement in his hearing since the injection;" Petitioner was "able to appreciate some tones such as a piano," but "[s]till struggle[d] with conversation/understanding." Id. at 23. Petitioner was not able to get his repeat audiogram prior to this visit. Id. A second transtympanic steroid injection was administered. Id. Petitioner was instructed to obtain a repeat audiogram and return for follow-up examination after testing. Id.

On November 15, 2019, Petitioner returned to his audiologist. Pet. Ex. 3 at 106. Petitioner reported that he had received a dose of intratympanic gentamycin and second dose of prednisone from Dr. Westbrook, and improvement in his vertigo. Id. The tympanic membrane appeared "slightly retracted" in the left ear and a "Type C tympanogram"[18] was noted in the left ear. Id. "Tone audiometry indicated normal hearing [in the] right ear and moderate to profound hearing loss [in the] left ear." Id. The audiologist noted "there [was] about [a] -25 to -30dB improvement below 6000 Hz [in the] left ear." Id.

Petitioner returned to otorhinolaryngologist, Dr. Anderson, on November 20, 2019. Pet. Ex. 20 at 19. Petitioner reported his recent audiogram showed a 20% improvement. Id. at 20. Petitioner still had difficulty with word recognition, which was not tested on his most recent audiogram, as well as vertigo, particularly in the morning. Id. He was walking with a cane and getting driven to work. Id. Upon review of Petitioner's most recent audiology report, Dr. Anderson concluded Petitioner's left ear was "essentially anacoustic."[19] Id. She further "suspect[ed]" his left ear was "unaidable" and recommended he try a Contralateral Routing of Signal ("CROS") hearing aid.[20] Id.

On December 11, 2019, Petitioner presented to the audiology clinic at William Beaumont Army Medical Center for "consideration of aural rehabilitation options." Pet. Ex. 2 at 17. Audiometry testing "indicate[d] moderate to profound primarily [SNHL] in the left ear and hearing within normal limits in the right ear." Id. Trial with CROS hearing aid was

[18] Type C tympanogram "demonstrates a highly negative pressure in the middle ear, correlating to a retracted tympanic membrane. A viral upper respiratory infection may impair the ventilatory function of the eustachian tube. Negative middle ear pressure develops and nasopharyngeal contents are aspirated into the middle ear, resulting in [acute otitis media]. This type of curve may indicate a transition between a normal ear and an ear that is full of fluid." Edward Onusko, Tympanometry, 70 Am. Fam. Physician 1713 (2004), https://www.aafp.org/pubs/afp/issues/2004/1101/p1713.html.

[19] Anacousia is "total deafness." Anakusis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=2535 (last visited Sept. 9, 2025).

[20] See Types of Hearing Aids, Clev. Clinic, https://my.clevelandclinic.org/health/articles/5122-hearing-aid-styles (last reviewed Sept. 12, 2022).

recommended as well as a hearing evaluation for possible cochlear implant candidacy or bone-anchored hearing aids ("BAHA")[21] in three to six months. Id.

Petitioner followed up with his audiologist on October 15, 2020. Pet. Ex. 8 at 95. Petitioner's diagnosis of moderate to profound SNHL in his left ear was unchanged. Id. Petitioner was fitted for a hearing aid on November 6, 2020. Id. at 94; Pet. Ex. 95 at 104.

On December 2, 2020, Petitioner established care at the occupational health clinic at William Beaumont Army Medical Center. Pet. Ex. 11 at 19. During this visit, it was documented that Petitioner had a "reaction to [flu] vaccine and lost hearing in [his] left ear and [had] diminished hearing in his right ear." Id.

On December 5, 2023, Petitioner's Allergy Profile was updated to include the flu vaccine "due to [history] of hearing loss." Pet. Ex. 95 at 9, 27. Petitioner's "previous contraindication" to the flu vaccine was noted and "[p]rovider review indicate[d] [Petitioner] should NEVER receive seasonal [flu] vaccine." Id. at 27.

2. Petitioner's Declaration[22]

At the time of vaccination, Petitioner was a member of the U.S. Army Reserve, where he was required to take a bi-annual physical fitness test that included a hearing test, vision test, and immunizations, which he completed on September 15, 2019. Pet. Ex. 13 at ¶¶ 1-2. He received a flu vaccination on this date and reported he "felt well." Id. at ¶ 2.

On the evening of September 21, 2019, Petitioner noticed tinnitus in his left ear. Pet. Ex. 13 at ¶ 3. The following morning, September 22, 2019, he noticed sudden hearing loss in his left ear. Id. "Symptoms of vertigo and nausea soon followed several hours later." Id. He was "incapacitated by vertigo for the first four days of [his] symptoms," after which the severity subsided. Id.

Petitioner first saw a provider for treatment on September 24, 2019. Pet. Ex. 13 at ¶ 4. After various tests were run, he was diagnosed with SSNHL. Id. He was started on a 10-day course of prednisone. Id. at ¶¶ 4-5. He underwent a repeat audiogram after completing his course of prednisone and no improvement was indicated. Id. at ¶ 5. Petitioner underwent a transtympanic injection, which "improved lower tonal frequencies," but "verbal word recognition and high tonal frequencies were still absent." Id. He explained he had "very little functional improvement as a result of the treatment." Id. And after an additional injection in October 2019, audiogram confirmed no improvement was seen. Id. at ¶ 6. His vertigo and tinnitus also remained unchanged. Id.

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[21] See Bone-Anchored Hearing Aid, Clev. Clinic, https://my.clevelandclinic.org/health/treatments/14794-bone-anchored-auditory-implant (last reviewed on Nov. 21, 2023).

[22] Although titled an affidavit, it was not notarized. Therefore, it is referenced as a declaration. Petitioner also submitted a declaration stating no prior civil action had been filed for his alleged vaccine injury. See Pet. Ex. 14.

At this time, in late 2019, Petitioner was unable to drive and required a cane to ambulate. Pet. Ex. 13 at ¶ 6. He also began using hearing aids, which allowed for "some improvement." Id. at ¶ 8. As of the date Petitioner executed his declaration, May 3, 2021, Petitioner "still rel[ied] on [] hearing aids for the slight boost they provide to [his] hearing deficit." Id.

D. Expert Reports and Medical Literature

1. Petitioner's Expert, Dr. Edwin Monsell[23]

a. Background and Qualifications

The parties stipulated that Dr. Monsell is an expert in otolaryngology. Joint Submission at 1. Dr. Monsell is "board-certified by the American Board of Otolaryngology—Head and Neck Surgery and hold[s] a Certificate of Added Qualifications from the same board in Neurotology (diseases and surgery of the inner ear and related skull base)." Pet. Ex. 19 at 1; see also Pet. Ex. 20 at 4. He obtained a Ph.D. in cell biology and neuroscience from Duke University in 1977 and an M.D. from University of North Carolina School of Medicine in 1979. Pet. Ex. 20 at 1. He then completed a surgical internship and a residency in the Otolaryngology—Head and Neck Surgery department at Northwestern University, followed by a fellowship at the House Ear Institute in Los Angeles, CA. Id. Since 1986, Dr. Monsell has held various hospital or other professional appointments and since 2000, he has also held faculty appointments. Id. at 1-2. He currently holds two hospital appointments in Seattle, Washington and teaches in the Otolaryngology—Head and Neck Surgery department at University of Washington in Seattle. Id. He is a member and has held various positions for the American Academy of Otolaryngology—Head and Neck Surgery, Association for Research in Otolaryngology, and other professional societies. Id. at 2-3; Pet. Ex. 19 at 1. In 2003, Dr. Monsell "received the Harris P. Mosher Award, the highest award for clinical and translational research in Otolaryngology by the American Laryngological, Rhinological[,] and Otological Society (a/k/a the Triological Society) for [his] research on mechanisms of hearing loss." Pet. Ex. 19 at 1. Throughout his career, he has published on the mechanisms of hearing loss, "treated many thousands of patients with hearing loss[,] and performed over 3,500 major ear operations to remove tumors or infections and restore hearing." Id.; see also Pet. Ex. 20 at 10-18.

b. Opinion

To provide context for his opinions, Dr. Monsell explained "the inner ear contains very small, complex cellular, molecular, vascular, immunologic, neural, and sensory structures surrounded by dense bone." Pet. Ex. 19 at 13.

He "propose[d] that [the] [flu] vaccine sometimes can and very rarely does trigger SSNHL." Pet. Ex. 19 at 14. He explained that identified risk factors for SSNHL (physiology, energy dependence, and blood supply) predispose the human ear to SSNHL. Id. These risk factors "lower[] the threshold for complex, interacting cascades of events triggered by [flu]

[23] Dr. Monsell provided two expert reports. Pet. Exs. 19, 91.

vaccination. The defenses of the inner ear are overwhelmed, resulting in cochlear damage and SNHL." Id. Thus, "in the right setting," the flu vaccine can initiate a sequence of events resulting in cochlear damage and SNHL. Id.

i. Althen Prong One

According to Dr. Monsell, "[m]ultiple mechanisms are implicated in the process of SSNHL." Pet. Ex. 19 at 20. The sudden onset seen in SSNHL suggests a vascular mechanism. Id. He proposed that the flu vaccine can cause SSNHL "based on idiosyncratic microvascular and inflammatory reactions." Id. at 23.

Dr. Monsell proposed a detailed mechanism: the "post-vaccination inflammatory response[] led to development of inflammation, reduced cochlear blood flow, injury/reperfusion of [the] left inner ear, creation of reactive oxygen species [("ROS")[24]], and activation of cellular signaling pathways and events, leading to cell death and resultant unilateral severe-to-profound sensorineural deafness." Pet. Ex. 19 at 23. According to Dr. Monsell, this mechanism contains seven steps to explain how the flu vaccine can trigger SSNHL. Id. at 15.

Each step is set forth below, along with a brief analysis of Dr. Monsell's supportive medical literature. See Pet. Ex. 19 at 14-19.

1. Seven-Step Mechanism and Supporting Medical Literature

I. Flu vaccination stimulates the Acute Phase Reaction ("APR"), which includes release of inflammatory cytokines into circulation, fibrinogen, tumor necrosis factor ("TNF"),[25] nuclear factor kappa B ("NFKB" or "NFκB"),[26] and others.

[24] Reactive oxygen species, or ROS, are "biologically active, partially reduced derivatives of molecular oxygen (O_2), including the superoxide radical, hydrogen peroxide, and the hydroxyl radical. They are produced by normal metabolic processes and may also be produced by the absorption of energy, such as ultraviolet or ionizing radiation, and can damage biological systems." Reactive Oxygen Species, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=107488 (last visited Sept. 9, 2025).

[25] TNF refers to "either of two lymphokines that are capable of causing in vivo hemorrhagic necrosis of certain tumor cells but not affecting normal cells; they have been used as experimental anticancer agents but can also induce shock when bacterial endotoxins cause their release." Tumor Necrosis Factor, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74613 (last visited Sept. 9, 2025).

[26] NFKB is "a transcription factor that plays an important role in the expression of proinflammatory genes that are associated with apoptosis, tumorigenesis, inflammation, arthritis, and some autoimmune conditions." Nuclear Factor κB, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=74572 (last visited Sept. 9, 2025).

The first step of Dr. Monsell's theory is that following flu vaccination, the APR is stimulated, leading to the release of inflammatory cytokines (fibrinogen, TNF, NFKB, and others). Pet. Ex. 19 at 16.

In support, Dr. Monsell cited Khalil and Al-Humadi,[27] who discussed types of "acute phase reactants" and their potential importance relative to vaccination. Pet. Ex. 19 at 16 (citing Pet. Ex. 46 at 1). Acute phase reactants are defined as "a group of 11 key proteins," eight of which have positive effects and three with potentially undesired effects. Pet. Ex. 46 at 6. The authors posited that changes in the expression of these proteins vary due to individual genetics, age, or other factors, and may alter the response to any given vaccine. Id.

Khalil and Al-Humadi listed the acute phase reactants associated with vaccines. Pet. Ex. 46 at 5 tbl.II. The acute phase reactants C-reactive protein ("CRP")[28] and transthyretin[29] were identified as associated with the flu vaccine based on a study published in 2006. Id. (citing Pet. Ex. 35).[30] CRP was identified as a "[p]ositive acute phase reactant[]." Id. at 3, tbl.I. It is a "plasma protein . . . produced as a result of pro-inflammatory cytokine signaling" that is "elevated during infection or inflammation as part of the innate immune response."[31] Id. at 2. The authors suggested that CRP has potential value as a "diagnostic marker for active inflammation and infection." Id. The authors did not identify any adverse effects of CRP caused by the flu vaccination.

Transthyretin (also known as prealbumin) "is a negative [acute phase reactant] synthesized and excreted by the kidneys and gastrointestinal tract." Pet. Ex. 46 at 5. The authors noted that it "functions as a biomarker for predicting poor short-term outcome and disease severity in patients with burn injuries or respiratory failure." Id. (internal citations omitted). Low levels have been associated with an "increase in mortality," and low preoperative levels are associated with "increased risk of postoperative infections." Id. Khalil and Al-Humadi did not

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[27] Rafaat H. Khalil & Nabil Al-Humadi, Types of Acute Phase Reactants and Their Importance in Vaccination (Review), 12 Biomed. Reps. 143 (2020).

[28] C-reactive protein is "a globulin that forms a precipitate with the somatic C-polysaccharide of the pneumococcus in vitro; it is the most predominant of the acute-phase proteins." C-Reactive Protein, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=100489 (last visited Sept. 9, 2025).

[29] Transthyretin is "an α-globulin secreted by the liver that transports retinol-binding protein and thyroxine in the blood." Transthyretin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=50705 (last visited Sept. 9, 2025).

[30] Cara L. Carty et al., Inflammatory Response After Influenza Vaccination in Men with and Without Carotid Artery Disease, 26 Arterioscler. Thromb. & Vasc. Biol. 2738 (2006).

[31] Innate immunity is "immunity based on the genetic constitution of the individual." Innate Immunity, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=81992 (last visited Sept. 9, 2025).

identify any risk of SSNHL or other hearing loss due to CRP or transthyretin. The specific proteins listed by Dr. Monsell in his expert report, including fibrinogen, TNF, and NFKB, were not identified in the list of reactants associated with vaccines in the paper by Khalil and Al-Humadi.

Dr. Monsell also cited a paper by Rogers et al.[32] for the proposition that the NFKB gene was associated with an inflammatory process and expressed in both flu infections and with flu vaccinations. Pet. Ex. 19 at 16 (citing Pet. Ex. 72). NFKB was found to be one of 334 genes common to both flu infection and vaccination. Pet. Ex. 72 at 6. The authors noted that the NFKB signaling pathway "is activated during [flu] infection which up-regulates antiviral genes and can regulate viral synthesis." Id. at 11. The authors did not implicate NFKB as playing a role in causing hearing loss or SSNHL after the flu vaccine.

Of note, Rogers et al. also found that genes unique to flu infection and vaccination were "involved in different processes," which "indicate[d] that with the actual infection the body undergoes different processes to [those] induced by vaccination." Pet. Ex. 72 at 15-16.

II. Acute Phase Reactants reduce cochlear blood flow and cause vascular damage, including breakdown of the cochlear blood-tissue barrier.

Second, Dr. Monsell proposed that acute phase reactants reduce cochlear blood flow and cause vascular damage and breakdown of the cochlear blood-tissue barrier.[33] Pet. Ex. 19 at 15-16. He posited that "[f]ibrinogen is released in response to [flu] vaccination and raises blood viscosity." Id. at 16. However, Khalil and Al-Humadi did not identify fibrinogen as an acute phase reactant associated with the flu vaccine. See Pet. Ex. 46 at 5 tbl.II. Fibrinogen was identified with *Staphylococcus* ("*S.*") *aureus* capsular polysaccharide vaccines, not flu vaccines. Id.

Dr. Monsell cited articles to support the proposition that fibrinogen levels have been found to be elevated in hearing loss patients compared to controls as well as animal studies inducing hearing loss following injection of fibrinogen. Pet. Ex. 19 at 16-17 (citing, e.g., Pet. Ex. 73 at 1 ("Fibrinogen plasma levels were significantly increased in [sudden hearing loss]

[32] Lavida R.K. Rogers et al., Microarray Gene Expression Dataset Re-Analysis Reveals Variability in Influenza Infection and Vaccination, 10 Front. Immunol. 1 (2019).

[33] For more information on the cochlear blood-tissue barrier, see Pet. Ex. 61 (Lingling Neng et al., Endothelial Cell, Pericyte, and Perivascular Resident Macrophage-Type Melanocyte Interactions Regulate Cochlear Intrastrial Fluid-Blood Barrier Permeability, 14 J. Ass'n for Rsch. Otolaryngol. 175 (2013)).

patients.");[34] Pet. Ex. 86 at 1 (inducing acute hearing loss by injecting fibrinogen in an animal study)).[35]

Dr. Monsell also cited two articles that he asserted "reported [] fibrinogen levels were higher in patients with SSNHL who did not recover hearing than in patients who did." Pet. Ex. 19 at 16. The first, by Weiss et al. (2014),[36] "found a significant association between lower plasma fibrinogen levels and good hearing recovery." Pet. Ex. 85 at 6. And the second article, from Oya et al. (2018),[37] found elevated fibrinogen was "a prognostic factor" in SSNHL and "reflect[ed] disturbance and damage of microcirculation in the inner ear." Pet. Ex. 64 at 5.

However, Dr. Monsell cited no foundational evidence to show that the flu vaccine at issue here contains fibrinogen or that fibrinogen is increased after administration of a flu vaccine. And Weiss et al (2014) and Oya et al. disagree as to whether fibrinogen is a cause of SSNHL. The Weiss et al. (2014) authors "suggest[ed] that higher fibrinogen is not only a surrogate marker of increased [idiopathic SSNHL] risk, but a pathogenetic factor in the development of [idiopathic SSNHL] itself." Pet. Ex. 85 at 6. However, Oya et al., published four years later, specifically stated that they were unable to confirm elevated fibrinogen as a direct cause of SSNHL. Pet. Ex. 64 at 5. They concluded elevated fibrinogen "might not directly connect with the cause of SSNHL but with the severity of SSNHL." Id.

Further, Khalil and Al-Humadi did not show that the flu vaccination was associated with an acute phase reaction relative to TNF. See Pet. Ex. 46 at 5 tbl.II. Dr. Monsell cited a paper by Sharaf et al. for the premise that "TNF decreases cochlear blood flow." Pet. Ex. 19 at 17. However, this article was not provided, and thus, the statement cannot be confirmed.

In addition to fibrinogen and TNF, Dr. Monsell also asserted that the flu vaccine activates NFKB. Pet. Ex. 19 at 17. He opined that hemagglutinin, which is in the flu vaccine, "strongly activates the binding and transcription of NFκB," which "induces production of [TNF], which in turn induces the production of NFκB, potentially creating a circular, accelerat[ed] pathway of cytokine secretion." Id. However, Dr. Monsell did not provide support for the premise that the

[34] Claudia Rudack et al., Vascular Risk Factors in Sudden Hearing Loss, 95 Thromb. & Haemost. 454 (2006).

[35] Bernhard G. Weiss et al., Drug-Induced Defibrinogenation as New Treatment Approach of Acute Hearing Loss in an Animal Model for Inner Ear Vascular Impairment, 38 Otol. & Neurotol. 648 (2017).

[36] Daniel Weiss et al., Platelet Glycoproteins and Fibrinogen in Recovery from Idiopathic Sudden Hearing Loss, 9 PLoS One e86898 (2014).

[37] Ryohei Oya et al., Serum Fibrinogen as a Prognostic Factor in Sudden Sensorineural Hearing Loss: A Meta-Analysis, 39 Otol. & Neurotol. e929 (2018).

flu vaccine[38] induces the binding and transcription of NFKB. Further, Dr. Monsell has not provided foundational evidence to establish that NFKB plays a role in hearing loss associated with the flu vaccine. Thus, the relevance of NFKB here is not clear.

The same is true of TNF. Dr. Monsell stated that TNF is "released in response to [flu] vaccination" and that "TNF activation" occurs as part of the immune response to the flu vaccine. Pet. Ex. 19 at 17. He cited a paper by Szyszko et al.,[39] published in 2006, reporting on the cytokine response to whole and split virus flu vaccines. Pet. Ex. 80 at 1-2. Four inflammatory cytokines were detected after vaccination, including TNF-α. Id. at 4. Higher levels of TNF-α were found in the whole virus vaccine, "whereas [the] split virus vaccine [was] dominated [by] the [cytokine] Il-1β response." Id. at 4, 7. The study does not appear to support a finding that the split virus vaccine[40]—"[t]he most commonly used formulation today"—significantly elevated TNF-α levels or that TNF activation occurred. Id. at 1.

The other articles cited by Dr. Monsell related to TNF do not show that the flu vaccination increases levels of TNF. Bertlich et al.,[41] for example, noted that impaired cochlear blood flow has been associated with SSNHL, and in their animal study, TNF decreased blood vessel diameter. Pet. Ex. 29 at 1; see also Pet. Ex. 34 at 5;[42] Pet. Ex. 61. But the authors did not discuss the flu vaccine or suggest that it increased or activated TNF so as to affect cochlear blood flow.

Regarding CRP, Dr. Monsell stated it "inhibits endothelial nitric oxide [] synthase[43] expression, which in turn reduces [nitric oxide] bioavailability and increases the production of

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[38] Dr. Monsell cited Pahl and Baeuerle for support; however, the authors reported the "flu *virus* hemagglutinin [] activated NF-κB DNA binding and transcriptional activation." Pet. Ex. 65 at 1 (emphasis added) (Heike L. Pahl & Patrick A. Baeuerle, Expression of Influenza Virus Hemagglutinin Activates Transcription Factor NF-κB, 69 J. Virol. 1480 (1995)). Flu vaccination was not discussed.

[39] E. Szyszko et al., Impact of Influenza Vaccine Formulation with a Detailed Analysis of the Cytokine Response, 64 Scand. J. Immunol. 467 (2006).

[40] The record does not specify whether the vaccine Petitioner received was a split-virus vaccine, as the package insert was not provided. See Pet. Exs. 1, 16.

[41] Mattis Bertlich et al., Cochlear Pericytes Are Capable of Reversible Decreasing Capillary Diameter In Vivo After Tumor Necrosis Factor Exposure, 38 Otol. & Neurotol. e545 (2017).

[42] Martin Canis & Mattis Bertlich, Cochlear Capillary Pericytes, 1122 Adv. Exp. Med. & Biol. 115 (2019).

[43] Synthase is "used in the trivial or recommended names of some enzymes . . . when the synthetic aspect of the reaction is dominant or emphasized." Synthase, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=48589 (last visited Sept. 9, 2025).

ROS." Pet. Ex. 19 at 17. He did not explain the relevance of this sentence but cited to several articles. Id. Carty et al. explained that the proteins CRP, serum amyloid A, and IL-6 are inflammatory markers that "are associated with an increased risk of cardiovascular disease." Pet. Ex. 35 at 1. These proteins are also described as "key components of the [APR] to injury or infection," and the APR is regulated by the NFKB pathway, "which is activated systemically by cytokines." Id. The authors studied APR in white men with severe carotid[44] artery disease (43) and in controls (61). Id. The study sought to determine "whether APR predicts or varies with vascular disease status." Id. Using the seasonal flu vaccine as "a standard immune stimulus," measurements were taken to determine carotid artery disease status and changes in plasma levels of the proteins known to be markers of inflammation, including CRP. Id. The study found that "APR may trigger [] systemic and local (at the vessel wall) inflammation" that "may contribute to cardiovascular disease." Id. at 6. However, the authors limited their discussion of the findings to cardiovascular disease. Id. They did not discuss the cochlear blood system, which, based on Dr. Monsell's description, is unique and quite complex. See Pet. Ex. 19 at 13.

Pepys and Hirschfield[45] also discussed CRP in the context of cardiovascular disease and provided information about the protein as an "acute-phase reactant," discussing its "possible role in pathogenesis of disease." Pet. Ex. 67 at 1. The article is limited to cardiovascular disease and again does not discuss the role of CRP as it relates to cochlear blood flow.

Falsey et al.[46] studied how the flu A infection affected CRP levels in seven young adults, 15 elderly patients, and 36 hospitalized adult patients and found CRP levels were elevated during acute flu illness in hospitalized patients. Pet. Ex. 41 at 1. The study further showed that symptoms of dyspnea (shortness of breath), wheezing, and fever, were associated with higher CRP levels. Id.

And Liuba et al.[47] studied levels of brachial artery endothelial function and levels of CRP and fibrinogen using the flu vaccine as an inflammatory stimulus in eight healthy men. Pet. Ex. 50 at 1. They found the "[flu] vaccination caused a slight elevation in CRP . . . and fibrinogen . . . at [two] days, which completely resolved at 14 days." Id. The authors noted that "[s]imilar to other vaccines, [the flu] vaccine causes a short-lived inflammatory response that resolves within 48-72 hours after vaccination. Slight elevation in CRP, fibrinogen, and proinflammatory cytokines occurs during the inflammatory phase." Id. at 7 (internal citation omitted). However,

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[44] Carotid "pertain[s] to the principal artery of the neck." Carotid, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=8129 (last visited Sept. 9, 2025).

[45] Mark B. Pepys & Gideon M. Hirschfield, C-Reactive Protein: A Critical Update, 111 J. Clinical Investigation 1805 (2003).

[46] Ann R. Falsey et al., Response of C-Reactive Protein and Serum Amyloid A to Influenza A Infection in Older Adults, 183 J. Infect. Dis. 995 (2001).

[47] Petru Liuba et al., Residual Adverse Changes in Arterial Endothelial Function and LDL Oxidation After a Mild Systemic Inflammation Induced by Influenza Vaccination, 39 Ann. Med. 392 (2007).

this study was limited to cardiovascular disease; these observations were not described in the context of the inner ear, cochlear blood flow, or hearing loss. Further, no injury was described in association with slight elevations in these proteins.

In summary, relative to steps one and two of his mechanism, Dr. Monsell has not shown that the flu vaccine causes a significant increase in fibrinogen, TNF, NFKB, or other reactants, or that these reactants cause adverse effects, specifically reduced blood flow to the inner ear, vascular damage to the blood vessels in the inner ear, or breakdown of the cochlear blood-tissue barrier.

III. Reduction of cochlear blood flow results in cochlear ischemia.

The third step of Dr. Monsell's mechanism is that reduction of cochlear blood flow results in cochlear ischemia. Pet. Ex. 19 at 17. He described how reduction of cochlear blood flow causes vascular damage (including breakdown of the cochlear blood-tissue barrier) and cochlear ischemia. Id. at 16-17.

Dr. Monsell, however, has not shown that the flu vaccine reduces blood flow in the inner ear, causes vascular damage to the blood vessels in the inner ear, or leads to breakdown of the cochlear blood-tissue barrier. Therefore, there is no foundational evidence underlying this step of his proposed mechanism.

Further, the medical literature cited by Dr. Monsell does not show that the flu vaccine reduces cochlear blood flow or causes cochlear ischemia. For example, Brown et al.[48] conducted an animal study showing that transient asphyxia[49] caused by "interrupting respiration for brief periods" caused reversible changes in all measured cochlear potentials. Pet. Ex. 30 at 1, 3. The relevance of this study is not clear, as there is no evidence here that the flu vaccine induced interrupted respiration or caused oxygen deprivation. Nor is there any suggestion that Petitioner's presenting signs and symptoms included shortness of breath or respiratory compromise.

Dr. Monsell cited several other articles in support of this step. Upon review, these papers also do not support the premise that the flu vaccination reduces blood flow or otherwise causes or contributes to cochlear ischemia. Onal et al.[50] explained that the cochlea is "very sensitive to alterations in blood circulation, and transient ischemia of the cochlea may result in . . . noise-induced [hearing loss] and [SSNHL]." Pet. Ex. 62 at 1. The authors discussed the concepts of

[48] M. Christian Brown et al., Cochlear Inner Hair Cells: Effects of Transient Asphyxia on Intracellular Potentials, 9 Hear. Rsch. 131 (1983).

[49] Asphyxia refers to the "pathologic changes caused by lack of oxygen." Asphyxia, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4494 (last visited Sept. 9, 2025).

[50] Merih Onal et al., Ozone Prevents Cochlear Damage from Ischemia-Reperfusion Injury in Guinea Pigs, 41 Artif. Organs 744 (2007).

oxidative stress and ischemia and reperfusion injury and their role in hearing loss; however, they did not identify the flu vaccination as a trigger for transient ischemia of the cochlea.

Randolf et al.[51] described how repeated compression of local blood flow to animal cochlea induces "transient cochlear hypoxia." Pet. Ex. 71 at 1. Compression of the blood flow caused vascular spasm and a permanent decrease in blood flow. Id. at 2. Again, this study describes facts and circumstances not present in this case.

Nakashima et al.[52] provided an overview of disorders of cochlear blood flow, noting that while there have been reports of hearing loss thought to be caused by disturbances of blood flow in the cochlea, "direct evidence . . . is still lacking in most of the cases." Pet. Ex. 60 at 1. Of note, the authors explained that noise exposure has been associated with impaired cochlear blood flow. Id. at 4. The authors identified many different causes or contributors to hearing loss in addition to noise exposure, including endolymphatic hydrops,[53] changes that occur with aging, and nitric oxide, low blood flow, cochlear hemorrhage, tumor, blood flow disturbances caused by inner ear diseases such as Meniere's disease, and abnormalities of the posterior communicating arteries of the circle of Willis. Id. at 4-8. Specific to SSNHL, the authors noted that hearing loss has been associated with "increased fluid pressure in the inner ear," which is "considered to be associated with reduced cochlear blood flow." Id. at 7. Hearing loss has also been associated with "acute reduction of cerebrospinal fluid pressure," diseases that provoke infarcts (i.e., Susac syndrome, Takayasu's disease, polyarteritis, etc.), blood disorders (leukemia, sickle cell anemia, and polycythemia), and microembolism (reported after cardiopulmonary bypass surgery). Id. at 7-8. The authors did not report an association between vaccination and disturbances of cochlear blood flow leading to hearing loss.

Dr. Monsell further wrote that "[a]nother aspect of the mechanism, especially for severe cases, is likely to be the involvement of the clotting mechanism in the setting of damage to the cochlear microvascular endothelium." Pet. Ex. 19 at 18. For support, he cited Esmon[54] and Neng et al. Id. (citing Pet. Exs. 40, 61). However, Esmon does not discuss ischemia in relation to the cochlea. See Pet. Ex. 40. Nor does Esmon discuss vaccination. See id. And although Neng et al. discusses the cochlear blood-tissue barrier, the authors' findings do not relate to

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[51] H.-B. Randolf et al., Cochlear Blood Flor Following Temporary Occlusion of the Cerebellar Arteries, 247 Eur. Arch. Otorhinolaryngol. 226 (1990).

[52] Tsutomu Nakashima et al., Disorders of Cochlear Blood Flow, 43 Brain Rsch. Revs. 17 (2003).

[53] "Endolymphatic hydrops is a condition in which too much endolymph is present." Pet. Ex. 60 at 5. Endolymphatic hydrops is also referred to as Meniere's disease, which is "hearing loss, tinnitus, and vertigo resulting from nonsuppurative disease of the labyrinth with edema." Meniere Disease, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=70588 (last visited Sept. 9, 2025).

[54] Charles T. Esmon, Reprint of Crosstalk Between Inflammation and Thrombosis, 61 Maturitas 122 (2008).

vaccination. See Pet. Ex. 61. Therefore, no foundational evidence underlying this step of Dr. Monsell's proposed mechanism was provided.

IV. Cochlear ischemia results in an injury/reperfusion situation and generation of ROS, creating oxidative stress in cochlear hair cells.

While the statement above may be supported by medical literature cited by Dr. Monsell, the articles cited by Dr. Monsell related to this step of his theory fail to show an association between vaccination and cochlear ischemia. And the literature cited by Dr. Monsell in support of this step does not bridge the causal gap between vaccination and cochlear ischemia.

Merchant et al.[55] discussed the pathology and pathophysiology of idiopathic SSNHL. Pet. Ex. 55. At the outset of their paper, they stated that the "cause and pathogenesis" of this type of hearing loss is unknown. Id. at 1. The authors described temporal bone histopathology from 17 subjects and found "the most common abnormality . . . [was] atrophy of hair cells and supporting cells of the organ of Corti,[56] with variable involvement of other structures." Id. at 8. The data did not support vascular occlusion or viral infection as a common etiology. Id. An alternative and novel hypothesis was proposed by the authors: an "abnormal activation of cellular stress pathway involving [NFKB] within the cochlea." Id. More specifically, they posited that activation of the NFKB pathway "can result in production of inflammatory cytokines and other stress-related proteins that can disrupt the homeostatic balance of a cell or tissue." Id. The authors issued a caveat, however, noting that while this idea was consistent with the histopathological findings, there was no "direct proof in support of [their] theory." Id. at 9.

Relative to NFKB, Dr. Monsell cited studies that he asserted show the flu vaccine could activate the NFKB system in the inner ear. Pet. Ex. 19 at 17. However, the studies cited by Dr. Monsell related to flu infections, not the flu vaccination.

For example, Pahl and Baeuerle showed that expression of "the [flu] virus hemagglutinin (HA)[] activates NF-κB DNA binding and transcriptional activation." Pet. Ex. 65 at 1. They explained that "NF-κB induces transcription of a variety of cytokines released during [flu] virus infection," and thus, activation of NFκB "represents one mechanism by which [flu] virus infection increases cytokine transcription." Id. at 4. The authors suggested several examples in which this information could be clinically relevant, but the examples involve viral infections, such as viral pneumonia. Id. The authors did not discuss vaccination. Dr. Monsell did not provide any literature showing the flu vaccination, not flu infection, can stimulate the NFKB pathway.

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[55] Saumil N. Merchant et al., Pathology and Pathophysiology of Idiopathic Sudden Sensorineural Hearing Loss, 26 Otol. & Neurotol. 151 (2005).

[56] The organ of Corti "rest[s] on the basilar membrane of the cochlear duct[] [and] contains the auditory hair cells, special sensory receptors for hearing, as well as several types of supporting cells. Organum Spirale, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=94965 (last visited Sept. 9, 2025).

V. Cochlear hair cells and ROS-capture mechanisms struggle with oxidative stress. Some hair cells may survive if the degree of injury is mild, in others, the regulatory systems (including NFKB) sense severe cellular damage. To mitigate widespread tissue destruction, caspase pathways are activated.

For support of this step, Dr. Monsell cited to the "injury/reperfusion model" by Yang et al.,[57] which "show[ed] an important link between ischemia and inflammation in the cochlea." Pet. Ex. 19 at 18 (citing Pet. Ex. 89). Yang et al. (2021) examined the role of cochlear spiral ganglion progenitor cell-derived exosomes ("CSGPC-exosomes") and determined "CSGPC-exos[omes] could protect cochleae damage from [ischemia-reperfusion injury], probably via inhibiting the inflammatory process." Pet. Ex. 89 at 1. The authors explained "[ischemia-reperfusion injury] can generate [ROS]-induced proinflammatory environment" and "the administration of CSGPC-exosomes could reduce the amount and apoptosis[58] of hair cells by down-regulating the proinflammatory mediators." Id. at 6-7. However, the foundational evidence in support of Dr. Monsell's previous steps of his mechanism is lacking. Further, Yang et al. does not discuss their study in terms of vaccination, instead focusing on ways to "reduce . . . apoptosis of hair cells by down-regulating proinflammatory mediators" via the administration of CSGPC-exosomes. Id. at 6-8.

In an earlier study from Yang et al. (2017),[59] the authors studied the effects of temporary occlusion of the carotid artery for 30 or 60 minutes on auditory brainstem responses in mice. Pet. Ex. 88 at 1. Significant hearing loss occurred after occlusions in both groups, although "the longer the ischemia lasted, the worse the resultant hearing impairment." Id. at 6. The authors concluded that autophagy[60] played a protective role in "cell survival in the cochlea," although "excessive [ischemia/reperfusion] damage overwhelms the beneficial" effects and can lead to cell death. Id. at 7. Again, the facts are not comparable to those presented here as there was no evidence the vaccine caused a temporary occlusion of the carotid artery.

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[57] Tao Yang et al., Exosomes Derived from Cochlear Spiral Ganglion Progenitor Cells Prevent Cochlea Damages from Ischemia-Reperfusion Injury Via Inhibiting the Inflammatory Process, 386 Cell & Tissue Rsch. 239 (2021).

[58] Apoptosis is "a morphologic pattern of cell death affecting single cells, marked by shrinkage of the cell . . . and fragmentation of the cell into membrane-bound apoptotic bodies that are eliminated by phagocytosis." Apoptosis, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=5011 (last visited Sept. 9, 2025).

[59] Haidi Yang et al., The Protective Effect of Autophagy on Ischemia/Reperfusion-Induced Hearing Loss: Implications for Sudden Hearing Loss, 28 NeuroRep. 1157 (2017).

[60] Autophagy is "the segregation and digestion of part of the cell's own cytoplasmic material within lysosomes." Autophagy, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=5011 (last visited Sept. 9, 2025).

Next, Dr. Monsell opined that activation of NFKB induces the production of cytokines and "a defensive cellular inflammatory response that may, if severe, include activation of the apoptotic cascade (programed cell death) and SSNHL," as described by Merchant et al. and Adams.[61] Pet. Ex. 19 at 18 (citing Pet. Exs. 21, 55). As previously described, Merchant et al. involved histopathology study of temporal bones to better understand the cause of hearing loss. Pet. Ex. 55 at 1. Merchant et al. also stated that the effects of NFKB activation include apoptosis as well as "protection from apoptosis," although the "nature of the response depends on the molecular mediators that trigger NFκB activation and the context in which the activation occurs." Id. at 8. This hypothesis was described as the "stress response hypothesis." Id. Vaccination was not discussed.

Adams discussed the role of NFKB in cochlear cells and the possibility that NFKB could play a role in "[SSNHL] associated with otosclerosis, otitis media, and central nervous system inflammation[] . . . by disrupt[ing] [] the normal balance of inflammatory cytokines within the spiral ligament." Pet. Ex. 21 at 1. But they did not discuss this possible mechanism in the context of vaccination.

The last article cited by Dr. Monsell in support of this step of his proposed mechanism was by Cabrera et al.,[62] which described how the NFKB1 gene may influence prognosis in patients with Meniere's disease. Pet. Ex. 32 at 1. The authors described two variants in the NFKB1 gene associated with rapid hearing loss progression in patients who have unilateral SNHL. Id. Immune-mediated inner ear disease and its pathogenesis was also discussed. Id. at 1-2. Dr. Monsell stated that the findings in Cabrera et al. illustrated how the genetic variants described could be affected by inflammatory challenges such as vaccination. Pet. Ex. 19 at 18. However, vaccination was not discussed by Cabrera et al. Moreover, an underlying genetic variant has not been identified as relevant here.

[61] Joe C. Adams, Clinical Implications of Inflammatory Cytokines in the Cochlea: A Technical Note, 23 Otol. & Neurotol. 316 (2002). In another article from Adams et al., systemic inflammatory stress was induced by injection of endotoxin and acoustic trauma was induced by intense noise exposure and resulted in NFKB activation. Pet. Ex. 22 at 1-2 (J.C. Adams et al., Selective Activation of Nuclear Factor Kappa B in the Cochlea by Sensory and Inflammatory Stress, 160 Neurosci. 530 (2009)). The authors stated, "[c]learly systemic inflammation[] do[es] not routinely cause unilateral hearing loss" and suggested that such hearing loss would require a secondary stress, resulting in compounding effects. Id. at 8. Vaccination was not discussed.

[62] Sonia Cabrera et al., Intronic Variants in the *NFKB1* Gene May Influence Hearing Forecast in Patients with Unilateral Sensorineural Hearing Loss in Meniere's Disease, 9 PLoS One e112171 (2014).

VI. Caspase pathways activate the mechanisms of apoptosis, resulting in programmed death of hair cells and irreversible SSNHL. If the damage is severe enough, cell death by necrosis may occur.

Dr. Monsell described apoptosis and necrosis as it relates to severe hearing loss. Pet. Ex. 19 at 18-19. In Van De Water et al.,[63] the authors described caspases and their role in "programmed cell death" of inner ear sensory cells. Pet. Ex. 82 at 2. Caspases are defined as a "family of cysteine proteases[64] . . . present within the cells of normal healthy tissue in inactive (procaspase) forms." Id. at 3. Some members of caspases are involved in "programmed cell death (apoptosis) of cells," while others are involved in the "regulation and execution of apoptosis of affected cells," which occurs as part of normal development or due to "a cell's response to a high level of internal injury (e.g. membrane lipid peroxidation) that results from exposure to oxidative stress." Id. The authors described the pathway of cell death that arises out of an "insult that generates oxidative stress with the creation of [ROS] and other free radicals [] that damage the cell's organelles and internal membranes resulting in mitochondrial membrane damage and loss of membrane potential." Id. at 4. While programmed cell death is a normal part of development of the inner ear and normal hearing, caspases may also be involved in "maldevelopment . . . and a hearing deficit." Id. at 6. Caspases also play a role in cell death in cases of ototoxicity, as demonstrated by gentamicin[65]-induced injury to vestibular hair cells resulting in hearing loss. Id. at 6-7. The authors also described how acoustic trauma could cause hair cell loss with cell death and activation of caspase. Id. at 9. Van De Water et al. did not describe cell death caused by vaccination or suggest that vaccination-induced activation of a caspase pathway results in death of hair cells or hearing loss.

In support of this step, Dr. Monsell again cited Merchant et al., specifically Case 2, which was a 57-year-old male with pre-existing Meniere's disease in the left ear who had sudden hearing loss in the right ear after having a head cold for three days. Pet. Ex. 19 at 20 (citing Pet. Ex. 55 at 5-6). The patient was hospitalized for anticoagulant treatment and subsequently had sudden left leg pain with loss of pulses. Pet. Ex. 55 at 6. Surgery was performed to remove clots, but his condition deteriorated, and he died six days later due to coronary artery occlusion. Id. A postmortem examination of his temporal bones showed "marked swelling with edema and

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[63] TR Van De Water et al., Caspases, the Enemy Within, Their Role in Oxidative Stress-Induced Apoptosis of Inner Ear Sensory Cells, 25 Otol. & Neurotol. 627 (2004).

[64] Cysteine protease (endopeptidase) is "any of a group of endopeptidases containing at the active site a cysteine residue involved in catalysis; the group includes papain and several cathepsins." Cysteine Endopeptidase, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=12267 (last visited Sept. 9, 2025).

[65] Gentamicin is "an aminoglycoside antibiotic complex" that "is effective against a wide range of aerobic gram-negative bacilli . . . and some gram-positive bacteria." Gentamicin, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=20074 (last visited Sept. 9, 2025). Gentamicin is known to be ototoxic and to cause hearing loss. Ototoxicity, Clev. Clinic, https://my.clevelandclinic.org/health/diseases/24769-ototoxicity (last reviewed Feb. 22, 2023).

vacuole formation within the cytoplasm and blurring of cell boundaries." Pet. Ex. 19 at 20 (quoting Pet. Ex. 55 at 6). Although the patient's history suggested a vascular cause of hearing loss, the histopathology did not support the etiology of vascular occlusion. Pet. Ex. 55 at 6-7. Instead, it showed "unusual and severe swelling of hair cells" as well as "swollen and shrunken areas of the organ of Corti," which was interpreted "as evidence that these cells were under severe osmotic stress." Id. at 7. The authors opined these findings were consistent with a "stress-induced" hypothesis. Id. They stated that "if such stress were irreversible, these cells would undergo apoptosis or necrosis, resulting in atrophy of the organ of Corti, which is the abnormality observed in the majority of cases of sudden deafness that do not recover." Id. Again, vaccination was not discussed.

VII. Microthrombosis in the cochlear capillaries and other events may exacerbate the injury process or impair recovery.

In describing the last step of his mechanism, Dr. Monsell offered a list of things that could worsen the "injury process or impair recovery" in the setting of inflammation: (1) upregulation of adhesion molecules on endothelial surfaces; (2) increased generation of oxygen species facilitated by deficient bioactivity of endothelial nitric oxide; (3) accelerated oxidative modification of low-density lipoprotein; (4) creation of micro-atherosclerotic emboli; and (5) activation of thrombosis in the microcirculation of the cochlea. Pet. Ex. 19 at 15, 19. For support, Dr. Monsell cited four articles.

Liuba et al. used the flu vaccine to simulate mild systemic inflammation to study the effect on vascular endothelium. Pet. Ex. 50 at 1. They reported that abnormalities "possibly involving decreased [nitric oxide] and increased intravascular oxidative burden" could occur and that these events "might be important" in playing a role in arterial disease. Id. at 7. Additional studies were recommended to "verify this hypothesis." Id. The vascular endothelium and vascular system of the inner ear were not studied or discussed.

Albertini et al.[66] discussed the role of lipoprotein oxidation in the development of atherosclerosis. Pet. Ex. 23. The article does not discuss vaccination or the inner ear. Esmon described the interplay between inflammation and thrombosis. Pet. Ex. 40. Esmon also does not discuss vaccination or the inner ear. Yang et al. described research about "stem cell[] related regenerative therapies to prevent degeneration and damage of hair cells and spiral ganglion neuron." Pet. Ex. 89 at 6. While the article mentions ROS and the proinflammatory environment it induces through activation of caspase-1 and proinflammatory cytokines, the context is not vaccination.

2. Additional Supporting Opinions and Medical Literature

After identifying his seven-part mechanism and citing medical literature, Dr. Monsell offered additional opinions explaining aspects of his proffered mechanism. Although there are

[66] R. Albertini et al., Oxidation of Low-Density Lipoprotein in Atherosclerosis from Basic Biochemistry to Clinical Studies, 2 Current Molecular Med. 579 (2002).

multiple mechanisms implicated in SSNHL, "SSNHL is rare in association with vaccination." Pet. Ex. 19 at 20. He explained that each step of his mechanism is "subject to individual differences in genetic predisposition, microvascular anatomy, and risk factors." Id. Dr. Monsell added that comorbidities, including hypertension, cardiovascular disease, obesity, and other factors such as aging, can "increas[e] susceptibility to vascular injury" as well as prognosis. Id. This individuality extends to "[i]ndividual immune responses and toxicity to vaccination," which "can vary significantly, including responses to [the flu] vaccination." Id. This range in individual responses is thought to be due to genetic differences. Id.

Dr. Monsell maintained SSNHL is "relatively rare, making it difficult to accumulate large numbers of patients for investigations and clinical trials." Pet. Ex. 19 at 13. He noted difficulty selecting appropriate control groups as well as ethical constraints with human subjects. Id. He acknowledged his proposed mechanism is "conceptual and general in many respects" and maintained that he provided "a reasonable and plausible mechanism whereby [flu] vaccination can cause SSNHL and could have in the case at hand." Id. at 14.

Dr. Monsell asserted inflammation has a critical role in the mechanism leading to SSNHL due to clinical response to anti-inflammatory steroid treatment. Pet. Ex. 19 at 21. He explained that "[c]orticosteroids may act by reducing inflammatory aspects of cell trauma caused by cell death and other potential intra-cochlear inflammatory pathways activated by ischemic injury." Id.

Lastly, Dr. Monsell discussed epidemiology studies. Pet. Ex. 19 at 22. He noted that Baxter et al., a study of more than eight million trivalent flu vaccinations, did not find an association between flu vaccinations and SSNHL. Id. (citing Pet. Ex. 27 at 1). According to Dr. Monsell, if a study involved "a larger population and sufficient power," an association might be found. Id.; see also Pet. Ex. 91 at 4. He opined that "[w]ithout a massive, controlled clinical trial," it is "not [] possible" to determine the accurate incidence of SSNHL after flu vaccination. Pet. Ex. 19 at 22. Reliance on Baxter et al., however, to "reject the possibility of occasional rare cases of SSNHL triggered by [flu] vaccination . . . would not be rational." Id.

He also cited a case report of SSNHL two days after flu vaccination.[67] Pet. Ex. 19 at 22 (citing Pet. Ex. 48).[68] Kolarov et al. described the case of a 79-year-old woman who developed bilateral SSNHL and vertigo two days after flu vaccination. Pet. Ex. 48 at 1. The patient had a significant medical history that included insulin-dependent diabetes, atrial fibrillation, and past thalamic stroke, but no recent infection. Id. at 1-2. Kolarov et al. also described a 17-year-old

[67] Dr. Monsell stated that the article by Alsanosi reported two cases of SSNHL after H1N1 vaccination. Pet. Ex. 19 at 22 (citing Pet. Ex. 24 (A.A. Alsanosi, Influenza A (H1N1): A Rare Cause of Deafness in Two Children, 126 J. Laryngol. & Otol. 1274 (2012))). However, Alsanosi described two reports in children after H1N1 infections, not vaccinations. Pet. Ex. 24 at 1. The first child had bronchopneumonia as a complication of her infection, and the second child had a high fever; both had bilateral hearing loss. Id. at 1-2.

[68] Claudia Kolarov et al., Bilateral Deafness Two Days Following Influenza Vaccination: A Case Report, 15 Hum. Vaccines & Immunother. 107 (2019).

girl with bilateral hearing loss, dizziness, and tinnitus 14 hours after receipt of an H1N1 vaccination. Id. at 2. The authors opined that an undetected infection or disease may have caused her hearing loss, or that it was speculatively related to her flu vaccination. Id.

ii. Althen Prong Two

Dr. Monsell opined that "more likely than not," Petitioner's flu vaccination had a "substantial causal role" in his development of left-sided SSNHL due to "a combination of complex factors, primarily idiosyncratic inflammatory and microvascular events resulting in ischemia and reperfusion injury, which were triggered by his [flu] vaccination." Pet. Ex. 19 at 8, 23. And but for this vaccination, Petitioner would not have developed SSNHL. Id. at 23.

Dr. Monsell opined Petitioner had "a strong treatment response" which "confirm[s] the role of inflammation in his case," despite the response being insufficient to restore functional hearing. Pet. Ex. 19 at 5, 23. Petitioner's "hearing loss was at the profound level on presentation and continued to be severe." Id. at 21. Petitioner was treated with oral and intratympanic corticosteroids, and subsequent hearing examinations (audiograms) showed "substantial improvement." Id. Dr. Monsell opined this "substantial improvement" after treatment with corticosteroids "is substantial evidence for inflammation as a critical component of the mechanism operating in [Petitioner's] case." Id.

Petitioner had numerous prior flu vaccinations. See Pet. Ex. 16 at 1-3. Dr. Monsell asserted that "prior [flu] vaccinations should not be used as a point to argue in favor or against the proposed mechanism." Pet. Ex. 19 at 21. He explained every vaccine is different and the human body is always changing, thus "susceptibility to SSNHL is not fixed." Id.

Dr. Monsell opined that alternative factors were not the cause of Petitioner's SSNHL. Pet. Ex. 19 at 8-13, 23. First, he ruled out age-related and noise-induced hearing loss as both typically occur gradually and cause bilateral hearing loss, while Petitioner's SSNHL was sudden and unilateral. Id. at 8-9. Additionally, Petitioner was not on ototoxic medication that could have explained his hearing loss. Id. at 9. Dr. Monsell also noted Petitioner was not diagnosed with Meniere's disease, nor did his clinical picture match that of a patient with Meniere's disease. Id. at 9; Pet. Ex. 91 at 1. Thus, he excluded Meniere's disease as a diagnosis for or cause of Petitioner's SSNHL. Pet. Ex. 19 at 9; Pet. Ex. 91 at 1.

Dr. Westbrook suspected Petitioner's hearing loss was related to a vestibular neuronitis; however, Dr. Monsell asserted this does not establish a diagnosis of vestibular neuritis. Pet. Ex. 19 at 9-10 (citing Pet. Ex. 2 at 20). He opined "[t]he diagnosis of vestibular neuronitis or neuritis can be discarded in [Petitioner's] case" because vestibular neuritis does not involve loss of hearing. Id. at 10; see also Pet. Ex. 91 at 1. According to Dr. Monsell, "[t]he only way that vestibular neuritis could account for [Petitioner's] clinical presentation and course would be if he had both SSNHL and vestibular neuritis unilaterally and simultaneously in the same ear, giving him [two] diseases when one disease is sufficient to explain his symptoms." Pet. Ex. 91 at 1-2.

Additionally, Petitioner's MRI documented "no evidence of acoustic schwannoma or other [internal auditory canal] lesion," excluding these as a cause of Petitioner's hearing loss.

Pet. Ex. 19 at 10 (citing Pet. Ex. 3 at 56). The MRI also excluded the possibility of acute brainstem, cerebellar, or vertebrobasilar stroke. Id. Dr. Rubinstein disagreed an acoustic neuroma was excluded because the MRI was not done with contrast. Pet. Ex. 91 at 2 (citing Resp. Ex. A at 4). In response, Dr. Monsell asserted Petitioner's treating physicians presumably thought the evidence was sufficient enough to rule out an acoustic neuroma, and "we must use the best evidence we have." Id.

Dr. Monsell next discussed metabolic syndrome, hypertension, and thromboembolic stroke. Pet. Ex. 19 at 10-11. He noted hypertension, diabetes, dyslipidemia, obesity, and smoking have been found to be associated with stroke and SSNHL. Id. at 10. Although Petitioner had some risk factors,[69] Dr. Monsell opined Petitioner's clinical presentation and MRI did not show signs consistent with a stroke. Id. at 11. And because Petitioner "had an incomplete loss of hearing[,] [] acute vascular occlusion and a stroke to the brainstem [could be] excluded as alternative causes in [Petitioner's] case." Id.

Dr. Monsell did not assert Petitioner had autoimmune inner ear disease ("AIED"). Pet. Ex. 16 at 12. He explained AIED occurs when "there is a breakdown in the regulation of the complex processes of the body's immune system such that it makes a mistake, attacking a normal organ or antigen molecule in the body as though it were a foreign invader." Id. Petitioner's presentation (sudden, unilateral, and profound from onset), according to Dr. Monsell, "does not correspond to clinical findings in AIED," and thus, this diagnosis can be excluded. Id. Nevertheless, Dr. Monsell asserted "AIED is not unrelated to [Petitioner's] case" since AIED "establishes that a disease process that is based on dysfunction of the immune system—but without infection—can cause SNHL." Id. (emphasis omitted).

Lastly, Dr. Monsell opined Petitioner "had no signs or symptoms of any viral illness, measles, mumps, Ramsay Hunt, or any other virus" that could explain his hearing loss. Pet. Ex. 19 at 12-13. Additionally, "there is no credible evidence that ordinary cold viruses, runny noses, sore throats, or sneezing can cause SSNHL." Id. Further, Petitioner's MRI was normal and did not show enhancement of the applicable cranial nerve which occurs with viral-related inflammation of the inner ear. Id. However, Petitioner's MRI was not done with contrast. See Pet. Ex. 3 at 56.

In his second expert report, Dr. Monsell opined that "Dr. Rubinstein asserted a hypothesis of viral infection but did not propose a specific virus to have caused [Petitioner's] SSNHL." Pet. Ex. 91 at 2. Dr. Monsell did not disagree that SNHL can occur during and following systemic infections. Id. at 3. But he maintained there was no evidence of an infection in Petitioner, and "histopathologic studies do not support asymptomatic viral infection of the inner ear as a cause of SSNHL." Id.

[69] Dr. Monsell noted Petitioner had diagnoses of hypertensive vascular disease and hypertensive heart disease. Pet. Ex. 19 at 10 (citing Pet. Ex. 3 at 4). According to his body mass index, he was obese. Id. at 10-11 (citing Pet. Ex. 3 at 116). And, less than one month prior to vaccination, on August 21, 2019, Petitioner had abnormally low high-density lipoprotein ("HDL") and abnormally high low-density lipoprotein ("LDL"). Id. at 11 (citing Pet. Ex. 3 at 10).

Dr. Monsell also disagreed with Dr. Rubinstein that SSNHL post-vaccination would always be bilateral, not unilateral, due to the systemic effects of vaccination. Pet. Ex. 91 at 3-4. For support, he noted systemic infections have affected only one ear and animal studies have also been unilateral/asymmetric. Id. at 3. Thus, he concluded "unilaterality of SSNHL does not rule out that it is vaccine-induced when that is the primary consideration." Id. at 4.

iii. Althen Prong Three

Petitioner received the flu vaccination at issue on September 15, 2019, and six days later, he experienced sudden hearing loss in his left ear, later diagnosed as SSNHL. Pet. Ex. 19 at 21, 23. Dr. Monsell opined this six-day onset post-vaccination is consistent with his proposed mechanism as it is "well within the period of maximal activity of the [APR] to his vaccination." Id. at 20-21, 23 ("Sudden onset suggests some form of vascular mechanism as with the proposed mechanism . . . .").

He further noted that the 1994 Institute of Medicine ("IOM") report[70] accepted the "timing of an immune-mediated response following vaccination up to 42 days after vaccination." Pet. Ex. 19 at 23 (citing Pet. Ex. 78 at 3). For further support, he cited Baxter et al., who "reported 95 cases of SSNHL 1-14 days following vaccination." Id. (citing Pet. Ex. 27). He acknowledged Baxter et al. did not detect any association between SSNHL and flu vaccination, but maintained that "[a] close examination of the data . . . shows a trend toward association between [flu] vaccination and SSNHL in the 1-14 day interval." Id. at 22.

Dr. Monsell concluded that his theory "is consistent with the medically accepted timeframe for vaccine-related adverse events." Pet. Ex. 19 at 23.

2. Respondent's Expert, Dr. Jay T. Rubinstein[71]

a. Background and Qualifications

The parties stipulated that Dr. Rubinstein is an expert in otolaryngology. Joint Submission at 1. He is board certified in Otolaryngology–Head and Neck Surgery and Neurotology. Resp. Ex. B at 2. Dr. Rubinstein obtained his M.D. and Ph.D. in Bioengineering from the University of Washington after which he completed a surgery internship at Beth Israel Hospital and otolaryngology residency at Massachusetts Eye & Ear Infirmary in Boston. Id. at 1. He has also completed three fellowships, one of which was a research fellowship at the Department of Otology and Laryngology at Harvard Medical School and another that was a clinical fellowship in Otology/Neurotology at the Department of Otolaryngology at The University of Iowa Hospitals and Clinics. Id. Since 1995, Dr. Rubinstein has held academic appointments in the Department of Otolaryngology at The University of Iowa Hospitals and Clinics and the University of Washington. Id. He currently works as the Virginia Merrill

[70] Inst. of Med., Adverse Events Associated with Childhood Vaccines: Evidence Bearing on Causality (Kathleen Stratton et al. eds., 1994).

[71] Dr. Rubinstein provided two expert reports. Resp. Exs. A, C.

Bloedel Professor and Director of the Virginia Merrill Bloedel Hearing Research Center at the University of Washington. Id.; Resp. Ex. A at 1. Dr. Rubinstein spends half of his time on clinical activities, treating both children and adults, and the other half of his time is "dedicated to research, administration, and teaching, all in the domain of hearing and vestibular disorders." Resp. Ex. A at 1-2; see also Resp. Ex. B at 33 (noting areas of research include "[f]unctional electrical stimulation of the inner ear;" "[t]reatment of hearing loss, tinnitus[,] and vestibular dysfunction;" and "[h]igh performance computing for neural modeling"). He has served as the President of the Association for Research in Otolaryngology, the American Auditory Society, and the President of the Politzer Society, an international otologic research organization. Resp. Ex. A at 2. Dr. Rubinstein has authored or co-authored over 150 publications throughout his career. Resp. Ex. B at 5-21.

b. Opinion

i. Althen Prong One

Dr. Rubinstein opined Dr. Monsell failed to show an association between vaccination and unilateral sudden hearing loss. Resp. Ex. A at 5-6. He explained that Dr. Monsell's proposed mechanisms involve systemic inflammatory/immune responses, which affect the entire body, and it is "very difficult to postulate a plausible mechanism by which [] a [systemic] response can only cause damage to one ear." Id. at 5. "The theories proposed by Dr Monsell would, by definition, be associated with bilateral hearing loss as they are systemic processes rather than a localized infection [which is] the most accepted mechanism of [idiopathic SSNHL]." Id. at 6. Although viral infections of the inner ear are thought to be the "leading" cause of SSNHL, the "precise pathway(s) viruses gain access to the inner ear to cause [idiopathic SSNHL] . . . are still unknown." Id. at 4-5.

Dr. Rubinstein addressed the epidemiologic studies relied upon by Dr. Monsell and noted the studies have not shown an association between vaccination and SSNHL. Resp. Ex. A at 5. Baxter et al., for example, included over eight million recipients of a flu vaccine and found no statistically significant increase in SSNHL. Id. (citing Pet. Ex. 27). Dr. Monsell argued Baxter et al. is underpowered, but Dr. Rubinstein disagreed. Id. According to Dr. Rubinstein, Dr. Monsell's bases his opinion "on the flawed assumption that [idiopathic SSNHL] is a rare disease," which it is not. Id. (citing Pet. Ex. 37 at 1 (indicating an incidence rate of 5-27 per 100,000 population annually)) (noting his clinic sees approximately one case of idiopathic SSNHL per week). And a study on eight million flu vaccinations "is indeed high powered[] and peer reviewed." Resp. Ex. C at 2.

He added that studies cited by Dr. Monsell are not applicable to this case. Resp. Ex. A at 5-6. For example, Dr. Monsell cited studies that support autoimmune/inflammatory cause of hearing loss when both ears are involved, which is not the case here. Resp. Ex. A at 5-6 (citing,

e.g., Pet. Ex. 53;[72] Pet. Ex. 74).[73] And case reports of sudden hearing loss following vaccination and flu infection show bilateral involvement of the ears. Id. at 6 (citing, e.g., Pet. Ex. 24; Pet. Ex. 45;[74] Pet. Ex. 48).

Lastly, Merchant et al. does not explain how a systemic process can lead to unilateral hearing loss. Resp. Ex. A at 6 (citing Pet. Ex. 55). In fact, two of the three patients in Merchant et al. had bilateral hearing loss which does not support a relationship between vaccination and unilateral hearing loss. Id.

ii. Althen Prong Two

Dr. Rubinstein opined Petitioner has a "classic case" of idiopathic SSNHL "accompanied by vestibular loss." Resp. Ex. A at 3.

Dr. Rubinstein agreed with Dr. Monsell that Petitioner does not have noise-induced or age-related hearing loss. Resp. Ex. A at 3. He also agreed that there was no evidence for ototoxic injury, metabolic syndrome, hypertension, thromboembolic stroke, or AIED. Id. at 4.

Dr. Rubinstein "mostly agree[d]" that Meniere's disease was "not likely" Petitioner's diagnosis as his "presentation was inconsistent with early Meniere's disease." Resp. Ex. A at 3. Although Petitioner did not meet the criteria for Meniere's disease, Dr. Rubinstein explained that it cannot be ruled out because it is "very common" for patients with SSNHL to later develop Meniere's disease. Resp. Ex. C at 1.

Although he "agree[d] with Dr Monsell that there was no evidence of stroke either on the MRI or in the clinical presentation," he "strongly disagree[d] that an acoustic neuroma [was] excluded" because Petitioner's MRI was not done with contrast. Resp. Ex. A at 4. He explained that the use of contrast "is the standard of care for identifying [] acoustic neuromas." Id. "It can be impossible to detect such lesions without contrast," although he acknowledged "tumors are occasionally missed even with contrast." Id. He maintained that "[w]ithout a contrasted scan, it is far more likely that an undiagnosed tumor is present than that a vaccine injury occurred." Resp. Ex. C at 1; see also Resp. Ex. A at 6 ("[T]here is still a reasonable probability that [Petitioner] has a small, undiagnosed vestibular schwannoma that caused his hearing loss and vertigo.").

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[72] Akihiro J. Matsuoka & Jeffrey P. Harris, Autoimmune Inner Ear Disease: A Retrospective Review of Forty-Seven Patients, 18 Audiol. & Neurotol. 228 (2013). This study focused on AIED, which the parties agree Petitioner does not have.

[73] Hitomi Sakano & Jeffrey P. Harris, Emerging Options in Immune-Mediated Hearing Loss, 4 Laryngoscope Investig. Otolaryngol. 102 (2019). This study also focused on AIED, not SSNHL.

[74] Tim V. Hulbert et al., Bilateral Hearing Loss After Measles and Rubella Vaccination in an Adult, 325 New Eng. J. Med. 134 (1991).

Because viral infections of the inner ear are the "leading hypothesis" for causation in idiopathic SSNHL, Dr. Rubinstein could not rule out infections as causative. Resp. Ex. A at 4-5. He acknowledged that the "causative viruses are still unknown" in SSNHL. Id. at 5. Yet he opined "viral infection . . . is almost certainly the cause here." Id. He agreed with Dr. Monsell that there was no evidence of mumps, measles, rubella or "obvious herpetic viral infection;" however, he could not rule out "unobvious herpetic or other common and potentially asymptomatic viral infections." Id. at 4. In his second expert report, he clarified that he "[did] not propose a particular virus as the cause in this case." Resp. Ex. C at 2.

Dr. Rubinstein opined "the evidence [for an infection] is far more credible than the evidence for a vaccine-related injury." Resp. Ex. A at 4. He explained that the assertion of vaccine-related hearing loss here is "weak . . . due to the inability to explain the pathophysiology of unilateral deafness as a result of a systemic immune or inflammatory response to vaccination." Id. at 5. According to Dr. Rubinstein, "[s]uch a response would affect both ears similarly and produce bilateral hearing loss," and epidemiologic data has rendered such a response as "exceedingly unlikely." Id. In his second expert report, he clarified that he does not suggest unilateral SSNHL rules out a systemic illness as causative, and instead, he "only concluded that it makes systemic illness far less likely [here] than in the setting of bilateral sudden hearing loss." Resp. Ex. C at 2.

iii. Althen Prong Three

Dr. Rubinstein did not provide any opinions as to the date of onset and whether this timeframe was medically acceptable.

IV. DISCUSSION

A. Standards for Adjudication

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

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, Petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

A petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a special master must decide what weight to give evidence including oral testimony and contemporaneous medical records). Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting 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))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of

Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike 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 v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[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 v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not rigidly bound by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (noting Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d. 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on his assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether a petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The undersigned must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence

presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying Petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

V. CAUSATION ANALYSIS

A. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d at 1359; see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 223 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

The undersigned finds Petitioner failed to provide preponderant evidence of a sound and reliable theory to explain how the flu vaccine can cause SSNHL. There are several reasons for this finding.

At the outset, the undersigned notes that the pathogenesis of SSNHL is not known. The cited literature is consistent on this point. See, e.g., Pet. Ex. 33 at 1 ("[T]he pathogenesis is still largely unknown even today and is subject to controversial discussion.");[75] Pet. Ex. 55 at 1 ("The

[75] Martin Canis et al., Fibrinogen/LDL Apheresis Is a Promising Rescue Therapy for Sudden Sensorineural Hearing Loss, 7 Clin. Rsch. Cardiol. Suppl. 36 (2012).

cause and pathogenesis of idiopathic [SSNHL] remain unknown."); Pet. Ex. 93 at 1 ("The etiology of the majority of [SSNHL] cases is unknown."); Pet. Ex. 94 at 9 ("The etiology of SSNHL remains unknown.").

Suggested causes of SSNHL are numerous and include infections, vascular insult, and inflammatory processes, among others. For each possible etiology, a different pathophysiological process is described. And there are inconsistencies between studies and papers about these posited etiologies. See, e.g., Pet. Ex. 49 at 1 ("Analysis of human temporal bones from patients with [] [SSNHL] does not support a vascular insufficiency but is more suggestive of a viral etiology.").[76] When the cause is vascular insufficiency, "it is usually in conjunction with some known systemic pathological process such as leukemia or intracranial lesions . . . or surgical interventions." Id. at 3-4. SSNHL has also been linked to risk factors such as diabetes and hypertension, but "findings are not consistent from study to study." Id. at 4.

Dr. Monsell's seven-step theory is an amalgamation of all the theories discussed in the numerous medical articles. When stacked on top of each other, the result is not a sound and reliable causal mechanism. Instead, it is a confusing puzzle. Pieces of the puzzle are not explained, foundational evidence is lacking, or the evidence is not relevant because the underlying facts and circumstances are different.

Moreover, Dr. Monsell did not provide evidence to support step one—that the flu vaccine stimulates the Acute Phase Reaction, or APR—of his seven-step mechanism. Since the six other steps of the seven-step theory rely on the first step, the entire mechanism fails.

For example, the first step of Dr. Monsell's theory asserts that the flu vaccine stimulates the APR, in which key proteins are released which may lead to a potentially undesired effect. He relies on Khalil and Al-Humadi who identify proteins occuring after flu vaccination, including CRP and transthyretin, and they note that CRP is a diagnostic marker for inflammation. But the authors did not attribute an adverse reaction to CRP or conclude that it caused pathology. Other examples of how the medical literature does not support the steps posited by Dr. Monsell are provided above, in the undersigned's review and analysis of the medical literature.

Dr. Monsell attempts to rely on inferences that a live virus flu infection causes the same inflammatory response as the inactivated flu vaccine. "An expert may 'extrapolate from existing data,' and use 'circumstantial evidence,' [b]ut the reasons for the extrapolation should be transparent and persuasive." K.O. v. Sec'y of Health & Hum. Servs., No. 13-472V, 2016 WL 7634491, at *12 (Fed. Cl. Spec. Mstr. July 7, 2016) (internal citations omitted) (first quoting Snyder v. Sec'y of Health & Hum. Servs., 88 Fed. Cl. 706, 743 (2009); and then quoting Althen, 418 F.3d at 1280). Dr. Monsell failed, however, to explain how data from the live flu virus could be extrapolated to the inactivated flu vaccine at issue.

Petitioner need not make a specific type of evidentiary showing or require identification of a specific antigenic trigger for an immune-mediated pathology to prove that a theory is sound

[76] Fred H. Linthicum et al., Idiopathic Sudden Sensorineural Hearing Loss: Vascular or Viral?, 149 Otolaryngol. Head & Neck Surg. 914 (2013).

and reliable by preponderant evidence. Given the state of current scientific knowledge, there is no way that a petitioner could satisfy such a requirement. Requiring proof of the identify of a specific antigen to prove causation would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program").

However, based on the current understanding of SSNHL as described in the literature filed herein, Dr. Monsell's proposed mechanism falls short of sound and reliable.

Overall, the undersigned finds that here, Petitioner's seven-step theory in part or as a whole is unsupported by medical or scientific facts, research, or any other reliable evidence. Moreover, his theories are speculative and/or conclusory in nature. When evaluating whether petitioners have carried their burden of proof, special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020). The undersigned will not rely on "opinion evidence that is connected to existing data only by the ipse dixit of the expert." Prokopeas v. Sec'y of Health & Hum. Servs., No. 04-1717V, 2019 WL 2509626, at *19 (Fed. Cl. Spec. Mstr. May 24, 2019) (quoting Moberly, 592 F.3d at 1315). Instead, special masters are expected to carefully scrutinize the reliability of each expert report submitted. See id.

Lastly, Vaccine Program case law does not support causation here. In Vanore, a case where compensation was denied, Petitioner's expert was Dr. Monsell and he offered the same seven-step theory to explain how the flu vaccine could cause SSHNL. Vanore v. Sec'y of Health & Hum. Servs., No. 21-0870V, 2024 WL 3200287 (Fed. Cl. Spec. Mstr. May 31, 2024). In Vanore, the Chief Special Master analyzed Dr. Monsell's seven-step theory and found Petitioner was not entitled to compensation, in part, because Dr. Monsell's theory was unreliable and not persuasive. Id. at *19-20. And more recently, the undersigned in Zikeli analyzed Dr. Monsell's seven-step theory and found it was not sound and reliable. Zikeli v. Sec'y of Health & Hum. Servs., No. 20-564V, 2025 WL 2306208, at *28-32 (Fed. Cl. Spec. Mstr. July 16, 2025).

Moreover, there are other flu vaccine and hearing loss cases where compensation has been denied.[77] See, e.g., M.R. v. Sec'y of Health & Hum. Servs., No. 16-1024V, 2023 WL 4936727 (Fed. Cl. Spec. Mstr. June 30, 2023) (determining the petitioner's acoustic neuroma/vestibular schwannoma, not the flu vaccine, was the most likely cause of his SSNHL); Donica v. Sec'y of Health & Hum. Servs., No. 08-625V, 2010 WL 3735707, at *1, 10 (Fed. Cl. Spec. Mstr. Aug. 31, 2010) (finding flu vaccine was not demonstrated to cause adult hearing loss

[77] In addition to the flu vaccine, cases involving other vaccines have also found against compensation. See, e.g., Hopkins v. Sec'y of Health & Hum. Servs., No. 00-745V, 2007 WL 2454038, at *1, *19-23 (Fed. Cl. Spec. Mstr. Aug. 10, 2007) (dismissing claim of SNHL due to *haemophilus influenzae* type B, diphtheria-tetanus-pertussis, and oral polio vaccines and finding the submitted literature failed to support the theory), aff'd, 84 Fed. Cl. 517 (2008).

in adult but not making a determination as to Althen prong one because prong three was not met).

Petitioner in Inamdar alleged the flu vaccine caused his SNHL. Inamdar v. Sec'y of Health & Hum. Servs., No. 15-1173V, 2019 WL 1160341, at *1 (Fed. Cl. Spec. Mstr. Feb. 8, 2019). Petitioner proposed the flu vaccine "could cause the production of proinflammatory cytokines immediately upon vaccine administration," and alternatively, that specific components of the vaccine "were structurally homologous with ganglioside receptors on the neuronal myelin contained in the inner ear tissue, and that antibodies generated in response to the vaccine could also cross-react with the self-myelin, resulting in tissue damage." Id. at *5-6. The Chief Special Master found the first theory relied too heavily on what was known about the wild virus rather than the vaccine, and further found that both theories were unsupported by the literature. Id. at *17-18. An alternative cause also existed, and a one-day onset was not shown to be medically acceptable. Id. at *18-19.

In Kelly, the Petitioner alleged the flu vaccine caused his SNHL. Kelly v. Sec'y of Health & Hum. Servs., No. 16-878V, 2021 WL 5276373, at *1 (Fed. Cl. Spec. Mstr. Oct. 18, 2021), mot. for rev. den'd, 160 Fed. Cl. 316 (2022). Petitioner alleged a significant aggravation claim, but the Chief Special Master noted his determination would have been the same even if Petitioner alleged a causation-in-fact claim. Id. at *24. Petitioner proposed a Type I sensitivity reaction and alternatively, an autoimmune response. Id. at *25-26. The Chief Special Master found limited support for the primary theory and found the autoimmune theory inconsistent with the facts presented, including the fact that Petitioner's hearing loss was unilateral, and the onset was two hours. Id. at *24-26. It was also noted that "an autoimmune origin would in most cases mean some other underlying systemic disease was occurring" and there was nothing in the record to support that. Id. at *26 (emphasis omitted).

The undersigned has also denied entitlement in two additional hearing loss cases. In Alsaadeh, the undersigned denied entitlement where Petitioner alleged the flu and Prevnar 13 vaccines caused his hearing loss. Alsaadeh v. Sec'y of Health & Hum. Servs., No. 19-1097V, 2024 WL 694072 (Fed. Cl. Spec. Mstr. Jan. 23, 2024). The undersigned found Petitioner's immune-mediated theory was not sound or reliable in part because the causal mechanism of immune-mediated hearing loss is unknown. Id. at *31-34. In Herms, the undersigned also denied entitlement where petitioner alleged that the diphtheria tetanus toxoid acellular pertussis vaccination that she received caused SNHL and tinnitus in her left ear. Herms v. Sec'y of Health & Hum. Servs., No. 19-70V, 2024 WL 1340669, at *1 (Fed. Cl. Spec. Mstr. Mar. 4, 2024), mot. for rev. den'd, aff'd, 173 Fed. Cl. 1, appeal docketed, No. 25-1007 (Fed. Cir. Sept. 27, 2024). The undersigned found Petitioner's theory was not sound or reliable because it was underdeveloped, conclusory in nature, and vague and because the causal mechanism of immune-mediated hearing loss is unknown. Id. at *19-20.

Although decisions of other special masters are not binding, the undersigned generally agrees with the reasoning of her colleagues in the above cases. See Boatmon, 941 F.3d at 1358; Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999).

While there is one reasoned decision where entitlement was granted to a petitioner who alleged the flu vaccine caused SSNHL, the undersigned notes that the facts and theory are different. Madigan v. Sec'y of Health & Hum. Servs., No. 14-1187V, 2021 WL 3046614 (Fed. Cl. Spec. Mstr. June 25, 2021).

Overall, the majority of Vaccine Program cases have resulted in denial in compensation to petitioners. This outcome largely tracks the medical literature, which upon review, illustrates that the cause of hearing loss is unknown. The lack of knowledge cannot be supplanted by supposition.

In summary, Petitioner has failed to offer a sound and reliable medical theory in support of his claim. Thus, the undersigned finds Petitioner has failed to provide preponderant evidence with respect to the first Althen prong.

B. Althen Prong Two

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

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

Since Petitioner failed to prove Althen prong one, it follows that he cannot prove Althen prong two. However, even if Petitioner had proven Althen prong one, the undersigned finds there is not preponderant evidence in the record to support a logical sequence of cause-and-effect showing the September 15, 2019 flu vaccine to be the cause of Petitioner's SSNHL. See Althen, 418 F.3d at 1278. There as three reasons for this finding.

First, the records and expert opinions show aspects of Petitioner's clinical course are not consistent with the mechanism posited by Dr. Monsell. Dr. Monsell opines Petitioner had "a strong treatment response" to steroids, which resulted in "substantial improvement" in hearing, "confirming the role of inflammation in his case." Pet. Ex. 19 at 5, 21, 23. However, the records

do not support a finding of "a strong treatment response" to steroids resulting in "substantial improvement" in hearing.

In all, Petitioner's audiograms document moderate to profound hearing loss. Dr. Monsell agrees Petitioner's "hearing loss was at the profound level on presentation and continued to be severe." Pet. Ex. 19 at 21.

Petitioner first saw an audiologist post-vaccination on September 24, 2019, and the audiologist documented "moderately severe to profound SNHL in the left ear." Pet. Ex. 3 at 120. Petitioner was given oral steroid on this date. On October 3, 2019, Petitioner had "profound hearing loss" in the left ear according to his audiologist. Id. at 106. The audiologist documented Petitioner's left-sided hearing loss was "stable." Id. at 107.

On October 10, 2019, Petitioner saw an otorhinolaryngologist, Dr. Westbrook who noted Petitioner's first audiogram showed "severe panfrequency SNHL" and his follow-up audiogram conducted after ten days of prednisone showed "profound panfrequency loss." Pet. Ex. 2 at 25. This supports a finding that Petitioner's hearing worsened after oral steroids.

Petitioner received his first transtympanic steroid injection at his visit with Dr. Westbrook on October 10. Petitioner returned and saw otorhinolaryngologist Dr. Anderson on October 23, 2019. She documented "subjective improvement in his hearing since the injection." Pet. Ex. 2 at 23. No objective findings as to Petitioner's hearing loss were documented. She administered a second transtympanic steroid injection.

Although a "-25 to -30dB improvement below 6000 Hz [in the] left ear" was documented by Petitioner's audiologist on November 15, 2019, Petitioner's hearing loss remained "moderate to profound." Pet. Ex. 3 at 106. Subsequent follow up examinations with audiology in December 2019 and October 2020 documented "moderate to profound" hearing loss in the left ear. Pet. Ex. 20 at 17; Pet. Ex. 8 at 95. Petitioner's audiologist did not provide any opinions on the improvement seen on audiogram in November 2019, nor did his audiologist opine as to whether corticosteroids were involved.

At a follow-up with Dr. Anderson on November 20, Petitioner reported his recent audiogram showed a 20% improvement. However, after Dr. Anderson was able to review the most recent audiology report, she concluded Petitioner's left ear was "essentially anacoustic." Pet. Ex. 20 at 20.

Thus, at all times, Petitioner's left-sided hearing loss appeared to be between moderate and profound. If there was an improvement after steroids, it was not significant enough to change this conclusion. High-dose steroids would be expected to improve his hearing if the etiology was inflammatory in nature. The level of improvement required to support this hearing loss as inflammatory is not clear from the record but no treating physician opined Petitioner's hearing loss was inflammatory, or that he benefitted from corticosteroids.

Further, steroids are strong inhibitors of the NFKB pathway, and the lack of treating physician support that Petitioner's hearing loss was inflammatory and/or improving on steroids weighs against Dr. Monsell's theory.

Additionally, Petitioner's hearing loss was unilateral, not bilateral. Dr. Rubinstein explained a systemic response seen with vaccination would affect both ears, leading to bilateral hearing loss, not unilateral.

Second, the undersigned finds that in this case, other causes of Petitioner's hearing loss were considered. Dr. Westbrook opined a "[p]ossible vestibular neuronitis as a cause" of Petitioner's SSNHL. Pet. Ex. 2 at 27. Additionally, Dr. Rubinstein considered two other causes, an acoustic neuroma and viral illness as more likely causes. Further. he opined that viral infections of the inner ear are the "leading hypothesis" for causation in idiopathic SSNHL, and although the specific "causative viruses are [] unknown," infection was more likely the cause of Petitioner's SSNHL than vaccination. Resp. Ex. A at 4-5.

The undersigned acknowledges that Petitioner is not required to eliminate other potential causes in order to be entitled to compensation. See Walther v. Sec'y of Health & Hum. Servs., 485 F.3d 1146, 1149-52 (Fed. Cir. 2007) (finding petitioner does not bear the burden of eliminating alternative independent potential causes). However, she finds it reasonable to consider "evidence of other possible sources of injury" in determining "whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question." Stone, 676 F.3d at 1379; see also Winkler v. Sec'y of Health & Hum. Servs., 88 F.4th 958, 963 (Fed. Cir. 2023) ("Such contemplation of a potential causative agent when evaluating whether or not a petitioner has established a prima facie case is in accordance with the law."); Flores, 115 Fed. Cl. at 162-63 ("[T]he special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case."). Here the record indicates two other causes, acoustic neuroma and viral illness. The undersigned finds it reasonable to consider these potential causes of Petitioner's condition in determining that Petitioner has failed to provide preponderant evidence of prong two.

Third, Petitioner's physicians did not attribute his hearing loss to vaccination. Petitioner saw various treating providers in his first year of hearing loss, including audiologists and otolaryngologists, and none opined Petitioner's flu vaccination caused or contributed to Petitioner's SSNHL.

Generally, treating physician statements are typically "favored" 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.'" Capizzano, 440 F.3d at 1326 (quoting Althen, 418 F.3d at 1280). "In weighing the persuasiveness of opinion testimony, special masters may consider the relative expertise of the witness." Koehn v. Sec'y of Health & Hum. Servs., No. 11-355V, 2013 WL 3214877, at *32 (Fed. Cl. Spec. Mstr. May 30, 2013), aff'd, 773 F.3d 1239 (Fed. Cir. 2014); see also Dwyer v. Sec'y of Health & Hum. Servs., No. 03-1202V, 2010 WL 892250, at *64 (Fed. Cl. Spec. Mstr. Mar. 12, 2010) (giving greater weight to M.D. epidemiologists' opinions on medical issues than to Ph.D. epidemiologist's opinion); Pafford, 451 F.3d at 1359 (affirming the special master's rejection of expert's testimony because

he lacked proper qualifications in the specialty areas in which he testified). Here, there are no contemporaneous opinions in favor of vaccine causation made by Petitioner's audiologist or the specialists who evaluated and treated his hearing loss.

Later-in-time records document Petitioner's hearing loss post-flu vaccination; however, the undersigned does not find this persuasive evidence to reach the level of preponderance. On December 2, 2020, while establishing care at an occupational health clinic, Petitioner reported that in the past he had a "reaction to [flu] vaccine and lost hearing in [his] left ear." Pet. Ex. 11 at 19. And on December 5, 2023, a nursing note stated that Petitioner's allergy profile was updated to include hearing loss post-flu vaccine and that "[p]rovider review indicate[d] [Petitioner] should NEVER receive seasonal [flu] vaccine." Pet. Ex. 95 at 27.

Both of these statements in the medical records appear to have been documented pursuant to a report made by Petitioner. Neither statement indicated that a treating physician had offered an opinion that the flu vaccine caused Petitioner's SSNHL. Thus, the statements are conclusory in nature and lack supportive evidence. Special masters consistently reject "conclusory expert statements that are not themselves backed up with reliable scientific support." Kreizenbeck v. Sec'y of Health & Hum. Servs., No. 08-209V, 2018 WL 3679843, at *31 (Fed. Cl. Spec. Mstr. June 22, 2018), mot. for rev. den'd, decision aff'd, 141 Fed. Cl. 138, aff'd, 945 F.3d 1362 (Fed. Cir. 2020).

Additionally, Petitioner offers no contemporaneous medical records with opinions by his treating physicians in support of vaccine causation. The undersigned finds later-in-time records less persuasive as contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras, 993 F.2d at 1528.

Moreover, the December 2023 statement was made more than four years after vaccination and onset, and after litigation was initiated. See, e.g., Zumwalt v. Sec'y of Health & Hum. Servs., No. 16-994V, 2019 WL 1953739, at *19 (Fed. Cl. Spec. Mstr. Mar. 21, 2019) (rejecting opinion from a treating provider when he presented an opinion two-and-one-half years after treatment and after litigation was initiated), mot. for rev. den'd, 146 Fed. Cl. 525 (2019).

Accordingly, the undersigned finds that Petitioner has not satisfied his burden under Althen prong two.

C. Althen Prong Three

Althen prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That term has been defined as a "medically acceptable temporal relationship." Id. The Petitioner must offer "preponderant proof that the onset of symptoms occurred within a time frame for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also be consistent with the theory of how the relevant vaccine can cause the injury alleged (under Althen Prong One). Id.; Koehn, 773 F.3d at 1243; Shapiro, 101 Fed. Cl. at 542; see Pafford, 451 F.3d at 1358.

Petitioner received a flu vaccine on September 15, 2019. On September 24, 2019, nine days post-vaccination, Petitioner presented to an audiologist for left-sided hearing loss, later diagnosed as SSNHL, that began September 22, 2019.

Dr. Monsell placed onset six days post-vaccination and maintained this onset was consistent with his proposed mechanism. Dr. Rubinstein did not provide an opinion as to the date of onset and whether this time frame is medically appropriate.

A temporal association, without more, is insufficient. Moberly, 592 F.3d at 1323; Grant v. Sec'y of Health & Hum. Servs., 956 F.2d 1144, 1148 (Fed. Cir. 1992) ("[A] proximate temporal association alone does not suffice to show a causal link between the vaccination and the injury."). Thus, even though Petitioner has provided preponderant evidence satisfying Althen prong three, Petitioner is not entitled to compensation.

VI. CONCLUSION

The undersigned extends her sympathy to Petitioner for all that he has suffered due to his hearing loss. Her Decision, however, cannot be decided based upon sympathy, but rather on the evidence and law.

For the reasons discussed above, the undersigned finds that Petitioner has failed to establish by preponderant evidence that the flu vaccination he received caused him to develop SSNHL. Therefore, Petitioner is not entitled to compensation, and the petition must be dismissed.

In the absence of a timely filed motion for review pursuant to Vaccine Rule 23, the Clerk of Court **SHALL ENTER JUDGMENT** in accordance with this Decision.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master