Tinnitus Etiology

Tinnitus is not a disease, but a condition that can result from a wide range of underlying causes. The most common cause is noise-induced hearing loss. Other causes include neurological damage (multiple sclerosis), ear infections, oxidative stress, emotional stress, foreign objects in the ear, nasal allergies that prevent (or induce) fluid drain, wax build-up, and exposure to loud sounds. Withdrawal from benzodiazepines may cause tinnitus as well.

Patients with idiopathic intracranial hypertension can have pulsatile tinnitus, a whooshing sensation in one or both ears (64–87%); this sound is synchronous with the pulse.

Tinnitus distress has been linked to increased beta oscillatory activity in the dorsal anterior cingulate cortex (dACC). The amount of distress is linked to alpha activity in the medial temporal lobe (amygdala and parahippocampal area), as well as the subgenual (sg)ACC and insula, and the functional connectivity between the parahippocampal area and the sgACC at 10 and 11.5 Hz.

Aortocranial fibromuscular dysplasia.

Chiari 1 malformation.

Intracranial hypotension.

Primary otalgia

Geniculate neuralgia.

Meniere’s disease.

Endolymphatic-subarachnoid shunts.

Glomus tumor

Vestibular schwannoma: see Vestibular schwannoma tinnitus.

Aminoglycoside toxicity: streptomycin, tobramycin (tinnitus precedes hearing loss)

Recent studies have adopted the Bayesian brain model to explain the generation of tinnitus in subjects with auditory deafferentation. That is, as the human brain works in a Bayesian manner to reduce environmental uncertainty, missing auditory information due to hearing loss may cause auditory phantom percepts, i.e., tinnitus. This type of deafferentation-induced auditory phantom percept should be preceded by auditory experience because the fill-in phenomenon, namely tinnitus, is based upon auditory prediction and the resultant prediction error. For example, a recent animal study observed the absence of tinnitus in cats with congenital single-sided deafness (SSD; Eggermont and Kral, Hear Res 2016). However, no human studies have investigated the presence and characteristics of tinnitus in subjects with congenital SSD. Thus, the present study sought to reveal differences in the generation of tinnitus between subjects with congenital SSD and those with acquired SSD to evaluate the replicability of previous animal studies. This study enrolled 20 subjects with congenital SSD and 44 subjects with acquired SSD and examined the presence and characteristics of tinnitus in the groups. None of the 20 subjects with congenital SSD perceived

tinnitus on the affected side, whereas 30 of 44 subjects with acquired SSD experienced tinnitus on the affected side. Additionally, there were significant positive correlations between tinnitus characteristics and the audiometric characteristics of the SSD. In accordance with the findings of the recent animal study, tinnitus was absent in subjects with congenital SSD, but relatively frequent in subjects with acquired SSD, which suggests that the development of tinnitus should be preceded by auditory experience. In other words, subjects with profound congenital peripheral deafferentation do not develop auditory phantom percepts because no auditory predictions are available from the Bayesian brain [1].

1)
Lee SY, Nam DW, Koo JW, De Ridder D, Vanneste S, Song JJ. No auditory experience, no tinnitus: Lessons from subjects with congenital- and acquired single-sided deafness. Hear Res. 2017 Aug 15;354:9-15. doi: 10.1016/j.heares.2017.08.002. [Epub ahead of print] PubMed PMID: 28826043.

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