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Pathophysiological differences in phantom sound: subtypes in tinnitus

Permanently affecting one in seven adults, chronic tinnitus lacks both widely effective treatments and adequate understanding of its brain mechanisms. Progress towards an effective treatment has been hampered by the fact that “tinnitus” represents a highly heterogeneous condition. It is suggested that there might be different subtypes of tinnitus that could explain the differences in treatment outcome. Based on a theoretical model, i.e. a multiphase compensation mechanism, we predict two very different kinds of bottom-up related tinnitus depending on the amount of hearing loss. The first form of tinnitus is not associated with severe hearing loss where the auditory cortex plays a key role. The second form of tinnitus is associated with severe hearing loss, in which the parahippocampus plays a key role while the auditory cortex might be of little relevance. The weaknesses of this multiphase compensation model are (1) that it does not explain why some people with severe hearing loss do not develop tinnitus or (2) why some people report tinnitus without measurable hearing loss. However, recent research has suggested that tinnitus can be the result of a deficient top-down auditory gating mechanism. This central gating mechanism evaluates the relevance and affective meaning of sensory stimuli. It modulates information via descending inhibitory pathways, starting from the pregenual anterior cingulate cortex to the thalamic reticular nucleus, which modulates the information flow between the thalamus and the auditory cortex by inhibiting specific thalamic neurons in a highly selective and frequency-specific manner. Interestingly, the COMT polymorphism is especially prominent in prefrontal cortex and has been associated with auditory gating of the tinnitus percept. Our research demonstrates that both the amount of hearing loss and the COMT polymorphism can increase the susceptibility to the clinical manifestation of tinnitus that goes together with not cancelling auditory information, leading to increased tinnitus loudness. In this presentation, I will talk about the potential subtypes of tinnitus and their underlying neural mechanisms.