The Neural Bases of Tinnitus: Lessons from Deafness and Cochlear Implants

Authors: Knipper M, van Dijk P, Schulze H, Mazurek B, Krauss P, Scheper V, Warnecke A, Schlee W, Schwabe K, Singer W, Braun C, Delano PH, Fallgatter AJ, Ehlis AC, Searchfield GD, Munk MHJ, Baguley DM, Rüttiger L.
Publisher: -
In: The Journal of Neuroscience, 16 September 2020

Abstract:

Subjective tinnitus is the conscious perception of sound in the absence of any acoustic source. The literature suggests various tinnitus mechanisms, most of which invoke changes in spontaneous firing rates of central auditory neurons resulting from modification of neural gain. Here, we present an alternative model based on evidence that tinnitus is: (1) rare in people who are congenitally deaf, (2) common in people with acquired deafness, and (3) potentially suppressed by active cochlear implants used for hearing restoration. We propose that tinnitus can only develop after fast auditory fiber activity has stimulated the synapse formation between fast-spiking parvalbumin positive (PV+) interneurons and projecting neurons in the ascending auditory path and coactivated frontostriatal networks after hearing onset. Thereafter, fast auditory fiber activity promotes feedforward and feedback inhibition mediated by PV+ interneuron activity in auditory-specific circuits. This inhibitory network enables enhanced stimulus resolution, attention-driven contrast improvement, and augmentation of auditory responses in central auditory pathways (neural gain) after damage of slow auditory fibers. When fast auditory fiber activity is lost, tonic PV+ interneuron activity is diminished, resulting in the prolonged response latencies, sudden hyperexcitability, enhanced cortical synchrony, elevated spontaneous γ oscillations, and impaired attention/stress-control that have been described in previous tinnitus models. Moreover, because fast processing is gained through sensory experience, tinnitus would not exist in congenital deafness. Electrical cochlear stimulation may have the potential to reestablish tonic inhibitory networks and thus suppress tinnitus. The proposed framework unites many ideas of tinnitus pathophysiology and may catalyze cooperative efforts to develop tinnitus therapies.

Related links:

Online publication
The Neural Bases of Tinnitus: Lessons from Deafness and Cochlear Implants (nih.gov)

Download complete PDF: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7534911/pdf/zns7190.pdf