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Sensory Systems

P229.-Activity of the α9α10 nAChR inversely correlates with the magnitude of acoustic injury.

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Noise induced hearing loss (NIHL) has become a major public health problem. In order to address the role of the efferent olivocochlear system in NIHL we made use of a mouse model in which the α9 nicotinic receptor subunit bears a mutation and leads to enhanced medial efferent activity (Chrna9L9'T knock-in (KI)) in addition to one lacking the a9 subunit of the nicotinic receptor (Chrna9 knockout (KO)).
We exposed WT, Chrna9L9'T KI and Chrna9 KO mice to loud sounds (1-16 kHz, 100 dB SPL, 1hr) and measured auditory brainstem responses (ABR), which reflect synchronized discharges from neurons along the auditory pathway. We tested outer hair cell function by recording the distortion product otoacoustic emissions (DPOAEs). Large auditory threshold shifts were found one day after exposure in WT and Chrna9 KO mice. However, one week later, thresholds returned to normal in WT, whereas the Chrna9 KO ears did not recover. In contrast, Chrna9L9'T KI mice were resistant to the same noise exposure. Finally, we used immunohistochemistry to visualize efferent neurons and found a reduction in the number of terminals after trauma in WT mice. Immunofluorescence against Ctbp2, a protein located in inner hair cell (IHC) ribbon synapse, revealed a decrease in the number of ribbon synapses per IHC after acoustic trauma in WT mice. These findings suggest a key role of α9α10 nAChRs in the efferent-mediated noise protection.