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Synaptic Transmission and Excitability

P244.-A point mutation in the α9α10 nAChR alters short-term synaptic plasticity of medial olivocochlear- hair cell (MOC-HC) synapses

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IHCs convey acoustic information to the central nervous system while OHCs are responsible for the mechanical amplification of sound. IHCs receive a transient MOC innervation since birth to the onset of hearing, while MOC fibers synapse onto OHCs from the first postnatal week throughout adulthood. The MOC-HC synapse is inhibitory and mediated by α9α10 nicotinic receptors (nAChRs). We analyzed the properties of synaptic transmission of a knock-in mouse (Kin) with a point mutation in the α9 nAChR subunit (L9´T) that prolongs MOC inhibition (Taranda et. al 2009). Synaptic currents (IPSCs) were recorded in IHCs and OHCs of isolated mouse cochleas at postnatal day 9-13 during electrical stimulation of MOC fibers. In previous studies we showed that high frequency stimulation causes synaptic depression in MOC-IHC synapses, whereas it causes facilitation in MOC-OHC synapses. We found that in both wt and Kin IHCs, 100Hz-trains applied to the MOC fibers caused depression of IPSC amplitudes (S10/S1: 21% and 10% in Kin and wt mice, respectively) whereas 10Hz-trains caused depression only in Kin mice (S10/S1:60%). Accordingly, the ready releasable pool size was smaller in Kin mice (wt:3.7±0.9 Kin:2.7±0.8). Preliminary experiments in OHCs show that high frequency stimulation (40-80 Hz) caused 3-fold more facilitation in Kin than in wt mice. These results show that a modification in the postsynaptic nAChR alters the short term plasticity pattern of MOC-HC synapses.

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