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ABSTRACTS

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0.47±0.16; BayK 0.22±0.07, n=6, p<0.05), indicating that Ca2+ influx through L-type VGCC is only activating BK channels at this stage. These results show that the VGCCs coupled to ACh release at the MOC-OHC synapse at P11-13 are the same as those at the MOC-IHC synapse at early stages (P4 to 7; Kearney et al., ARO Abstracts 2014), while at P20-22, they resemble those of the MOC-IHC synapse at P9-11 (Zorrilla de San Martin et al., 2010). These results suggest that the MOC-OHC synapse is still immature at the onset of hearing. Support: UBA& ANPCyT to EK and ABE

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Enhanced Hair Cell Postsynaptic Responses Alter Release from Presynaptic Efferent Neurons to Prolong Inhibition of the Cochlea

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Gain control of the auditory system operates at multiple levels. Cholinergic medial olivocochlear (MOC) fibers that originate in the brainstem and make direct synaptic contacts at the base of the outer hair cells (OHCs) are the final targets of several feedback loops from both the periphery and higher processing centers. Efferent activation inhibits somatic electromotility of OHCs, an active amplification system within the mammalian cochlea. This is mediated by the activation of a calcium permeable α9α10 ionotropic cholinergic nicotinic receptor (nAChR) functionally coupled to calcium activated SK potassium channels. The strength of cochlear inhibition is driven by the rate of MOC activity and short term facilitation at the MOC-OHC synapse (Ballestero et al., 2011).The present work shows that a knockin mouse with a mutation in the α9α10 nAChR (L9';T) with increased channel gating (Taranda et al., 2009) greatly prolongs hair cell evoked inhibitory postsynaptic currents (IPSCs). Long-term presynaptic compensatory mechanisms lead to reduced quantum content (IHC wt =1.29 ± 0.21; L9';T= 0.83 ± 0.12, n=5-6. OHC wt =0.23 ± 0.04, L9';T = 0.14 ± 0.02, n=12-15). However, upon high frequency stimulation of MOC-OHC synapses, L9';T mice exhibited more facilitation leading to greatly prolonged synaptic responses (S_2/S_{1-40Hz}: wt = 1.37 ± 0.16, L9';T = 3.47 ± 0.44, n = 6-8, p< 0.05). At the cochlear physiology level, these synaptic changes were matched by a longer time course of efferent MOC suppression of DPOAEs. Thus, the maximal suppressive effect of electrical shocks (70-s, 200 Hz) at the base of the IVth ventricle was doubled both at 16 (p < 0.01) and 22 kHz (p < 0.05), reached much more slowly (16 kHz: wt = 5.3 ± 1.0 s, L9';T = 30.8 ± 4.1 s; 22 kHz: wt = 1.5 ± 0.4 s, L9';T = 44.1 ± 3.1 s) and persisted for a longer time after the shocks for both 16 and 22 kHz in L9';T mice (> 5 min) as compared to their wt littermates (≤1 s). These results indicate that the properties of the MOC-OHC synapse directly determine the efficacy of the MOC feedback to the cochlea being a main player in the "gain control" of the auditory periphery.

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Immunohistochemical Identification of Human Spiral Ganglia Neurons: Implications in Sensorineural Hearing Loss and Cochlear Implantation

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Background

Human spiral ganglia neurons (hSGNs) persist in the human cochlea after hair cell loss, in contrast to SGNs in the cochlea of animal models. We hypothesize that the persistent immunolocalization of structural and functional proteins in hSGNs suggest that they may be active, even in the absence of hair cells. Here we investigate the immunolocalization of three specific structural and functional proteins in hSGNs in normal aging and inner ear pathologies, and in patients who have undergone cochlear implantation.

Methods

Temporal bones from 38 patients (age: 8-89 years; n=11 normal hearing, n=27 hearing loss, n=7 received cochlear implants) were identified. Celloidin-embedded human cochleas were immunostained using mouse monoclonal antibodies against pan-neurofilaments, acetylated-tubu-