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SENSORY AND MOTOR SYSTEMS

GABA and ACh are co-released from olivocochlear efferent terminals

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During development, inner hair cells (IHCs) in the mammalian cochlea are unresponsive to acoustic stimuli but instead present intrinsic electrical activity, crucial for the normal development of the auditory pathway. During this same period, neurons originating from the medial olivocochlear complex (MOC) transiently innervate IHCs. This innervation is mediated by acetylcholine (ACh), activating α9α10 nicotinic receptors and is responsible for controlling IHC excitability during this period. Even though this is a cholinergic synapse, previous evidence indicates the presence of abundant GABA and GABAB receptors in MOC fibers in the inner spiral bundle. Moreover, the application of GABAB agonists reduces ACh release. Transgenic mice expressing channelrodhopsin (ChR2) under the control of either GAD (GABAergic) or ChAT (cholinergic) promoters were used in this study. Here we show for the first time, that optogenetically activated fibers in GAD-cre/ChR2 mice (n=7) produced postsynaptic responses that were blocked with cholinergic antagonists (n=3). In addition, pharmacological experiments in ChAT-cre/ChR2 mice indicate GABAB activation, suggesting GABA release by cholinergic neurons (n=4). ChAT-cre/TdTomato cochleas, co-stained with antibody against GAD, showed a co-localization of GABAergic and cholinergic terminals in the inner spiral bundle. Altogether these results strongly suggest that ACh is being co-released with GABA from MOC fibers.