P1.13.

Cholinergic control of GABA release by carbachol at perisomatic inhibitory synapses in the CA3 region of mouse hippocampus

Szabó, G. G.[1]; Holderith, N.[1]; Gulyás, A. I.[1]; Freund, T. F.[1]; Hájos, N.[1*]

1: Institute of Experimental Medicine, Hungarian Academy of Sciences, Szigony u. 43, 1083 Budapest, Hungary

Action potential generation of hippocampal pyramidal neurons is controlled by perisomatic inhibition, originating from fast-spiking basket (FSBC)- and axo-axonic cells (AACs) as well as from regular spiking basket cells (RSBCs). These neurons play a role in feedforward and feedback inhibition and are tuned by cholinergic receptor activation. We aimed to clarify the impact of cholinergic receptor activation on synaptic communication between these perisomatic inhibitory neurons (PINs) and pyramidal cells (PCs). By performing paired recordings between the three types of PINs and PCs in mouse hippocampal slices, we investigated the release properties and their sensitivity to the cholinergic receptor agonist carbachol (CCh). Synaptic currents originating from the distinct types of PINs had different properties recorded in CA3 PCs. Analyzes of the decay kinetics suggested that at AAC-PC pairs synaptic cross-talk may occur between adjacent release sites. RSBCs showed robust asynchronous release, whereas the other two types of PINs did not. CCh decreased the GABA release significantly from all the three types of PINs to a different extent and via different receptor activation. In addition, CCh significantly reduced the short-term depression of synaptic transmission at FSBC- and AAC-PC pairs.These findings suggest that the contribution of different types of PINs to feedforward and feedback inhibition might be distinct that could be altered by cholinergic receptor activation.