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Neuroscience 2015

Presentation Abstract

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Presentation Title:	Rapid dynamic changes of dendritic inhibition in the dentate gyrus by presynaptic activity patterns
Location:	Hall A
Presentation time:	Sunday, Oct 18, 2015, 8:00 AM -12:00 PM
Presenter at Poster:	Sun, Oct. 18, 2015, 9:00 AM - 10:00 AM
Topic:	++B.07.e. Modulation: ACh, amino acids and GABA
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Abstract:	The dentate gyrus (DG) serves as a primary gate to control information transfer from the cortex to the hippocampus. Activation of incoming cortical inputs results in rapid synaptic excitation followed by slow GABA-mediated (GABAergic) synaptic inhibition onto DG granule cells (GCs). GABAergic inhibitory interneurons (INs) in the DG comprise fast-spiking (FS) and non-fast-spiking (non-FS) cells. Anatomical analyses of DG INs reveal that FS cells are soma-targeting INs, whereas non-FS cells are dendrite-targeting INs. These two IN classes are differentially recruited by excitatory inputs and in turn provide exquisite spatiotemporal control over GC activity. Yet, little is known how FS and non-FS cells transform their presynaptic dynamics into varying postsynaptic response amplitudes. Using paired recordings in rat hippocampal slices, we show that inhibition in the DG is dominated by somatic GABAergic inputs during periods of sparse presynaptic activity, whereas dendritic GABAergic inputs are rapidly shifted to powerful and sustained inhibition during periods of intense presynaptic activity. The variant dynamics of dendritic inhibition is dependent on presynaptic IN subtypes and their activity patterns and is attributed to Ca2+-dependent increases in the probability of release and the size of the readily releasable pool. Furthermore, the degree of dynamic GABA release can be reduced by blocking voltage-gated K+ channels, which increases the efficacy of dendrite-targeting IN output synapses during sparse firing. Such rapid dynamic modulation of dendritic inhibition may act as a frequency-dependent filter to prevent overexcitation of GC

dendrites and thus set the excitatory-inhibitory synaptic balance in the DG circuits.

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