



Seminar/Talk

A cooperative mechanism involving Ca^{2+} -permeable AMPA receptors and retrograde activation of GABAB receptors in interpeduncular nucleus plasticity

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The medial habenula-interpeduncular nucleus (MHb-IPN) pathway which connects the limbic forebrain to the midbrain, has recently been implicated in aversive behaviors. The MHb-IPN circuit is characterized by a unique topographical organization, an excitatory role of GABA and a prominent co-release of neurotransmitters and neuropeptides. However, little is known about synaptic plasticity in this pathway. An application of high-frequency stimulation resulted in a long-lasting potentiation of glutamate release in IPN neurons. This plasticity required the activation of post-synaptic Ca^{2+} -permeable AMPA receptors (CPARs) and pre-synaptic GABAB receptors. Strikingly, adolescent IPN neurons lacked CPARs and exhibited an inability to undergo plasticity. Our experiments reveal that CPAR-dependent release of GABA from IPN neurons and retrograde activation of GABAB receptors on MHb terminals result in a long-lasting enhancement of glutamate release. This plasticity at the MHb-IPN synapses might be critical for the regulation of aversive behaviors, which is altered during adolescence.

Friday, February 24, 2017 04:00pm - 05:00pm

Seminar Room, Lab Building East



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