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MEETING ABSTRACT Open Access

Neuropeptide Y Y_2 receptors modulate trace fear conditioning and spatial memory in the dorsal hippocampus

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Background

Neuropeptide Y (NPY), a highly conserved 36 amino acid peptide is widely distributed in the central nervous system. Besides its functions in various metabolic processes NPY has attracted considerable attention in modulating emotional-affective behavior. NPY exerts a pronounced anxiolytic effect most likely mediated by Y_1 receptors, whereas stimulation of predominantly pre-synaptic Y_2 receptors results in increased anxiety. The role of NPY Y_2 receptors in the processing of emotional learning, however, remains still elusive.

Methods

The current study aims to investigate the role of NPY Y_2 receptors in Pavlovian fear conditioning, a simple form of associative learning and in a spatial memory task, the Barnes maze. Y_2-KO mice were subjected to delay (amygdala-dependent) and trace (hippocampus-dependent) fear conditioning paradigms.

Results

While in delay fear conditioning Y_2-KO mice performed similar to wild-type controls, recall of a trace fear memory was significantly increased in Y_2-KO mice. Furthermore, Y_2-KO mice exhibited an improved long-term memory in the Barnes maze test, a paradigm investigating spatial learning. Trace fear conditioning and spatial memory are predominantly mediated by the dorsal hippocampus. For investigating the specific contribution of Y_2 receptors in the adult dorsal hippocampus in trace fear conditioning and spatial memory formation we locally deleted hippocampal Y_2 receptors in conditional Y_2-KO mice by injection of a rAAV-CreGFP vector. Moreover we over-expressed NPY_{3-36}, an Y_2 receptor preferring agonist, at the same brain sites.

Conclusions

Our data indicate that while Y_2 receptors are not involved in amygdala-dependent delay fear conditioning, they seem to play an inhibitory role on the acquisition of trace fear memories. Moreover, Y_2 receptors in the dorsal hippocampus are crucial for spatial memory formation. These actions are probably mediated by inhibition of glutamate release in dorsal hippocampal circuitries.

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