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Poster Presentation

Long-Term Potentiation: The Mechanisms of CaMKII in Lerarning and Memory

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Abstract

Long-term potentiation (LTP) is a form of activity dependent plasticity that induced by high-frequency stimulation or theta burst stimulation and results in synaptic transmission. Several Studies have been shown that LTP is one of the most important processes in the CNS that plays an important role in learning and memory formation. Ca2+/calmodulin-dependent protein kinase II (CaMKII) is a major synaptic protein that involved in many signaling cascades and has an important role in the induction of LTP and certain forms of learning. This kinase consist of 12 subunits (alpha and beta) and activated by calcium-calmodulin and expressed presynaptically and postsynaptically. In one of the most important pathways, CaMKII is phosphorylated α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors (AMPARs) and increased conductance of the ion-channels. On the other hand, CaMKII increased the insertion of AMPARs at the postsynaptic membrane and enhanced the density of receptors at the neurons. Therefore, CaMKII seems prone to be a mediator of essential significance in connecting transient calcium signs to neuronal plasticity.

Keywords: Long-Term Potentiation, Calcium-Calmodulin-Dependent Protein Kinase Type 2, Neuronal Plasticity.

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