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Lecture presentation



# CaM kinases and AMPA receptor subunit recomposition in hippocampal synaptic plasticity

Victor Derkach

Address: Vollum Institute, Oregon Health and Science University, Portland, OR 97201-3098, USA

Email: Victor Derkach - derkachv@ohsu.edu

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It is broadly believed that synaptic plasticity is a neuronal mechanism for learning and memory in the mammalian brain. In the mature hippocampus, the expression of long-term potentiation (LTP) in Schaffer collateral-CA1 synapses requires a postsynaptic $Ca^{2+}$ influx and the GluR1 subunit of the AMPA subtype of glutamate receptor (AMPAR). New findings indicate that the pattern of synaptic activity associated with exploratory behavior can induce LTP by changing the quality of synaptic AMPARs. This process is dynamic and requires activity of $Ca^{2+}$/calmodulin dependent protein kinases (CaMKs), key transducers of postsynaptic $Ca^{2+}$ changes into LTP. The two CaMKs, CaMKI and CaMKII target AMPARs and regulate synaptic strength differently, however. Under basal conditions, AMPARs in these synapses are heteromers composed of GluR1 and GluR2 subunits. CaMKI enhances synaptic strength by trafficking to synapses more functionally efficient and highly $Ca^{2+}$-permeable GluR2-lacking AMPARs through a regulated actin dynamics. In contrast, CaMKII can enhance functional properties of these GluR2-lacking AMPARs by a direct phosphorylation of the C-terminus of GluR1 subunit. Taken together, these results argue for two distinct but orchestrated mechanisms in modification of synaptic strength during LTP. Results are discussed in terms of the role of AMPAR subunit recomposition for synaptic plasticity.