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Titel des Beitrags:
Role of hippocampal Cav1.2 Ca2+ channels in NMDA receptor-independent synaptic plasticity and spatial memory.

Abstract:
Current knowledge about the molecular mechanisms of NMDA receptor (NMDAR)-independent long-term potentiation (LTP) in the hippocampus and its function for memory formation in the behaving animal is limited. NMDAR-independent LTP in the CA1 region is thought to require activity of postsynaptic L-type voltage-dependent Ca2+ channels (Cav1.x), but the underlying channel isoform remains unknown. We evaluated the function of the Cav1.2 L-type Ca2+ channel for spatial learning, synaptic plasticity, and triggering of learning-associated biochemical processes using a mouse line with an inactivation of the CACNA1C (Cav1.2) gene in the hippocampus and neocortex (Cav1.2(HCKO)). This model shows (1) a selective loss of protein synthesis-dependent NMDAR-independent Schaffer collateral/CA1 late-phase LTP (L-LTP), (2) a severe impairment of hippocampus-dependent spatial memory, and (3) decreased activation of the mitogen-activated protein kinase (MAPK) pathway and reduced cAMP response element (CRE)-dependent transcription in CA1 pyramidal neurons. Our results provide strong evidence for a role of L-type Ca2+ channel-dependent,
NMDAR-independent hippocampal L-LTP in the formation of spatial memory in the behaving animal and for a function of the MAPK/CREB (CRE-binding protein) signaling cascade in linking Cav1.2 channel-mediated Ca2+ influx to either process.