Alpha1E-containing Ca2+ channels are involved in synaptic plasticity

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Long-term potentiation (LTP) is the most prominent model for the molecular and cellular mechanisms of learning and memory. Two main forms of LTP have been distinguished. The N-methyl-D-aspartate-receptor-dependent forms of LTP have been studied most extensively, whereas much less is known about N-methyl-D-aspartate-receptor-independent forms of LTP. This latter type of LTP was first described at the mossy fiber synapses in the hippocampus and subsequently at parallel fiber synapses in the cerebellum as well as at corticothalamic synapses. These presynaptic forms of LTP require a rise in the intraterminal calcium concentration, but the channel through which calcium passes has not been identified. By using pharmacological tools as well as genetic deletion, we demonstrate here that alpha1E-containing voltage-dependent calcium channels (VDCCs) shift the threshold for mossy fiber LTP. The channel is not involved in the expression mechanism, but it contributes to the calcium influx during the induction phase. Indeed, optical recordings directly show the presence and the function of alpha1E-containing VDCCs at mossy fiber terminals. Hence, a previously undescribed role for alpha1E-containing VDCCs is suggested by these results.
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