GS03-3 Zinc-mediated long-term potentiation in the hippocampus

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The hippocampus plays an important role in learning, memory and recognition of novelty, in which glutamatergic neurons are involved. Synaptic plasticity such as long-term potentiation (LTP) has been widely studied as a model of memory. LTP induction at Schaffer collateral-CA1 pyramidal cell synapses involves the increase in postsynaptic calcium concentration via N-methyl-D-aspartate (NMDA) receptor activation. In contrast, LTP induction at mossy fiber-CA3 pyramidal cell synapses is dependent on the increase in presynaptic calcium concentration, which activates the calcium-calmodulin-sensitive adenyl cyclase I. On the other hand, zinc exists in the synaptic vesicles of glutamatergic neurons and is released with glutamate in a calcium- and impulse-dependent manner. However, the role of synaptic Zn^{2+} in learning and memory is poorly understood. We have reported that Zn^{2+} released from neuron terminals may be a negative-feedback factor against presynaptic activity during tetanic stimulation. In the present study, the role of synaptic Zn^{2+} in LTP at Schaffer collateral-CA1 pyramidal cell synapses and mossy fiber-CA3 pyramidal cell synapses were examined in hippocampal slices prepared from rats. $ZnCl_2$ (5 μM) potentiated CA1 LTP, while attenuated CA3 LTP. Zn^{2+} released from mossy fibers may be taken up into the same terminals and inhibit the process of exocytosis. On the other hand, Zn^{2+} released from Schaffer collateral may enhance intracellular LTP signaling via NMDA receptor activation. The present study suggests endogenous Zn^{2+} multi-functionally modulates LTP in the hippocampus.