S26-3 Modulation of hippocampal long-term potentiation by zinc

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The hippocampus plays an important role in learning, memory and recognition of novelty, in which glutamatergic neurons are involved. A large portion of the total zinc in the hippocampus exists as zinc metalloproteins. The rest is histochemically reactive, exists in the presynaptic vesicles, and is released with glutamate; all giant boutons of mossy fibers contain zinc in the presynaptic vesicles, while approximately 45% of Schaffer collateral/commissural pathway is zinc-positive. Zn^{2+} released from neuron terminals may serve as a negative-feedback factor against presynaptic activity via crosstalk to Ca^{2+} and modulate postsynaptic neuronal activity. However, the role of Zn^{2+} in synaptic plasticity, a widely studied model of memory, is poorly understood. Long-term potentiation (LTP) at mossy fiber-CA3 pyramidal cell synapses, which is expressed by enhancement of glutamate release, is attenuated in the presence of 5 μM $ZnCl_2$. In contrast, LTP at Schaffer Collateral-CA1 pyramidal cell synapses, which is expressed when postsynaptic Ca^{2+} concentration reaches the threshold, is potentiated in the presence of 5 μM $ZnCl_2$. The present study is the first to demonstrate that low micromolar concentrations of Zn^{2+} differentially modulate mossy fiber LTP and CA1 LTP. Memory seems to be multifunctionally modulated by endogenously released Zn^{2+}.