S30-1 **Anxiety, fear and zinc signaling**

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The hippocampus plays an essential role in learning and memory, while the amygdala plays an important role in emotional memory. The accepted mechanisms of memory formation are synaptic plasticity. Long-term potentiation (LTP) is known as a cellular model of synaptic plasticity. Zinc is released into the synaptic cleft with glutamate, which plays a pivotal role in memory. In the hippocampus, Zn^{2+} attenuates mossy fiber LTP, while enhances CA1 LTP. Zn^{2+} is also required for LTP in the lateral amygdala. The modulation of LTP by Zn^{2+} may be linked to memory formation. On the other hand, the environment influences memory formation. The stressful environment affects synaptic plasticity and memory formation, while the enriched environment improves them. When rats are subjected to acute stress, zinc homeostasis in the extracellular compartment is significantly altered in the hippocampus and amygdala. This alteration may be involved in the attenuation of LTP in the hippocampus. In exposure to inescapable fear stress, freezing behavior in the retrieval of fear experience is increased in the presence of zinc chelator in the amygdala. Zinc released in the lateral amygdala may modulate the acquisition and retrieval of fear memory. Significance of zinc signaling in the control of memory will be discussed.