Contribution of Ca2+ release channels to hippocampal synaptic plasticity and spatial memory: Potential redox modulation

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Significance: Memory is an essential human cognitive function. Consequently, to unravel the cellular and molecular mechanisms responsible for the synaptic plasticity events underlying memory formation, storage and loss represents a major challenge of present-day neuroscience. Recent Advances: This review article first describes the wide-ranging functions played by intracellular Ca2+ signals in the activity-dependent synaptic plasticity processes underlying hippocampal spatial memory, and next, it focuses on how the endoplasmic reticulum Ca2+ release channels, the ryanodine receptors, and the inositol 1,4,5-trisphosphate receptors contribute to these processes. We present a detailed examination of recent evidence supporting the key role played by Ca2+ release channels in synaptic plasticity, including structural plasticity, and the formation/consolidation of spatial memory in the hippocampus. Critical Issues: Changes in cellular oxidative state particularly affect the function of Ca2+ r