Stress hormone modulation of plasticity in the amygdala

Abstract

The amygdala is necessary for the consolidation of emotional memory. A large body of evidence exists showing that stress and glucocorticoids produce an enhancement in the performance of memory tasks which require activation of the basolateral amygdala (BLA). Stress effects that aid learning in the BLA produce a deficit in hippocampal dependent memory and synaptic plasticity. The role of synaptic plasticity in the BLA during stress or glucocorticoid exposure has not been extensively explored. In this study I looked in the rat BLA at effects of the stress hormone corticosterone (CORT) on long-term potentiation (LTP), a cellular model for memory formation. Recording field potentials from the BLA via stimulation of the lateral amygdala in vitro. I found that both rapid and delayed effects of CORT produced an enhancement in LTP. This enhancement was mediated in the rapid effect by non-genomic mechanisms, and protein synthesis and glucocorticoid receptor (GR) dependent mechanisms in the delayed effect. In addition, I found that mineralocorticoid receptor (MR) blockade caused an enhancement in LTP, and B-adrenergic blockade concurrent with rapid CORT exposure increased the CORT enhancement of LTP. These results are opposite to those found in the hippocampus. The findings of this study help to understand the effects of stress on synaptic plasticity in the amygdala. In addition, these results not only provide a framework in which to interpret the behavioral effects of stress in the amygdala, but also shed light on why these effects differ from those seen in the hippocampus.