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Neuropeptide Induced Synaptic Plasticity: Structure and Function

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MODULATION OF DENDRITIC DIFFERENTIATION BY CORTICOTROPIN-RELEASING FACTOR IN THE DEVELOPING HIPPOCAMPUS

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The stress-activated neuropeptide corticotropin-releasing factor (CRF) is released from hippocampal interneurons by environmental signals, including stress (Chen et al., 2004. Neuroscience). In the adult hippocampus, the peptide increases synaptic efficacy in hippocampal principal cells, promoting stress-evoked enhancement of learning and memory. In the early postnatal hippocampus, we have previously characterized an additional, transient population of CRF-expressing Cajal-Retzius-like cells (Chen et al., 2001. J. Neurosci., 2001). Here we queried whether this stress-activated neuromodulator influences synaptic plasticity or even synapse formation in the developing hippocampal network. Using mice deficient in the principal hippocampal CRF receptor (CRF1 [-/−]) and organotypic cultures grown in the presence of synthetic CRF, or CRF receptor antagonists, we found robust effects of CRF on dendritic differentiation in hippocampal neurons. In CRF1 (-/-) mice, the dendritic trees of hippocampal principal cells were exuberant, an effect that was induced in normal hippocampi in vitro by the presence of CRF1 antagonists. In both cases, total dendritic length and dendritic branching were significantly increased. In contrast, exogenous synthetic CRF blunted dendritic growth in hippocampal organotypic cultures. Taken together, these findings suggest that endogenous CRF, if released excessively by pre- or early postnatal stress, might influence neuronal connectivity–and thus function–of the immature hippocampus.