Long-Term Changes (LTC) of Evoked Field Potentials in the Amygdala: A Model of Emotional Memory.

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Abstract: The identification of the amygdala as an essential neural substrate for fear conditioning has permitted neurophysiological examinations of synaptic processes in the amygdala that may mediate fear conditioning. We examined synaptic plasticity in the thalamo-amygdala pathway, known to mediate acoustic fear conditioning and the hippocampal-amygdala pathway, involved in contextual fear conditioning. Results indicate that theta burst high frequency stimulation can induce either LTP or LTD in both pathways. The mechanisms responsible for long-term changes (LTC) in the amygdala are, to a great extent, elusive and further work is planned to elucidate them.

Keywords: Amygdala / long-term potentiation / long-term depression / emotional memory / fear conditioning / rat.
1. INTRODUCTION

It is well established that emotionally neutral stimuli can acquire the capacity to evoke striking emotional reaction following temporal pairing with an aversive event. Conditioning does not create new emotional responses but instead simply allows new stimuli to serve as triggers capable of activating existing, often hard-wired, species-specific emotional reactions. In the rat, for example, a pure tone previously paired with footshock evokes a conditioned fear reaction consisting of freezing behavior accompanied by a host of autonomic adjustments, including increases in arterial pressure and heart rate (5,24).

In addition to its role in appetitive and perhaps attentional processes (e.g. ref. 14), converging evidence now indicates that the amygdala, and its many efferent projections, may represent a central fear system involved in the acquisition and expression of learned fear, both in experimental animals and humans (4, 11). The amygdala receives highly processed sensory information from all modalities through its lateral and basolateral nuclei (2, 25). In turn, these nuclei project to the central amygdaloid nucleus (1, 2, 32), which then projects to a variety of hypothalamic and brainstem target areas that directly mediate specific signs of fear and anxiety (cf ref. 11). Amygdala lesions, particular of the central nucleus, reduce or abolish various conditioned fear-related behaviors in a number of mammalian species. Acquisition and retention