

NEURAL MECHANISMS UNDERLYING AFFECTIVE MEMORY OF THE INHIBITORY AVOIDANCE RESPONSE IN RATS

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This review intends to integrate our findings on neural processes underlying acquisition, consolidation and retrieval of inhibitory avoidance memory in rats, and to discuss their implications on some fundamental issues concerning the neural substrate of affective memory. Our results suggest that enduring memory for emotional events relies on memory-enhancing effects of stress-released hormones or neurotransmitters, such as epinephrine, corticotropin releasing factor, and central norepinephrine, which exert their influences on memory directly or indirectly via the amygdala, hippocampus, locus coeruleus, etc. These structures are involved not only in formation of emotional memory but also in routine processing of threatening stimuli and initiating emotional responses. Blocking AMPA or NMDA receptors in the amygdala impaired inhibitory avoidance memory, suggesting that glutamate fibers provide the amygdala with sensory information and may induce some form of neural plasticity such as long-term potentiation. However, formation of emotional memory is not exclusively mediated by NMDA-dependent neural plasticity, because norepinephrine could attenuate the amnesia caused by blockade of NMDA receptors. Retrieval of recent (1-day) and remote (21-day) emotional memory depended upon the amygdala and the medial prefrontal cortex, respectively. Because the medial prefrontal cortex was also engaged during training and played a role in establishing a long-term trace, it is suggested that for an emotional experience, multiple memory traces with dynamic courses are formed in the brain to retain the experience for various lengths of retention intervals. These findings suggest that an affective experience engages multiple brain structures, activates various forms of neural plasticity, and establishes compound traces with different temporal dynamics. Such information on how the brain processes and stores emotional events in rats may contribute to our construction of functional models for human memory.

Keywords: Classical conditioning, Operant conditioning, Amygdala, Hippocampus, Locus coeruleus, Stress-related hormones, Glutamate, Long-term potentiation.