13. ABSTRACT (Maximum 200 words)

Research during this funding period has focused on the role of a particular brain area, the amygdala, in fear conditioning, using increased acoustic startle amplitude in the presence of a stimulus previously paired with shock as a measure of fear in rats. We have found that a) electrical stimulation of the amygdala increases startle; b) mechanical or chemical lesions of the amygdala prevent either footshock or stimuli paired with footshock from elevating startle; c) there is a direct anatomical connection between the central nucleus of the amygdala and a specific point along the acoustic startle pathway; d) lesions at several levels of this connection between the amygdala and the startle circuit block both conditioned and unconditioned fear; e) local infusion of specific receptor antagonists (N-methyl-D-aspartate receptors) into the amygdala prevent the development of fear conditioning and f) presentation of a conditioned fear stimulus activates early expression genes (c-fos) in the amygdala. The data strongly implicate the amygdala as a critical brain structure for both the acquisition and expression of conditioned and unconditioned fear. Drugs that reduce anxiety in humans may act by interacting with specific receptors in the amygdala.
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Final Technical Report

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AFOSR-87-0336

Publications:


Boulis, N.M. and Davis, M. Blockade of the spinal excitatory effect of cAMP on the startle reflex by intrathecal administration of the isoquinoline sulfonamide H-8: Comparison to the C-kinase inhibitor H-7. *Brain Research*, 1990, 525, 198-204.


