



Use of a Gene and Related Mouse Model for the Study and Development of Therapeutics for Neuropsychiatric Disorders

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INNOVATION

UCLA investigators have characterized an association between the expression of a single gene and behavioral learning associated with forebrain function. The gene is preferentially expressed in the cell bodies and dendrites of post-natal neurons of the forebrain. Since the gene is not expressed until after birth, it does not appear to be involved in development. To assess the physiological role of the gene, knockout (gene disruption) mice were developed. The null mice appear normal and reproduce normally, but show enhanced amygdala-dependent long-term memory consolidation with a concomitant elevation in amygdala, but not hippocampal, long-term potentiation (LTP). Hippocampal-dependent learning and motor skills appear normal, but mutant mice showed specific enhancement of amygdala-dependent learning of fear as assessed by cued (tone) conditioning and taste aversion protocols. These findings represent a rare instance of elevated learning and memory that is localized and experimentally accessible to both in vitro and in vivo analyses, and is the only such case associated with fear and emotion learning. Mutations of this gene may be a contributing factor in human neuropsychiatric disorders characterized by increased excitatory activity in the amygdala or in amygdala dependent circuits. This gene, and reagents developed from it, should provide a useful model system for the development of therapeutic interventions.

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OTHER INFORMATION

KEYWORDS

therapeutics

CATEGORIZED AS

- Medical
 - Disease: Central Nervous System
 - Research Tools
- Research Tools
 - Animal Models

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