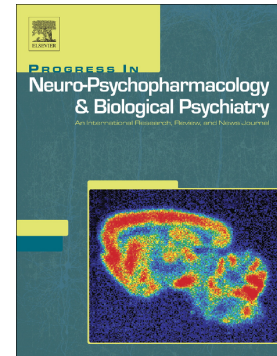


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Ketamine-induced hypnosis and neuroplasticity in mice is associated with disrupted p-MEK/p-ERK sequential activation and sustained upregulation of survival p-FADD in brain cortex: Involvement of GABAA receptor



Glòria Salort, María Álvaro-Bartolomé, Jesús A. García-Sevilla

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Ketamine-induced hypnosis and neuroplasticity in mice is associated with disrupted p-MEK/p-ERK sequential activation and sustained upregulation of survival p-FADD in brain cortex: Involvement of GABA_A receptor

Glòria Salort, María Álvaro-Bartolomé, Jesús A* jesus.garcia-sevilla@uib.es.
García-Sevilla*

Laboratory of Neuropharmacology, Institut Universitari d'Investigació en Ciències de la Salut (IUNICS), University of the Balearic Islands (UIB), and Institut d'investigació Sanitària Illes Balears (IdISBa), Palma de Mallorca, Spain

*Corresponding author at: Laboratori de Neurofarmacologia, IUNICS/UIB, Cra. de Vallemossa km 7.5, E-07122 Palma de Mallorca (Balears), Spain.

Abstract

Ketamine (KET) is an antidepressant and hypnotic drug acting as an antagonist at excitatory NMDA glutamate receptors. **The working hypothesis postulated that KET-induced sleep in mice results in dysregulation of mitogen-activated protein kinases (MAPK) MEK-ERK sequential phosphorylation and upregulation of survival p-FADD and other neuroplastic markers in brain.** Low (5-15 mg/kg) and high (150 mg/kg) doses of KET on target proteins were assessed by Western immunoblot in mouse brain cortex. During the time course of KET (150 mg/kg)-induced sleep (up to 50 min) p-MEK was increased (up to +79%)

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