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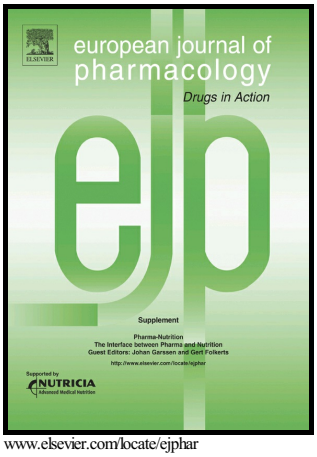

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Very low doses of muscimol and baclofen ameliorate cognitive deficits and regulate protein expression in the brain of a rat model of streptozocin-induced Alzheimer's disease

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Abstract

Recent studies devoted to neuroprotection have focused on the role of the gamma-aminobutyric acid (GABA) system in regulating neuroinflammatory processes which play a key role in the neurodegenerative processes observed in Alzheimer's disease (AD) by inducing glial cell overactivation and impairing neurotransmission.

Data on the efficacy of classical GABA-A and GABA-B receptor agonists (muscimol and baclofen, respectively) in animal models of AD are not available. Moreover, no published studies have examined the ability of optimal doses of these compounds to prevent neuroinflammation, the alterations in neurotransmission and cognitive deficits. In the present study, we used a non-transgenic rat model of AD obtained by intracerebroventricular streptozocin (STZ) injection and assessed the effects of muscimol and baclofen at very low doses (0.01 – 0.05 mg/kg) on spatial memory and the expression of cortical and hippocampal proteins related to neuroinflammation, namely proteins involved in astroglial functions (glial

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