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Methyl jasmonate attenuates memory dysfunction and decreases brain levels of biomarkers of neuroinflammation induced by lipopolysaccharide in mice

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Highlights

- MJ reversed LPS-induced memory deficits in mice.
- The increased brain levels of PGE2, TNFα and IL1β in LPS-treated mice were reduced by MJ
- MJ suppressed the expression of COX2, iNOS and NFκB in LPS-treated mice
- Increased brain level of Aβ in LPS-treated mice was suppressed by MJ

Abstract

Neuroinflammation plays a central role in the etiology and progression of Alzheimer's disease (AD), a neurodegenerative disorder, characterized by a gradual loss of memory functions. Thus, it has been proposed that agents that could reduce inflammatory processes in AD brains might be useful for the treatment of the disease. Methyl jasmonate (MJ) is a bioactive compound, which has been reported to exhibit anti-amnesic and *in vitro* anti-inflammatory activities. In this study,