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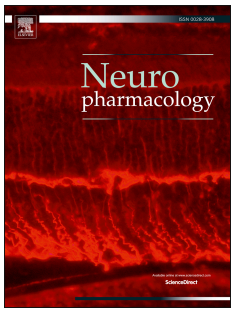

Neuroinflammation produced by heavy alcohol intake is due to loops of interactions between toll-like 4 and TNF receptors, peroxisome proliferator-activated receptors and the central melanocortin system: A novel hypothesis and new therapeutic avenues

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Neuroinflammation produced by heavy alcohol intake is due to loops of interactions between Toll-like 4 and TNF receptors, peroxisome proliferator-activated receptors and the central melanocortin system: A novel hypothesis and new therapeutic avenues

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ABSTRACT

Excessive alcohol intake induces an inflammatory response in the brain, via TNFα, TLR4 and NF-κB signaling pathways. It has been proposed that neuroinflammation would play a very important role in the development of alcohol addiction. In addition to stimulating the synthesis of inflammatory mediators such as IL-6, IL-1β and TNFα, NF-κB is capable of reducing the anti-inflammatory activity of PPARα and PPARγ. Reciprocally, PPARα, PPARγ and melanocortin 4 receptor (MC4R) can decrease the proinflammatory activity of NF-κB, establishing an interplay of inactivations between such