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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 TNF α , TLR4 and NF- κ B 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-1 β and TNF α , NF- κ B is capable of reducing the anti-inflammatory activity of PPAR α and PPAR γ . Reciprocally, PPAR α , PPAR γ and melanocortin 4 receptor (MC4R) can decrease the proinflammatory activity of NF- κ B, establishing an interplay of inactivations between such

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