Protective effect of curcumin against chronic alcohol-induced cognitive deficits and neuroinflammation in the adult rat brain.

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Abstract
Chronic alcohol intake is known to induce the selective neuronal damage associated with increase oxidative-nitrosative stress and activation of inflammatory cascade finally resulting in cognitive deficits. In the present study, we investigated the protective effect of curcumin, a potent natural anti-oxidant and anti-inflammatory molecule against chronic alcohol-induced cognitive dysfunction and nuclear factor kappa beta (NF-κβ) mediated inflammatory signaling in the brain of rats chronically administered ethanol. Male Wistar rats were given ethanol (10 g/kg; oral gavage) for 10 weeks, and treated with curcumin (15, 30 and 60 mg/kg) for the same duration. Ethanol-exposed rats showed impaired spatial navigation in the Morris water maze test and poor retention in the elevated plus maze task which was coupled with enhanced acetylcholinesterase activity, increased oxidative-nitrosative stress, cytokines (tumor necrosis factor alpha (TNF-α) and interleukin-1 beta (IL-1β)), NF-κβ and caspase-3 levels in different brain regions (cerebral cortex and hippocampus) of ethanol-treated rats. Co-administration with curcumin significantly and dose-dependently prevented all the behavioral, biochemical and molecular alterations in rats chronically administered ethanol. Thus, findings from the current study demonstrates the possible involvement of oxidative-nitrosative stress mediated cytokine release and inflammatory signaling in chronic alcohol-induced cognitive dysfunction and also suggests the effectiveness of curcumin in preventing cognitive deficits associated with chronic alcohol consumption.

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