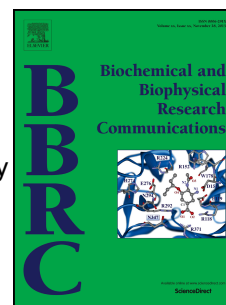


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Hepatoprotective effects of Methyl ferulic acid on alcohol-induced liver oxidative injury in mice by inhibiting the NOX4/ROS-MAPK pathway

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Abstract

Aims: The present study aimed to investigate the hepatoprotective effects of Methyl ferulic acid (MFA) against oxidative stress and apoptosis as well as inflammation in mice with liver injury induced by alcohol and its underlying mechanisms.

Methods: C57BL/6 mice were divided into a control group, a model group, and Methyl ferulic acid with high dosage (20 mg/kg), moderate dosage (10 mg/kg) and low dosage (5 mg/kg) groups. The general condition and organ index of each group were investigated. Histopathological analysis was performed to determine the degree of hepatic injury. Biochemical analyses of functional liver enzymes, lipid peroxidation enzymes and lipid content in each group. The levels of inflammatory cytokines were measured by enzyme-linked immunosorbent assay (ELISA). The mechanisms were investigated by detecting levels of NADPH Oxidase 4 (NOX4), p22phox, cytochrome P4502E1 (CYP2E1), Bax, B-cell lymphoma 2 (Bcl-2), cleaved-caspase 3 and 9 and phosphorylated extracellular regulated protein kinases (ERK), phosphorylated c-Jun N-terminal kinase (JNK), and phosphorylated p38 mitogen-activated protein kinase (MAPK) using real-time polymerase chain reaction (PCR) and Western blotting.

Results: MFA treatment significantly decreased serum enzymatic activities of alanine aminotransferase (ALT) and aspartate aminotransaminase (AST). MFA markedly increased levels of superoxide dismutase (SOD), catalase (CAT), glutathione (GSH), glutathione peroxidase (GSH-Px) and total antioxidative capacity (T-AOC), and reduced the concentration of malondialdehyde (MDA) and reactive

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