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In vivo hepatoprotective effect of *Salvia miltorrhiza* Bunge against ethanol-induced oxidative stress

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The long-term heavy consumption of alcohol results in the development of alcohol-related liver disease, which is the second leading cause of death among all liver diseases[1–2]. Oxidative stress is considered as one of the key mechanisms responsible for alcoholic liver damage[3–4]. In the present study, the protective effects of 5% ethanol extract (SME) from *Salvia miltorrhiza* Bunge. against alcoholic liver damage were investigated in male C57BL/6 mice. Mice (*n* 9 per group), which received SME (100 or 400 mg/kg b.w./d) with ethanol revealed complete prevention of alcohol-induced hepatotoxicity as evidenced by the significant reductions of serum aspartate aminotransferase and alanine aminotransferase activities, compared with ethanol-alone administered mice (5 g ethanol/kg b.w./d). When compared with the ethanol-alone treated group, the mice receiving ethanol plus SME exhibited significant increases in hepatic antioxidant activities, including superoxide dismutase, catalase, glutathione-*S*-transferase, glutathione peroxidase, glutathione reductase and glutathione. Furthermore, the amelioration of malondialdehyde levels indicated SME's protective effects against liver damage mediated by alcohol *in vivo*. Also, the pre-treatment with SME significantly suppressed ethanol-induced increase in the expression of cytochrome P-450 2E1 (CYP2E1), a major contributor in generating a state of oxidative stress, which results in hepatotoxicity[5]. These results suggest that 5% ethanol extract of *S. miltorrhiza* Bunge. has protective action against alcohol-induced toxicity in the liver by suppressing the expression of CYP2E1 and recovering the antioxidant status.

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