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L-carnitine inhibits ethanol-induced gastric mucosal injury in rats

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Abstract:
L-carnitine is a quaternary amine that is essential for the normal oxidation of long-chain fatty acids by mitochondria. It is known that L-carnitine and its derivatives prevent the formation of reactive oxygen species, scavenge free radicals and protect cells from peroxidative stress. Oxygen-derived free radicals and lipid peroxidation products play a critical role in the pathogenesis of ethanol-induced gastric mucosal injury. The aim of the present study was to determine the effect of L-carnitine on lipid peroxidation induced by ethanol in the rat stomach. In our study, gastric mucosal injury was induced by the intragastric administration of 1 ml of absolute ethanol. Test compounds were given to rats by gavage 30 min before the ethanol administration. The animals were killed 60 min after the administration of ethanol. The stomach of each animal was removed. Mucosal damage was evaluated by macroscopic examination, histological analysis and by measurement of lipid peroxidation and glutathione activity. The intragastric administration of ethanol induced hyperemia and hemorrhagic erosions in the rat stomachs. L-carnitine significantly prevented gastric ulcerogenesis induced by ethanol and decreased the ulcer index. Plasma and gastric lipid peroxidation that was increased significantly by ethanol was decreased after treatment with L-carnitine. Ethanol treatment decreased significantly the gastric glutathione levels, and pretreatment with L-carnitine increased them significantly. Based on these data, the beneficial effects of L-carnitine on ethanol-induced gastric mucosal injury may be attributed to its antiperoxidative effects.

Key words:
carnitine, ethanol, gastric mocosal injury, lipid peroxidation