

Alcohol hepatic toxicity in rat: evidence of the utility of gastric ethanol metabolism

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Abstract

Since a fraction of ingested ethanol (EtOH) is metabolized by gastric mucosa, different amounts of alcohol should reach the liver, when the same dose is administered by oral or intravenous route. Accordingly, we demonstrated that the hepatic depletion of glutathione induced by EtOH is lower when it is administered orally rather than intraperitoneally (i.p.). In the present study we investigated, after EtOH load, the time-course of common serum liver damage tests and of α -glutathione-S-transferase (α GST) levels as a new indicator of hepatocellular injury in rats. The tests were also performed in Cimetidine-treated rats. Oral EtOH administration was followed by a less pronounced decrease and by a quicker recovery of hepatic glutathione than after i.p. route. After oral EtOH load, Cimetidine, a potent inhibitor of gastric alcohol-dehydrogenase, produced a decrease of hepatic glutathione significantly ($P < 0.005$) more pronounced than in controls. Serum α GST increased significantly ($P < 0.05$) 6 h after i.p. EtOH administration, whereas no modifications were found after oral EtOH load. Common liver damage tests did not show any modification. In Cimetidine-treated rats, oral EtOH load was followed by an increase of serum α GST similar to that after i.p. administration. This study demonstrates the beneficial effects of gastric

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