The administration of ethionine induced the damage to liver parenchyma as indicated by SGOT value. Ethanol could protect against this effect presumable by competing with ATP trapping process of ethionine and thereby help to provide the continual, even though minimal, supply of adenine nucleotides. The toxicity of ethionine decreased after 8 weeks of feeding. The induction of drug metabolizing enzyme, particularly microsomal cytochrome $P_{450}$ was postulated to be responsible to this effect.

The carcinogenicity of ethionine was significantly enhanced by ethanol administration. The lesions were range from islands of the oval cells to hyperplastic nodules or cholangiomas. These lesions were amplified both in size and incidence after partial hepatectomy. The enhancing effect of ethanol was proposed as through the activation of S-adenosyl methionine synthetase enzyme which would result in increase the ethylation precursor, SAE.
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