Alcohol ingestion, liver glutathione and lipoperoxidation: Metabolic interrelations and pathological implications

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Data reviewed here indicate that acute and chronic ethanol ingestion induce a decrease in the concentration of GSH and an increase in lipoperoxidation in the liver both in experimental animals and in man, changes that are closely interrelated GSH depletion is suggested to be due to an oxidation in the liver tissue and to a translocation into the extrahepatic medium as free glutathione and/or as conjugates with ethanol-derived acetaldehyde. As a result, the hepatic GSH/GSSG ratio is drastically reduced. Lipoperoxidation seems to be related to the metabolism of ethanol and acetaldehyde by secondary pathways that are known to generate oxygen-related free radicals. Being lipoperoxidation a process associated with cell damage and death, its stimulation by ethanol ingestion could play a role in the production of alcoholic liver damage in man. The involvement of several contributory factors in the development of a high lipoperoxidative index in the liver in this situation is discussed. © 1982