REVERSAL OF ETHANOL-INDUCED HEPATIC STEATOSIS AND LIPID PEROXIDATION BY TAURINE: A STUDY IN RATS

STEFAN BLEICH and DETLEF DEGNER

Department of Psychiatry, Georg-August-University of Göttingen, Von-Siebold Str. 5, 37075 Göttingen, Germany

(Received 6 October 1999; accepted 1 November 1999)

We read with interest the study reported by Kerai et al. in the July–August (1999) issue of Alcohol and Alcoholism. The authors concluded that hepatic steatosis and lipid peroxidation caused by chronic alcohol consumption in rats can be reversed by administration of taurine. Furthermore, an increased urine excretion of homocysteine during alcohol withdrawal was found. Recently, it has been shown that chronic alcoholism is associated with hyperhomocysteinaemia (Hultberg et al., 1993; Cravo et al., 1996). Hyperhomocysteinaemia was observed in chronic alcoholics who underwent withdrawal from alcohol (Bleich et al., 1999), whereas no significant changes of taurine concentrations in alcohol-dependent patients were observed (Badawy et al., 1998). It has been suggested that withdrawal symptomatology could result from increased activity of excitatory mechanisms (e.g. NMDA receptor) and from reduced functioning of inhibitory receptors (e.g. GABA \(_\alpha\) receptor) and it has been shown that chronic treatment with ethanol leads to an increase of the NMDA receptor number at transcriptional and post-transcriptional levels (Grant et al., 1990). In addition to the excitatory role played by the amino acid transmitters glutamate and aspartate in the central nervous system, their sulphur-containing analogues homocysteic acid (HCA) and cysteine sulphinic acid (CSA) may also play a similar role. These latter compounds are oxidation products of homocysteine and cysteine, are putative neurotransmitters and are endogenous agonists at the NMDA receptor, a subtype of the glutamate receptor (Cuenod et al., 1990). In addition, it has been shown that homocysteine itself acts as an agonist at the glutamate binding site of the NMDA receptor (Lipton et al., 1997), whereas taurine and hypotaurine may act as inhibitory transmitters (Saransaari and Oja, 1999).

In summary, in the metabolic pathway from methionine to taurine and in its branch pathways, excitatory sulphur-containing amino acids are formed. There is growing evidence that chronic alcoholism is associated with a derangement in the sulphur amino acid metabolism. Ethanol-induced hyperhomocysteinaemia with subsequent accumulation of the excitatory neurotransmitters may partly mediate the variety of symptoms which are seen in alcohol withdrawal. Hyperhomocysteinaemia is a treatable condition taking into account that folate therapy will reliably reduce plasma homocysteine levels. Furthermore, the administration of taurine or taurine-like agents acting as inhibitory neurotransmitters might be beneficial in patients undergoing alcohol withdrawal. In conclusion, taurine appears to be useful in ameliorating ethanol-induced hepatic steatosis and could also be effective against alcohol-withdrawal symptomatology. Furthermore, the inhibitory amino acid taurine may constitute an important protective mechanism against excitotoxicity, which leads to neuronal damage. Further investigations and controlled studies are therefore needed to clarify the role of homocysteine and taurine in patients with alcoholism.

REFERENCES


Editor's Note — Dr Catherine J. Waterfield has seen this Letter and welcomes the comments of Dr Bleich (AA-BB).