

## ZINC, ALCOHOL AND FREE RADICAL DAMAGE

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The possibility has been recognized for several decades that alcohol may disturb the balance of certain essential nutrients in the body, and human and animal studies now link the consumption of ethanol to changes in the body status of several essential trace elements. Little is known concerning the functional significance of these changes, but some possibly reflect underlying biochemical adaptations that are important during alcohol-related pathology or in relation to alcohol-induced cellular defence mechanisms. With regard to zinc, an especially interesting situation has arisen, following suggestions that the hyperzincuria and low serum and liver zinc levels, often reported to accompany prolonged alcohol abuse, may reflect an induced zinc deficiency; which in turn might be responsible for some aspects of the chronic alcohol syndrome and possibly for the morphological abnormalities of the fetal alcohol syndrome.

It seems unlikely however that alcohol damage occurs entirely due to an alcohol-induced zinc deficiency. It is more likely that each factor exerts its influence independently in the body, but that their effects may overlap at a common biochemical locus. The suggestion was therefore made (Dreosti 1984) that at least some aspects of alcohol and zinc deficiency related toxicity may lie in the role played by zinc in the scavenging of free radicals released during the metabolism of ethanol, and in membrane lipid antioxidant mechanisms. Certainly, earlier studies in our laboratory pointing to increased levels of superoxide dismutase in adult and fetal rat livers exposed to gestational alcoholism suggest enhanced cellular free radical defence in response to treatment with alcohol.

The proposal was examined directly in our laboratory by measuring the lipid peroxidation product, malondialdehyde, in liver microsomes from zinc-deficient, adult and fetal rats treated with ethanol. Malondialdehyde levels were found to be sharply higher (60%) in zinc-deficient adult animals than in controls and slightly higher (21%) in the alcohol-treated group. Augmentation of peroxidation was not evident when the treatments were combined. In fetal liver, malondialdehyde levels were significantly higher (30%) in zinc-deficient pups, but unchanged in alcohol-treated animals. When in utero alcoholism and zinc deficiency were superimposed, levels rose more than 300% above the zinc-supplemented, alcohol treated pups and a little more than 200% above zinc-deficient pups not exposed to maternal alcoholism. Taken together with our earlier data concerning superoxide dismutase, the present findings suggest an involvement of the fetus in the metabolism of alcohol and a degree of zinc-sensitive pathology in fetal livers which is probably related to free radical damage of membrane lipids.

DREOSTI, I.E. (1984). In 'Mechanisms of Alcohol Damage in utero', Ciba Foundation Symposium 105, p 103, (Pitman; London).