

EFFECTS OF ALCOHOL AND ZINC DEFICIENCY ON TWO RAT ZINC METALLOENZYMES

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The teratogenic effects of zinc deficiency (Hurley & Swenerton, 1966) and chronic alcoholism (Jones & Smith, 1975) have been well established. Following a report by Wang & Pierson (1975) which showed decreased levels of zinc in plasma, liver and muscle tissue of alcoholic rats, it was decided to examine the individual and combined effects of alcohol ingestion and zinc deficiency on two zinc metalloenzymes in rat foetal (20 day) and maternal tissues.

Maternal liver alcohol dehydrogenase (A.D.H.) and foetal liver and brain alkaline phosphatase (A.P.) were selected, as A.D.H. is an important enzyme in the detoxification of ethanol, and A.P. activity has been shown to parallel maturation of rat brain (Cohn & Richter, 1956).

The activity of A.D.H. in the liver fell by approximately 60% in the dams deprived of zinc throughout gestation. Zinc deficient rats receiving 20% alcohol in their drinking water over this period displayed a slightly higher level of A.D.H. Animals supplemented with 500 ppm zinc, and receiving 20% alcohol, had normal levels of the enzyme.

The levels of A.P. in the livers and brains of zinc deficient fetuses were approximately 50% of those found in the controls. When the dams received 20% alcohol, with or without 500 ppm zinc during pregnancy, the levels in both tissues returned to normal.

Zinc balance studies (Dreosti *et al*, 1978) suggest that initially the ingestion of alcohol mobilizes zinc from certain body tissues and leads to a slight elevation in plasma zinc levels. It would appear from these results that some of this plasma zinc is available for the synthesis of hepatic alcohol dehydrogenase as well as for synthesis of alkaline phosphatase in the foetus.

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