

ASPECTS OF ZINC DEFICIENCY IN THE HIPPOCAMPUS AND CEREBELLUM
OF SUCKLING RATS

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Severe dysmorphogenesis of the CNS occurs in fetal rats exposed to prenatal zinc deficiency (Hurley and Shrader 1972; Adeloye and Warkany 1976). Postnatal zinc depletion in suckling rats affects behaviour, but not the overall development of the CNS (Buell *et al.* 1977).

In rats, the hippocampus and the cerebellum develop in the first few weeks postnatally, during which time zinc is accumulated specifically in the terminal boutons of the hippocampal mossy fibres (Crawford and Connor 1972). The metal has been suggested to be involved in synaptic transmission in these neurons, possibly through the action of the zinc metalloenzyme L-glutamic acid dehydrogenase (EC 1.4.1.3) (Crawford and Connor 1973). Indeed, Hesse (1979) has reported impaired electrophysiological activity in hippocampi from chronically zinc-deficient adult rats.

In the present study, rat pups were rendered zinc-deficient from birth by restricting the zinc intake of the lactating dam. At 18 days of age cerebella and hippocampi were removed and examined histologically and biochemically. The intensity of Timm's silver sulphide staining indicated considerably less zinc in the mossy fibre layer of the hippocampi in zinc-deprived animals than in the controls. Total hippocampal zinc levels were also perceptibly diminished.

DNA synthesis, as measured by the incorporation of ^3H -thymidine into DNA, was unaffected by postnatal zinc restriction in either brain region, but the activity of the enzymes 2',3'-cyclic nucleotide 3'-phosphohydrolase and L-glutamic acid dehydrogenase were both significantly lower in zinc-deprived hippocampi.

The present data demonstrate a specific biochemical lesion in the hippocampus associated with zinc restriction during early postnatal development in rats. The defect may contribute in part to the behavioural anomalies reported to accompany zinc deficiency during suckling in certain experimental animals.

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