

GESTATIONAL ALCOHOLISM AND HIPPOCAMPAL DEVELOPMENT IN ZINC-DEFICIENT FETAL RATS

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In animals, zinc deficiency and alcohol individually affect morphological development of the central nervous system. Furthermore, even in the absence of overt teratology, behavioural and learning deficits are a common feature of both conditions (Henderson et al. 1981, Hurley 1981). Little is known, however, concerning the possibility of an interaction of these factors at more subtle levels of neural organisation, although a case exists for consideration of an alcohol/zinc interaction specifically involving the hippocampus and possibly localized in the mossy fiber pathway (Dreosti et al. 1981).

Accordingly, in the present study an examination was made of the cellular development of the hippocampus in 20-day-old rat fetuses exposed to either gestational alcoholism or to maternal zinc deficiency and in some cases to both. Zinc deficiency reduced cell density in both the granule cell layer of the dentate gyrus and the pyramidal cell layer of the Horn of Ammon. Ethanol, alone, reduced the total number and the density of cells in the granule cell layer in the dorsal arm of the dentate gyrus and, when imposed concurrently with zinc deficiency, the deleterious effects of both treatments appeared to be additive. Cells in the pyramidal layer were not affected by gestational alcoholism but, together, the treatments led to reduced cell numbers in the CA3c and CA4 subregions of the hippocampus. The possibility is raised that some aspects of the behavioural deficits associated with gestational zinc restriction and with alcoholism in animals may arise from impaired development of the fetal hippocampus. Attention is drawn to the possibility of an interaction between treatments at this level.

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