COMPOSITIONAL DEVELOPMENT OF THE ZINC-DEFICIENT FETAL RAT

I.R. RECORD,* I.E. DREOSTI,* S.J. MANUEL* and R.A. BUCKLEY*

Previously other workers (eg Hurley 1972) have extensively described the teratogenic effects of zinc deficiency during the development of the fetal rat. The range of abnormalities produced by a deficit of this trace element extends from relatively minor skeletal defects, such as fusion of the digits through to severe malformations of the central nervous system and intra-uterine death. In our laboratory teratological defects, principally hydrocephalus and anophthalmia, were seen in a number of zinc-deficient fetuses, but not in the control offspring.

At this time it is not known whether zinc deficiency exerts its primary effect on cell division and fetal development by inhibition of DNA, RNA or protein synthesis, or by interference with some other general control point in the cell cycle.

As part of our investigations into the mechanism whereby a lack of dietary zinc produces its teratogenic effects, we have monitored the accretion of DNA, RNA, protein and zinc by zinc-deficient and replete fetal brains and livers throughout the last third of gestation.

Body, liver and brain weights of the zinc deficient fetuses were only marginally less than those of the zinc replete controls until day 20 of gestation, when they became significantly less. Accretion of RNA, DNA and protein did not appear to be adversely affected until day 19.

Despite the low maternal plasma zinc values obtained from all zinc-deficient dams, the zinc content of fetal livers and brains was not lower than the control tissues until just prior to parturition.

These results suggest that fetuses surviving to day 14 are able to acquire zinc from even a zinc-deficient dam for most of the latter part of pregnancy, which allows normal compositional development to continue over the next few days. In the period just prior to parturition when the fetuses exert a large demand for zinc due to their rapid growth, the dam becomes unable to mobilize sufficient zinc from her own stores to accommodate fetal requirements with a resultant retardation in the rate of cell division.

HURLEY, L S (1972) in "Neurobiology of the Trace Elements Zinc and Copper" (C C Pfeiffer, ed) Academic Press, New York.

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* CSIRO Division of Human Nutrition, Kintore Avenue, Adelaide, SA 5000.