DEVELOPMENT OF ZINC-DEFICIENT RAT EMBRYOS IN CULTURE

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In rats, a maternal dietary zinc deficiency has been shown to be highly teratogenic, resulting in developmental abnormalities in every organ system. Many of these abnormalities arise during the critical periods of organogenesis, especially during the time of closure of the neural tube (about d 11 in the rat). Advances in technique have now made it possible to remove rat embryos from the dam after 9.5 days gestation, and to culture egg cylinders in vitro for up to 48 h, during which time development proceeds at the same rate as in vivo. Use has been made of this technique to study the development of the zinc deficient rat embryo during the time when the embryo is most susceptible to teratogenic stimuli.

Normal 9.5 d embryos obtained from Sprague-Dawley rats grew and developed normally in sera obtained from both zinc-deficient and replete rats. Embryos from some dams fed the zinc-deficient diet since mating were found to be morphologically normal, whilst other dams provide embryos which appeared stunted and/or malformed. These latter embryos developed abnormally in culture, regardless of the serum in which they were incubated. Morphologically normal zinc-deficient embryos, however, grew and developed to the same extent as control embryos, even in zinc-deficient sera. Inclusion of ^{65}Zinc in the medium showed that all embryos were able to obtain sufficient zinc from the media to maintain growth over this period. The results of these studies suggest that abnormal embryonic development due to zinc deficiency cannot be induced in vitro, however, it would appear that a maternal zinc deficiency can exert its effect prior to day 9.5 of gestation, and that these effects are not easily reversible.

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