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Iron deficiency in maternal rats influences cell membrane function in both the mother and neonate

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Iron (Fe) deficiency is one of the most common nutritional disorders worldwide. It is especially serious during pregnancy, with short- and long-term health consequences for both the mother and her developing fetus. These include fetal growth retardation, obesity and increased blood pressure in the adult offspring. We have designed a rat model to investigate why fetal growth and development are adversely affected by maternal Fe deficiency. In this abstract, we examine the effect of maternal Fe deficiency on the osmotic sensitivity of erythrocytes taken from both mothers and their neonates. We correlate the data with the maternal Fe status, as estimated by Fe levels in both the maternal and neonatal livers. Weanling Rowett Hooded Lister female rats were fed a control diet for 2 weeks. Thereafter, one group continued on control and another on Fe-deficient diets for 4 weeks. They were mated and kept on the same diet throughout their pregnancy. At term, the dams either continued on the same diet or switched to a control diet. The dams and their neonates were killed at different times after birth. At 7 days after birth, the neonates from Fe-deficient dams were smaller than those from the control dams (10.1 g (SE 1.10) and 13.5 g (SE 0.48); $n = 4$ and 6, $P < 0.01$). No effect of Fe deficiency was observed on the maternal weights. Dams on Fe-deficient diet had lower haematocrits (38% (SE 0.65) and 28% (SE 0.75); $n = 6$ and 4, $P < 0.001$). Similar reductions were recorded in the neonates (37% (SE 0.48) and 21% (SE 2.12); $n = 6$ and 4, $P < 0.001$). Fe deficiency decreased the erythrocytes' sensitivity to low osmotic pressure, measured by determining the concentration required to produce 50% cell lysis (LC_{50}). In the dams, the LC_{50} dropped from 149 (SE 2.66) to 136 (SE 5.07) mOsm/l ($n = 6$ and 4, $P < 0.03$). The same effect was seen in non-pregnant animals (152 (SE 1.53) and 138 (SE 3.72) mOsm/l, $n = 3$ and 5; $P < 0.03$). These data suggest that the differences in the LC_{50} values between the Fe deficient and control dams were due to the Fe deficiency and not as consequence of pregnancy. The data also show that Fe deficiency not only influences growth and development of the neonates, but also alters cell membrane function. This may be a consequence of membrane lipid composition; this is currently being investigated and may, at least in part, explain the consequences of maternal Fe deficiency on the health of their offspring.

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