

MATERNAL FOETAL IRON RELATIONSHIP

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The relationship between maternal and foetal iron nutrition has evoked considerable interest and debate. In pregnancy the demand for iron is increased by the mother's need to expand her red cell mass to fulfill the requirements of the developing foetus¹. Foetal iron is derived solely from the mother through the active transport function of the placenta², and the quantity transferred might reasonably be supposed to be influenced by how much is available³. Earlier observations suggested that iron deficient mothers produce babies who later develop iron deficiency^{2,4,5}. Several subsequent studies using biochemical values reflecting iron nutrition, such as Serum Iron, TIBC and percent saturation of transferrin, failed to show this correlation^{6,7}. The advent of sensitive radioimmunoassay for ferritin^{8,9} offered an additional approach to the problem, which has produced conflicting results. Some workers found that mothers with very low ferritin concentrations produced infants whose cord serum ferritin concentrations were low^{1,4,10}, while others were unable to demonstrate such a correlation¹¹⁻¹³. The rapidly growing human foetus requires a large supply of iron², which is obtained from the iron stores of the mother. Iron is transported from mother to foetus against a concentration gradient^{2,4,14,16}. The placenta plays a key role in regulating the supply of iron in the foetus. The amount of iron passing through the placenta increases as gestation progresses^{15,17}. The concentration of maternal haemoglobin is also greatly reduced in the latter part of pregnancy even in women who are not anaemic². These findings indicate that iron is mobilized from the stored iron pool in the mother as gestation progresses in order to meet the increasing demand of iron for haematopoiesis in the foetus and placenta^{2,3,17}. At delivery the umbilical cord blood had a significantly higher content of serum iron than the maternal blood^{2-4,17}. This provides evidence that at least in the terminal stage of gestation iron is actively transported from mother to child against a concentration gradient. The cord blood also contains a significantly higher amount of serum ferritin than does the maternal blood¹⁷. The relationship between maternal and foetal iron stores is controversial. Some found a positive correlation^{1,3,10,17} while others were unable to do so^{2,4,11,12,14}. Babies born to the mother with low iron stores had a lower amount of storage iron than babies born to the mothers with normal iron stores^{1,3,4,18}, but an overall correlation between maternal and cord serum was not found^{1,11,12,17}. This suggests that iron storage in the mother and iron metabolism in the foetus are not directly related². The mother and foetus have independent systems for the control of iron metabolism. The foetus and placenta are able to take iron from the maternal circulation in spite of maternal iron deficiency^{4-7,19,20}. Thus, although the mother is iron deficient, foetal iron supply is usually adequate. However, there seem to be a level of maternal iron storage, below which the newborn was endowed with decreased iron stores^{4,19,20}.

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