The causes and effects of fetal macrosomia in mothers with type 1 diabetes

The mechanisms and physiology of in utero fetal nutrition are not understood, and the proportions of carbohydrate, fat, and protein contributing to fetal energy uptake are unknown. Lipids are energy rich and potentially a valuable source of energy for the fetus. Little intact triglyceride crosses the placenta but non-esterified fatty acids (NEFAs) do cross easily. However, the plasma concentration of maternal NEFAs is too low to sustain the total energy supply to the fetus and, therefore, monosaccharides have been assumed to be the major energy source for the growing fetus. There are considerable species variations in placental fatty acid transfer. In general, the fewer the numbers of cell layers contributing to the placental barrier the higher the net flux. Most experimental biology of materno-fetal energy transport has been performed in species with non-haemochorial placentaion. Human placentaion is haemochorial—maternal blood is in direct contact with a thin layer of fetal cells. The guinea pig also has a haemochorial placenta and is able to hydrolyse triglyceride, producing NEFAs that cross to the fetus.1

Human experimentation is more difficult, but inutrition may give insights into normal physiology. In this issue Merzouk et al describe mothers with type 1 diabetes mellitus who were delivered of “macrosomic” neonates with higher glucose concentrations and higher triglyceride concentrations than mothers with type 1 diabetes mellitus and appropriate for gestational age neonates.2 The “fetal insulin hypothesis” is the concept that insulin mediated fetal growth in utero, as well as insulin resistance in childhood and adulthood. Low birth weight, measures of insulin resistance in life, and ultimately glucose intolerance, diabetes, and hypertension, would all be phenotypes of the same insulin resistant genotype. Central to this “fetal insulin hypothesis” is the concept that insulin mediated fetal growth will be affected by fetal genetic factors that regulate either fetal insulin secretion or the sensitivity of fetal tissues to the effects of insulin.

Merzouk et al have found dyslipidaemia in infants of mothers with poorly controlled type 1 diabetes. Does the genetic predisposition increase the risk of type 1 diabetes in the infant as in their mother; or does the dysnutrition increase the risk of insulin resistance and type 2 diabetes, unlike their mother, with impaired glucose tolerance in adulthood, or neither, or both? Although it may be premature to suggest that these results have implications for metabolic diseases in these infants, these studies may in time give insights into the differential effects of the metabolic milieu predisposing to impaired glucose tolerance in adulthood and also the genetic predisposition to impaired glucose tolerance in adulthood.

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