Fetal growth and placental physiology: From cell to community

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Fetal growth is absolutely dependent on supply of nutrients from mother *via* the placenta. It follows that when fetal growth is deficient, i.e. in intrauterine growth restriction (IUGR, a condition which markedly increases the risk of mortality and morbidity of the baby), then the supply of nutrients by the placenta must have been reduced. The interesting question is whether the reduction in placental supply capacity is a cause or a consequence of IUGR. Placenta supply capacity is dependent on a number of variables: i. uterine and umbilical blood flow; ii. placental dimensions including exchange barrier surface area, thickness and pore size; iii. nutrient transporter kinetics, including number/m^2, turnover and affinity. There has been a focus on the relationship between placental blood flow reductions and IUGR but it is now clear that placental exchange barrier dimensions and nutrient transporter activity are also affected. Rat and mouse studies suggest that some of these placental transporter changes, e.g. the decreased System A activity, may be one cause of IUGR in some pregnancies. Studies on placentas from pregnant teenagers, who are at greater risk of an IUGR baby, also suggest relationships with maternal growth. These observations show that the placental phenotype in IUGR needs to be defined in terms of all the determinants of supply capacity. We propose that a rigorously defined placental phenotype be taken as the starting point for defining and diagnosing IUGR. Furthermore, we suggest, and provide data to show, that magnetic resonance imaging might provide one component of tests which non-invasively diagnose a placenta with the IUGR phenotype early in pregnancy.

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