Hindawi Publishing Corporation
Experimental Diabetes Research
Volume 2011, Article ID 171376, 5 pages
doi:10.1155/2011/171376

Research Article

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growth restriction by upregulating its nutrient supply to the fetus such that body weights are maintained [4]. By day 16, this compensation helps limit the fetal growth restriction to a large extent despite the placenta being around a third underweight, whereas later in the pregnancy, the placenta appears to become overwhelmed and the fetal weight restriction approaches that of the placenta.

The compensatory increase in nutrient supply relative to placental weight that occurs around the start of the third week of the mouse pregnancy in this model includes a greater than 50% increase in placental glucose transfer rates on day 16 of pregnancy when they are expressed relative to placental weights [4, 5]. This increase appears to be at least partly mediated by increased placental *Slca3* (glut3) expression. The fact that glucose transfer is normalised at this stage of pregnancy despite having a severely growth restricted placenta is emphasised by the fact that the placental glucose transfer rates in affected fetuses are similar to those of unaffected litter mates when expressed relative to fetal weights. By day 19, when both the fetus and the placenta are severely growth restricted there is still increased placental glucose transport but it is of a smaller magnitude of around 30% above that of control values [5]. *Igf2*, at least in the first half of the third week of pregnancy in the mouse, therefore appears to be able to upregulate maternal nutrient supply to meet fetal demand [6]. We have recently suggested that such fetal demand could be affected by fetal genotype effects on maternal metabolism [7]. We therefore performed the following study to test the hypotheses that the relative maintenance of fetal weight and the increased placental glucose transport at day 16 of pregnancy in this mouse model is related to increased maternal glucose concentrations and