Placental metabolism of carbohydrates

Normal gravid state

In state of pregnancy, maternal physiology undergoes changes to facilitate constant stream of glucose to the infant; including maternal insulin resistance and increased gluconeogenesis. Glucose is the main substrate for fetal, and placental metabolism. The majority of the glucose supplied to the uteroplacental unit is from maternal circulation (Gallo L.A. et al, 2017). The normal transfer of maternal glucose into the placenta is through GLUT family transporters. The GLUT1 transporter is in greater concentration on the maternal face of the placenta, in microvillus structures, and transfers glucose via facilitated diffusion (Laugeu et al 2013). Within the normal physiological range of blood glucose, there is more GLUT1 on the maternal villous side than on the basal/fetal side (laugue edt al 2013). There appears to be greater saturation of GLUT1 transporters early in the pregnancy, with levels stagnating as gestation progresses (Gallo 2017). Because maternal glycemia is higher than that of the fetus, even in normal pregnancy and the transporters are of passive mechanism, it was previously supposed that maternal blood glucose was the only driver in glucose transport out of maternal circulation. There is burgeoning evidence that fetal and placental needs, as well as fetal insulinemia also play a critical role in driving the transfer of glucose (Gallo 2017). There are some GLUT4 transporters present in placental tissues, but they do not appear to have a key role in nutrient transfer, as insulin dependent signaling is not crucial in the placenta (laugue et al, 2013). The normal placenta also holds an isoform of glucose 6 phosphatase, implying it is able to hydrolyse and use the glycogen that it stores (Laugue, 2013).

Obese gravid state

In pregnancies of women who have obesity, there is increased insulin signaling. This insulin biding to its receptor activates mTORC1 signalling, and affecting macronutrient metabolism (laugue et al 2013).

Gestational diabetes

In pregnancies complicated by GDM, there is evidence of increased expression of the GLUT1 transporter on the fetal interface of placental tissue (Laugue et al 2013).