The spiny mouse: a novel species for studying fetal and placental development and improving obstetric outcomes

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The spiny mouse is a precocial rodent species native to regions of the Middle East. The long gestation of the spiny mouse (39 days) relative to other rodents renders this species useful to study precocial fetal and neonatal development, as pregnancy ends after major organogenesis is complete. We have an extensive research program using the spiny mouse to understand the causes of major clinical obstetric complications such as preterm birth; intrauterine growth restriction (IUGR); maternal stress/illness; and birth asphyxia, and develop effective treatment/preventative strategies for clinical application.

The major circulating glucocorticoid in the spiny mouse is cortisol, and the adrenal gland produces dehydroepiandosterone (DHEA). The gestation length is 39 days, and litter sizes range from 1-5 (mean is 2-3). The spiny mouse represents an interesting species in which to study comparative placentation. The placenta is described as discoid hemotrichorial with the emergence of the labyrinth at mid-gestation. The spongy zone is made up of glycogen cells, spongiotrophoblast, giant cells and large maternal blood spaces. Unlike conventional rodents, micro CT imaging reveals a large vascular contribution to the fetal membranes originating from the umbilical vessels close to the fetal surface of the placenta.

Maternal cortisol levels rise with advancing gestation in the spiny mouse, but fetal cortisol levels fall towards term. We have recently described the glucocorticoid barrier in the spiny mouse and show high expression of the p-glycoprotein isoforms Abcb1a and -1b in the labyrinth zone of the placenta toward term, which may explain the fall in fetal cortisol levels towards term. We have shown that placental development differs for a male and female fetus and that maternal exposure to high concentrations of glucocorticoids at mid pregnancy in the spiny mouse compounds these differences, resulting in sexually dimorphic alterations of placental structure and gene expression and poorer outcomes for male offspring.

We have recently developed a model of IUGR in the spiny mouse, which is associated with dramatic changes in gross placental morphology.