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In This Issue

This issue of the *Journal of Developmental Origins of Health and Disease* contains two excellent Editorials as well as seven original articles. The four articles related to human studies includes analysis of effects of exposure to hyperemesis gravidarum, the association of placental shape with placental efficiency, fetal heart rate responses in relation to birth weight and vascular programming. The animal studies include an investigation of the mechanism of cardiovascular programming and two studies exploring fetal/renal development. The Editorial by Dr Hansen highlights critical public health issues related to developmental origins of health and disease while the review of Dr Bagby serves as the predecessor of the upcoming International DOHaD Meeting to be held in Portland, 18–21 September 2011.

Original Articles

Prenatal exposure to hyperemesis gravidarum linked to increased risk of psychological and behavioral disorders in adulthood. Mullin *et al.*, examined 279 participants by survey, comparing emotional and behavioral diagnoses of adults born to pregnancies complicated by hyperemesis gravidarum to those of normal controls. The authors demonstrated that offspring exposed to hyperemesis gravidarum had a markedly greater incidence of psychological and behavioral disorders. The authors speculate that this association may be due to maternal stress, malnutrition, vitamin deficiencies or altered hormone levels.

Variable placental thickness affects placental functional efficiency independent of other placental shape abnormalities. Yampolsky *et al.* examined placental disk thickness as a reflection of deviations in placental vascular growth, hypothesizing that increased variability of thickness is associated with reduced placental efficiency (as reflected by smaller baby for given placental weight). The authors examined 587 placentas, determining that the variability of disk thickness was correlated with reduced placental efficiency. The authors propose that placental shape and size may serve as markers for screening infants at risk of programmed adult diseases.

Patterns of fetal heart rate response at ~30 weeks of gestation predict size at birth. Sandman *et al.* examined the fetal heart rate response to a startle stimulus at 30 weeks of gestation. There was a significant association between fetal heart rate patterns and birth weight, with the lowest birth weight group demonstrating an immediate fetal heart rate deceleration followed by an immediate acceleration. In contrast, the highest birth weight group demonstrated an immediate and fast acceleration in response to a fetal startle. These findings suggest that fetal neurologic manifestation, particularly with regard to cardiovascular regulation, may be influenced by factors regulating growth and birth weight.

Aortic growth arrest after preterm birth: a lasting structural change of the vascular tree. Schubert *et al.* performed a cohort study of 50 infants comparing 21 born very preterm, with 29 born at term. Using ultrasonographic measures through 3 months after term (equivalent age), the authors demonstrated impaired aortic and carotid artery growth following preterm birth. These findings have significant implications for cardiovascular morbidity in adult life.

Maternal undernutrition upregulates apoptosis in offspring nephrogenesis. Tafti *et al.* hypothesized that the association of fetal growth restriction with reduced nephron numbers may be a consequence of apoptotic signaling during renal development. Utilizing undernourished rat dams, the investigators demonstrated the upregulation of pro-apoptotic factors and downregulation of anti-apoptotic factor in the developing kidney. These findings indicate that low birth weight associated nephropenia may be a result, in part, of enhanced apoptosis during renal development.

The lack of impact of peri-implantation or late gestation nutrient restriction on ovine fetal renal development and function. Braddick *et al.* examined renal development in ewes receiving moderate undernutrition during peri-implantation period or late gestation periods. Maternal undernutrition in either of these periods had no effect on fetal kidney weight or nephron number. These findings suggest that moderate undernutrition, to a level that does not result in fetal low birth weight, does not impact significantly on fetal renal development, despite programming offspring cardiovascular dysfunction.

Brief Report

Feeding a protein-restricted diet during pregnancy induces altered epigenetic regulationof peroxisomal proliferator-activated receptor-α in the heart of the offspring. Slater-Jefferies *et al.* examined the effects of maternal protein restriction in rats on genes related to lipid and carbohydrate metabolism. Within the cardiac tissue of adult offspring, reduced nutrition before birth resulted in a dysregulation of lipid metabolism in the heart and increased TAG concentration, in association with increased mRNA expression of PPARα. These findings indicate that maternal nutrition may alter the composition and cardiac energy metabolism of the offspring.

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