Journal of Developmental Origins of Health and Disease (2014), **5**(2), 69–70.
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doi:10.1017/S2040174414000166

In This Issue

This issue of the *Journal of Developmental Origins of Health and Disease* contains a tribute and obituary of David Barker, written by our DOHaD members who were closest to him, both professionally and personally. The Journal includes one brief report and eight original articles of studies in animals and humans.

Editorial

A Tribute to Professor David J.P. Barker (FRS CBE) – 29 June 1938–27 August 2013 by Caroline Fall. Obituary: David James Purslove Barker: clinician, scientist and father of the 'Fetal Origins Hypothesis', C. Cooper, J. Eriksson, C. Fall, C. Osmond, D. Phillips.

Brief Report

Increased placental neurosteroidogenic gene expression precedes poor outcome in the preterm guinea pig. Cumberland *et al.* examine aspects of placental steroidogenesis, focusing on neuroactive steroids produced via placental 5α-reductase (5αR). Among preterm guinea pigs, higher levels of placental 5αR2 were associated with reduced neonatal survival suggesting that an induction of the neurosteroidal pathway may contribute to poor outcome. The authors discuss mechanisms by which 5αR2 may be mediated and impact on neonatal survival.

Original Articles

The effect of periconceptional undernutrition of sheep on the cognitive/emotional response and oocyte quality of offspring at 30 days of age. Abecia *et al.* examine the effect of periconceptional undernutrition in sheep on reproductive and behavioral offspring effects. At 1 month of age, offspring of undernourished ewes demonstrated a decrease in walking as compared with control ewes. However, the undernourished group evidenced a population of oocytes more than two-fold greater than those of controls.

Maternal high-fat diet induces follicular atresia but does not affect fertility in adult rabbit offspring. Léveillé *et al.* examine the effect of maternal and offspring high-fat diet on fertility and follicular number. Although there was no effect on fertility, exposure to high-fat diet during one of the periods (gestation/lactation, post-weaning) was associated with a higher number of atretic follicles. If extrapolated to the human, these findings may have important consequence for the impact of maternal obesity on offspring reproductive health.

Acute and chronic immunomodulatory changes in rat liver after fetal and perinatal asphyxia. Vlassaks *et al.* assess the effects of fetal and/or perinatal asphyxia and hepatic inflammatory responses. Both fetal and perinatal asphyxia independently induced hepatic inflammatory responses, though prior fetal asphyxia attenuated the inflammatory response to perinatal asphyxia.

Short- and long-term effects of maternal perinatal undernutrition are lowered by cross-fostering during lactation in the male rat. Wattez *et al.* examine the effects of maternal undernutrition on offspring metabolic and cardiovascular parameters. Cross-fostering offspring from undernourished to normally nourished dams mitigated offspring alterations. These findings indicate the importance of newborn and infant feeding regiments, including both breast *v.* bottle as well as nutrient composition.

Perinatal bisphenol A exposure beginning before gestation enhances allergen sensitization, but not pulmonary inflammation, in adult mice. O'Brien *et al.* examined the influence of maternal bisphenol A (BPA) exposure on the pathogenesis of asthma in offspring. Offspring exposed to perinatal BPA had an increase pulmonary inflammatory response and elevated serum IgE levels to ovalbumin. These finding suggest there are critical periods in gestation in which exposure to BPA can increase allergen sensitization in adulthood.

Placental lipoprotein lipase DNA methylation levels are associated with gestational diabetes mellitus and maternal and cord blood lipid profiles. Houde *et al.* examined the expression of placental lipoprotein lipase, an enzyme crucial for placental lipid transfer, in relation to maternal metabolic status. Placental samples were obtained at delivery from women with control and gestational diabetes mellitus (GDM) pregnancies. The authors demonstrated that methylation of the lipoprotein lipase (LPL) promoter was lower in GDM placenta and inversely correlated with placental and methylation levels inversely correlated with placental LPL mRNA expression. These findings indicate that maternal metabolic status may strongly influence fetal and cord lipid newborn metabolic profile.

The effects of prenatal oxidative stress levels on infant adiposity development during the first year of life. Loy *et al.* utilize the University Sains Malaysia Birth Cohort Study to examine the effects of oxidative stress on infant adiposity. The results demonstrated that maternal DNA damage was associated with reduced infant and offspring weight. The authors hypothesize that oxidative damage during pregnancy may

https://doi.org/10.1017/S2040174414000166 Published online by Cambridge University Press

program newborn/infant growth restriction and impact subsequent catch-up growth.

Cardiovascular diseases in grandparents and the risk of congenital heart diseases in grandchildren. Wijnands *et al.* performed a case-control family study utilizing questionnaires among families with and without children with congenital heart disease. The results demonstrated that cardiovascular disease among grandparents was associated with increased risk of congenital heart disease in offspring. These findings suggest that altered metabolic pathways may be associated with fetal cardiovascular development.

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https://doi.org/10.1017/S2040174414000166 Published online by Cambridge University Press