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In This Issue

This issue of the *Journal of Developmental Origins of Health and Disease* includes two review articles focusing on early life nutrition and environmental metal exposure, a brief report related to renal development in Australian indigenous groups, and four original articles examining both animal models and human studies.

Reviews

The role of early life nutrition in programming of reproductive function. Chadio and Kotsampasi provide an interesting review of the role of early life nutrition on reproductive development and adult reproductive function. The authors discuss mechanisms including an alteration in neuroendocrine control mechanisms, follicle and spermatogonia development, the association with obesity, and the role of epigenetics.

Lead, cadmium and mercury levels in pregnancy: the need for international consensus on levels of concern. Taylor *et al.* provide data on pregnant women blood levels of lead, cadmium and mercury from the UK Avon Longitudinal Study of Parents and Children (ALSPAC). The authors emphasize that there are few recommendations regarding levels of concern for heavy metals, however, there is significant exposure during pregnancy, and evidence for placental transfer and adverse fetal effects.

Brief Report

Reduced nephron endowment in the neonates of Indigenous Australian peoples. Kandasamy and colleagues examine a potential mechanism for the markedly high rates of chronic kidney disease among Australian indigenous groups. The authors provide evidence that indigenous neonates have a markedly lower total kidney volume in likely reduced nephron number, though similar glomerular filtration rates. These findings would result in a higher single nephron filtration rate, contributing to long-term kidney damage.

Original Articles

Sex-specific effects of low protein diet on *in utero* programming of renal G-protein coupled receptors. Cooke and co-authors examined low protein-induced IUGR rats to assess the effects on the G-protein coupled receptor, GPR91. The authors demonstrate that IUGR rats exhibit upregulation of GPR91 in conjunction with increased angiotensin converting enzyme and renin, which may predispose the development of hypertension in adult offspring.

Unlimited access to low-energy diet causes acute malnutrition in dams and alters biometric and biochemical parameters in offspring. Nascimento and colleagues examine the effect of the maternal low energy diet on pregnant dams and offspring. Pups born to low energy dams exhibited reduced body weight and through 2 months of age. In addition, pups had reduced levels of VLDL triglycerides and glucose, indicating that low-energy maternal diet impairs offspring growth development and metabolic regulation.

Antenatal betamethasone increases vascular reactivity to endothelin-1 by upregulation of CD38/cADPR signaling. Lee and associates utilize pregnant sheep to examine the effects of prenatal betamethasone on offspring vascular reactivity. The authors demonstrate an increased response to endothelin, likely mediated via the CD38/cADPR pathway. These studies may provide insights into the impact of antenatal glucocorticoids and/or maternal stress on offspring hypertension.

Early gestation screening of pregnant women for iodine deficiency disorders and iron deficiency in urban center in Vadodara Gujarat, India. Joshi *et al.* examine the prevalence of iron deficiency and iodine insufficiency among the population of pregnant urban Indian women. The authors reported a nearly ubiquitous iron deficiency and a high incidence of iodine insufficiency indicating that early screening and supplementation programs would be of benefit.

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