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In This Issue

This issue of the *Journal of Developmental Origins of Health and Disease* contains an excellent review article of the impact of early-life stress and five original articles. Four of the original articles focus on human studies, with two articles addressing issues of environmental exposures, including selenium, copper and arsenic. The DOHaD studies are increasingly addressing environmental exposures and environmental toxins and their impact during pregnancy and neonatal life on offspring disease. This interest has been highlighted by the recent Gordon conference on environmental toxins and the upcoming Environmental Stressors in Disease and Implications for Human Health 'PPTOX' conference in Boston, MA, October 26–29, 2014.

Several of the papers in this issue demonstrate gender-specific impacts of early-life exposures. These findings illustrate importance of the recently published NIH policies to insure that pre-clinical research in cells and animals considers both genders.

Review Article

Early stress and human behavioral development: emerging evolutionary perspectives. Del Giudice discusses the impact of early-life stress as being an adaptive rather than maladaptive response. The author includes an evolutionary perspective examining how maternal fetal interactions influence the physiology of prenatal stress and its impact on postnatal development.

Original Articles

Association of selenium and copper with lipids in umbilical cord blood. Wells *et al.* examined singleton births from Baltimore, MD, assessing umbilical cord selenium and copper levels in relation to serum lipids. Copper, but not selenium, was significantly associated with increased triglycerides, potentially raising the risk for future cardiovascular disease.

Arsenic exposure in early pregnancy alters genome-wide DNA methylation in cord blood, particularly in boys. Broberg *et al.* examined 127 mother/infant pairs, assessing arsenic concentration in maternal urine in relation to DNA methylation in mononuclear cell cord blood. Three CpG sites in boys but not in girls were significantly associated with arsenic exposure. The authors discussed that early prenatal arsenic exposure induction of reduced DNA methylation may increase expression of cancer-related genes.

Postprandial metabolism and inflammatory markers in overweight adolescents. Schauren *et al.* examined overweight and normal weight adolescents (aged 11–18 years) for postprandial metabolic and inflammatory responses. Overweight adolescents demonstrated increased cholesterol and inflammatory markers, indicating an increased risk for chronic disease in this group of young individuals.

The effect of known cardiovascular risk factors on carotid-femoral pulse wave velocity in school-aged children: a population-based twin study. McCloskey *et al.* examined the relationship between birth parameters on carotid–femoral pulse rate velocities (PWV) in twin pairs (aged 7–11 years). The authors demonstrated association between markers of adiposity and insulin resistance with PWV. The results suggest both genetic and environmental contributions to increased PWV, a marker of arterial stiffness and long-term cardiovascular events.

Growth restriction in the rat alters expression of cardiac JAK/STAT genes in a sex-specific manner. van der Linde *et al.* studied the expression of cardiac JAK/STAT signaling genes in growth-restricted rats. In the heart there was a significant effect of age for males with divergent effects on JAK/STAT signaling targets in females. These findings indicate that growth restriction alters cardiac JAK/STAT signaling, potentially contributing to cardiovascular disease in males.

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