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In This Issue

As we begin the second volume of the *Journal of Developmental Origins of Health and Disease*, we introduce our first themed issue, "Role of Environmental Stressors in the Developmental Origins of Disease," with Guest Editor Jerry Heindel. This issue incorporates an Editorial introduction by Dr. Heindel *et al.* highlighting the critical impact of developmental exposure to environmental chemicals on adult health and disease. Dr. Heindel *et al.* describe the Prenatal Programming and Toxicity II (PPTOX II) conference (Miami, Florida) which was held in December 2009. This issue of J DOHaD incorporates select highlights of the conference, and will serve to both disseminate critical information, and hopefully stimulate further research into the effects of environmental exposures on human development and health. The issue contains two themed Reviews, focusing on both obesity and cancer, as well as four Original Articles. Consistent with our policy to incorporate non-themed content within themed issues, we include two additional Original Articles examining developmental aspects of arterial elasticity and placental development.

Themed Content: Role of Environmental Stressors in the Developmental Origins of Disease

Reviews

Obesogens, stem cells and the maternal programming of obesity. Blumberg presents a review of an environmental obesogen model in which chemicals which stimulate adipogenesis and fat storage may alter adipose tissue homeostasis. The authors review the potential for environmental exposure and the mechanisms, including PPARγ signaling, by which organotins may program the development of obesity.

Cancer as development gone awry: the case for bisphenol-A as a carcinogen. In this review, Sonnenschein *et al.* examine the impact of xenoestrogens on the development of neoplasms, utilizing studies from animal models and human epidemiologic data. The authors compare the current studies to the unfortunate experience with the use of maternal DES during the 1950's and 1960's to prevent spontaneous abortion. The results provided insight into the possible contribution of xenoestrogens to the increased incidence of breast cancer over the past 50 years.

Original Articles

Effects of early low-level lead exposure on human brain structure, organization and functions. Cecil utilizes the results of the Cincinnati "Lead Study," in which infants from a birth cohort underwent MRI examinations through six years of age. Higher mean childhood blood lead levels are associated with structural, organizational, and functional changes in the brain, emphasizing the adverse consequences of both pregnancy and childhood lead exposure.

The ovarian dysgenesis syndrome. Louis *et al.* present a paradigm in which environmental exposures, particularly endocrine disrupting chemicals, may be associated with alterations in ovarian structure and function, ultimately manifest as gynecologic disorders or diseases. The authors emphasize the "exposome" paradigm, as the assessment of the impact of exposures during critical windows of development, as a complement to genomic and epigenetic research.

Choice of animal feed can alter fetal steroid levels and mask developmental effects of endocrine disrupting chemicals. Ruhlen *et al.* expose in utero mice to maternal diets containing low and high doses of DES, which significantly increase fetal serum estradiol at low dose but reduce serum estradiol at the high dose. In utero DES exposure effects on female onset of puberty and male sperm production are dependent upon the background soy-based Purina diet, emphasizing the potential interaction of environmental toxigens and underlying diets.

Developmental basis of disease: environmental impacts. Collman describes the biologic and epidemiologic issues pertinent to research on developmental impacts of environmental exposures. The author examines the public health implications of both research, communication, and public information of the impact of the developmental effects of environmental chemicals.

Non-themed Content

Original Articles

The impact of maternal cortisol concentrations on child elasticity. Rondó *et al.* examine 130 pregnant women and their children at 5–7 years of age, comparing maternal salivary cortisol concentration with offspring elasticity at the radial artery. The finding that exposure to higher glucocorticoid concentrations during the prenatal period is associated with lower arterial elasticity, suggests that excess glucocorticoids may be associated with offspring cardiovascular disease.

A maternal high fat diet in rat pregnancy reduces growth to the fetus and the placental junctional zone but not placental labyrinth zone growth. Mark *et al.* demonstrate that maternal high fat diet results in a reduction in male and female fetal weights and the weights of the junctional zone of the rat placenta. As many of the placental derive endocrine signals originate from the junctional zone, these findings emphasize the potential effect of maternal diet on fetal and placental endocrine homeostasis.

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