A well-documented and accepted hypothesis of the origins of diseases that occur with a high frequency in adulthood is the causal relationship between an adverse intrauterine environment during fetal life and development of diseases including hypertension, diabetes, obesity, insulin resistance, and metabolic syndrome [1, 2]. Epidemiological studies provided the impetus for experimental studies to identify cell and molecular mechanisms that may underpin this relationship. The precise mechanisms, however, remain only partially described. It is anticipated that on-going studies will provide data that more rigorously test this hypothesis and provide insights into gestational age-dependent programming effects and the consequences of alterations in intrauterine life on risk profiles for adult diseases. To date, few studies have addressed whether the preconception period is also involved in this phenomenon [3]. Thus, it is clear that on-going investigative effort in this field for a better understanding of the cellular and molecular mechanisms underlying fetal programming of adult diseases is required. This special issue includes contributions over a wide area of expertise but particularly studies concerning the vascular physiology/pathophysiology of diseases of pregnancy that could result in programming of the fetus leading to adult diseases.

The articles included in the present issue include both clinical and basic science studies. The alterations caused by exposure to elevated D-glucose environment are discussed in chapters by J. B. Moreli et al. and D. C. Damasceno et al. These articles reported that hyperglycaemia causes damage to the maternal genetic material without a clear consensus regarding the impact of this adverse environmental condition on fetal cells. Since DNA repair mechanisms may be important to prevent the deleterious effects of hyperglycaemia in both the maternal and fetal DNA, it may be preventive of the development of diseases in adulthood. Studies in animal models support the targeting of the development of new therapeutics that minimise or prevent diabetes-induced DNA damage due to increased oxidative stress.

It is now clearer that maternal obesity is also a condition that impacts on fetal growth and development [4]. The prevalence of obesity, especially in women of childbearing age, and a supraphysiological increase in gestational weight gain during pregnancy is associated with adulthood diseases. This is clearly summarized in the chapter by S. A. Segovia et al. In addition, these authors highlight the fact that the predisposition of offspring to obesity and metabolic and cardiovascular disorders in later life occurs via poorly described mechanisms including programming of metabolic disorders. In this review, the authors discussed the possibility that maternal obesity-related inflammation may program insulin sensitivity of tissues in offspring. Interestingly, in the
proposes that docosahexaenoic acid supplementation early in pregnancy may help to reduce the incidence of deep placental disorders. Thus, this compound could be a strategy for primary prevention of preeclampsia. More studies are required to confirm or refute this proposal.

D. I. Chiarello et al. report the effects of hypoxia on placental function, in particular, lipid peroxidation state of syncytiotrophoblast plasma membranes. The authors conclude that increased membrane calcium content interacts with phospholipids leading to exposure of hydrocarbon chains of fatty acids to free radicals, where magnesium might play a protective role. These findings could be key for understanding the abnormal function of the human placenta in states of hypoxia such as preeclampsia. In another set of articles in this issue the effect of hypoxia on pregnancy is reviewed by L. Wang et al. and A.-C. Peyter et al. Interestingly, studying rats exposed to hypoxia, L. Wang et al. described maternal hypoxia increased collagen (I and III) expression in the left ventricle of adult offspring. Another study regarding hypoxia by A.-C. Peyter et al. reported that inhaled nitric oxide administered simultaneously to perinatal hypoxia in mice has beneficial effects on the adult pulmonary circulation. Since inhaled nitric oxide is the therapy of choice in neonates with pulmonary hypertension, this article shows an interesting set of results documenting that relaxation of adult mouse pulmonary arteries to acetylcholine is restored following inhaled nitric oxide.

This series of articles and reviews identifies putative mechanisms that may impact on intrauterine life and predispose the newborn to adulthood diseases such as obesity, diabetes, preeclampsia, and metabolic syndrome. Further insights in the abnormal function of fetal cells, including endothelium, placenta, and circulating blood cells, is required to understand and to correlate these specific mechanisms with adult diseases.

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