Developmental programming of cardiovascular dysfunction by prenatal hypoxia and oxidative stress

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Fetal hypoxia is a common complication of pregnancy. It has been shown to programme cardiac and endothelial dysfunction in the offspring in adult life. However, the mechanisms via which this occurs remain elusive, precluding the identification of potential therapy. Using an integrative approach at the isolated organ, cellular and molecular levels, we tested the hypothesis that oxidative stress in the fetal heart and vasculature underlies the molecular basis via which prenatal hypoxia programmes cardiovascular dysfunction in later life. In a longitudinal study, the effects of maternal treatment of hypoxic (13% O 2) pregnancy with an antioxidant on the cardiovascular system of the offspring at the end of gestation and at adulthood were studied. On day 6 of pregnancy, rats (n = 20 per group) were exposed to normoxia or hypoxia ± vitamin C. At gestational day 20, tissues were collected from 1 male fetus per litter per group (n = 10). The remaining 10 litters per group were

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