The Impact of Prenatal Hypoxia and Mitochondrial Dysfunction on the Fetal and Postnatal Heart

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An adverse intrauterine environment has been shown to increase fetal morbidity and lead to asymmetric fetal growth restriction. Prenatal hypoxia is one of the most challenging intrauterine stressors during pregnancy and contributes to a wide variety of maternal, placental and fetal complications. Its impact on the fetus is dependent on the severity and duration of hypoxia, as well as, the gestational age of exposure. While the growth restricted fetus has compensated to the adverse conditions, it comes at a cost, resulting in an increased vulnerability to disease after birth. This talk will discuss the role of the mitochondria in contributing to heart dysfunction in both the fetus and offspring. We hypothesize that the mitochondria and oxidative stress play important roles in increasing the vulnerability of the offspring exposed to prenatal hypoxia to cardiovascular disease.