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Metabolic Syndrome and Morphological changes in the Hearts of Offspring Born to Mothers under Stress Conditions

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Annotation: This article examines the morphological alterations in the cardiac tissue of offspring born to mothers affected by metabolic syndrome and stress during pregnancy. Core components of metabolic syndrome, such as insulin resistance, dyslipidemia, arterial hypertension, and obesity, are shown to exert adverse effects on fetal development. Additionally, elevated stress hormones (cortisol, catecholamines) contribute to structural remodeling of cardiomyocytes, disturbances in microcirculation, and myocardial weakness at the morphological level. The article highlights the mechanisms by which maternal metabolic and stress-related disorders program long-term vulnerability of the offspring's cardiovascular system.

Keywords: metabolic syndrome, stress, pregnancy, offspring, heart, morphology, myocardium, fetal programming.

Introduction

In recent decades, the prevalence of metabolic syndrome has risen sharply worldwide, posing a major public health concern. This condition not only affects adults but also significantly influences fetal development. During pregnancy, maternal metabolic disturbances combined with stress factors may induce irreversible changes in the formation of the offspring's cardiovascular system. According to the concept of "fetal programming," any deviation in maternal homeostasis during pregnancy may predispose the offspring to cardiovascular diseases in later life.

Metabolic syndrome exerts its influence on the fetal heart through several mechanisms. Insulin resistance disrupts glucose metabolism in cardiomyocytes, thereby reducing energy availability. Maternal arterial hypertension limits placental blood flow, leading to chronic hypoxia of the developing heart. Dyslipidemia, characterized by elevated triglyceride and cholesterol levels, predisposes the fetal vascular system to early atherosclerotic changes. Obesity-related inflammatory mediators, such as interleukin-6 and tumor necrosis factor-α, stimulate fibroblast activation and excessive collagen deposition, resulting in myocardial fibrosis.

Stress exposure during pregnancy further exacerbates these effects. Elevated maternal cortisol and catecholamine secretion alters myocardial structure by enhancing apoptosis, impairing mitochondrial energy metabolism, and increasing myocardial oxygen demand. Excess cortisol leads to alterations in cellular membranes, while catecholamines accelerate cardiac workload, aggravating tissue injury. Microcirculatory disturbances caused by stress restrict adequate capillary development, impairing oxygen and nutrient delivery to fetal cardiomyocytes.

Consequently, offspring born to stressed mothers with metabolic syndrome display significant morphological changes in the heart. These include hypertrophy and dystrophy of myocardial cells, increased fibrosis due to collagen accumulation, narrowing of vascular lumens, endothelial dysfunction, and mitochondrial impairment. Such structural changes compromise cardiac function and increase susceptibility to ischemic heart disease, hypertrophic cardiomyopathy, and chronic heart failure in postnatal life.

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In conclusion, maternal metabolic syndrome and stress during pregnancy play a decisive role in shaping the morphology of the offspring's heart. These alterations begin prenatally and significantly elevate the risk of cardiovascular diseases later in life. Early detection and management of metabolic syndrome in pregnant women, combined with strategies to reduce stress exposure, represent crucial preventive approaches to safeguard the cardiovascular health of future generations.

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