CHRD 2024: Abstract Submission Form

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Role in the project Design Perform Experiments Analyze Data Write Abstract Presenter Status Masters Student

Research Category Basic Science

Title

Maternal Resveratrol (RESV) Supplementation and the Effects on Cardiac Hypertrophy, Mitochondrial Metabolism, and Calcium Flux

Background

Gestational diabetes mellitus (GDM) is a condition that manifest in the later stages of pregnancy and is characterized by maternal insulin resistance, glucose intolerance, and hyperglycemia and impacts maternal and offspring health. Medications have shown effectiveness but have associated risk of adverse pregnancy outcomes and the long-term effects on the offsprings are unknown.

Objective

Our previous studies in rats observed that GDM-exposed offspring exhibit hypertrophy, mitochondrial dysfunction and impaired calcium flux in rat cardiomyocytes. In this study, we hypothesize that Resveratrol (RESV) in the maternal GDM diet will mitigate mitochondrial dysfunction, cardiac hypertrophy and improve calcium flux in GDM-exposed offspring.

Methods

Female Sprague-Dawley rats were fed a low-fat (Lean, 10% kcal fat) or high-fat and sucrose (GDM, 45% kcal fat) diet six weeks before mating to induce GDM. A subgroup of GDM dams were switched to a diet containing RESV (GDM+RESV, 45% kcal + 4g/kg RESV). At e18.5 fetal echocardiography was performed to assess cardiac structure. To determine the effects of RESV on GDM-offspring, e20 pups were sacrificed for fetal cardiomyocyte isolation and measurements of mitochondrial respiration and calcium flux were assessed

Results

Fetal echocardiography revealed maternal RESV attenuated GDM-induced cardiac hypertrophy. GDMexposed offsprings showed 1.4X larger intraventricular septal and left ventricular posterior wall thickness compared to lean and GDM+RESV offspring (Lean vs. GDM, p<0.05) (Lean vs. GDM+RESV p<0.05). cardiomyocytes isolated from GDM-offspring had decreased mitochondrial respiration, but higher glycolytic activity compared to lean and GDM+RESV offspring (Lean vs. GDM, p<0.05) (Lean vs. GDM+RESV, p<0.05). Furthermore, cardiomyocytes isolated from GDM-offsprings exhibited delayed calcium flux cycles compared to lean and GDM+RESV offsprings upon angiotensin II stimulation

Conclusion

Our data replicates the previous findings that GDM-offspring exhibit cardiac hypertrophy and mitochondrial dysfunction. Maternal RESV attenuated GDM-induced cardiac hypertrophy in GDM-offspring and improved mitochondrial respiration which contributed to impaired calcium flux upon angiotensin II stimulation.

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