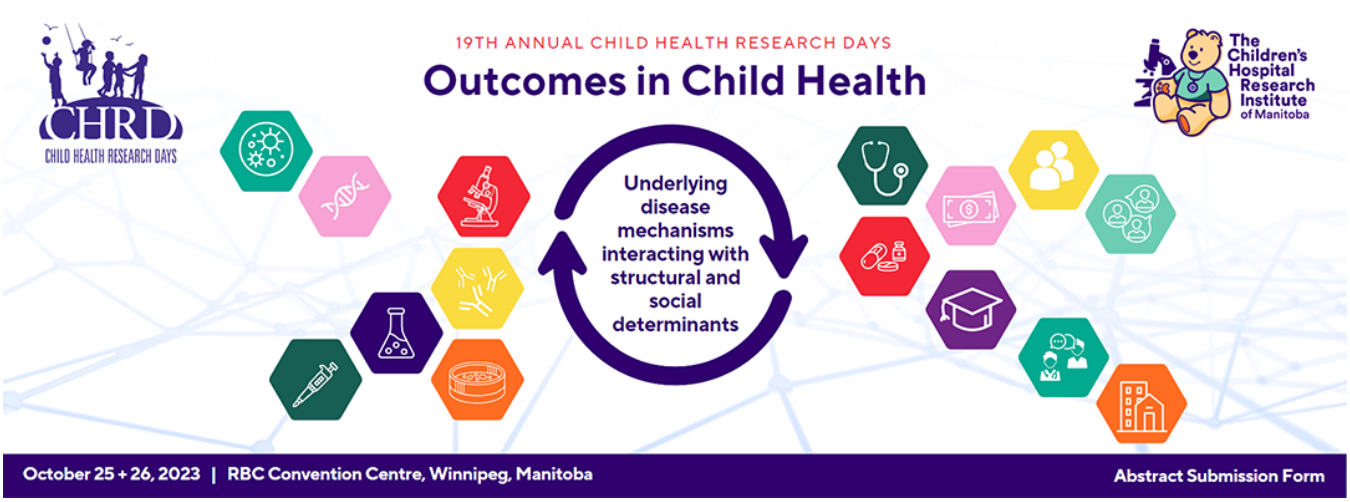

CHRD 2023: Abstract Submission Form

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Presenter Name
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Presenter Status
Masters Student

Research Category
Basic Science

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Title
Maternal Resveratrol (RESV) Supplementation and the Effects on Cardiac Hypertrophy in the Offspring

Background
Gestational diabetes mellitus (GDM) is a pregnancy condition that is characterized by maternal insulin resistance, glucose intolerance, and hyperglycemia, and impacts maternal and offspring health. Sometimes medications are used, but are associated with the risk of adverse pregnancy outcomes and the long-term effects on the offspring are unknown.

Objective
Our previous studies 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. Measurements of mitochondrial respiration were performed using the Agilent-Seahorse XFe24. Measurements of calcium flux were performed using fluo-4 on the Cytation-5.

Results
Fetal echocardiography revealed maternal RESV attenuated GDM-induced cardiac hypertrophy. GDM-exposed offspring showed 1.4-fold increased 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 form GDM-offspring had approximately 20% lower levels of maximal respiratory capacity 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-offspring exhibited delayed calcium flux cycles compared to Lean offspring.

Conclusion
Our data replicates the previous findings that GDM-offspring exhibit cardiac hypertrophy and mitochondrial dysfunction. Maternal RESV supplementation improved mitochondrial respiration which contributed to impaired calcium flux. Importantly, maternal RESV supplementation attenuated GDM-induced cardiac hypertrophy in GDM-offspring.

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