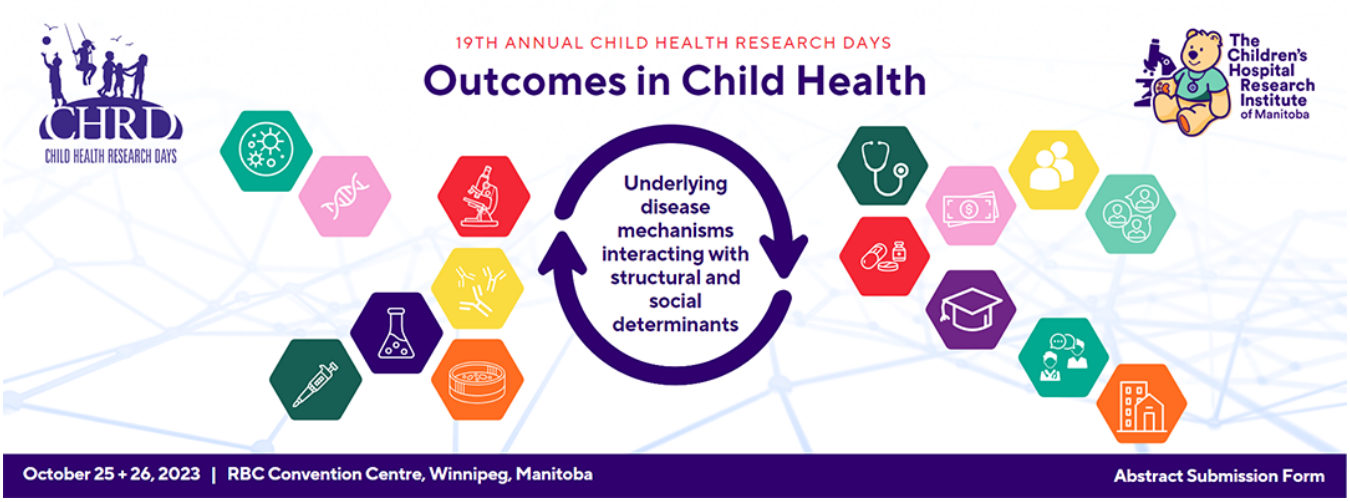

CHRD 2023: Abstract Submission Form

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Presenter Status
PhD Student

Research Category
Basic Science

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Title
Does prenatal EtOH reduce placental lipid droplet and n-3 fatty acids content? A potential link with negative fetal developmental outcomes

Background
Prenatal Ethanol (EtOH) has been shown to impact placental nutrient transport, specifically of lipids. The placenta can store lipids in the form of triglycerides (TG) or shunt them towards the fetus. Whether EtOH-induced defects in lipid metabolism affect fetal development is unclear.

Objective
This study aims to determine the impact of prenatal EtOH on placental morphology, lipid transport and storage, and consequential fetal outcomes.

Methods
Pregnant Sprague-Dawley rats were placed in control or EtOH (20%, v/v in water) groups. At gestational day 20 (GD20), developmental measurements were completed, while placentas were collected for morphological measurements, lipid analysis, and protein expression.

Results
Prenatal EtOH reduced maternal food intake ($P<0.05$) and pregnancy weight gain ($P<0.05$). Prenatal EtOH affected the fetal outcomes: reduced litter size and fetal body weights ($P<0.0001$), increased fetal reabsorption points ($P<0.05$), and increased placental weight leading to a 17% reduction in placental efficiency ($P<0.0001$). Compared to the control, prenatal EtOH increased both the placental labyrinth and

junctional zone thickness (P<0.05), while increasing lipid droplets in the labyrinth zone (fetal-side) and a decrease in lipid droplets in the decidual zone (maternal-side). Total placental lipid fractions followed the trend of phospholipids>TG>Cholesterol esters in both groups; EtOH decreased total saturated, monounsaturated, and polyunsaturated lipids in all lipid classes (P<0.05). Prenatal EtOH also reduced placental TG omega-3 fatty acids (- 42%, P<0.05) and DHA (-37%, P<0.05) compared to control. These lipid changes were accompanied with increased amounts of lipid-related transport and storage proteins in placenta (P<0.05).

Conclusion
These new findings indicate that EtOH-induced alterations to placental morphology may reduce its capacity to mobilize lipids (like n-3 PUFA and DHA) to the developing fetus via lipid transport and storage proteins. Ultimately, this study provides insight on how prenatal EtOH impacts placental lipid metabolism and may play a role in fetal alcohol spectrum disorder development.

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