CHRD 2024: Abstract Submission Form

Presenter Name
Sriyani Ranatunga

Presenter Status
Masters Student

Role in the project
Design
Perform Experiments
Analyze Data
Write Abstract

Research Category
Basic Science

Title
Studying the relationship between exposure to maternal diabetes and sensitivity to cigarette smoke in adulthood in mice

Background
Chronic obstructive pulmonary disease (COPD) is Canada's fifth leading cause of death and is primarily caused by smoking. Early-life exposures may also increase COPD risk by impairing lung development and altering response to cigarette smoke (CS). Maternal diabetes (MD) increases the risk for premature birth and childhood asthma, which are risk factors for COPD. However, the direct impact of MD on offspring susceptibility towards CS and COPD remains unexplored.

Objective
To understand whether exposure to MD worsens CS induced lung damage in offspring.

Methods
Six-week-old C57BL/6NJ female mice were fed with a high-fat diet (HFD-45% kcal) to induce diabetes or low-fat (LFD-10% kcal) control-diet for 6-weeks, and throughout pregnancy and weaning. Weaned offspring were fed standard research chow-diet until 8-weeks of age, at which point they were exposed to CS/Room air for 50 mins, twice/day, for four days. Lung function and inflammation were assessed on day five. Data analyzed in Prism GraphPad using two-way ANOVA, significance set at $p<0.05$.

Results
Offspring from diabetic pregnancy were significantly heavier at 3-weeks than controls. At 8-weeks the body weight difference maintained only in males. CS exposure significantly increased total lung resistance (14%) and airway resistance (18%) in HFD male offspring compared to control. HFD influenced CS induced immune cell infiltration in a sex-specific manner. Female HFD-CS offspring had significantly decreased neutrophils, CD4+ T-cells and NK cells compared to control-smokers. Male HFD-CS offspring had significantly elevated B-cells and NKT cells compared to HFD-control. At baseline, HFD female offspring had elevated Eotaxin and MIG but reduced IL-1α. CS induced cytokine changes in control animals, which were sex specific, were blunted in HFD exposed offspring.

Conclusion
MD alters offspring susceptibility towards CS-induced lung dysfunction and inflammation, in a sex-specific manner. MD may modulate response to cigarettes in early adulthood, suggesting a propensity to future COPD.

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No

Authors

Name	Email	Role	Profession
Sriyani Ranatunga	ranatuns@myumanitoba.ca	Presenting Author	Graduate
Dina H.D. Mostafa	dina.mostafa@umanitoba.ca	Co Author	Other
Sujata Basu	sujata.basu@umanitoba.ca	Co Author	Other
Shana Kahnamoui	kahnamos@myumanitoba.ca	Co Author	Other
Chinonye D. Onuzulu	onuzuluc@myumanitoba.ca	Co Author	Graduate
Jignesh Vaghasiya	vaghasij@myumanitoba.ca	Co Author	Graduate
Meaghan J. Jones	meaghan.jones@umanitoba.ca	Co Author	Assistant Professor
Christopher D. Pascoe	christopher.pascoe@umanitoba.ca	Co Author	Assistant Professor