CHRD 2024: Abstract Submission Form

Presenter Name
Harleen Kaur

Presenter Status
Undergraduate Students

Role in the project
Perform Experiments
Analyze Data
Write Abstract

Research Category
Basic Science

Title
Developmental origins of Chronic Obstructive Pulmonary Disease (COPD): Maternal diabetes and Inflammatory biomarkers as they are related to the disease

Background
COPD, the third leading cause of death worldwide, is a chronic inflammatory disease characterized by tissue destruction and difficulty breathing. Long considered a disease of smokers, only <20% of smokers develop COPD, suggesting that other factors are important, specifically the early-life environment. Maternal diabetes causes lung dysfunction in childhood, but a potential link with COPD risk remains unexplored.

Objective
We hypothesize that prenatal exposure to maternal diabetes increases cigarette smoke (CS)-induced Myeloperoxidase (MPO) and Serum Amyloid A (SAA) levels, two important inflammatory biomarkers in COPD, in offspring lungs

Methods
Mice prenatally exposed to diabetes (or no diabetes) were exposed to CS or room air at 8 weeks of age, twice a day for four days, and then lung tissue and lavage were collected. Enzyme-linked immunosorbent assays (ELISAs) were performed to quantify SAA and MPO levels in protein from lavage and tissue. Biomarker concentrations for tissue were normalized to tissue weight. Data were analyzed using GraphPad Prism with significance set at $p<0.05$ and presented as mean ± standard error of the mean (SEM).

Results
Male offspring from diabetic pregnancy had significantly increased SAA levels in their lavage following cigarette exposure (0.27±0.01ug/ml) compared to control CS (0.19±0.02ug/ml), whereas no significant difference was found in females. No significant differences in MPO lavage concentration were found in response to CS in control and diabetes exposures for both sexes. In males, lavage MPO concentration was positively correlated with absolute neutrophil count ($R^2 = 0.6433$). In lung tissue, MPO abundance was high after CS exposure in diabetes exposed offspring compared to control with no differences between sexes (345.6±16.4 vs. 308.0±12.3pg/mg of tissue).

Conclusion
Male offspring from diabetic pregnancy exhibit an enhanced inflammatory biomarker level (SAA) following cigarette exposure, and both sexes had higher tissue MPO levels. This suggests a potential sex-specific impact of maternal diabetes on future COPD risk.

Do you have a table/figure to upload?
No

Authors

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