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Abstract Title

Effects of Maternal Smoking on Offspring Susceptibility to Cigarette Smoke- Induced Gene Expression

Authors

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Background

Chronic Obstructive Pulmonary Disease (COPD) is an incurable illness, primarily caused by smoking, that is the 2nd leading cause of hospitalizations in Canada. However, only 15-20% of smokers develop COPD, suggesting other factors contribute to COPD risk. Early life exposures can increase COPD risk by reducing lung function and changing responses to cigarette smoke (CS). For example, people who smoke are more likely to develop COPD if their mothers smoked during pregnancy.

Objective

To investigate how prenatal CS exposure influences offspring susceptibility to CS-induced gene expression.

Methods

10-week-old C57BL/6NJ female mice were exposed to CS/room air for 50 minutes, twice a day, five days per week. Exposures started 2 weeks before breeding, continuing throughout pregnancy and until 3-weeks of age. At 8-weeks of age, offspring were re-exposed to 4-day CS/room air. 24-hours after the last exposure, lung tissues was collected, and gene abundance was measured using qPCR. Data were

analyzed using 2-way ANOVA.

Results

Offspring from smoking mothers had lower levels of the detoxifying enzymes Cytochrome P450 Family 1 Subfamily A Member 1 (CYP1A1) and Glutathione S-Transferase Mu 1 (GSTM1) compared to the control animals before postnatal CS exposure. Regardless of prenatal exposure, CYP1A1 increased in males following postnatal CS exposure by 6.3-fold. GSTM1 in prenatal CS females increased by 2.2-fold following postnatal CS exposure, but was still 1.5 fold lower than control animals. Reduced Insulin Receptor (IR) and Insulin-Like Growth Factor 1 (IGF1) in prenatally CS-exposed offspring suggest maternal CS impairs growth signaling. IR and IGF1 decreased to 0.63-fold of control levels in prenatal CS males.

Conclusion

Maternal smoking changes the expression of genes involved in CS detoxification and lung growth and repair, which may provide mechanistic context for the change in COPD risk associated with prenatal smoking.