CHRD 2022: Abstract & Poster Submission Form

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Presenter Status

○ Undergraduate Students

○ Masters Student

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○ Post-Doctoral Fellows

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○ Non-Trainee

Research Category

◉ Basic Science

○ Clinical

○ Community Health / Policy

Role in the project

☐ Design

☑ Perform Experiments

☑ Analyze Data

☑ Write Abstract

☐

Title

Smoking in adulthood reestablishes DNA methylation changes set by early life cigarette smoke

Background

The early life period is highly sensitive to the environment. Studies have shown that cigarette smoke (CS) exposure in early life causes lung function defects, and that DNA methylation (DNAm) changes may be involved. However, previous human studies have only shown that prenatal CS is associated with DNAm changes in blood and peripheral tissues, but not in lungs, and have not accurately excluded the effects of postnatal smoke exposure on offspring.

Objective

Therefore, this project investigates the effects of prenatal and early postnatal CS exposure on offspring lung DNAm and lung phenotype at different periods: immediately after smoke exposure, after smoking cessation and after re-exposure to CS in adulthood.

Methods

We exposed female mice to CS for 9 weeks, beginning three weeks prior to mating and ending at weaning, and cross-fostered pups at birth to generate prenatal CS only, postnatal CS only, prenatal and postnatal CS, and control exposure groups. We collected offspring lungs at birth and at 16 weeks (early adulthood) following lung function measurement. At one year of age, we re-exposed half the remaining middle-aged offspring to CS, then collected tissues again. DNAm in the lungs was measured at a candidate gene Cytochrome P450 1A1 (Cyp1a1), via pyrosequencing.

Results

At birth, lung Cyp1a1 expression was elevated 25-fold in CS-exposed offspring compared to controls. At 16 weeks, while offspring in the prenatal CS only and postnatal CS only groups had higher Cyp1a1 DNAm levels compared to controls, offspring exposed to both prenatal and postnatal CS showed significantly lower Cyp1a1 DNAm levels. Re-exposure of middle-aged offspring to CS re-established the Cyp1a1 DNAm pattern observed at 16 weeks of age, accompanied by significant reduction in lung function.

Conclusion

These results demonstrate that early life smoke exposure primes offspring lungs by causing transient phenotypic and DNAm changes which are re-established upon a secondary exposure in adulthood.

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