CHRD 2021: Abstract & Poster Submission Form

Submitter Name

Doris
First

Onuzulu
Last

Email

onuzuluc@myumanitoba.ca

Research Category:

- ◉ Basic Science
- ○ Clinical
- ○ Community Health / Policy

What was your role in the project?

- ☐ Design
- ☑ Perform Experiments
- ☑ Analyze Data
- ☑ Write Abstract
- ☐

Presenter Status:

- ○ Undergraduate Students
- ○ Masters Student
- ◉ PhD Student
- ○ Post-Doctoral Fellows
- ○ Residents
- ○ Non-Trainee

Title

Maternal cigarette smoke exposure alters detoxification mechanisms in offspring lungs

Background

Though the rate of smoking is decreasing globally, smoking remains a public health concern, as it has been linked to development of diseases like obesity, cancers, and respiratory issues. A phenomenon called DNA methylation reflects past environmental exposures, and thus studying how maternal smoking alters offspring DNA methylation, can help us understand how early life cigarette smoke (CS) exposure leads to disease development in later life. Human studies of early life smoke exposure are challenging due to the difficulty of distinguishing between prenatal and postnatal CS effects, and accessing primary lung tissue, but the use of mouse models can overcome these limitations.

Objective

We hypothesized that maternal smoking would alter the offspring DNA methylation of genes involved in CS detoxification in developing lungs, and that this alteration would persist even after CS cessation.

Methods

We exposed 16 female Balb/c mice to CS beginning two weeks prior to mating and ending at weaning, and cross-fostered pups at birth to generate prenatal only, postnatal only, prenatal and postnatal, and control exposure groups of pups. We collected tissue samples at 16 weeks in a subset of offspring, and at one year re-exposed half the remaining pups to CS, then collected tissue samples once again.

Results

At 16 weeks, prenatal CS exposure increased methylation of the Cyp1a1 gene, involved in detoxification of CS components. In contrast, pups which had both prenatal and postnatal CS exposure showed decreased Cyp1a1 methylation. One year after smoking cessation, we observed no differences between CS exposure groups in the non-re-exposed animals, but the same pattern from 16 weeks was reestablished in pups re-exposed to CS.

Conclusion

These results support our hypothesis by showing that the effects of prenatal smoking linger into adulthood, and our further work will investigate this effect across the genome.

Authors

- For each author, please click "**[+] Add Item**" and provide the author's information

Name	Email	Role	Profession
Doris Onuzulu	onuzuluc@myumanitoba.ca	Presenting Author	PhD student
Sujata Basu	sbasu@chrim.ca	Co Author	Technician
Samantha Lee	umlee285@myumanitoba.ca	Co Author	PhD student
Nikho Hizon	nikho.hizon@umanitoba.ca	Co Author	Technician
Dr Christopher Pascoe	christopher.pascoe@umanitoba.ca	Co Author	Assistant Professor
Dr Meaghan Jones	meaghan.jones@umanitoba.ca	Co Author	Assistant Professor