CHRD 2022: Abstract & Poster Submission Form

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Presenter Status

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

Research Category

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

Role in the project

- ☐ Design
- ☑ Perform Experiments
- ☑ Analyze Data
- ☑ Write Abstract
- ☑ Design in consultation with supervisor

Title

Prenatal air pollution exposure alters newborn cord blood DNAm in the racially diverse CANDLE study

Background

Previous studies examining the effect of prenatal air pollution exposure on fetal DNA methylation (DNAm) have mainly been conducted in White populations, limiting applicability of findings to other populations.

Objective

This investigation uses a subset (N=515; 53% Black) of racially diverse CANDLE study participants to identify DNAm changes in newborn cord blood associated with prenatal NO2, PM2.5, and PM10 exposure, and examines whether altered DNAm mediates the association between prenatal pollutant exposures and asthma, wheeze, and atopy at age four.

Methods

Average prenatal NO2, PM2.5, and PM10 exposures were estimated at the individual address-level using land-use regression models with spatial smoothing. Associations between pollutants and individual DNAm sites were examined using robust linear regression. We also identified whether pollutants were associated with altered DNAm across several nearby sites ("regions") using a method that spatially averages p-values. For significant sites and regions, we used causal mediation analysis to explore whether DNAm mediates the association between pollutant exposures and child health outcomes, and we used gene set enrichment analysis to identify impacted biological pathways. All models were adjusted for covariates to remove unwanted biological variation.

Results

At a false discovery rate of 5% and effect size cut-off of 1% DNAm, we identified altered DNAm sites in IGFB3 and MYO7B associated with NO2 and PM10, and 21, seven, and 19 regions associated with NO2, PM2.5, and PM10, respectively. Regions associated with NO2 were enriched for gene ontology terms related to cell adhesion. One region associated with PM10 localized to XYLT1. Causal mediation analysis suggested this region mediates the associations between prenatal PM10 and asthma, wheeze, and atopy.

Conclusion

This study provides new insight into robust DNAm changes that are shared across racially diverse CANDLE participants and deepens our understanding of mechanisms underlying the previously observed associations between prenatal air pollution exposure and pediatric asthma.

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Authors

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