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Basic Science

Abstract Title

The influence of developmental cannabis smoke exposure on the responses to re-exposure in adulthood: evidence for DOHaD in lung function and epigenetics.

Background

Prenatal cannabis use has risen with legalization, yet its effects on fetal lung health remain unclear. While prenatal tobacco exposure is strongly linked to childhood asthma, cannabis studies have focused mainly on neurodevelopment, leaving lung outcomes understudied. Epigenetic mechanisms, particularly DNA methylation (DNAm), can reveal how early exposures shape long-term health. The Developmental Origins of Health and Disease (DOHaD) hypothesis proposes that such exposures “prime” the epigenome, creating lasting molecular memory. We investigated whether early-life cannabis smoke (CS) exposure primes the lung to alter responses to re-exposure in adulthood.

Objective

We hypothesized that early-life CS exposure primes the lung, such that adult re-exposure produces synergistic effects on lung function, potentially linked to DNAm changes.

Methods

Female Balb/c mice (8 weeks old) were exposed to CS for 40 minutes, five days per week, over nine weeks spanning mating, pregnancy, and weaning. Age-matched room air-exposed mice served as controls. Once offspring were born, they were aged to 36–39 weeks, after which half of each group was re-exposed to CS for three weeks, generating four groups ($n = 3\text{--}4/\text{group}$): Early-life exposed + re-exposed, Early-life exposed only, Control + re-exposed, and Control only. Lung function was then assessed, and whole-lung DNAm profiled using Illumina microarrays.

Results

Male offspring in the Early-life exposed + re-exposed group showed non-significant increases in tissue damping (G , mean = 5.2, $p = 0.74$) and elastance (H , mean = 27, $p = 0.66$), suggesting a potential synergistic effect. Female offspring showed a significant decrease in tissue damping (G , mean = 3.2, $p = 0.017$), indicating sex-specific vulnerability. DNAm analysis is ongoing.

Conclusion

Lung function changes were modest and largely non-significant but suggest possible sex-specific susceptibility to early-life cannabis smoke exposure. Ongoing DNAm profiling will clarify underlying epigenetic mechanisms within the DOHaD framework.

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