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Graduate

Poster Submissions

Poster Title

Molecular impact of electronic cigarette exposure on airway epithelium

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Please describe the extent of your work in this research

I oversee the project including all in vitro exposures, as well as running and analyzing the data from all in vitro assays following exposure.

Abstract Submission

- [Evans-Day-Abstract-2015-ECIG_v3.docx](#)

Would you like your abstract to be considered for an oral presentation (students and post docs only)?

Yes

Molecular impact of electronic cigarette exposure on airway epithelium

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Objective: Electronic cigarettes (ECIGs) simulate cigarette smoking by delivering nicotine via an aerosol and are advertised as an alternative to tobacco cigarette (TCIG) smoking. However, there are few studies evaluating the physiological impact of ECIG use. Our objective was to determine the cellular and molecular impact of ECIG aerosol on human bronchial epithelial cells (1) *in vivo* and (2) *in vitro*.

Methods: For the *in vivo* arm of this study, we profiled bronchial epithelial cells from the mainstem bronchus of former TCIG smokers (n=10) and former smokers who have transitioned to ECIGs (n=8). For the *in vitro* arm, human bronchial epithelial cells (HBECs) differentiated at an Air Liquid Interface (ALI) were directly exposed to ECIG vapor or TCIG smoke. ECIG exposures examined the effects of variations in dose, flavoring, and nicotine content.

Results: We identified a signature of 199 genes ($p < 0.005$) associated with ECIG use *in vivo*. These gene-expression changes are enriched for genes that are induced with TCIG exposure, which suggests that components of the host response to ECIG and TCIG exposure are concordant. *In vitro*, we found that while there is partial overlap between gene expression changes induced by ECIG and TCIG exposure, there are also unique effects of ECIG exposure related to cell cycle and proliferation. In addition, ECIG exposure, like TCIG exposure, induces genes involved in response to oxidative and xenobiotic stress.

Conclusions: Together, these results indicate that ECIG vapor can induce cellular stress and molecular alterations within airway epithelium that share similarities with the effects of TCIG smoke. These findings underscore the urgent need for physiologically relevant studies to fully determine the type and magnitude of the cellular and molecular response to ECIG exposure in order to determine whether their use may lead to some of the same deleterious health outcomes as those caused by tobacco smoke exposure.