

TITLE

Gene Regulation and Viability of Cell Cultures Exposed to E-Cigarette Refill Solutions

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ABSTRACT

Marketed as smoking cessation aids, E-cigarettes have become increasingly popular in the past decade. One of the primary tobacco alkaloids, nicotine, is a measured and reported within an e-cigarette refill solution. However, other tobacco alkaloids are present as well and are not disclosed on the packaging. Using an *in vitro* culture system, we tested of the effects of these chemicals on the growth of human lung cells in culture. Cell viability was measured as a function of metabolic ATP activity, using the Cell-Titer Glo Luminescent assay. Overall, the presence of alkaloids generally decreased cell as compared to control (non-challenged) cultures.

There have been a number of studies that have linked changes in gene expression to tobacco-related diseases in humans. However, the effects of e-cigarettee solutions have not been well documented. Our group used qRT-PCR to analyze gene expression of cell cultures challenged with alkaloids. We hypothesize that differential gene regulation associated with cells exposed to these alkaloids is similar to that found in regular tobacco-related disease. To test this hypothesis, we used implicated in carcinogenesis and tumorigenesis including: adhesion (CEACAM6, CX3CL1), immune response (TLR4, CX3CL1, CEACAM6), oxidative stress (GPX2, ALDH3A1), putative oncogenes (PIR, CEACAM6), a putative tumor suppressor genes (SLIT1), and xenobiotic metabolism (CYP1A1, AHR, ALDH3A1). Expression of these markers were compared between cells treated with the aforementioned alkaloids and control cultures. Our results show significant ($p < 0.05$) differential regulation of ALDH3A1, CEACAM6, PIR, and TLR4 in response to alkaloid exposure. This study illustrates the need to better understand the impact that electronic cigarettes are having on the health of the individuals that use this project as a smoking cessation aid.