Electronic Cigarettes Induce Chronic Obstructive Pulmonary Disease in a Pre-Clinical Model
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ABSTRACT
Background: Chronic obstructive pulmonary disease (COPD) is currently listed as the 4th leading cause of death and is projected to be the 3rd leading cause of death by 2020. Cigarette smoking is considered to be the leading cause of COPD in the developed world; however, with the emerging popularity of electronic cigarettes (e-cigarettes), the impact of e-cigarette vapor on the development of COPD requires further attention. COPD is characterized by limitations in expiratory airflow, emphysematous destruction of the lungs, bronchitis, and chronic inflammation of the lung tissue. Most of the e-cigarettes are introduced as a healthier tool to help people to quit the traditional cigarette. This is a device that effectively transports evaporated liquid nicotine to the lungs. Users can choose the nicotine concentration of the e-cigarette liquid (e-liquid) that loaded into the device's cartridge. When inhaled, the e-liquids (nicotine) is heated to produce vapor that enters the lungs.

Hypothesis: We hypothesized that nicotine-containing e-cigarettes induce the exacerbation of COPD in a pre-clinical model.

Methods: Twenty Scnn1b-Tg+ mice were exposed to nicotine-containing e-cigarette vapor for ten days, each mice was exposed two hours per day. This transgenic animal model exhibits mucus hypersecretion and defective mucus clearance in the lung closely mimicking human COPD onset and progression. After treatment, bronchoalveolar lavage (BAL) fluid, lung tissues, and serum were collected to assess for inflammation, fibrosis, and mucus accumulation.

Results: Inflammatory cytokines such as CXCL1, MMP-2, and CX3CL1 concentrations were significantly higher in animals exposed to e-cigarette vapor. Moreover, e-cigarette exposure increased mucus accumulation and fibrosis production in the bronchioles of the Scnn1b-Tg+ mice.

Conclusion: These results suggest that nicotine-containing e-cigarette vapor induce the exacerbation of COPD in our animal model. Future studies will focus on chronic exposure of e-cigarette vapor to evaluate the mechanism of nicotine-containing e-cigarettes on the pathogenesis of COPD.