

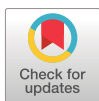


# Nicotine promotes e-cigarette vapour-induced lung inflammation and structural alterations

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Shareable abstract (@ERSpublications)

**E-cigarette use, particularly with nicotine-containing vapour, is a harmful alternative to tobacco smoking. Nicotine-containing e-cigarette vapour increases pulmonary endothelial permeability, induces inflammation and causes airway and parenchymal alterations.** <https://bit.ly/40s24n9>

**Cite this article as:** Roxlau ET, Pak O, Hadzic S, *et al.* Nicotine promotes e-cigarette vapour-induced lung inflammation and structural alterations. *Eur Respir J* 2023; 61: 2200951 [DOI: 10.1183/13993003.00951-2022].

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This article has an editorial commentary:  
<https://doi.org/10.1183/13993003.00886-2023>

Received: 9 May 2022  
Accepted: 19 March 2023

## Abstract

**Background** Electronic cigarette (e-cigarette) vapour is gaining popularity as an alternative to tobacco smoking and can induce acute lung injury. However, the specific role of nicotine in e-cigarette vapour and its long-term effects on the airways, lung parenchyma and vasculature remain unclear.

**Results** *In vitro* exposure to nicotine-containing e-cigarette vapour extract (ECVE) or to nicotine-free e-cigarette vapour extract (NF ECVE) induced changes in gene expression of epithelial cells and pulmonary arterial smooth muscle cells (PASMCs), but ECVE in particular caused functional alterations (*e.g.* a decrease in human and mouse PASMC proliferation by 29.3±5.3% and 44.3±8.4%, respectively). Additionally, acute inhalation of nicotine-containing e-cigarette vapour (ECV) but not nicotine-free e-cigarette vapour (NF ECV) increased pulmonary endothelial permeability in isolated lungs. Long-term *in vivo* exposure of mice to ECV for 8 months significantly increased the number of inflammatory cells, in particular lymphocytes, compared to control and NF ECV in the bronchoalveolar fluid (BALF) (ECV: 853.4±150.8 cells·mL<sup>-1</sup>; control: 37.0±21.1 cells·mL<sup>-1</sup>; NF ECV: 198.6±94.9 cells·mL<sup>-1</sup>) and in lung tissue (ECV: 25.7±3.3 cells·mm<sup>-3</sup>; control: 4.8±1.1 cells·mm<sup>-3</sup>; NF ECV: 14.1±2.2 cells·mm<sup>-3</sup>). BALF cytokines were predominantly increased by ECV. Moreover, ECV caused significant changes in lung structure and function (*e.g.* increase in airspace by 17.5±1.4% compared to control), similar to mild tobacco smoke-induced alterations, which also could be detected in the NF ECV group, albeit to a lesser degree. In contrast, the pulmonary vasculature was not significantly affected by ECV or NF ECV.

**Conclusions** NF ECV components induce cell type-specific effects and mild pulmonary alterations, while inclusion of nicotine induces significant endothelial damage, inflammation and parenchymal alterations.

