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E.T. ROXLAU ET AL.

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

Elsa T. Roxlau[1,10], Oleg Pak[1,10], Stefan Hadzic[1], Claudia F. Garcia-Castro[1], Marija Gredic[1], Cheng-Yu Wu[1], Julia Schäffer[1], Balachandar Selvakumar[1,2], Alexandra Pichl[1], David Spiegelberg[1], Janik Deutscher[1], Mariola Bednorz[1], Katharina Schäfer[1], Simone Kraut[1], Djuro Kosanovic[1,3], Esraa M. Zeidan[1,4], Baktybek Kojonazarov[1,5], Susanne Herold[1], Ievgen Strielkov[1], Andreas Guenther[1], Jochen Wilhelm[1,5], Mohamed M.A. Khalifa[4], Ashraf Taye[6], Ralf P. Brandes[7], Matthias Hecker[1], Friedrich Grimminger[1], Hossein A. Ghofrani[1,8], Ralph T. Schermuly[1], Werner Seeger[1,5,9], Natascha Sommer[1] and Norbert Weissmann[1]

[1]Justus Liebig University, Excellence Cluster Cardio-Pulmonary Institute (CPI), Universities of Giessen and Marburg Lung Center (UGMLC), Member of the German Center for Lung Research (DZL), Giessen, Germany. [2]Sharjah Institute of Medical Research (SIMR), College of Medicine – University of Sharjah (UoS), Sharjah, United Arab Emirates. [3]I.M. Sechenov First Moscow State Medical University (Sechenov University), Moscow, Russia. [4]Department of Pharmacology and Toxicology, Faculty of Pharmacy, Minia University, El-Minia, Egypt. [5]Institute for Lung Health (ILH), Giessen, Germany. [6]Department of Pharmacology and Toxicology, Faculty of Pharmacy, South Valley University, Qena, Egypt. [7]Institute for Cardiovascular Physiology, Goethe University, Cardio-Pulmonary Institute (CPI), Frankfurt, Germany. [8]Department of Medicine, Imperial College London, London, UK. [9]Max Planck Institute for Heart and Lung Research, Bad Nauheim, Germany. [10]Joint first authors.

Corresponding author: Natascha Sommer (natascha.sommer@innere.med.uni-giessen.de)

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

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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^{-1}; control: 37.0±21.1 cells·mL^{-1}; NF ECV: 198.6±94.9 cells·mL^{-1}) and in lung tissue (ECV: 25.7±3.3 cells·mm^{-3}; control: 4.8±1.1 cells·mm^{-3}; NF ECV: 14.1±2.2 cells·mm^{-3}). 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.