



CoMBoS2 – the Second Congress of Molecular Biologists of Serbia, Abstract Book – Trends in Molecular Biology, Special issue 06-08 October 2023, Belgrade, Serbia Online Edition https://www.imgge.bg.ac.rs/lat/o-nama/kapacitet-i-oprema/istrazivackadelatnost

https://indico.bio.bg.ac.rs/e/CoMBoS2

IMPRESSUM

PUBLISHER: Institute of Molecular Genetics and Genetic Engineering (IMGGE), University of Belgrade

FOR THE PUBLISHER: Dr. Sonja **Pavlović**

EDITOR: Dr. Zorana **Dobrijević**

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DESIGN: Ivan **Strahinić**

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ELECTRONIC CIGARETTE VAPOUR CONDENSATE AFFECTS MITOCHONDRIAL POTENTIAL IN BEAS2B CELLS

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Introduction: Cigarette smoke exposure is a known risk factor for development of lung diseases and electronic cigarettes (e-cigarettes) were introduced as a popular and safer alternative to combustible tobacco products. Increasing number of studies are reporting their adverse biological effects both in vivo and in vitro. Aim of this study was to evaluate the effect of e-cigarettes on mitochondrial function in lung bronchial epithelial cells.

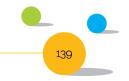
Methods: Electronic cigarette vapor condensate (ECC) was generated using an e-cigarette device on a suction trap cooled in a dry ice/ethanol bath. We used unflavoured and flavoured e-cigarette liquids with and without nicotine. Human bronchial epithelial BEAS2B cells were seeded in 96well plates and treated with 2% e-cigarette vapour condensate for 24h. Mitochondrial membrane potential was measured using 50nM TMRE (Tetramethyl rhodamine ethyl ester) and cells were visualized on ImageXpress[®] Pico Automated Cell Imaging System (Molecular Devices, San Jose, CA, USA) with a 10x objective.

Results: We found a significant reduction of TMRE fluorescence in treated cells compared to the control. Imaging of treated cells also revealed changes in cell morphology and the presence of mitochondria in TNT-like structures.

Conclusion: Mitochondrial dysfunction has been associated with various pathological conditions including lung diseases such as asthma, COPD and lung cancer. Due to their relative novelty, the role of electronic cigarette use in development of chronic lung diseases is still relatively unknown. Our findings contribute to the growing list of studies pointing to their adverse biological effects and imply their involvement in processes contributing to chronic lung diseases.

Key words: electronic cigarettes; mitochondria; lung disease

Acknowledgments: This study was supported by the Ministry of Science, Technological Development and Innovation of the Republic of Serbia (Agreement no. 451-03-47/2023-01/ 200042).



Session MOLECULAR MECHANISMS OF CELL FUNCTIONS