OCCUPATIONAL SECONDHAND SMOKE EXPOSURE MAY MODIFY THE PROTEOMA EXPRESSION OF HUMAN NASAL EPITHELIUM

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The tobacco is one of the biggest public health threats, smoking kills more than 7 million people/year worldwide and more than 890,000 are deaths resulting from exposure to Second Hand Smoke (SHS). In adults, SHS is associated to cardiovascular and respiratory diseases, including coronary heart disease and lung cancer, through pathological and molecular mechanisms not yet understood [1]. In this study, we aimed to investigate the SHS effects on airway proteome in exposed workers.

Nasal epithelium was collected from hospitality workers (non-smokers=40; smokers=12) long-term exposed and non-exposed to SHS at the workplace. Samples were analyzed by shotgun proteomics using an *ESI-LQT Orbitrap XL* mass spectrometer. The generated MS raw data was submitted to 'PatternLab for Proteomics 4.0' for peptide identification and relative quantification by label-free - extracted ion chromatograms (XIC). Golden rules were applied to obtain reliable data such as the inferred proteins must have at least one unique peptide identified to be considered and be detected in at least 80% of the cohort.

Two proteins were found to be differentially expressed in the no-smokers exposed to SHS compared with the control: *BPI fold-containing family A member 1* (BPIFA1) and *Heat shock Protein Beta-1* (HSPB1). The first protein plays a role in the airway inflammatory response after exposure to irritants substances [2] and the second is associated as a regulator of actin filament dynamics [3].

Our findings support the indication that in non-smokers the prolonged exposure to SHS can lead to airway proteome modulation. When validated, the uncovered proteins can be promising candidates to "susceptibility/risk" and/or "response" biomarkers for SHS exposure.

References
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