The effect of particulate matter air pollution on human bronchial epithelial cell lines

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Background:
Air pollution, particularly the fine and ultrafine particulate matter ($PM_{2.5}$, and $PM_{0.1}$, i.e. particles with aerodynamic diameter <2.5 μm and 0.1 μm, respectively), is one of the first risk factor for the development of non-communicable diseases (NCDs), due to the ability of $PM_{2.5}$ and $PM_{0.1}$ to reach the pulmonary alveoli (1). Recent data demonstrate that freshly emitted $PM_{2.5}$ and $PM_{0.1}$ induce DNA damage and oxidative stress after 24h of exposure (2, 3).

Methods and Results:
To understand the physiological molecular mechanisms involved in the early response to $PM_{2.5}$, after developing a cell culture model, we are studying the effects of $PM_{2.5}$ on human epithelial cells, with a specific focus on genes of the inflammation and oxidative stress processes. In particular, fresh $PM_{2.5}$ and $PM_{0.1}$ are able to induce a clear response event at lower times of exposure (5, 10, 20 min, 1h), with a wave of activation of the pathways related to detoxification (i.e., Nrf2, NFkB, HIF1α, HO1, CAT) and inflammation (i.e., TNFα).

Conclusions and Significance:
The project, included in the activities of NBFC-Spoke 6, highlights the role of oxidative stress, detoxification and inflammation as a response of human bronchial cells to exposure to $PM_{2.5}$ and $PM_{0.1}$in short timepoints, opening new scenarios that investigate the regulation of these functional pathways (4).

Keywords: particulate matter, lung epithelial cells, exposome, inflammation, oxidative stress.

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