Cellular responses after exposure of lung cell cultures to secondary organic aerosols

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Ambient fine and ultrafine particles have a variety of adverse health effects. The chemical and physical properties of aerosol particles causing these effects remain unclear. A major fraction of the ambient aerosol particle mass is composed of secondary organic aerosol (SOA). Within the interdisciplinary POLYSOA project (Baltensperger et al. 2008) this work aimed to examine in vitro the response of target lung cells to SOA particles with the goal to eventually identify particle components that are responsible for cell responses.

SOA particles were deposited on the air-liquid interface of cultured porcine and human lung epithelial cells (micro-dissected tracheal epithelium, primary cultures and cell lines) and lung surface macrophages in a recently constructed particle deposition chamber (Savi et al. 2008). Particles were applied under realistic ambient air and physiological conditions occurring when particles are inhaled by mammals.

Cellular responses were examined within 24 hrs after exposure to SOA. Ultrastructural changes of cells were assessed by transmission electron microscopy. Necrotic cell death was tested by measuring lactate dehydrogenase release. Phagocytic activity of macrophages was tested by post-exposure treatment with 6-μm polystyrene particles. Inflammatory responses were assessed by measuring TNF-α, IL-6 and IL-8 release. In addition, epithelial repair function was tested by measuring the closure of mechanically wounded alveolar epithelial cell monolayers using a computerized imaging technique (Geiser et al., 2000).

Analyses of the lung cells indicate that a short time exposure to realistic concentrations of SOA does not induce cytotoxicity but leads to subtle changes in cell function that are essential for lung homoeostasis. We found decreased phagocytic activity in macrophages (Fig. 1) and cell type specific increases in IL-8 release. The alveolar epithelial wound repair was affected mainly due to alterations of cell spreading and cell migration at the edge of the wound.

Figure 1. The phagocytic activity of human macrophages, i.e. the number of phagocytosed particles per macrophage (mph), was significantly decreased (*p<0.05) after exposure to SOA from α-pinene (2 experiments) as compared to untreated cells (incubator control) and cells exposed to particle-free air.

These analyses of cellular responses induced by organic aerosols will greatly help to understand the particle properties as well as the cellular mechanisms responsible for biological effects.

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