Effects of secondary organic aerosols from gasoline exhaust on healthy and diseased respiratory epithelia

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Keywords: secondary organic aerosols, SOA, gasoline exhaust, health effects
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Gasoline exhaust is a major anthropogenic source of atmospheric particles (Bahreini, 2012) and potentially contributes to respiratory diseases from inhalation exposure. Individuals with pre-existing lung diseases are more vulnerable than healthy people.

The aim of this study was to investigate acute responses of cultured respiratory epithelium cells after exposure to photochemically aged aerosols from gasoline exhaust using advanced technologies from aerosol science and in-vitro toxicology.

To achieve this aim, exhaust of a gasoline car (EURO 5 standard) was diluted and injected into a smog chamber consisting of a 27 m^3 fluorinated ethylene propylene bag suspended in a temperature-controlled enclosure, resulting in aerosol concentrations of typically 10-30 μg m^{-3} before and 100-250 μg m^{-3} after aging. A combination of instruments monitoring gas- as well as particle phase allowed precise measurements of several parameters. These included chamber temperature, relative humidity, oxides of nitrogen, ozone, total hydrocarbon, black carbon, as well as the total particle number and volume concentration. In addition an aerosol mass spectrometer was used to monitor chemical composition during the aerosol aging process.

After injection of exhaust and homogenisation/equilibration of the chamber, secondary organic aerosol (SOA) was produced through photochemical processes initiated by irradiation with artificial sunlight (4 xenon arc lamps and 80 blacklights). Then, the aged aerosol was transferred from the smog chamber through a versatile aerosol concentration enrichment system (VACES) (Wang, 2013) into an aerosol deposition chamber (Mertes, 2013). By enriching particle concentrations up to a factor of 50, the dose of particles deposited on cell cultures could be varied over a broad range.

The total particle concentration was permanently measured after the enrichment system. Harmful gases were removed using a charcoal denuder and SOA was deposited out of a continuous air-flow (0.6 L/min) simultaneously on 12 individual cell cultures through electrostatic deposition. The conditions in the aerosol deposition chamber were continuously recorded and could be adjusted to a relative humidity of 85-95 % and a temperature of 37 °C. Therefore, particle-cell interactions could be simulated at physiological conditions while minimizing stress on the cell cultures. A detailed knowledge of particle deposition characteristics on the cell cultures allowed evaluating reliable dose-response relationships (Mertes, 2013).

Re-differentiated human bronchial epithelia (HBE) cultured at an established air-liquid interface and the bronchial cell line BEAS-2B were exposed to the aerosol for 2 hours. Biological endpoint tests determined the dose-response relationships in the exposed cells. Several tests were performed including (i) necrotic cell death assessed by release of lactate dehydrogenase from damaged cells, (ii) induction of apoptosis by activation of caspase-3, (iii) inflammatory responses by expression and release of inflammatory mediators and (iv) oxidative stress by induction of mRNA synthesis of respective markers.

Results demonstrate cytotoxicity as well as inflammatory responses of HBE and BEAS-2B cells after exposure to SOA from gasoline exhaust. Different responses of cell cultures from a donor with lung disease were observed compared to HBE from a healthy donor. In addition, there is evidence for substantial differences between responses of BEAS-2B cells and HBE cells after aerosol exposure, which challenges the use of cell lines to study effects of inhaled particles in vitro.

The combination of aerosol science and in-vitro toxicology technologies provides a highly realistic and reproducible system for testing toxicity of environmental aerosols and determining dose-response relationships.

This work was supported by the Swiss National Science Foundation (CR3213-140851).
Special acknowledgements go to the University of Southern California (USC) Viterbi School of Engineering.

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