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Differential impact of biogenic and anthropogenic secondary organic aerosol compounds adsorbed on soot particles in lung cell models at the air-liquid interface

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BACKGROUND & MOTIVATION: Secondary organic aerosols (SOA) formed from anthropogenic or natural gaseous precursors are substantially contributing to the ambient $PM_{2.5}$ burden, which is known to correlate with adverse human health effects ^[1]. However, our knowledge is mostly limited to the effects of collected airborne particles under submerged exposure conditions rather than the direct deposition and interaction of aerosols with cell cultures ^[2]. Moreover, a direct link between single aerosol compounds and their health hazards remains largely unknown ^[3, 4]. Therefore, we were interested in differentiating the toxicological effects of combustion-derived soot particles (SP) from the effects induced by the soot photochemically aged together with a biogenic (β -pinene) or an anthropogenic (naphthalene) volatile organic compound in two different lung cell models exposed at the air-liquid interface (ALI).

METHODS: Monoculture of lung epithelial cells (A549) and a co-culture model with A549 and endothelial cells (EA.hy926) were exposed at the ALI for 4 h to different aerosol concentrations of pure SP or a photochemically-aged mixture of primary combustion SP and β -pinene (SOA_{βPIN}-SP) or naphthalene (SOA_{NAP}-SP). The internally mixed soot/SOA particles were comprehensively characterized in terms of their physical and chemical properties. We conducted toxicity tests to determine cytotoxicity, intracellular oxidative stress, primary- and secondary-genotoxicity as well as inflammatory and angiogenic effects.

RESULTS & DISCUSSION: Both investigated SOA types caused significant toxicological effects, while the nano-sized soot cores alone showed only minor toxic effects under the current experimental settings. The toxicological assays furthermore indicated greater adverse effects of SOA_{NAP}-SP compared with SOA_{βPIN}-SP in both cell models. At the functional level, we showed that SOA_{NAP}-SP augments the secretion of e.g. malondialdehyde and interleukin-8, and may induce the activation of endothelial cells in the co-culture system. This activation was confirmed by comet assay suggesting secondary genotoxicity and an increased angiogenic potential. Chemical characterization of PM revealed distinct qualitative differences in the composition of the two secondary aerosol types. It is shown that SOA-compounds can increase the toxicity of primary SP, which are ubiquitous in inhabited and wildfire influenced areas. Aromatic precursors, such as naphthalene caused the formation more oxidized, more aromatic SOA of higher oxidation potential with higher toxicity compared to an aliphatic precursors, such as β -pinene. The influence of atmospheric chemistry on the chemical PM composition thus can play a crucial role for the adverse health outcome of emissions.

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