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Molecular adsorption at PM-surfaces

Recent studies reported here show particulate matter (PM)-lung fluid interactions result in selective molecular adsorption at PM surfaces. Such adsorption onto nonbiological PM surfaces deposited in fluid lining the lung may mediate the toxicity of PM. Little is currently known about specific interactions at the PM/fibre-lung interface and how these determine downstream health effects.

In one series of PM studies, the surfaces of different types of collected PM_{2.5} (urban, “clean air”, and tobacco smoke) were found to vary in composition as measured by XPS (X-ray photo spectroscopy) and ToFSIMS (time-of-flight secondary ion mass spectrometry). Both XPS and ToF-SIMS detected PM_{2.5} surface species and observed changes in surface concentrations after treatment with human lavage fluid. XPS analysis showed the surface of untreated urban PM_{2.5} was dominated by carbon and 10-16% oxygen, with smaller contributions of N, S, Si, and P. A wider variety of other inorganic and organic species (including metals, aliphatic and aromatic hydrocarbons, and nitrogen-containing molecules) was detected with ToF-SIMS. Comparable species and quantities were identified in a previous study of London PM_{2.5}, where PM_{2.5} surface chemistry differed considerably depending on the source. After treatment with BALF, the N-C signal increased (detected by both XPS and ToF-SIMS), indicating significant surface adsorption of protein or other N-containing biomolecules. ToF-SIMS also indicated an adsorption of phospholipid on the treated PM_{2.5} surfaces. Analyses of the soluble species NH₄⁺, NO₂⁻, Si, and S indicated decreases in both saline- and BALF-treated samples, showing that these species may be bioavailable in the lung. ToF-SIMS additionally suggested the bioavailability of Na⁺ and Al⁺. Such surface modifications are likely to be important in mediating PM toxicity.

In further studies of PM adsorption using surrogate particles, colloidal suspensions of three types of carbon black (CB) particles with different surface chemistry and decreasing surface areas were generated. Single lung lining fluid components spanning physiological concentrations were added. For DPPC combinations with particles, visible particle agglomeration occurred within 1 h and reduction of DPPC occurred in a surface- and size-dependent manner. This indicated that surface adsorption was responsible for the observed agglomeration and the gross reductions in phospholipid concentrations. Combination of particles with fibrinogen and albumin, revealed little agglomeration/precipitation at the protein concentrations chosen. However, one surfactant protein (SP) was completely eliminated from suspension upon combination with all three-particle types. The reaction between SP and particles was therefore concluded to be independent of surface chemistry. If such surface modifications occur in lung-deposited PM, they are also likely to mediate PM toxicity.

Finally, experiments using atomic force microscopy (AFM) showed that lung lining liquid modified the attractive forces at the surface of PM_{2.5}, leading to enhanced particle aggregation. We proposed that this was an important protective mechanism that aids particle clearance in the lung. Further work is currently being undertaken to determine how individual molecules and specific mixtures influence PM agglomeration, and how surface chemistry affects cellular responses to PM.

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