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### **The effects of ultrafine or nano-particles on lung cells**

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Particulate air pollution (PM10) consists of a complex mixture of components including primary particles, derived predominantly from man-made sources such as traffic and industrial emissions. Such emissions include mainly ultrafine or nanoparticles (less than 100 nm diameter) consisting of carbon associated with metals and organic matter. In addition particulate air pollution contains biological materials such as bacteria, and secondary particles formed through photochemical reactions.

A toxicology study was conducted to identify which components of PM10 might induce an adverse reaction in the rat lung. Inflammation (activation of immune cells such as macrophages and neutrophils) is implicated in many diseases such as bronchitis, smokers lung disease (COPD) and cardiovascular disease, and these diseases are known to be enhanced by air pollution. In this study inflammation within the lung airspaces was measured as a marker of the response to PM10. The study compared PM10 collected from 6 different UK locations and found that their ability to induce inflammation was linked to (in decreasing order of importance) the mass dose to which the animals were exposed, the metal content (mainly the zinc and iron content), and the primary particle content. Secondary particles and markers of bacterial content were not associated with the induction of inflammation. Organic components were not included in the analysis.

In parallel studies using low toxicity particles of different chemical composition (carbon, TiO<sub>2</sub> or polystyrene), particles with a diameter of less than 100 nm were found to be more potent at inducing inflammation than the same mass of larger particles (200-260 nm diameter). The ability of the particles to induce inflammation was directly related to the surface area dose introduced into the lung. In a chemical assay the smaller particles were also found to generate more toxic free radicals and reactive oxygen species (ROS) than the larger particles.

Since PM10 contains metals that also generate (ROS), nanoparticle carbon particles were mixed with either iron chloride, iron sulphate or copper sulphate in order to investigate their potential to interact. All three metal salts induced a potentiation of the ROS production by the particles as well as a potentiation of their ability to induce inflammation in the lung.

Treatment of macrophage cells in culture with either PM10 or the nanoparticles, but not the larger particles, induced a dose dependent increase in the production of a protein, tumour necrosis factor alpha (TNF- $\alpha$ ), that drives inflammation. PM10 was more potent than the pure nanoparticles suggesting that other factors within the mixture also activate the macrophages. Co-treatment of the macrophages with carbon nanoparticles and iron salts did not alter TNF- $\alpha$  production, however, zinc chloride synergistically increased the production of this protein. This data suggests that both metals and nanoparticles are important in driving inflammation, and that the two components interact to enhance their toxicity in the lung.

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