Exposure and harm to combustion-derived wood particles

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The human respiratory system is the gateway of entry for inhaled detritus from anthropogenic (e.g. combustion-derived (CD) particulate matter (PM; e.g. diesel exhaust and wood-burning PM). Adult humans inhale 20m^3 of air and suspended debris (gases and particles) into the airways daily. Inhalation exposure to CDPM (Figure 1) is known to increase the risk of morbidity and mortality of lung and heart diseases in all exposed individuals

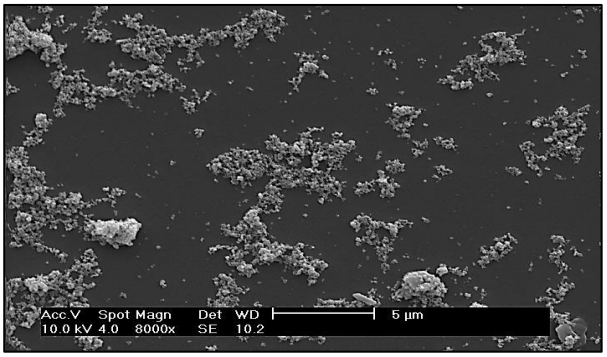

Figure 1. Scanning electron microscopy of spruce wood CDPM.

The physicochemical properties of size, surface area and presence of transition metals have been implicated as drivers of the oxidative capacity of CDPM. However, the precise role of reactive organic compounds (ROC) in ambient aerosols, present either in the gas or particle phase has not been fully-investigated for their relevance in the induction of the observed adverse health effects.

When addressing the toxicity of inhalation hazards such as wood smoke CDPM, a model that resembles the human lung responding to toxic challenges is required. In our *in vitro* exposure studies, we utilised normal human bronchial epithelial (NHBE) cells grown at the air-liquid interface (ALI) using filter-well technology (Prytherch *et al* 2011), to create an *in vivo*-like 3-dimensional lung model.

This model is a fully-differentiated, pseudo-stratified, muco-ciliary epithelium containing basal, serous, Clara, goblet and ciliated cells. NHBE cells were exposed to wood smoke derived from Spruce, Beech and Birch at a dose of 152µg/cm^2: carbon black (CB; negative control; Monarch 120, Cabot UK; DQ12 quartz (positive control). Following exposure (24 hours), tissue integrity (i.e. transepithelial electrical resistance (TEER) was measured to reveal minor disruption to bronchial tissue integrity (Figure 2). However, changes in cellular energy levels (i.e. ATP) between the types of wood smokes (Figure 3), could infer the smoke acted as an irritant to the lung environment. Wood smoke exposure can depress the immune system and damage the layer of cells in the lungs that protect and cleanse the airways.

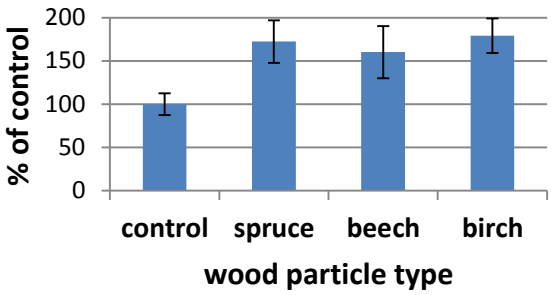

Figure 2. TEER readings for types of wood-exposed cells.

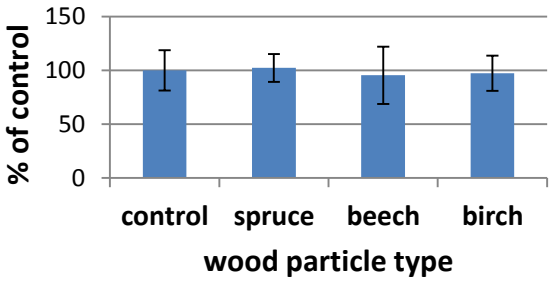

Figure 3. ATP readings for types of wood-exposed cells.

Further work on the biological and histological impacts of wood smoke will allow us to reveal mechanisms behind the changes observed, as well identifying biomarkers of cell damage by specific CDPM ROCs.

For vulnerable populations, such as people with asthma, chronic respiratory disease and those with cardiovascular disease, wood smoke is particularly harmful, even at short exposures it can prove dangerous. Wood smoke interferes with normal lung development in infants and children. It also increases children's risk of lower respiratory infections such as bronchitis and pneumonia.

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