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Toxicity of wood smoke particles in human lung epithelial cells: the role of PAHs, soot and zinc

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The health impact of emissions from domestic burning of biomass and coal is estimated to contribute to over 4 million premature deaths per year worldwide. Wood is the main fuel source for biomass combustion and the shift towards renewable energy sources will further increase emissions from wood combustion even in developed countries. However, little is known about the constituents of wood smoke and biological mechanisms responsible for adverse health effects.

We exposed human A549 and BEAS-2B lung epithelial cells to freshly generated wood smoke at the air-liquid-interface (ALI) using a continuous flow exposure system. Toxicity at ALI was compared to submerged exposure with collected wood smoke particles (WSP). To address critical constituents of WSP, we exposed A549 cells under submerged conditions individually to benzo[a]pyrene (B[a]P), carbon black nanoparticles (CB14, Printex 90®) and zinc oxide nanoparticles (ZnO) to represent the polycyclic aromatic hydrocarbons (PAHs), soot and metal fraction of WSP, respectively.

At the ALI, in both cell lines, the 1:10 diluted wood smoke did not induce cell death. However, enhancing the particle dose by use of an electrostatic field, led to an increase of toxicity, while this method had no effect on cells when exposed to clean air.

Under submerged conditions, even particle doses greatly exceeding the toxic dose at the ALI provoked no signs of acute toxicity. However, WSP induced formation of cellular reactive oxygen species (ROS) as well as a response to bioavailable PAHs. We thus tested the contribution of B[a]P, ZnO and CB14 to the observed effects of WSP.

ZnO and CB14 were able to induce ROS formation in cells, measured as enhanced H_2DCF oxidation. However, the magnitude of the effects differed considerably. ZnO increased ROS formation only at high concentrations, which are well beyond the levels of Zn present in the tested WSP samples. CB14, however, potently induced H_2DCF oxidation, in comparable magnitude to WSP when EC content is used as dose metric. Of note, augmented ROS formation by WSP or CB14 did not trigger an adaptive anti-oxidative stress response in A549 cells, evidenced by a lack of heme oxygenase-1 up-regulation at the protein and mRNA level.

As expected, the PAH response induced by WSP could be mimicked by B[a]P. Strikingly, PAHs adsorbed to WSPs were even more potent in activating target gene expression than B[a]P individually applied in suspension. As PAHs are adsorbed on the particle surface, particles might serve as a vehicle to deliver PAHs to the cell either due to particle sedimentation and/or increasing PAH uptake via a "trojan horse" mechanism, where particles act as carriers for PAHs.

In conclusion, this study demonstrates cytotoxicity of WSP specifically at the air-liquid-interface. Mechanistic investigations employing classical submerged cell culture methods indicated a critical role of soot, metals and especially PAHs in hazardous effects of WSP (Figure 1). As metabolic activation of PAHs is critically linked to genotoxicity, mutagenesis and carcinogenesis, the effect of WSPs on these endpoints needs to be critically evaluated in the future. Furthermore, a systematic comparison of toxicological responses at the ALI and in conventional cell culture systems is warranted and needs to be scrutinized in animal studies.

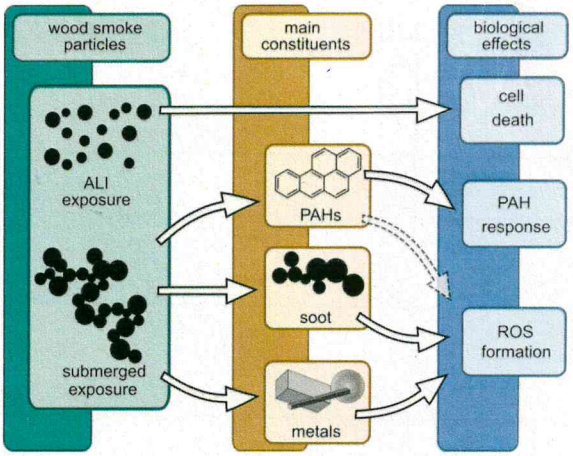

Figure 1. WSP lead to cell death at the air-liquid-interface and induce ROS formation as well as a strong PAH response under submerged conditions. These effects can be mimicked by individual substances representing the PAH, metal and soot fraction of WSPs.

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