ULTRAFINE PARTICLES ARE NOT MAJOR CARRIERS OF CARCINOGENIC PAHS AND THEIR GENOTOXICITY IN SIZE-SEGREGATED AEROSOLS

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Abstract

Recent research indicates gaps of the current policy framework, leaving health relevant size fractions of particulate matter (PM) of ambient air pollution unregulated, thus, jeopardizing public health by particles of aerodynamic diameter - dae of submicron (dae <1um) or even ultrafine (dae <0.1um) fractions. Genotoxic effects of the combustion related PM are mainly induced by carcinogenic polycyclic aromatic hydrocarbons (c-PAHs) and their derivatives forming organic fraction of the ambient outdoor air PM. This study aimed to quantify the c-PAHs content and the genotoxicity of the organic extracts (EOMs) from various PM fractions, including particles of dae <0.17 um formed mostly by the ultrafine fraction. We focused on the high size (4 fractions) and time (26 consecutive days) variability.

Coarse (1 < dae < 10 um), upper accumulation (0.5 < dae < 1 um), lower accumulation (0.17 < dae < 0.5 um) aerosol particles were collected on polyurethane foam (PUF) and the smallest aerosol particles of dae < 0.17 um, in this study termed ultrafine, were trapped on PTFE-coated Glass Micro-Fiber Absolute filters (Pallflex 70 TX40). Aerosol was sampled by means of a HiVol cascade impactor (BGI 900, USA). Aerosol samples were collected from January 26th to February 21st 2012 in residential area of Ostrava-Radvanice. The concentrations of seven of the PAHs regarded as carcinogenic were analyzed in each EOM sample. Calf-thymus DNA (1 mg/ml) was incubated with various EOM samples (EOM corresponding to 3 m3 of the air/ml) for 24 h at 37 °C with and without metabolic activation by use of an S9 fraction from rat liver. DNA adducts were analyzed by 32P-postlabelling with use of the nuclease P1 treatment for adduct enrichment.

The results suggest that concentrations of c-PAHs in Ostrava-Radvanice during the sampling period were generally very high and represent significant health risk. In agreement with the mass distribution among aerosol size fractions, carcinogenic PAHs are bound mainly on the upper accumulation mode of PM (0.5-1 um). Ultrafine particles (< 0.17 um) bound only 11-15% of total PM10 c-PAHs, while accumulation mode bounds everytime more than 60% of c/PAHs. Carcinogenic PAHs are mostly responsible for PM genotoxicity, the contribution of directly acting (without S9) is 2-5-fold lower. Accumulation mode (0.17-1um) is predominantly responsible for of PM genotoxicity (48.3-72.0 %) while ultrafine particles (< 0.17 um) represent only 2.9-20.6 of total DNA adduct levels detected. Our study suggests that relative genotoxicity of the aerosol (DNA adducts/mg PM) is higher when PM levels are lower. This finding also indicates that monitoring of PM10, PM2.5 or PM1 is not sufficient to assess genotoxic/carcinogenic potential of the particulate air pollution. [Support: Czech Science Foundation: P503/12/G147]

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