DOCTORAL THESIS

Risk assessment of human exposure to persistent organic pollutants associated with air particulates and settled dust in two urban centers of Pearl River Delta

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Risk Assessment of Human Exposure to Persistent Organic Pollutants Associated with Air Particulates and Settled Dust in Two Urban Centers of Pearl River Delta

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Abstract

The major objectives of this research were to analyze concentrations of persistent organic pollutants (POPs) (polycyclic aromatic hydrocarbons (PAHs), polybrominated diphenyl ethers (PBDEs), organochlorine pesticides (OCPs), polychlorinated biphenyls (PCBs) and phthalates esters) in air particulate and settled dust samples in urban areas of Guangzhou (GZ) and Hong Kong (HK), in the Pearl River Delta; investigate the factors governing POPs distribution in air particles and dust; evaluate the exposure risks of POPs via non-dietary routes of inhalation and ingestion; and assess the biological effects of settled dust.

∑PAHs in the urban surface dust ranged from 0.84 to 12.3 μg/g with a mean of 4.80 μg/g and ∑PBDEs from 40.6 to 8.30×10^3 with a mean of 998 ng g^{-1} for PBDEs. Two major PAHs inputs to urban surface dust were identified as vehicle emissions (51.9%) and coal combustion (26.8%), while the major source for PBDEs was the commercial deca-BDE mixtures. The Incremental Lifetime Cancer Risk (ILCR) due to human exposure to PAHs associated with urban surface dust in GZ was 3.03×10^{-6} for children and 2.92×10^{-6} for adults. PBDE exposure via street dust (2.64×10^{-2} μg/kg-bw/day for children, 2.87×10^{-3} μg/kg-bw/day for adults) was lower than that of house dust and dietary intake.

Home $PM_{2.5}$ bound PAHs in GZ ranged from 10.0 to 61.9 ng/m^3, significantly higher than those in HK (0.72 to 4.47 ng/m^3). A similar PAH pattern was found in home $PM_{2.5}$, TSP and hair (dominated by Nap and Phe), but different from home dust. Pyrene (Pyr) and fluoranthene (Flu) in home dust significantly correlated with that in hair ($r = 0.69$; 0.55, $p < 0.05$), but no significant correlation was found between PAHs in hair and that in $PM_{2.5}$. The lung cancer risk based on home $PM_{2.5}$ bound PAHs exposure was high in homes of GZ (10^{-4}), exceeding the excess cancer risk (10^{-5}) associated with home dust intake. Non-dietary exposure (air and dust) explained for PAHs exposure 1-3 times higher than fish consumption and contributed to at least 12.2-33.5% and 0.7-14.5% of total $TEQs_{-PAHs}$ exposure for children and adults, respectively.

∑PBDEs (53 to 2025 mean 240 pg m^{-3}) and OCPs (28.7 to 273 mean 142 pg m^{-3}) in home $PM_{2.5}$ of GZ were significantly higher than those of HK (PBDEs: 0.25-160, mean 44 pg m^{-3}; OCPs: 19.9-125, mean 39 pg m^{-3}). The constant $C_{particle}/C_{dust}$ values suggest that sorption is the dominant mechanism through which PBDEs/OCPs are associated with settled dust and airborne particles. Total pesticides in hair samples ranged from 65.6 to 405 ng g^{-1}, with *p,p'*-DDE being the dominant congener (nd to 274 ng g^{-1}). No significant correlation was found for OCPs in air particulates/dust with hair samples. Dust ingestion (48.6-80.9%) and dietary intake (59.4-96.4%) were the two predominant PBDEs exposure routes respectively for toddlers and adults. Although non-dietary exposure resulted in a low exposure of OCPs under low dust intake (4.16 and 55 mg d^{-1} for adults and children), however explained for at least comparable daily intake with seafood intake, especially for children (100 and 200 mg d^{-1}).

The levels of ∑OCP indoor and outdoor dust of GZ (median 520, 171 μg kg^{-1}), were significantly higher than those of HK (115, 130). ∑PCBs, varied between 51.9-264.3 and 4.02-227.9 ng g^{-1} in indoor and outdoor dust of GZ, were significantly higher than those of HK (17.4-137.4; 7.8-113.7 ng g^{-1}). Different cytotoxic effects on human hepatacellular live carcinoma cell (HepG2) and human skin keratinocyte cell (KERTr) lines were observed based on the extracts of outdoor and indoor dust. POPs enrichment for both PCBs and OCPs decreased with the increased particle size. Dust ingestion contributed to significantly higher exposure dose for children than adults, due to the high dust intake rate. Dust intake predominated in non-dietary OCPs exposure and had a high contribution to total OCP exposure for adults (3.8%) and children (24.4%). The cancer risks related to OCPs associated with indoor dust were moderate (below 10^{-4}), while more alarmingly, 42% of residents in GZ registered risks $>10^{-5}$. However, when bioaccessible OCPs/PCBs were used in risk assessment, daily intake and health risk were found to be significantly lower than the solvent-extractable levels.

Phthalates varied from 4.95 to 2217 μg g^{-1} in indoor dust, significantly higher than those of outdoor dust (1.75 to 869 μg g^{-1}), collected from GZ and HK.

Di-2-ethylhexyl phthalate (DEHP) dominated in all dust samples. The cancer risks associated with DEHP via indoor dust were high (10^{-5}-10^{-4}), with 10% of houses of GZ and HK estimated with unacceptable cancer risks (>10^{-4}). ∑PAHs in indoor dust of GZ homes varied from 1.2-22.2 μg g^{-1} and 52% of homes were estimated with unacceptable cancer risks (>10^{-4}) via dust exposure, which should be of concern. Particle size was negatively correlated to both PAHs and phthalate accumulation in dust. Significantly higher bioaccessible PAHs and phthalates were found in 0-63 μm than 280-2000 μm fraction. Particle size was found as a missing factor which exerted a significant impact on PAHs related cancer risks. *In vitro* cytotoxicity of dust extracts on Human T cell lymphoblast leukemic cell line (CCRF-CEM) indicated by the Lethal Concentration 50 (LC_{50}) decreased with the increase of particle size of dust. Particle size would be a missing factor impacting greatly on risk assessment of dust associated with PAHs/Phthalates.

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