Risk Assessment of Human Exposure to Persistent Organic Pollutants via Indoor Dust in Hong Kong

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ABSTRACT

The major objectives of this research were to analyze concentrations of persistent organic pollutants in indoor dust in Hong Kong and identify typical pollutants contained in the dust which might lead to biological effects such as cytotoxicity, mutagenicity, genotoxicity and immunotoxicological effect.

In the present study, settled workplace dust from commercial offices, secondary schools, shopping malls, hospitals, electronic factories and manufacturing plants in Hong Kong and settled house dust from Hong Kong, Shenzhen and Guangzhou were collected. Results of chemical analyses showed that the total PBDEs in workplace dust ranged from 397 to 40,200 ng/g, with the dust samples from electronic factories having the highest levels. In general, settled dust sample from houses contained lower concentrations (ranging from 685 to 18,400 ng/g). The most abundant BDE congeners found were BDE-209 in both workplace dust and home dust, followed by BDE-99 and BDE-47. BDE-47, -99, -100 and -183 were detected in most of the hair samples collected from occupants of these homes with BDE-47 being the most dominant congener. The concentration of BDE-183 in house dust was significantly correlated with that in human hair (r=0.55, p<0.05, n=18). Risk assessment indicated that daily intake of PBDEs for children via non-dietary ingestion of dust was higher than that via food consumption.

Total PAHs concentrations of workplace dust ranged from 1170 to 25,500 ng/g, with the dust samples from manufacturing plant having the highest concentration. The total
PAHs concentrations of settled house dust from three major cities ranged from 1630 to 29,200 ng/g, which were significantly correlated with house age \((r=0.55, p<0.05)\). 3-[4, 5-dimethylthiazol-2-yl]-2, 5-diphenyltetrasodium bromide (MTT) assay was performed to evaluate the cytotoxicity of organic dust extracts using human hepatocellular liver carcinoma cell line (HepG2) and human skin keratinocyte cell line (KERTr). Significant negative correlations were observed between the total PAHs concentration in workplace dust and \(LC_{50}\) of both HepG2 \((r=-0.65, p<0.01)\) and KERTr \((r=-0.63, p<0.01)\) cell lines. Source analyses demonstrated that the PAHs in indoor dust were mainly derived from pyrogenic origins.

Workplace dust samples were selected for AhR-mediated EROD assay and chemical analyses of known AhR-agonists including polychlorinated dibenzo-\(p\)-dioxins and dibenzofurans (PCDD/Fs), several PAHs and dioxin-like PCBs. All dust organic extracts showed remarkable ability of induction of 7-ethoxyresorufin O-deethylase (EROD) activity, especially those from manufacturing plant. The \(\text{TEQ}_{\text{cal}}\) of samples derived from chemical analyses (PCDD/Fs+PAHs+PCBs) were significantly correlated with the bioassay derived TEQ of 2, 3, 7, 8-TCDD (TEQ\(_{\text{bio}}\)) of total dust extracts, ranging from 4.64 to 21.6 ng/g \((r=0.98, p<0.01)\). PAHs which was also significantly correlated with TEQ\(_{\text{bio}}\) \((r=0.98, p<0.01)\), contributed 87.5-98.9% to the TEQ\(_{\text{cal}}\). The results indicated that PAHs were the dominant AhR agonists and risk stressor in the dust. When the organic dust extracts were treated with sulfuric acid, TEQ\(_{\text{cal}}\) of samples derived from chemical analyses (PCDD/Fs+PCBs) were significantly \((r=0.83, p<0.05)\) correlated with the bioassay derived TEQ of 2, 3, 7, 8-TCDD (TEQ\(_{\text{bio}}\)) of samples.
varying from 320 to 730 pg/g. Health risk assessment indicated that indoor dust was an important medium for human exposure to dioxin-like compounds.

The mutagenicity and genotoxicity of indoor dust were assessed. Results indicated that indoor dust contained both frameshift and base pair substitution mutagens. TA100 (–S9) mutagenic potency was significantly correlated with genotoxicity expressed as SOSIP (–S9) of workplace dust ($r^2 = 0.37$, $p<0.01$). Linear regression analyses indicated that the PAHs likely accounted for about 45% of the TA98 with S9 mutagenic activity of workplace dust. To achieve a more accurate cancer risk assessment, the oral bioaccessibility of B(a)A, Chry, B(b+k)F, B(a)P, D(ah)A and I(cd)P in different dust (ranging from 1.3% to 17%) was taken into account. When moderate and high estimations of dust ingestion rates were considered, about 26% and 57% of house dust samples resulted in unacceptable cancer risk ($> 1 \times 10^{-6}$) for preschool children, respectively.

Human cytokine array was used to investigate the cytokine profile of U937 and KERTr after exposure to indoor dust or dust extracts. The release of MCP-1 was increased, while release of IL-8 and IL-1β on U937 was decreased after exposure to indoor dust. The releases of RANTES, IL-8 and VEGF from KERTr after exposure to dust extract were increased. The results of IL-8 ELISA assay were consistent with the cytokine array. Real-time RT-PCR was performed to analyze relative changes in gene expression. The MCP-1 mRNA levels were increased after U937 exposure to 18 indoor dust samples, whereas, IL-8 and IL-1β mRNA level showed both up-regulation and down-regulation. The dose-dependent increase and decrease response was observed on
MCP-1 and IL-8, respectively. Most indoor dust extracts increased RANTES, IL-8 and VEGF mRNA levels on KERT\textit{r}. The dose-dependent increase response was observed both on RANTES and IL-8. A significant correlation ($r = 0.48$, $p < 0.05$) was obtained between the total PAHs concentration in dust extracts and the induction of RANTES mRNA. It can be concluded that PAHs in indoor dust are the major causative agents of the observed biological effects including cytotoxicity, mutagenicity, genotoxicity and immunotoxicological effects.
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