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# Chemical and bioanalytical characterization of dioxins in indoor dust in Hong Kong

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#### ABSTRACT

In the present work, air-conditioner filter dust samples collected from commercial office, secondary school, shopping mall, electronic factory and manufacturing plant in Hong Kong were collected for 7-ethoxyresorufin O-deethylase (EROD) assay using a hepatoma cell line (H4IIE) and chemical analysis of dioxins including polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) and PCBs with dioxin-like structure. The result of EROD assay showed that bioassay derived TEQ of 2,3,7,8-TCDD (TEQ<sub>bio</sub>) of dust samples varied from 320 to 730 pg/g. Chemical analyses revealed that chemical derived TEQ of 2,3,7,8-TCDD (TEQ<sub>cal</sub>) of dust samples ranged from 134 to 531 pg/g. In addition, the TEQ<sub>cal</sub> of samples were significantly correlated with TEQ<sub>bio</sub> of samples (R=0.83, P<0.01). The average daily doses (ADDs) of dioxins via indoor dust with the estimated ADDs of dioxins via air and food were compared. The results showed that indoor dust is an important medium of exposure to dioxins.

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#### 1. Introduction

Most of studies related to air pollution focused on sources of pollutants and human exposures that occurred in outdoor environment. Nowadays, people are likely to spend more than 90% of their time indoor including house and office (Butte and Heinzow, 2002; Graham and McCurdy, 2004). Therefore, the potential health risks posed by chemical contaminants in the indoor environment are of significant concern and the potential hazards of indoor pollutants contained in the indoor dust are now being widely acknowledged.

As Hong Kong lies within the subtropical region, most of the indoor environments are air-conditioned in summer or even throughout the entire year. The dust on the air-conditioner filter reflects the indoor dust and indoor air quality because the air conditioner recirculates the air through the filter (Saeed et al., 1998). In addition, the tightly packed high-rise building environment together with the mountainous terrain in Hong Kong further limited its air circulation pattern and thus increased the probability of accumulation of pollutants in indoor environment.

Indoor dust sources include polycyclic aromatic hydrocarbons (PAHs) from heating, smoking, cooking and parquet floor glue; polychlorinated biphenyls (PCBs) from plastics and sealing

\*\* Corresponding author. Fax. +852 3411 7/45. \*\* Corresponding author. Fax: +852 3411 7348. E-mail addresses: zwcai@hkbu.edu.hk (Z.W. Cai), mhwong@hkbu.edu.hk (M.H. Wong). materials; organochlorines and organophosphates from pesticides; and polybrominated diphenyl ethers from flame retardants (Graham and McCurdy, 2004; Riechelmann et al., 2007). The principal use of PCBs is in "closed systems," such as electrical transformers, capacitors and other equipment where the PCBs are encased. They were also used in a range of "open system" products, including building materials, from which they may seep into their surrounding environment (Herrick et al., 2004). Pentachlorophenol (PCP) in the textile industry has been identified as a contaminant source of PCDD/Fs (McLachlan et al., 1996). PCDD/Fs can be transferred from the textiles including clothes, carpets and upholstery to human skin and released into the indoor environment (Horstmann and McLachlan, 1994). Indoor dust is contaminated with PCBs, dioxin-like PCBs (Vorhees et al., 1999) and polychlorinated dibenzo-p-dioxins and dibenzofurans (PCDD/Fs) (Wittsiepe et al., 1997; Saito et al., 2003).

Activation of aryl hydrocarbon receptor (AhR) and induction of the cytochrome P450 system, specifically CYP1A1 in mammals and fish, have been commonly used as a parameter for assessing the potency of AhR agonists (Jones and Anderson, 1999). The CYP1A1-induction potency of dioxin-like compounds relative to 2,3,7,8-TCDD has contributed to the toxicological information used in development of toxic equivalency factors (TEFs) for use in risk assessment (Delistraty, 1997). Accordingly, the total dioxin-like toxic potency of a compound mixture can be expressed as toxic equivalency (TEQ<sub>cal</sub>). The TEQ<sub>cal</sub> has been used as an indicator of health risk assessment of human exposure to these contaminants (Luo et al., 2009; Shen et al., 2009).

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Considering that chemical analyses provide little information on the possible adverse biological effects, the in vitro and in vivo bioassays based on activation of the AhR and induction of the CYP1A1 have been developed and established (Villeneuve et al., 2002). The rat hepatoma (H4IIE), human breast carcinoma (MCF-7), human hepatoma (HepG2) and desert topminnow (Poeciliopsis lucida) hepatoma (PLHC-1) cells have been employed to determine the relative potency of dioxin-like compounds from the induction of EROD activity in vitro system (Willett et al., 1997; Jones and Anderson, 1999; Zeiger et al., 2001; Villeneuve et al., 2002). Although the link between the CYP1A1 induction and specific toxicological effects is not fully known, the EROD activity of induction of CYP1A1 is commonly used as a biochemical indicator to characterize the existence of these contaminants in the complicated environmental samples (Luo et al., 2009; Shen et al., 2009). A combination of bioassays and chemical analyses has been used to identify the stressor in environmental samples including sediment and soils (Qiao et al., 2006; Shen et al., 2008). However, for indoor dust samples, most of the studies conducted so far focused on the concentration level survey of PCBs and PCDD/Fs in indoor dust (Wittsiepe et al., 1997; Vorhees et al., 1999). No investigation has been attempted to characterize the biological effects of causative agents present in the indoor dust, using a combination of chemical analyses and bioassays.

The three main pathways of human exposure to indoor contaminants include ingestion, inhalation and dermal contact. A number of reports indicated that daily intake of organic pollutants such as PAHs and PBDEs via indoor dust are typically high (Jones-Otazo et al., 2005; Maertens et al., 2008). Health risk of exposure to indoor dust by non-dietary ingestion has also been evaluated and the results show that indoor dust is an important route of human exposure to the PAHs and PBDEs (Jones-Otazo et al., 2005; Maertens et al., 2008). However, there seems to be a severe lack of information focusing on the human exposure to dioxin and dioxin-like compounds via indoor dust.

The major objectives of this study were (1) to measure the concentrations of PCDD/F and dioxin-like PCBs in different types of workplace dust in Hong Kong; (2) to characterize the dominant

AhR agonist in different types of workplace dust in Hong Kong based on the combination of EROD assay and chemical analyses (PCDD/Fs and dioxin-like PCBs); and (3) to evaluate the potential health risk on human.

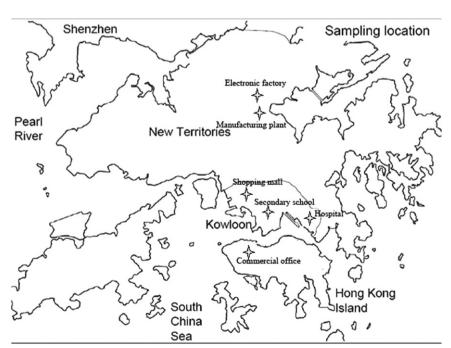
#### 2. Methods

#### 2.1. Sampling and preparation

The collection of air-conditioner filter sample was described in detail in our previous study (Kang et al., 2010). Briefly, the air-conditioner filter dust samples were obtained from six kinds of building: commercial offices (COMO, n=20), secondary schools (SCHL, n=4), shopping malls (SHOM, n=5), hospitals (HOSP, n=16), electronic factories (ELCF, n=6) and manufacturing plants (MANP, n=4) in Hong Kong. In the present study, one pooled sample from each kind of building was selected for analyses (Fig. 1). All collected samples were filtered through a stainless-steel sieve ( $<100 \, \mu m$ ) onto solvent-rinsed aluminum foil to remove large particles. The dust particle size used in different studies varied considerably. In the present study, we assessed the human exposure to dioxins via non-dietary intake of dust. Dust particles less than 100 µm can be effectively retained by skin or other surface (Lewis et al., 1999). Non-dietary ingestion of indoor dust occurred by occasionally ingestion of dust particles adsorbing on food or skin (Maertens et al., 2004). Therefore, the particle size less than 100 µm was selected for analyses. In total, 1.5 g sieved dust was then extracted with 100 ml acetone/ dichloromethane/n-hexane (1:1:1, v/v/v) in a Soxhlet apparatus. The extracts were concentrated on a rotary evaporator and were solvent-exchanged to 1.5 ml n-hexane. 1 ml of n-hexane extracts was stored at -20 °C for PCBs analyses. Another 0.5 ml n-hexane extract was treated with sulfuric acid, applied to sodium sulfate anhydrous/florisil column and eluted with n-hexane. The eluant was then concentrated to 0.5 ml, solvent-exchanged to 0.25 ml dimethyl sulfoxide (DMSO) and stored at -20 °C before EROD bioassay.

#### 2.2. Chemical analyses

For PCBs, 1 ml concentrated extracts was cleaned up using glass chromatography columns packed with 8 g florisil and a top layer of 4 g anhydrous sodium sulfate. The columns were subsequently eluted with 80 ml of a mixture of n-hexane (U.S.EPA., 1996a). The eluant was concentrated to 1 ml prior to analysis. Deuterated PAHs (acenaphthene-d<sub>10</sub>, phenanthrene-d<sub>10</sub>, chrysene-d<sub>12</sub> and perylene-d<sub>12</sub>) were used as internal standards. Internal standards were used to correct for the loss of analyte during sample storage and sample inlet or slight variation of instrument response from run to run. <sup>13</sup>C-PCBs were considered as ideal internal standards for PCBs analyses, but deuterated PAHs could be used as alternative



**Fig. 1.** The detailed information of sampling sites. Commercial office: located in the commercial building. Secondary school: located in residential area. Shopping mall: located in residential area. Hospital: located in residential area. Electronic factory: assembling electronics equipment such as computer; located in industrial area. Manufacturing plant: all producing furniture, toys and textiles; located in industrial area.

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