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Phosphorus flame retardants and Bisphenol A in indoor dust and $PM_{2.5}$ in kindergartens and primary schools in Hong Kong☆

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ABSTRACT

Organophosphate flame retardants (PFRs) and bisphenol A (BPA) were measured in indoor dust and $PM_{2.5}$ samples from nine kindergartens and two primary schools in Hong Kong. The average levels of $PM_{2.5}$ ranged from 4.0E+03 ng/m^3 to 1.5E+04 ng/m^3. Average levels of PFRs (from 1.5 ng/m^3 to 20 ng/m^3 in $PM_{2.5}$; from 8.0E−02 μg/g dw to 2.4 μg/g dw in dust) and BPA (from 6.4E−01 ng/m^3 to 1.0 ng/m^3 in $PM_{2.5}$; from 1.0E−02 μg/g dw to 2.0E−01 μg/g dw in dust) were detected in most of the sampling sites. Tri-(2-Chloroethyl) phosphate (TCEP), tris(1,3-Dichloro-2-propyl) phosphate (TDCP), tris-(chloroisopropyl) phosphate (TCPP), and triphenyl phosphate (TPHP) were present in low levels in $PM_{2.5}$ with medians of 16, 14, 8.7, and 3.2 ng/m^3, respectively. In dust, the medians were 1.5E−01, 5.5E−02, 5.9E−01, 8.6E−01, and 8.5E−02 μg/g dw for TCEP, TCPP, TDCPP, TPHP, and 2-ethylhexyl diphenyl phosphate, respectively. The medians of BPA were 6.4E−01 ng/m^3 and 7.4E−02 μg/g dw for $PM_{2.5}$ and dust, respectively. A positive correlation was found between indoor $PM_{2.5}$ and dust in the levels of TCEP ($r = 0.85$; $p = .05$). In the individual classroom in this survey, the predominant PFRs were similar, that is, TDCP and TCEP in indoor $PM_{2.5}$ while TPHP and TDCP in dust. TPHP and TCEP in primary schools were obviously lower than those in kindergartens. The estimated daily intakes via $PM_{2.5}$ and dust for all selected PFRs ranged from 1.3E−4 μg/kg/d to 2.0E−02 μg/kg/d, and the value of less than the detection limit at 3.5E−4 μg/kg/d was found for BPA. The EDI values of TPHP in dust non-dietary intake fraction were higher than those in the others. Calculated hazard indices (EDI/RfD) ranged from 4.8E−06 and 5.5E−03, showing that PFRs and BPA in $PM_{2.5}$ and dust presented no health risks to children.

1. Introduction

Many (semi) volatile organic compounds (Hwang et al., 2008; Takigami et al., 2009) have been detected in indoor environments and may be 2–5 times higher than those in outdoor environments (Kohler et al., 2005). Indoor pollution is closely related to human health as most city residents spend more than 70% (even up to nearly 90% in Hong Kong) of their time in indoor environments (WHO, 2006). Most gas pollutants, such as $PM_{2.5}$ (particulate matter less than 2.5 μm), can be easily absorbed by air particles and can thus deposit deep into the lungs (Ohura et al., 2005; WHO, 2010). These pollutants can also be deposited into dust by dry and wet deposition process, thereby posing risk to exposed people. At present, deterioration of indoor environments is a rising issue in developed and developing countries (WHO, 2006).

Among indoor pollutants, flame retardants (FRs) have attracted much attention. Several FRs, such as polybrominated diphenyl ethers (PBDEs), have potential toxicity, such as interfering with human endocrine and nervous systems (Herbstman et al., 2010; Kuriyama et al., 2007). As PBDEs are gradually prohibited because of high toxicity and bioaccumulation (SSCCH, 2011), an increasing number of other halogenated FRs, such as phosphorus flame retardants (PFRs), are being used as alternatives to PBDEs. PFRs are good substitute of PBDEs and have been widely used for building, computers, cars, furniture, carpet, upholstery, cable coatings, and plastic roofing materials in recent years because of their non-persistent and simple biodegradation characteristics (van der

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Veen and de Boer, 2012). PFRs are widely used and possess high production volume; thus, they can be detected in water, sediment, soil, air, biota, human breast milk; their detection levels in indoor environments are high, thereby presenting higher exposure to urban residents than PBDEs (Brandsma et al., 2015; Kim et al., 2013; Main et al., 2007; Staaf and Ostman, 2005; Sundkvist et al., 2010; van der Veen and de Boer, 2012). Current understanding on the adverse health effects of PFRs is insufficient; however, an increasing number of studies have revealed some toxic effects of PFRs, including nuclear receptor disrupting activities, neurotoxicity, disrupting normal growth, and liver toxicity (Liu et al., 2013a, 2012, 2013b; McGee et al., 2012; Meeker and Stapleton, 2010). A few chlorinated PFRs, such as tris (1,3-Dichloro-2-propyl) phosphate (TDCP) and tris (2-Chloroethyl) phosphate (TCEP), have been classified as potential human carcinogens (ECHA, 2010; Regnery and Püttmann, 2010). Thus, the effect of PFRs on human health has caused wide public concern over the recent years.

Children may have higher probability to suffer from the adverse effects of these pollution than adults; the reason is that children have higher inhalation rates per unit of body weight than adults, high chance to contact with dust, incomplete endocrine and nervous system, and critical period of growth and development (Schug et al., 2011). Although FRs can pose potential threats to child health, most works have focused on investigating brominated FRs—PBDEs (Kang et al., 2014). Few studies have focused on PFRs in $PM_{2.5}$ and dust in kindergartens and primary schools, especially the quantity uptake of children and the resulting health risk. A preliminary study by our group in five kindergartens from May 2014 to August 2014 showed that the levels of PBDEs in school indoor dust are 2—5 times higher than the levels in outdoor environments, thereby causing a high health risk of daily intake of PBDEs by kindergarten students via non-dietary ingestion of dust (Deng et al., 2016). Jones-Otazo et al. (2005) also reported that 82% of PBDE exposure is attributed to indoor dust exposure. However, small amount of information is known about the levels of PFRs in indoor environment in kindergartens and primary schools, especially on co-exposure with indoor $PM_{2.5}$ in Hong Kong of warm year-round climate. PFRs are partitioned into air readily in warm summer months from the materials containing PFRs and present high inhalation exposure (Xu et al., 2016).

Another highly exposed pollution in indoor environments is plasticizer. Among all plasticizers, bisphenol A (BPA) has been extensively used worldwide for several decades. BPAs are mainly used as plasticizers or stabilizers in polyester, polycarbonates, or epoxy resins and processed into children's toys, food-packaging materials, or thermal papers (Tsai, 2006). The health effect of BPA has drawn wide attention. Studies have shown that low-level exposure of BPA can lead to endocrine disrupting activities, reproductive harm, increased risk of cancer, and obesity (Rochester, 2013). Human can be exposed to BPA through extensive usage and multiple pathways, mostly by food or water ingestion, air inhalation, or dermal exposure to dust (Tsai, 2006).

PFRs and BPAs, which are potentially endocrine disruptors and neurotoxins, may increase the risk of childhood diseases during gestation, infancy, and childhood (Braun, 2017). This study aimed to quantify the levels of PFRs and BPAs in indoor $PM_{2.5}$ and dust and fully understand the species and amount of PFR uptake though indoor $PM_{2.5}$ and dust by using child population in the kindergartens and primary schools of Hong Kong. After the contents of PFRs were determined, daily exposure models would be applied to assess children's health risk. High-performance liquid chromatography electrospray ionization tandem mass spectrometry and gas chromatography spectrometry (GC-MS) technologies were used. The result would provide information on child exposure to PFRs and BPAs in Hong Kong and help regulate their usage.

2. Material and methods

2.1. Chemicals

Triphenyl phosphate (TPHP, 115-86-6, 99%), TCEP (115-96-8, 99%), 2-Ethylhexyl diphenyl phosphate (EDHPP, 1241-94-7, 97%), TDCP (13674-87-8, 98%), BPA (80-05-7, 99%), 13C12-BPA, and Tris (phenyl) phosphate-D15 (TPHP-D15) were purchased from Sigma—Aldrich (St. Louis, MO, USA). Other chemicals used in this research were of analytical purity and were purchased from Sinopharm Chemical Reagent Co., Ltd (Shanghai, China).

2.2. Sampling sites

The investigation was conducted from June 2015 to May 2016 in Hong Kong. Indoor air and dust samples were obtained from nine kindergartens (K1—K9) and two primary schools (P1 and P2). The geographically dispersed sampling sites included two classrooms from each of the nine kindergartens and two primary schools. All classrooms had PVC floorings (except K8 and P1) and were wall-painted. The detailed description and characteristics of the classrooms are presented in S1 of the Supporting Information. The building conditions, fittings, and decoration materials were similar in kindergartens, whereas few fittings and furnishing materials were found in primary schools.

2.3. Sampling and chemical analysis

Dust-Trak (TSI Model 8532) was used to measure the real-time $PM_{2.5}$ level and was placed 1.0 m above the floor level. MiniVol portable air sampler (Airmetrics, USA) was used to collect $PM_{2.5}$. School dust sampling was performed with air sampling by using a vacuum cleaner (Black & Decker Dust buster, USA) in the air conditioner (A/C) filters. All samples were sealed in polyethylene bags (Ziploc) while wearing a powder-free nitrile glove, transported to the laboratory, and immediately stored at −20 °C upon arrival at the laboratory.

For PFRs, dust and membrane samples were extracted with *n*-hexane and acetone (1:1, V/V, 20 mL). Florisil SPE cartridges were used for clean-up. The internal standard was TPhP-D15. PFR analysis was performed on Agilent GC-MSD system (CA, USA). A DB-5MS capillary column (30 m × 0.25 mm × 0.25 μm) was used. For BPA, dust and membrane samples were weighed and extracted with acetonitrile (6 mL). Strata NH2 cartridge and RapidTrace SPE workstation (Caliper Life Sciences, Inc., Hopkinton, MA, USA) were used for clean-up. Final eluate was evaporated and adjusted to 0.5 mL with methanol. The internal standard was 13C12-BPA. The limit of detections (LODs) of dust PFRs ranged from 1.0E−03 μg/g to 2.0E−03 μg/g, and the recoveries ranged from 82% to 88%. The LODs of $PM_{2.5}$ PFRs ranged from 5.0E−05 ng/m^3 to 2.0E−04 ng/m^3, and the recoveries ranged from 83% to 105%. The LODs of $PM_{2.5}$ PFRs in the present study were slightly lower than those reported by Liu et al. (2016). Meanwhile, the LODs of dust PFRs were at the same level as those reported by Mizouchi et al. (2015).

BPA concentrations were measured using HPLC/MS/MS (Agilent 1200 series and Agilent 6410 Triple Quadrupole, Agilent, Santa Clara, CA, USA) with an Agilent Eclipse plus C18 column (3.5 mm, 2.1 mm × 100 mm). The LOD of dust BPA was 1.0E−04 μg/g, and the recovery was 103%. The LOD of $PM_{2.5}$ BPA was 1.0E−01 ng/m^3, and the recovery was 91%. The LODs of BPA were close to those reported by Moreau-Guigon et al. (2016) and Wang et al. (2015).

The details of sampling and chemical analysis are described in S2 (The detailed procedures of sample collection, preparation, extraction, and instrument analysis) of the Supporting Information.

2.4. Data analysis

Statistical test was carried out using SPSS 22.0 software (SPSS, Inc.). Shapiro–Wilk test was used for checking the normality of data. For data less than the LOD, a value of LOD/2 was used. Kolmogorov–Smirnov test was used for testing the distribution of probability. One-way ANOVA test was used to compare different schools. Pearson regressions and Spearman correlations were used to investigate the relationships of the target chemicals in different environmental media or sampling areas.

2.5. Health risk

The estimated risk assessment was represented by the estimated daily intake (EDI). $PM_{2.5}$ inhalation, dust dermal contact, and dust non-dietary intake were calculated. The oral reference doses (RfDs) were used to evaluate the non-cancer risks. The RfDs of TCEP (22 μg/kg/d), TPHP (70 μg/kg/d), TCPP (80 μg/kg/d), and TDCP (15 μg/kg/d) were used (ATSDR, 2012). The calculated hazard indices (HIs) (EDI/RfD) for non-cancer risks were used. The details of the calculation, which are presented clearly in our previous publication (Deng et al., 2016), are described in S3 and S4 of the Supporting Information.

3. Result and discussion

3.1. PFRs in indoor $PM_{2.5}$ and dust

3.1.1. Levels and patterns

Table 1 and Fig. 1 show the concentrations of five target PFRs in the indoor $PM_{2.5}$ and dust samples in nine kindergartens and two primary schools. Among the five target PFRs, TCEP, TCPP, and TDCP were found in 100% of indoor $PM_{2.5}$ samples collected, whereas TPHP and EHDPP were detected in 82% and 18% of the $PM_{2.5}$ samples, respectively. The medians of PFRs ranged as TCEP (16 ng/m^3) > TDCP (14 ng/m^3) > TCPP (8.7 ng/m^3) > TPHP (3.2 ng/m^3) > EHDPP (data were extremely small to obtain the median).

Different from indoor $PM_{2.5}$ samples (TDCP and TCEP were the predominant target PFRs), indoor dust was found with TPHP and TDCP as the predominant target PFRs. TPHP was found in 100% of the indoor dust samples collected and was followed by TDCP (86%). The medians of TPHP and TDCP at 8.6E−01 and 5.9E−01 μg/g dw, respectively, were much higher than those of the others. TCEP, EHDPP, and TCPP were detected in 71% of the dust samples with medians of 1.5E−01, 8.5E−02, and 5.5E-02 μg/g dw, respectively. Among the target PFRs, the values of TPHP spanned a wide range of 3.9E−02 μg/g dw to 7.8 μg/g dw in dust. The levels of TPHP in primary schools, as well as the levels of TCEP, were lower than the levels in kindergartens of a large number of furnishing materials, toys, and electronic equipment.

Fig. 2 provides a visual comparison of the occurrence of PFRs in indoor $PM_{2.5}$ and dust. A positive correlation was found between indoor $PM_{2.5}$ and dust samples in the levels of TCEP ($r = 0.85$; $p = .05$). For the other analytes, no significant correlations were found.

Table 1
Concentrations of five target PFRs and BPA in indoor $PM_{2.5}$ (ng/m^3) and dust (μg/g dw) in nine kindergartens and two primary schools.

	Chemicals	Min	Max	Median	Average
$PM_{2.5}$	TCEP	4.7	49	16	20
	TCPP	3.5	19	8.7	9.1
	TDCP	1.5	38	14	15
	TPHP	1.2	8.4	3.2	3.5
	EDP	9.2E−01	2	n.d.	1.5
	BPA	6.4E−01	1	6.4E−01	8.2E−01
Dust	TCEP	2.6E−02	8.4E−01	1.5E−01	2.5E−01
	TCPP	2.1E−02	1.9E−01	5.5E−02	8.0E−02
	TDCP	5.3E−02	3	5.9E−01	1
	TPHP	3.9E−02	7.8E−01	8.6E−01	2.4
	EDP	3.2E−02	2.9E−01	8.5E−02	1.5E−01
	BPA	1.0E−02	2.0E−01	7.4E−02	7.9E−02

"n.d.": not available.

3.1.2. Comparison with other areas

Investigations on PFRs in bulk air (total levels of gas and particles) or gas in daycare centers or schools worldwide (Bergh et al., 2011; Carlsson et al., 1997; Fromme et al., 2014; Marklund et al., 2005; Tollbäck et al., 2006) have been conducted, and their results are listed in Table 2. The medians of target PFRs in bulk air or gas, which are listed in Table 2, were comparable to the current levels of PFRs in indoor $PM_{2.5}$ surveyed in Hong Kong. The predominant PFRs in indoor $PM_{2.5}$ in this study in Hong Kong were TCEP and TDCP, which differed from those in the above-mentioned areas. Compared with the levels in bulk air samples in daycare centers in Sweden (median concentration of 8.4 ng/m^3) (Bergh et al., 2011), the medians of TCPP in indoor $PM_{2.5}$ in this study in Hong Kong (8.7 ng/m^3) were of the same order of magnitude whereas the medians of TCEP were lower. On the contrary, we found lower medians for TCPP and TDCP and higher medians for TCEP than the levels in bulk air samples in Sweden by Marklund et al. (2005). Compared with the results found in indoor air from daycare centers and early child care education centers in North America (mean concentration of 5.9E−01 ng/m^3) (Bradman et al., 2012), the values of TDCP in indoor $PM_{2.5}$ in this study in Hong Kong were higher (mean concentration of 15 ng/m^3). The levels of PFRs in indoor $PM_{2.5}$ in this research were significantly lower than the levels detected in outdoor $PM_{2.5}$ in 10 urban sites in China in the period of 2013–2014 (Liu et al., 2016).

Considering the geographic location of Hong Kong, A/C are working for long periods every year, and the exposure of pollutes in A/C filter dust is vital in this region. Thus, A/C filter dusts were surveyed in this research. PFRs in A/C filter dust have been rarely explored. The levels of PFRs in the current research were similar to the levels found in A/C filter dust in houses in Jeddah, Kingdom of Saudi Arabia with ∑PFR medians (1.5E+01 μg/g dw) (Ali et al., 2016). The levels of TCEP, TPHP, and TDCP in A/C filter dust in the present research were similar to the levels found in indoor floor/settled dust of daycare centers or schools in Norway, Japan, and Spain (Cequier et al., 2014; Cristale et al., 2016; Mizouchi et al., 2015), whereas the levels of EHDPP and TCPP were 1–2 orders of magnitude lower. Compared with the levels of PFRs in 0.8 m above floor dust or non-floor dust in Sweden and Denmark (Bergh et al., 2011; Langer et al., 2016), the levels in the current research were lower. The highest concentration of TDCP in the present study was found at the same level of TDCP in Sweden and Denmark daycare centers mentioned above (Bergh et al., 2011; Langer et al., 2016) with medians of 9.1 and 7.1 μg/g dw. The highest concentration of TPHP was found at the same level of TPHP in UK and Sweden schools or daycare centers (Langer et al., 2016; Marklund et al., 2003) with medians of 4.1 and 4.5 μg/g dw. The predominant PFRs in A/C filter dust in this study in Hong Kong were TPHP and TDCP, which were accordance with those in Japan and Sweden (Cristale et al., 2016; Marklund et al., 2003). In general, the levels of PFRs in indoor A/C filter dust in Hong Kong were slightly lower than the levels in floor or non-floor dust in other areas in schools or daycare centers.

The levels and patterns were shown to vary across daycare centers because of variation in number and emission characteristics of buildings, fittings, and the furnishing materials containing PFRs (Ali et al., 2012). However, in the individual classroom in this survey in Hong Kong, the predominant PFRs were similar, that is, TDCP and TCEP in indoor $PM_{2.5}$ while TPHP and TDCP in indoor dust.

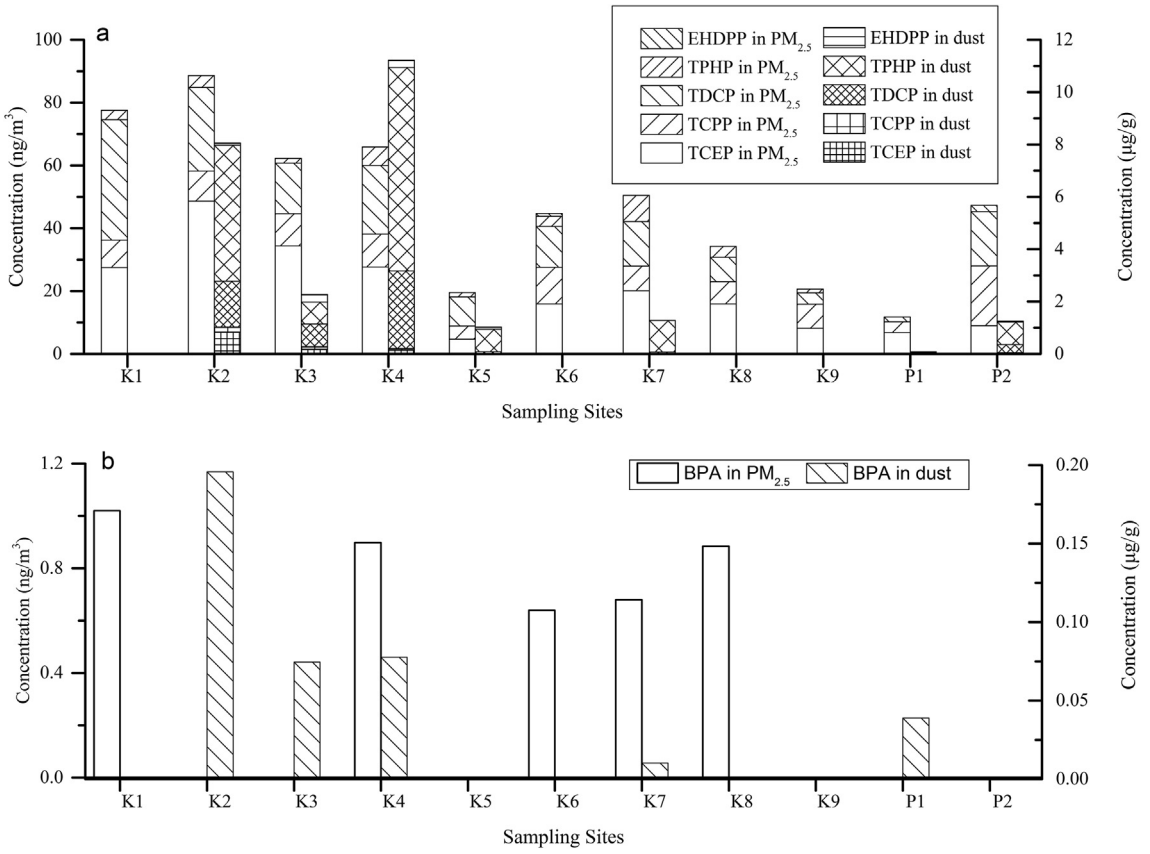

Fig. 1. Distribution of five PFRs and BPA in indoor $PM_{2.5}$ and dust.

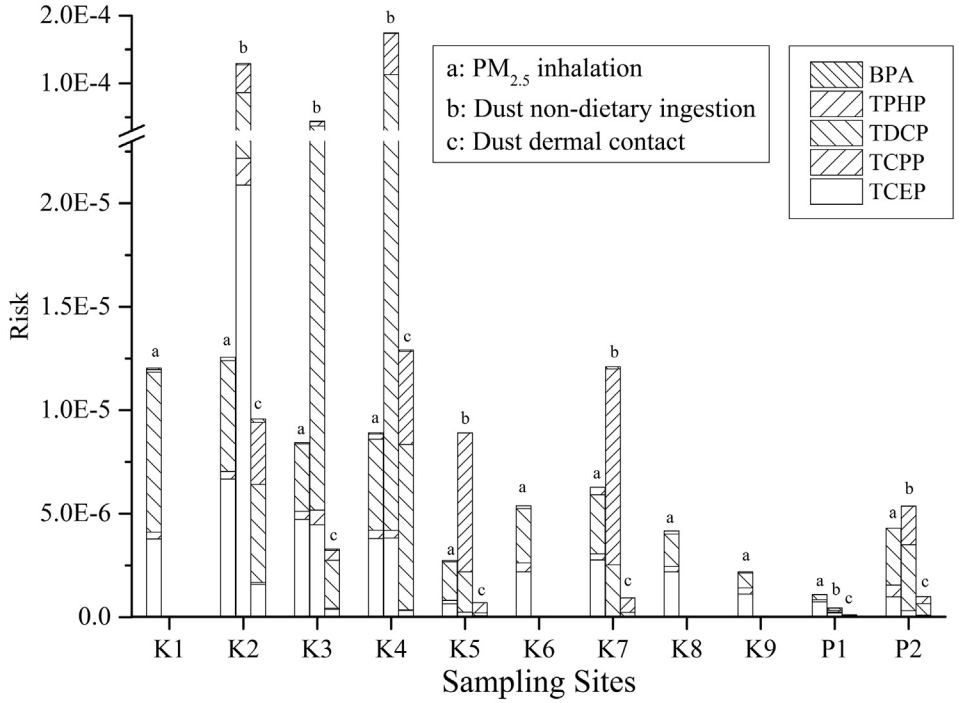

Fig. 2. Relative mass proportion of each congener to total risk.

Table 2
PFRs reported in indoor air (ng/m^3) or indoor dust (μg/g dw) in daycare centers and schools with the medians.

	TCEP	TCPP	TDCP	TPHP	EHDPP	Area	Location	References
Dust	30	3.1	9.1	1.9	8.0E−01	Sweden	Daycare centers	(Bergh et al., 2011)
	1.2	2	1.5	1.5	2.3	Norway	Classrooms	(Cequier et al., 2014)
	8.6E−01	16	5.1E−01	4.1	29	UK	Schools, daycare centers	(Brommer and Harrad, 2015)
	4.0E−01	2.7	—[a]	5.0E−01	1.1	Germany	Daycare centers	(Fromme et al., 2014)
	16	5.6	7.1	2	2.1	Denmark	Daycare centers	(Langer et al., 2016)
	5.0E−01	6.3E−01	7.4E−01	2.2	4.0E−01	Japan	Elementary school	(Mizouchi et al., 2015)
	1.4E−01	2.2	1.7E−01	6.0E−01	7.7E−01	Spain	schools	(Cristale et al., 2016)
	8.0E−01	2.5	1.8	4.5	–	Sweden	Daycare centers	(Marklund et al., 2003, 2005)
	1.5E−01	5.5E−02	5.9E−01	8.6E−01	8.5E−02	Hong Kong	Kindergartens and primary schools	This study
Air	25	8.4	n.d.[b]	n.d.	n.d.	Sweden	Daycare centers	(Bergh et al., 2011)
	<2.0	2.7	–	–	–	Germany	Dayare centers	(Fromme et al., 2014)
	1.4E+02	–	–	<0.5	–	Sweden	schools	(Carlsson et al., 1997)
	2.5	28	59	1.1	–	Sweden	Day-care centers	(Marklund et al., 2003, 2005)
	3.0	7.2E+02	–	3.0E−01	–	Sweden	schools	(Tollbäck et al., 2006)
	16	8.7	14	3.2	n.d.	Hong Kong	Kindergartens and primary schools	This study

[a] Not available/investigated.
[b] Not detected.

3.1.3. EDI estimation and risk assessment of PFR

The EDI values of PFRs via $PM_{2.5}$ and dust were calculated in each kindergarten and primary school. The concentrations of PFRs in $PM_{2.5}$ and dust were used to calculate the EDI values from three non-dietary sources, namely, dust non-dietary intake, dermal contact, and $PM_{2.5}$ inhalation.

The EDI values of all selected PFRs were found ranged from 1.5E−04 μg/kg/d to 2.1E−02 μg/kg/d. The EDI values of PFRs via $PM_{2.5}$ inhalation were approximately one-order lower than those via dust. As shown in S5 of the Supporting Information, the EDI values were significantly lower than RfD. HIs (EDI/RfD) for non-cancer risks of PFRs ranged from 1.6E−06 to 1.8E−03 (HQ < 1) in five kindergartens and two primary schools ($PM_{2.5}$ and dust were both surveyed) and for four other kindergartens (only $PM_{2.5}$ was surveyed). In general, HIs for non-cancer risks of PFRs via $PM_{2.5}$ and dust were all below 1.2E−05 (S6 of the Supporting Information).

Among all the PFRs in this study, dust non-dietary intake fraction was higher than the other routes. Relative mass proportion of individual PFR to the total mass is shown in Fig. 2. Human intake of PFRs from dust non-dietary ingestion accounted for 37.6%–90.3% of the total intake estimation in this research (Fig. 2), and TPHP in dust non-dietary intake fraction was the highest in the EDI via $PM_{2.5}$ and dust in this research in Hong Kong. TPHP, which was the most abundant PFR in this research, is the most frequently detected PFR in environmental and biota worldwide and can be used as FR and plasticizer in PVC, cellulosic polymers, or synthetic rubber (van der Veen and de Boer, 2012). According to a large-scale study in the USA by Stapleton et al., TPHP is the main PFR detected in house dusts (up to 1.8E+03 μg/g) and is higher than other target FRs (Stapleton et al., 2009).

In our previous work, $\sum_{13}$PBDE (from 1.0E−01 ng/m^3 to 6.4E−01 ng/m^3 in $PM_{2.5}$; from 2.9E−04 μg/g dw to 1.9E−01 μg/g dw in dust) were found in kindergartens in Hong Kong, and the EDI values of PBDEs via $PM_{2.5}$ inhalation by kindergarten children were higher than those via dust (Deng et al., 2016). In the present study, the levels of PFRs were higher than those of PBDEs in $PM_{2.5}$ and dust, and the EDI values of PFRs via $PM_{2.5}$ inhalation were approximately one magnitude lower than the values through dust.

3.2. BPA in indoor $PM_{2.5}$ and dust

The other target analyte BPA was observed in 45% of the air $PM_{2.5}$ samples and in 71% of the dust samples, with medians of 6.4E+02 ng/m^3 and 7.5E−02 μg/g dw, respectively (Table 1 and Fig. 1). The levels of BPA in indoor dust ranged from 1.0E−02 μg/g dw to 2.0E−01 μg/g dw, and they ranged from 6.4E−01 ng/m^3 to 1.0 ng/m^3 in the air $PM_{2.5}$ samples. The levels of BPA in air or dust samples in kindergartens or schools have also been rarely examined. The levels of BPA in this research in Hong Kong were in agreement with the levels detected in dust samples (up to 1.5E−01 μg/g dw in house dust from carpets) and air samples (up to 9.0 ng/m^3) from daycare centers in the USA (Wilson et al., 2007).

3.2.1. EDI estimation and risk assessment of BPA

The EDI values via $PM_{2.5}$ and dust were less than 3.5E−04 μg/kg/d for BPA. For PFRs, the EDI values of dust non-dietary intake were higher than those of the other routes. The HIs for non-cancer risk from BPA were all below 1.76E−03, which was significantly lower than 1 in the kindergartens and primary schools. Therefore, PFRs in $PM_{2.5}$ and dust did not present a potential health risk.

3.3. Levels of indoor $PM_{2.5}$

The concentrations of indoor air $PM_{2.5}$ measured in nine kindergartens and two primary schools are shown in S7 of the Supporting Information. The average indoor $PM_{2.5}$ concentrations in the nine kindergartens ranged from 4.0E+03 ng/m^3 to 1.5E+04 ng/m^3, which were lower than our previous results in five kindergartens in Hong Kong (from 1.3E+04 ng/m^3 to 4.2E+04 ng/m^3) (Deng et al., 2016). The average indoor $PM_{2.5}$ concentrations were 7.0E+03 and 9.0E+03 ng/m^3 in two primary schools, respectively, which were similar to those in kindergartens. In general, the average $PM_{2.5}$ levels of the nine kindergartens and two primary schools were lower than the WHO guidelines (an average concentration of 2.5E+04 ng/m^3 for 24 h).

PM can be generated from outdoor and indoor sources, and the major sources of PM in urban areas are automobile exhaust, road traffic dust, industrial production processes, and individual activities, such as cooking, smoking, and cleaning (Chan, 2002). In the kindergartens and primary schools, indoor cleaning activities or children activities can increase the PM concentration. Apart from leading to acute or chronic diseases, high levels of $PM_{2.5}$ or PM_{10} of large surface area to bear organic or inorganic air pollution can also transport pollution to humans (Daisey et al., 2003; Mendell and Heath, 2005; Ohura et al., 2005). Compared with coarse particles, fine particles penetrate deeper in the lungs.

In summary, no health risk was presented to the children according to the data of this survey. The long-term and low-dose toxicity data of PFRs and BPA to humans, particularly to young children, are still limited. The health risk of co-exposure of $PM_{2.5}$

and pollution are still unclear. Given that children spend much time indoor, the potential health risks of indoor air PFRs need urgent attention.

4. Conclusion

This research analyzed the concentrations of emerging PFRs and BPA via indoor $PM_{2.5}$ and dust in kindergartens and primary schools. The exposure levels of young children during school hours via indoor $PM_{2.5}$ and dust were also estimated. PFRs and BPA were detected in most of the sampling sites. No health risk was presented to the children according to the data of this survey. This study could provide policy makers with a valuable insight into indoor environments in this global city and help them formulate appropriate preventive strategies, such as searching the main pollutive source.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at https://doi.org/10.1016/j.envpol.2017.12.093.

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