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Levels and profile of several classes of organic contaminants in matched indoor dust and serum samples from occupational settings of Pakistan

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ABSTRACT

Dust ingestion is an important route of human exposure to organic contaminants, especially for flame retardants (FRs) in occupational settings. Several classes of organic contaminants were analyzed in matched dust and serum samples from academics and workers in electronics and clothing stores of Faisalabad, Pakistan. The concentrations of contaminants varied in dust as follow: organophosphate FRs (∑PFRs) > novel brominated FRs (∑NBFRs) > polybrominated diphenyl ethers (∑PBDEs) > organochlorine pesticides (∑OCPs) > polychlorinated biphenyls (∑PCBs), while, in serum, concentration varied: ∑OCPs > bromophenols (∑BPs) > ∑PCBs > ∑HO-PCBs ≈ ∑PBDEs. Two NBFRs, namely 1,2-bis(2,4,6-tribromophenoxy)-ethane (BTBPE) and bis(2-ethylhexyl) tetrabromophthalate (TBPH), were detected in <10% of the serum samples. *p,p′*-DDE was the major contaminant in serum contributing to ~75% of the total contaminant burden. Levels of Penta-BDE congeners in serum and dust were significantly correlated ($r = 0.64$, $p < 0.01$) for the academics, suggesting dust ingestion as an important determinant for their serum levels.

1. Introduction

Organic contaminants including polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs) and organochlorinated pesticides (OCPs), are widely occurring in the environment and animal tissues (Dirtu and Covaci, 2010; Law et al., 2008; Srogi 2008). Recent reports on the occurrence of novel brominated flame retardants (NBFRs) and organophosphate flame retardants (PFRs) in different environmental compartments, including the indoor environment (Covaci et al., 2011; Stapleton et al., 2011; Van den Eede et al., 2011) provided critical information about their current status and ecological risks. Many of these contaminants are lipophilic, highly stable in the environment and can induce adverse health effects to both wildlife and humans due to mutagenic, teratogenic and carcinogenic properties (ATSDR, 2000, 2002; 2004). Considered as an international issue, the use of many of these chemicals has been restricted and several are listed under Stockholm Convention, e.g. PCBs, most OCPs and Penta- and Octa-BDEs (Stockholm Convention on POPs, 2009).

While the importance of various routes of human exposure is still unclear, studies have shown that humans are exposed to these chemicals *via* food, air and dust intake (Dirtu and Covaci, 2010; Mercier et al., 2011). Indoor dust is often used as a marker of indoor exposure due to its importance as a sink and repository for semi volatile organic compounds and particle-bound matter (Butte and Heinzow, 2002). Recently, the role of dust as potential human non-dietary exposure source to organic contaminants has been suggested as an attractive area of research (Ali et al., 2013a; Dirtu et al., 2012; Mercier et al., 2011). Human exposure to organic contaminants *via* indoor dust ingestion and food consumption has been the focus of several studies from North America (Imm et al., 2009), Europe (Dirtu and Covaci, 2010) and Japan (Inoue et al., 2006).

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To date, there has been no to limited information on organic contaminants in Pakistan or an evaluation of correlations between dust intakes and concentrations in humans (Ali et al., 2013a,b; Eqani et al., 2012). Serum can be good biomarkers to assess human exposure, while indoor dust is a good indicator to indoor organic pollution (Ali et al., 2013a,b). Therefore, serum and indoor dust were considered to assess concentrations of several classes of legacy and emerging contaminants (Table 1) in different occupational settings of Faisalabad, Pakistan. The city of Faisalabad is located in the province of Punjab, Pakistan and in past decades was characterized by intensive industrial activities, such as textile industry, and urban development. Occupational settings have always been a source of chemical exposure to workers, but due to the dearth of information and lack of routine surveillance systems, large numbers of workers are routinely exposed to various indoor chemicals especially in developing countries like Pakistan (Kamal et al., 2012). Many of the organic contaminants are associated with human health effects, but few studies are available on their direct link with the health condition. For instance, hepatitis C has a high prevalence in Faisalabad, Pakistan (Ahmad et al., 2007).

Therefore, the objectives of the study were: (i) to study the occurrence and profiles of selected organic contaminants in matched indoor dust and serum from different occupational settings, (ii) to study the dust ingestion as an exposure pathway for these contaminants, and (iii) to study the effects of organic pollutant on the marker for liver health. This investigation fills an existing gap related to the lack of routine surveillance systems in developing countries. It also sets fundaments for future biomonitoring studies on human exposure in occupational settings.

2. Materials and methods

2.1. Reagents and materials

Individual standards of OCPs, PCBs, PBDEs, HO-PCBs, and HO-PBDEs were obtained from Dr. Ehrenstorfer Laboratories (Augsburg, Germany). Standards of NBFRs, BPs were purchased from Wellington Laboratories (Canada), while PFRs standards, except for TCPP (Pfaltz & Bauer, Waterbury, CT, USA), were supplied by Chiron AS (Trondheim, Norway). Several internal standards (ISs) BDE 77 and BDE 128 (AccuStandard Inc, USA), $^{13}C_{12}$-BDE 209, ε-HCH, 2,2′,3,4,5,6′-hexachlorobiphenyl (CB 143), 4′,hydroxylated-2,3′,3,4,4′,6′-hexachlorobiphenyl (4′HO-CB 159) (Wellington Laboratories), tri-amyl-phosphate (TAP) (TCI Europe, Zwijndrecht, Belgium) and triphenyl-phosphate-d15 (TPP-d15) (Sigma, Aldrich) were used. All solvents and chemicals used during the analysis were of pesticide-grade purchased from Acros Organics (Geel, Belgium) and Merck (Darmstadt, Germany). Anhydrous sodium sulfate (Na_2SO_4) and silica gel (Merck) were washed with *n*-hexane and used after heating overnight at 160 °C. A positive pressure manifold (3M Company, St. Paul, MN, USA), Oasis® HLB extraction cartridges (6 mL/500 mg, Waters, Milford, MA, USA) and silica Bond Elut (3 mL/500 mg, Agilent Technologies, Palo alto, CA, USA) were used for solid-phase extraction (SPE). Empty polypropylene columns for clean-up (3 mL) were purchased from Supelco (Bellefonte, PA, USA).

Table 1
Organic contaminants studied in the indoor dust and human serum during present study.

NBFRs	1,2-Bis(2,4,6-tribromophenoxy)ethane (BTBPE), Hexabromobenzene (HBB), Decabromodiphenylethane (DBDPE), Hexachlorocyclopentadienyl-dibromocyclooctane (HCDBCO), 2-Ethylhexyl-2,3,4,5-tetrabromobenzoate (TBB), Bis(2-ethylhexyl)-3,4,5,6-tetrabromophthalate (TBPH), Bromophenols (mono–penta)
OCPs	Hexachlorobenzene (HCB), cis-Chlordane (CC), trans-Chlordane (TC), Oxychlordane (OxC), trans-Nonachlor (TN), Hexachlorohexane (α-HCH, β-HCH, γ-HCH), Dichlorodiphenyldichloroethane (*p,p′*-DDD), Dichlorodiphenyl-dichloroethylene (*p,p′*-DDE), Dichlorodiphenyltrichloroethane (*p,p′*-DDT).
PCBs	99, 101, 105, 118, 138, 153, 156, 170, 180, 183, 187, 194, 199, 206, 209
HO-PCBs	4′HO-CB 79, 4′HO-CB 120, 3HO-CB 118, 4′HO-CB 107, 4′HO-CB 146, 4′HO-CB 127, 3HO-CB 138, 3′HO-CB 153, 4HO-CB 130, 4HO-CB 163, 4′HO-CB 162, 4HO-CB 177, 4HO-CB 187, 4HO-CB 193, 4′HO-CB 172, 3′HO-CB 180, 4′diHO-CB 202, 4′HO-CB 208.
PBDEs	28, 47, 99, 100, 153, 154, 183, 196, 197, 203, 209
HO-PBDEs	3HO-BDE28, 5HO-BDE47, 6HO-BDE99
PFRs	Tri-ethyl-phosphate (TEP), Tris–(2-chloroethyl)-phosphate (TCEP), Tris–(1,3-dichloro-isopropyl)-phosphate (TDCPP), Tri–n-propyl-phosphate (T*n*PP), Tri-iso-butyl-phosphate (T*i*BP), Tris–(1-chloro-2-propyl)-phosphate (TCPP), Tri-(2-butoxyethyl)-phosphate, Tri-cresyl-phosphate (TCP), Tri-phenyl-phosphate (TPhP), Tri-n-butyl-phosphate (T*n*BP).

2.2. Study participants, serum and indoor dust sampling

A total of 61 paired samples of blood and dust were collected from Faisalabad, Pakistan in December 2011. Of these, 30 paired samples were collected from people (age ranged 17–55 years, mean 30 years) working in old and new electronic (computer, home appliances and mobile) stores. The rest of the samples included people (age ranged 17–55 years, mean 29 years) working in old and new clothing stores ($N = 15$), and academics (age ranged 18–32 years, mean 25 years) ($N = 16$, young lecturers and post graduate students) from the University of Agriculture, Faisalabad, Pakistan. All individuals participating in this study were volunteers who signed an informed consent. For blood samples, baseline data, including age, gender and occupational history were collected from each participant. A volume of 7–8 mL blood was collected by venipuncture at a local clinic following overnight fasting. Blood was collected into sterile glass collection tubes without anticoagulant and after 1 h at room temperature; the serum was separated by centrifugation at 4000 g for 10 min, transferred to new tubes and kept at −20 °C until analysis. The serum lipid content was determined from enzymatic measurements of cholesterol and triglycerides (Phillips et al., 1989), which were done at the same clinic. Serum glutamic-pyruvic transaminase (SGPT) and serum glutamic oxaloacetic transaminase (SGOT), clinically a marker for liver health, were also measured to study the pollutants burden in people with normal and elevated enzymes. The study was approved by the Quiad-i-Azam University-Ethical Review Committee.

Dust samples were collected by brushing the floor surface (4–8 m^2) and, to avoid cross contamination, the brushes from the respective store/office/hostel room were used to collect samples. After brushing, dust was swept onto the aluminum foil, rapped and sealed in polyethylene zip bags. General information about the indoor inventories (details of electronics, foam chairs and other possible emission articles for chemicals) and date of last cleaning were collected. Before transfer to the laboratory, samples were kept in the dark to avoid photo-degradation. Each dust sample was sieved through a 500 μm mesh sieve pre-cleaned with acetone and stored in polypropylene recipients in a dark place. To prevent cross-contamination, sieves were scrubbed with acetone between homogenization of the samples.

2.3. Sample preparation and instrumentation

The procedure for extraction and clean-up for serum was used with minor modifications of previously described method (Covaci and Voorspoels, 2005; Dirtu et al., 2010). Details about the serum sample preparation are given in Ali et al. (2013b) and briefly in supplementary information (SI). The extraction and purification method of dust is described in detail in Van den Eede et al. (2012) and briefly in SI.

Details about the instrumental analysis of dust samples are described elsewhere (Ali et al., 2012; Van den Eede et al., 2012). Briefly, the analysis of PCBs, OCPs, NBFRs and PBDEs was performed by 6890 Agilent (Palo Alto, CA, USA) gas chromatography (GC) coupled to a 5973 mass spectrometer (MS) operated in electron capture negative ionization (ECNI). A DB–5 column (15 m × 0.25 mm × 0.10 μm) was used for separation and the MS was deployed in selected ion monitoring (SIM) mode. The ion source, quadrupole and interface temperatures were set at 200, 150 and 300 °C, respectively. The analysis of PFRs was performed by GC–MS in electron ionization (EI) mode. A HT–8 column (25 m × 0.22 mm × 0.25 μm) was used and the MS was operated in SIM mode with two characteristic ions acquired for each compound. The ion source, quadrupole and interface temperatures were set at 230, 150 and 300 °C, respectively.

For 1st fraction of serum samples, the analysis was performed on GC-ECNI/MS using a DB-5 column (15 m × 0.25 mm × 0.10 μm) was used. The ion source, quadrupole and interface temperatures were set at 170, 150 and 300 °C, respectively. For 2nd fraction of the serum samples, a DB-5ms column (30 m × 0.25 mm × 0.25 μm) was used. The ion source, quadrupole and interface temperatures were set at 200, 150 and 300 °C, respectively. More details about method are available in Ali et al. (2013b), the quantification and identification ions for analytes and their corresponding ISs are given in SI (Table S1).

2.4. Quantification and quality assurance

Multi-level calibration curves were created for the quantification and good linearity ($r^2 > 0.995$) was achieved for the whole concentration range found in the samples. The analytes identification was based on relative retention times and ion chromatograms to the standards. A deviation of the ion intensity ratios within 20% of the mean values of the calibration standards was considered acceptable. Method

limits of quantification (LOQs) were calculated as 3 × standard deviation of the analytes value in procedural blanks. For analytes that were not detected in procedural blanks, LOQs were calculated for a signal-to-noise ratio equal to 5 based on the signal obtained in the standard. To ensure method accuracy, matrices (a pooled serum and pre-washed Na_2SO_4 for dust samples) spiked with known concentrations of chemicals were analyzed in parallel with our samples. Indoor dust standard reference material (SRM 2585) from National Institute of Standards & Technology was analyzed to evaluate method accuracy. To avoid possible photodegradation of analytes, extraction and clean up steps were performed using amber glass under fume hood without light. The values of NBFRs, PBDEs, and PFRs measured in SRMs were in agreement (RSD <15%) with published values (Ali et al., 2011; Van den Eede et al., 2011; Wise et al., 2006). Recoveries ranged between 65% and 105% (RSD <15%) for OCPs, PCBs, HO-PCBs and PBDEs in serum (Covaci and Voorspoels, 2005; Dirtu et al., 2010), while for NBFRs, except TBPH, recoveries ranged between 84 and 117%, RSD <15% (Table S2).

2.5. Statistical analysis

Descriptive statistics was done using Microsoft Excel 2003. Values below LOQ were replaced by *df * LOQ*, where *df* is the detection frequency, e.g. number of samples >LOQ (James et al., 2002). Mann–Whitney test was applied to study differences in the levels of contaminants among different occupational settings, while correlations between concentrations of serum and dust was tested using the Spearman rank-order correlation. Using Minitab 15, Pearson correlation was performed to study the correlation among different chemicals and their correlation with age. Unpaired *t*-test was applied using Graph Pad to test differences in the serum concentrations of people with elevated and normal liver enzymes. The level of significance was set at $p < 0.05$, unless specified otherwise.

3. Results and discussion

3.1. Levels and profile of selected pollutants in serum and dust samples

In this study, levels and profiles of different classes of organic pollutants are reported from 61 paired serum and dust samples collected from different occupational settings of Faisalabad, Pakistan. The occurrence patterns of contaminants was markedly different in the two matrices i.e. serum and dust. PFRs were not assessed in the associated serum samples, because the analytical method used was not suitable for their analysis.

3.1.1. Neutral compounds in serum samples

Regarding the quantification of analytes in the serum samples, the levels of ∑OCPs were noticeably higher than the rest, while ∑PCBs, ∑NBFRs and ∑PBDEs had comparably smaller contribution (Fig. 1, Table 2). In serum samples of all groups, *p,p′*-DDE was the major OCP, followed by PCP > ∑HCHs > HCB. The order of occurrence for OCPs is similar to those reported recently in the human serum from the Punjab, Pakistan (Ali et al., 2013b), which might suggest similar past OCP usage pattern across the region. Moreover, the dominance of OCPs among all contaminants might demonstrate the still on-going current usage and unintentional leakage from pesticide obsolete storage places (Eqani et al., 2013). The higher OCP levels in the serum samples can also be attributed to the high lipophilicity (K_{OW}) of these pollutants, which ultimately lead to accumulation in humans *via* food chain transfer and dust ingestion for longer duration. The results of recent studies also confirm the high OCP residual levels observed in different environmental samples, including water, soil and biota, collected from same region (Eqani et al., 2012, 2013).

Lower levels BDE 47, −99, and −153 were detected in >90% of the serum samples and two important NBFRs, namely BTBPE and TBPH were detected, albeit in <10% of the serum samples. The low levels of these compounds suggest lower usage of NBFRs in the region. The levels of different classes of pollutants, as indicated in Table 3, were lower or similar to those reported in the literature. PCBs and OCPs, particularly DDTs, were lower than those reported in human serum from Bangladesh (Mamun et al., 2007). Background information revealed that all Bangladeshi donors eat fresh fish everyday and dry fish several times in a week which may contain high levels of DDT and its metabolites (Mamun et al., 2007). Eating contaminated food, e.g., fatty fish, accidental exposure, and living closer proximities to a contamination source, have been established as major exposure pathways for OCPs and PCBs (Wang et al., 2013a). In the present study, the donors had no accidental exposure, nor were fish a part of their regular diet. This could be a possible explanation for the lower levels of these chemicals in these serum samples. Indoor dust ingestion is considered as one of the major exposure route for PBDEs (Dirtu et al., 2012). The median levels of PBDEs in indoor dust from Pakistan (28 ng/g) were lower than those reported from New Zealand (495 ng/g), UK (2900 ng/g), US (3500 ng/g) and China (mean 2660 ng/g) (Ali et al., 2012; Harrad et al., 2008a; Huang et al., 2010). This is a possible explanation for the low levels of ∑PBDEs in human serum from Pakistan compared to other countries (Table 2).

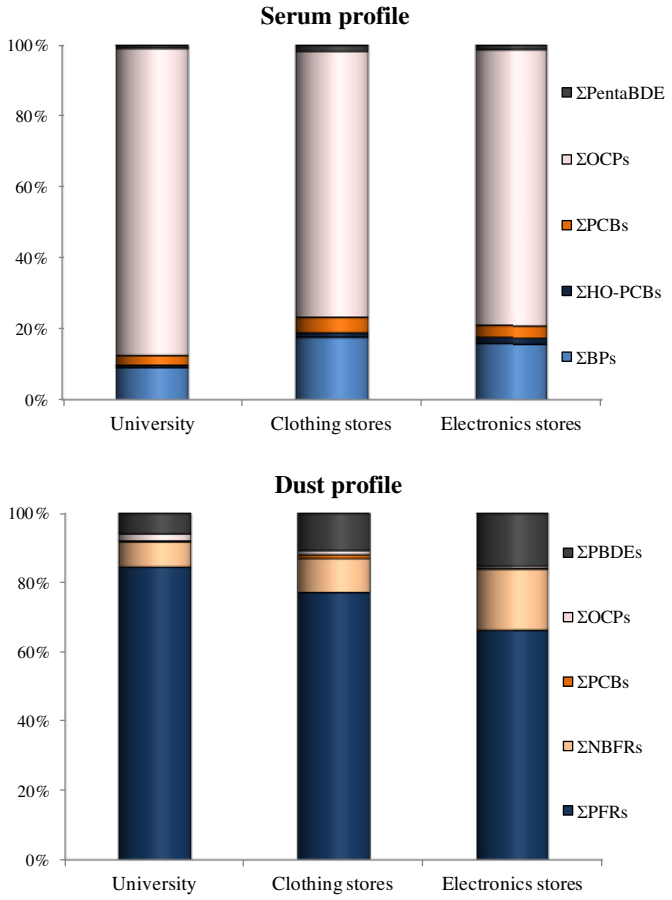

Fig. 1. Profiles of organic contaminants in serum and dust sample. *Y*-axis represents the percentage contribution of each class of contaminants.

3.1.2. Phenolic compounds in serum samples

Hydroxylated metabolites of PBDEs and PCBs were detected at (much) lower levels and detection frequencies than their respective parent compounds (Table 2). Among selected HO-PBDE only 6′HO-BDE 99 was detected and quantified in ~20% serum samples. 4HO-CB 79 (median 0.8 ng/g lw), 4HO-CB 107 (median 0.6 ng/g lw), and 4HO-CB 187 (median 0.2 ng/g lw) were the major HO-PCBs detected in ~70% of the samples. Pearson correlation was applied on log transformed data of ∑PCBs and ∑HO-PCBs. Significant correlation ($r = 0.42$, $p < 0.01$) (Table 5) was obtained between them, indicating PCBs metabolism as a major source for HO-PCBs along with other sources, such as food etc.

Table 2
Concentration (ng/g lw) of studied organic contaminants in serum samples from different occupational settings.

	University ($N = 16$)		Clothing stores ($N = 15$)		Electronics stores ($N = 30$)	
Analytes	Mean ± SD	Median (range)	Mean ± SD	Median (range)	Mean ± SD	Median (range)
CB 118	2 ± 1.6	1.8 (0.5–7)	2 ± 0.8	1.7 (0.9–3.6)	1.5 ± 1.5	1.2 (0–8)
CB 153	2 ± 0.8	2 (1–3.5)	2.5 ± 1	2.2 (1.5–4.5)	2.8 ± 2.9	2 (0–13)
CB 138	1.5 ± 0.8	1.3 (0.0–3.5)	2 ± 1.1	1.5 (1–4.5)	2.0 ± 2.3	1 (0–11)
CB 180	1 ± 0.5	1.2 (0.3–1.7)	1.1 ± 0.6	0.8 (0.6–2.7)	1.8 ± 2.2	1.1 (0–12)
CB 170	0.5 ± 0.2	0.5 (0.1–1)	0.5 ± 0.2	0.5 (0.3–1)	0.8 ± 0.9	0.5 (0–5)
∑PCBs	9 ± 5	7.5 (3–23)	9.5 ± 3.5	8.0 (5–18)	11 ± 11	7 (0–50)
∑HO–PCBs	5.5 ± 7.5	2.2 (0.6–27)	4 ± 5	2.0 (0.8–17)	5.5 ± 4.5	3.5 (1–18)
PCP	30 ± 18	24 (10–82)	57 ± 31	53 (17–140)	60 ± 70	40 (<10–275)
∑HCH	6.5 ± 5.8	4 (1–22)	13 ± 9	12 (3–40)	7.8 ± 6	5.2 (1–24)
HCB	4 ± 2.5	3 (2–13)	5 ± 3.5	4 (1.5–16)	4 ± 3.5	3 (0–18)
OxC	1 ± 0.7	0.5 (<0.5–6.5)	1.5 ± 1	1.3 (<0.5–3.5)	0.8 ± 1.2	0.6 (<0.5–5)
TN	0.6 ± 1.1	<0.5 (0–4.5)	0.5 ± 0.3	0.5 (0–1.2)	0.5 ± 0.5	<0.3 (<0.5–2)
p,p′-DDE	115 ± 70	105 (30–275)	200 ± 170	185 (37–690)	130 ± 120	92 (0–580)
p,p′-DDD	<1	<1	0.4 ± 1.6	<0.5 (<0.5–6.5)	0.3 ± 1.5	<0.5 (<0.5–8)
p,p′-DDT	4 ± 3	3.5 (<0.5–11)	10 ± 20	4 (1–85)	8.0 ± 28	2.5 (<0.5–155)
∑OCPs	130 ± 75	120 (40–295)	230 ± 180	210 (50–725)	155 ± 130	105 (15–620)
BDE 47	1.7 ± 2.5	1.0 (<0.5–11)	0.9 ± 0.7	0.8 (<0.5–3)	1.0 ± 1.1	0.9 (<0.5–4)
BDE 99	1.5 ± 2.5	0.8 (<0.4–11)	0.5 ± 0.3	0.5 (<0.4–1)	0.9 ± 0.6	0.7 (<0.4–2.8)
BDE 153	1.1 ± 0.4	1.1 (0.4–2.2)	0.9 ± 0.5	0.8 (0.2–2)	1.1 ± 0.9	0.8 (<0.2–3.7)
Penta–BDE	4.5 ± 6	3 (1–25)	2.3 ± 1.2	2.5 (0.5–5)	3.0 ± 2.2	2.5 (0–11)
6′HO–BDE99	0.3 ± 0.4	<0.5 (<0.5–1)	<0.5	<0.5	1.1 ± 3.2	<0.5 (<0.5–18)
BTBPE	0.2 ± 0.6	<1 (<1–2)	0.1 ± 0.4	<1 (<1–1.5)	0.5 ± 2.6	<1 (<1–14)
TBPH	<1	<1	<1	<1	1.5 ± 8	<1 (<1–45)
2,4,5-TBP	2.5 ± 2	3 (<1–5)	1.5 ± 2	<1 (<1–5)	2.5 ± 4	<1 (<1–18)
2,4,6-TBP	25 ± 12	22 (7–50)	20 ± 6	19 (11–33)	41 ± 60	21 (8–320)
∑BPs	32 ± 12	30 (10–60)	25 ± 6	26 (15–40)	53 ± 65	32 (13–340)

∑BPs were the 2nd dominant class of pollutants in serum, with 2,4,5- tribromophenol (2,4,5-TBP) and 2,4,6-TBP being the major BPs for all three groups of individuals. The presence of BPs in human serum might indicate the ether bond cleavage of PBDE congeners (BDE 47, −99, −100 and −154), which is an important metabolic pathway for PBDEs metabolism (Qiu et al., 2007). Higher levels of 2,4,6-TBP in the serum samples (median 20 ng/g lw) compared to PBDEs indicate other sources, such as its usage as reactive FR intermediate and/or as wood preservative (HSDB, 2003). In a recent study, dietary factors have been suggested to be good predictors of 2,4,6-TBP concentrations in human blood (Dallaire et al., 2009). PCP, a major OCP and phenolic compound, may be also formed to some extent through HCB metabolism. Pearson's correlation on log transformed data of HCB and PCP revealed significant correlation ($r = 0.49$, $p < 0.01$). This suggests that HCB metabolism is a contributing factor for PCP in serum along with other sources such as its use as biocides and wood preservatives (USEPA, 2008). No significant correlation ($p > 0.05$) was observed between PCP and 2,4,6-TBP, which indicates diverse emission source for these chemicals along with their common use as a wood preservative. This suggest sources of exposure of 2,4,6-TBP in human plasma are not clear, therefore it's accumulation in human blood might result from both dietary intake and biotransformation of PBDEs.

Table 3
Comparison of median concentrations (ng/g lw) of organic contaminants in serum sample with data from different countries.

Country	*N*	∑PCBs	*p,p′*-DDE	*p,p′*-DDT	β-HCH	HCB	$\sum_{3-7}$ PBDEs	Reference
Pakistan	University = 16	7.5	105	3.5	3	3	3.0	Present study (2013)
	Clothing = 15	8	185	4	9.5	4	2.5	
	Electronics = 30	7	92	2.5	3.8	3	2.5	
Pakistan	85	12.6	222	7.5	15.6	8	2.0	Ali et al., 2013b
Korea	40	127	224	18.6	49	16.7	–	Kang et al., 2008
Japan	80	166	221	–	93.2	–	–	Tsukino et al., 2006
China	26	52	540	37	11	39	–	Bi et al., 2007
	21	63	1800	340	38	31	–	
Bangladesh	Adult = 10	92	3600	380	160	7.7	–	Mamun et al., 2007
	Young = 8	–	3900	370	180	11	–	
Hong Kong	Male = 63	–	412	89.3	346	2.91	–	Wang et al., 2013a
	Female = 54	–	224	75.2	221	1.78	–	
Romania	142	383	1975	339	923	30	1.0	Dirtu et al., 2006
UK	154	170	100	2.9	12	11	4.7	Thomas et al., 2006
Spain	33	–	270	40	60	90	–	Lopez et al., 2007
Mexico	150	–	9500	1700	3100	–	–	Waliszewski et al., 2010
USA	341	212	204	–	–	14.9	–	Meeker et al., 2007
Korea	720	–	–	–	–	–	5.0	Kim et al., 2012
France	48	–	–	–	–	–	3.46	Brasseur et al., 2014
China	26	–	–	–	–	–	2.9	Zhu et al., 2009
Netherlands	33	–	–	–	–	–	8.7	Leijs et al., 2008
New Zealand	85	–	–	–	–	–	6.1	Harrad and Porter 2007
Hong Kong	117	–	–	–	–	–	5.36	Wang et al., 2013b

Table 4
Concentration (ng/g dust) of studied organic contaminants in dust samples from different occupational settings.

	University ($N = 16$)		Clothing stores ($N = 15$)		Electronics stores ($N = 30$)	
Analytes	Mean ± SD	Median (range)	Mean ± SD	Median (range)	Mean ± SD	Median (range)
CB 118	0.8 ± 1.1	0.6 (<0.2–4.5)	0.6 ± 0.5	0.5 (<0.2–2)	0.6 ± 0.9	0.3 (<0.2–5)
CB 153	1.2 ± 1.0	1.0 (<0.2–2.7)	1.1 ± 0.7	1.0 (<0.2–2.5)	0.7 ± 0.9	0.3 (<0.2–4)
CB 138	0.4 ± 1.6	<0.1 (<0.1–6.5)	0.3 ± 0.6	<0.1 (<0.1–2.2)	0.3 ± 0.9	<0.1 (<0.1–4)
CB 180	0.3 ± 0.1	0.3 (<0.1–0.5)	0.5 ± 0.2	0.5 (<0.1–1)	0.3 ± 0.7	0.1 (<0.1–4)
CB 170	0.3 ± 0.1	0.2 (<0.1–0.5)	0.4 ± 0.2	0.4 (<0.1–1)	0.4 ± 0.8	0.1 (<0.1–4)
∑PCBs	4.8 ± 2.8	4.6 (1.0–10)	5.5 ± 3.5	5.5 (1–12.5)	4 ± 5.5	1.3 (0.7–27)
∑HCHs	2.2 ± 2.8	1.6 (0.2–12.5)	0.6 ± 0.4	0.7 (0.2–1.5)	1.2 ± 3	0.4 (<0.1–17)
HCB	0.3 ± 0.2	0.1 (<0.1–0.6)	0.2 ± 0.1	0.3 (<0.1–0.5)	0.1 ± 0.1	<0.1 (<0.1–0.5)
TC	3.6 ± 6.3	2.0 (0.2–26.5)	3.5 ± 9.5	0.9 (<0.1–38)	5.1 ± 13	0.8 (<0.1–60)
CC	0.6 ± 1.2	<0.2 (<0.2–5.0)	0.5 ± 1.0	<0.2 (<0.2–4)	0.6 ± 2	<0.2 (<0.2–11)
TN	0.4 ± 0.9	0.2 (<0.1–3.7)	0.3 ± 0.7	<0.1 (<0.1–3)	0.3 ± 0.6	<0.1 (<0.1–2.5)
p,p′-DDE	65 ± 180	0.2 (<0.2–720)	<0.2	<0.2	16 ± 52	<0.2 (<0.2–225)
p,p′-DDD	6.3 ± 10	3.0 (<0.2–36)	0.1 ± 0.2	<0.2 (<0.2–1)	1.0 ± 3.5	<0.2 (<0.2–18)
p,p′-DDT	35 ± 50	18 (1.0–190)	4.5 ± 3	4 (0.5–11)	7 ± 13	3.5 (0.5–70)
∑OCPs	113 ± 190	35 (2–730)	10 ± 14	6.5 (1–60)	31 ± 73	6 (1–325)
BDE 47	3.5 ± 3.2	2.2 (1–12.5)	2.0 ± 1.8	1.7 (<0.2–6.5)	17 ± 66	3 (<0.2–365)
BDE 100	1.0 ± 1.5	0.5 (<0.2–5)	0.5 ± 0.5	0.3 (<0.1–1.6)	3.3 ± 11	0.3 (<0.2–62)
BDE 99	5.5 ± 6.0	3.5 (1–23)	2.8 ± 2.6	2.0 (<0.2–8.8)	20 ± 65	3 (<0.2–345)
BDE 154	0.7 ± 0.7	0.4 (<0.2–3)	0.3 ± 0.2	0.3 (<0.2–0.8)	3.2 ± 11	0.5 (<0.2–60)
BDE 153	1.4 ± 1.3	1 (0.5–5)	0.7 ± 0.4	0.6 (<0.2–1.5)	10 ± 30	1.2 (<0.2–150)
Penta–BDE	12 ± 12	7.5 (2.5–50)	6.5 ± 5.5	5 (0.8–19)	59 ± 210	10 (1–1150)
BDE 183	2.5 ± 4.0	1.1 (<0.2–17)	1 ± 0.8	0.8 (<0.2–3)	29 ± 73	2 (<0.2–300)
BDE 197	1.1 ± 1.4	0.6 (<0.2–6)	0.5 ± 0.3	0.4 (<0.2–1.5)	11 ± 26	1 (<0.2–105)
BDE 203	0.7 ± 0.7	0.5 (<0.5–3)	0.3 ± 0.2	<0.5 (<0.5–0.7)	8 ± 24	0.5 (<0.5–120)
BDE 196	1.0 ± 1.2	0.8 (<0.2–3)	0.4 ± 0.3	0.4 (<0.2–1)	10 ± 25	0.6 (<0.2–120)
Octa–BDE	5 ± 7	3.0 (1–30)	2 ± 1.5	2 (<0.5–6)	57 ± 145	4 (0.7–650)
BDE 209	75 ± 50	65 (12–205)	65 ± 60	45 (<2–195)	3200 ± 10,900	155 (<2–51,500)
HBB	1.3 ± 1.1	1.0 (<0.2–4.5)	2 ± 5	0.6 (<0.2–20)	11 ± 25	0.8 (<0.2–100)
TBB	2 ± 3.5	1.3 (<0.2–16)	0.6 ± 0.4	0.7 (<0.2–1.2)	3 ± 4	1 (<0.2–15)
TBPH	35 ± 50	19 (3–225)	11 ± 10	9 (<0.2–35)	100 ± 200	20 (0.6–950)
BTBPE	15 ± 27	9.5 (2–125)	7.5 ± 6	6.5 (0.5–20)	1000 ± 3450	17 (0.5–17,150)
DBDPE	62 ± 35	60 (10–120)	36 ± 37	31 (<2–155)	7100 ± 15,300	140 (2–52,150)
TCPP	35 ± 75	11 (<5–320)	35 ± 35	21 (<5–95)	120 ± 160	75 (6–620)
TBEP	825 ± 2685	115 (<15–11,900)	31 ± 20	32 (<15–70)	29 ± 29	<15 (<15–100)
TEHP	265 ± 930	50 (<5–4100)	38 ± 43	25 (<5–175)	110 ± 225	35 (<5–1025)
TPhP	1850 ± 5550	170 (10–23,450)	88 ± 70	62 (<2–220)	750 ± 1200	195 (10–5000)
EHDPP	105 ± 85	88 (5–285)	45 ± 37	45 (<2–120)	100 ± 185	45 (2–830)
TTP	70 ± 40	75 (15–150)	52 ± 39	50 (<15–120)	330 ± 920	57 (13–4750)
TDCPP	40 ± 45	20 (<5–185)	11 ± 10	7 (<5–30)	235 ± 465	29 (<5–1475)
∑PFRs	3290 ± 6100	1060 (120–24,600)	345 ± 200	395 (60–650)	1830 ± 2400	710 (75–9150)

3.1.3. *Profile of pollutants in dust samples*

PFRs were the dominant class of contaminants measured in the dust samples, contributing with >60% to the total load, followed by ∑NBFRs > ∑PBDEs > ∑OCPs > ∑PCBs (Fig. 1 and Table 4). For all occupational settings, TPhP, BDE 209, *p,p′*-DDT, and DBDPE were the major contributors for their respective group of chemicals (Table 4). These findings are similar with our earlier studies in Pakistan, where these chemicals were reported in household indoor dust (Ali et al., 2012, 2013a). The higher levels of FRs (PFRs, NBFRs and PBDEs) than OCPs and PCBs are not surprising, since these chemicals are used as additive materials in large number of consumer products and the use of PCBs and selected OCPs has been banned in Pakistan (Malik et al., 2011). The higher levels of PFRs in dust compared to PBDEs and NBFRs are also consistent with the literature (Ali et al., 2012; Dirtu et al., 2012; Kanazawa et al., 2010; Stapleton et al., 2009; Van den Eede et al., 2011), which suggests their application in a large range of polymers as a replacement to regulated PBDEs. In comparison to literature data, the median concentrations of PBDEs in this study was comparable to the levels reported in office dust from Belgium (D'Hollander et al., 2010) and was lower than those reported in indoor settled office dust from USA (Batterman et al., 2010), UK (Harrad et al., 2008b), Australia (Toms et al., 2009), Sweden (Thuresson et al., 2012) and Japan (Suzuki et al., 2006). Similarly, levels of PFRs were lower than those reported in different occupational settings of Sweden (Marklund et al., 2003). These differences in the levels of BFRs in indoor dust from various countries could be attributed to the country's fire safety regulations, which are generally more stringent in North America and Western Europe, more specifically UK (Guillaume et al., 2008; Zota et al., 2008). Another hypothesis might be the different timing for the replacement of PBDEs in various countries. Neither the use of BFRs in consumer products is regulated, nor they are produced in Pakistan. The occurrence of BFRs is assumed to arise *via* the presence in imported furniture and electronic stuff.

3.2. *Differences of organic contaminants among three groups*

Most OCPs, namely HCHs, PCP, OxC and *p,p′*-DDE, were present at higher levels in the serum samples of university volunteers compared to the other two groups. Information from the questionnaire could not explain any specific reason for these differences, since the volunteers lived in the same city and have similar age. Our data revealed higher OCPs levels in dust from the student hostel rooms/offices compared to dust from clothing and electronic stores, which might be a contributing factor for higher OCP levels in the serum. However, no statistically significant correlation between the levels of OCPs in dust and serum suggested the existence of additional factors/sources besides indoor dust ingestion. Most of the student donors who live in the hostel rooms are residents of other geographic areas (urban and rural), where these OCPs might have

Table 5
Pearson correlation among different organic contaminants in serum and age. Bold values indicate significantly positive associations.

Analyte		Age	∑PCBs	PCP	∑HCHs	∑DDTs	∑OCPs	Penta-BDE	∑HO-PCBs	2,4,6-TBP
∑PCBs	*r*	**0.46**								
	p	**<0.01**								
PCP	*r*	<0.01	0.23							
	p	0.98	0.08							
∑HCHs	*r*	<0.01	**0.39**	**0.53**						
	p	1	**<0.01**	**<0.01**						
∑DDTs	*r*	0.15	**0.44**	0.21	**0.54**					
	p	0.24	**<0.01**	0.11	**<0.01**					
∑OCPs	*r*	0.1	**0.47**	0.18	**0.49**	**1**				
	p	0.42	**<0.01**	0.16	**<0.01**	**<0.01**				
Penta-BDE	*r*	0.02	**0.33**	0.2	0.21	**0.29**	**0.28**			
	p	0.88	**0.01**	0.14	0.11	**0.03**	**0.03**			
∑HO-PCBs	*r*	0.08	**0.42**	0.18	0.08	0.16	0.05	0.12		
	p	0.55	**<0.01**	0.17	0.55	0.23	0.7	0.38		
2,4,6-TBP	*r*	0.19	**0.27**	<0.01	0.21	0.18	0.15	0.19	0.08	
	p	0.14	**0.04**	0.98	0.11	0.18	0.26	0.15	0.53	
∑BPs	*r*	0.2	**0.29**	0.01	0.19	0.14	0.1	0.22	0.13	**0.98**
	p	0.12	**0.03**	0.95	0.14	0.3	0.45	0.09	0.32	**<0.01**

been used historically and contributed significantly towards total OCP body burdens. All other contaminants in serum samples had similar patterns between each of three studied groups and no significant differences ($p < 0.05$) were observed.

Our results showed significantly higher ($p > 0.05$) levels of ∑HCHs and ∑DDTs in dust from university hostels rooms/offices. The dust brought in the room *via* the ventilation (opening of windows and/or doors) and *via* soil from outside the hostel/office on the shoes could be a reason for these higher levels in hostel dust. The hostels/offices are situated in an agricultural university campus with large surrounding area covered by agricultural fields in the close proximity; a possible use of these chemicals in the past could be an argument for these higher levels. Expectedly, levels of all FR classes were significantly higher ($p > 0.05$) in dust from electronic stores compare to clothing stores. Interestingly, most of the FRs, except for BTBPE and TCPP, were present at similar levels in dust from electronic stores and university hostels rooms/offices. An argument could be the more regular dusting of floor surface in electronic stores (on average daily) compares to the hostels rooms (on average once a week). A closer look at the data revealed that high levels of FRs were present in stores which were not vacuumed during the last 5 days and had bulk of old repairable electronics.

3.3. Associations between serum and dust levels

Spearman rank-order correlation coefficients for each pollutant, detected in at least 50% of samples, between dust and serum are provided in Table S3. Levels of Penta-BDE congeners in dust and serum were significantly correlated ($r = 0.64$, $p < 0.01$) (Table S3) for the university group. A significant correlation ($r = 0.54$, $p < 0.01$) (Table S3) was observed for BDE 99 in the electronics group, but no correlation was observed for other Penta-BDE congeners in both electronics and clothing groups. A possible explanation for this difference in the profile patterns among selected groups could be the number of hours/day spent by individuals in their respective indoor environment. Students are more closely associated with their belongings compared to the people working (8–10 h/day) in the stores. These findings are similar with the handful of studies who demonstrated various relationships between the levels of PBDEs in human serum and dust collected from the donor's homes (Johnson et al., 2010; Watkins et al., 2011; Wu et al., 2007).

Combining all groups together, we computed Pearson correlations among contaminants and results showed significant correlations among different analytes (Table 5). Levels of ∑PCBs were positively correlated with ∑OCPs, Penta-BDE, ∑HO-PCBs and ∑BPs ($p < 0.05$). Similarly, concentrations of ∑OCPs and Penta-BDE were also significantly correlated ($p < 0.05$). In the literature, strong correlations between serum concentrations of PCBs and OCPs are reported (Bi et al., 2007; Kang et al., 2008; Petrik et al., 2006) which indicates that humans are exposed to these chemicals *via* similar exposure routes; this seems logical as both groups of chemicals were widely used in past. Levels of PCBs were 2–3 orders of magnitude lower than OCPs, and the scenario might indicates the wider past application of OCPs in the region for malaria control and other industrial or agricultural purposes (Eqani et al., 2012). The situation addressed in current study is similar to other studies conducted in Asia, e.g. China, where relatively higher concentrations of OCPs contrasted with low concentrations of PCBs (Bi et al., 2007; Lee et al., 2007).

3.4. Associations of contaminants with age and effects on liver function parameters

We computed Pearson's correlation between age and levels of contaminants in serum. Only levels of ∑PCBs were significantly correlated ($r = 0.46$, $p < 0.01$) with age, suggesting that the exposure to most of the contaminants is independent of age. Various studies have reported positive associations between age and levels of PCBs and OCPs in human serum (Dirtu et al., 2006; Kang et al., 2008). Yet, in the present study, no significant associations were present between OCPs and age. No particular information and factors associated with these samples could explain the observed differences. Since levels of ∑PCBs in serum showed significant correlations (Table 5) with different OCPs, this suggests similar exposure pathways for these persistent contaminants. Lower levels of PCBs compare to higher levels of OCPs might suggest that the levels of PCBs are decreasing, while OCPs are still present at higher levels in the local food chain (Eqani et al., 2012).

Considering all three groups together, we divided them in two groups with (i) normal SGPT (male up to 45 U/l; female up to 34 U/l) and SGOT (up to 38 U/l) and (ii) higher SGPT and SGOT. A two tailed *t*-test showed the concentrations of ∑HCHs and 2,4,6-TBP were significantly higher ($p < 0.05$) in the group with elevated liver enzymes. These findings are in disagreement with our earlier findings, where ∑BPs was higher in the group with normal SGPT and SGOT (Ali et al., 2013b). The contrasting results of BPs in these two studies might be achieved just by chance. No studies on humans have documented BPs effect on liver enzymes or toxic effect on liver. A study on lab rats demonstrated increase in absolute and relative liver weights, but at high dose of 1000 mg/kg body weight per day (WHO, 2005). Although available human data are limited, increased values for the liver enzymes have been observed in factory workers involved in the production of technical-grade HCH (Kashyap, 1986). The higher levels of ∑HCHs in the group with elevated liver enzymes might be associated with some type of liver toxicity. Even though HCHs toxicity data on human is scarce, but studies on laboratory animal have reported some degree of liver toxicity such as increased microsomal activity, increased liver weight, mild-to-moderate liver necrosis and fatty degeneration following the ingestion of α-, β-, and γ-HCH isomers, individually or as technical-grade HCH (ATSDR, 2005).

4. Conclusions

This is the first study reporting several classes of organic contaminants in matched human serum and indoor dust samples from different working environments of Pakistan. ∑PFRs in dust and ∑OCPs in serum samples were the major contaminants in the three investigated occupational settings. Levels of Penta-BDEs were positively correlated in serum and dust samples from the university group, suggesting that dust is an important exposure pathway to the PBDE present in consumer articles for this group. NBFRs were detected in serum samples suggesting similar to PBDEs alternative BFRs also bioaccumulate in humans. Levels of ∑PCBs were positively correlated with age and ∑HCHs were significantly higher in people with increased liver enzymes. Although there were minimal differences among the levels of selected contaminants in the three groups, their occurrence in human serum and indoor dust suggests on-going exposure to some groups of chemicals. This study provides an important benchmark for bio-monitoring and exposure assessment of organic contaminants in Pakistan.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.envpol.2014.07.009.

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