Burdens of PBBs, PBDEs, and PCBs in tissues of the cancer patients in the e-waste disassembly sites in Zhejiang, China

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ABSTRACT

This study was conducted to explore the burdens of PBBs, PBDEs, and PCBs among cancer patients living in the e-waste disassembly sites. The contents of 23 PBB congeners, 12 PBDE congeners, and 27 PCB congeners in kidney, liver, and lung samples were measured by GC–MS. The results showed that low-brominated PBBs and PBB153 were the predominant congeners. PBDE47 were the most predominant PBDE congeners. PBDE209 were detected in >70% of the samples, with geometric means ranging from 64.2 to 113.9 ng g^{−1} lipid. Among the three subfamilies of PHAHs, PCB concentrations were the highest. The detected levels of PHAHs were in the same order of magnitude in the three tissues, which indicated that any of the three tissues could be the suitable indicator for assessing body burdens of PHAHs. PBB contents (181–192 ng g^{−1} lipid) were obviously higher than those reported in the general USA population (3–8 ng g^{−1} lipid). PBDE levels (174.1–182.3 ng g^{−1} lipid) were comparable to those reported in the USA population, but significantly higher than those of the European population. PCBs levels were comparable to those of the European population. The high cancer incidence in the disassembly sites may be related to higher burdens of PBBs, PBDEs, and PCBs in tissues.

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1. Introduction

Waste electrical and electronic equipment, or “e-waste” for short, has become the fastest growing stream of all solid waste found in China in that huge amounts of such waste are constantly being generated from (legal or illegal) imports and domestic use (Hicks *et al.*, 2005). A cluster of small villages in the littoral zone in the Zhejiang province has become a booming recycling center for e-waste, but nonetheless at the expense of having thousands of village workers engaged in primitive recycling operations without the use of adequate protective equipment (Zhao *et al.*, 2008). Such primitive operations include, but are not limited to, stripping of metals in open pit acid baths, removing electronic components from circuit boards by heating over a grill, and recovering metals by burning cables in (or near) the cropland (Deng *et al.*, 2007). Yet as a result of these operations and through leakage, evaporation, runoff, and leaching, many toxic chemicals, such as polybrominated biphenyls (PBBs), polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs), have been reported to be released into the local environmental and human matrices such as air (Deng *et al.*, 2007), sediment (Wang *et al.*, 2005; Luo *et al.*, 2007), soil (Liu *et al.*, 2006; Zhao *et al.*, 2008), cord blood (Qu *et al.*, 2007), and milk (Zhao *et al.*, 2007a). These pollutants can

be bioaccumulated in the aquatic and the terrestrial food chains and biomagnified in humans via food due to their lipophilicity (Zhao *et al.*, 2007a,b). These earlier findings suggest that the residents living near the e-waste disassembly site might be at high health risk.

Polyhalogenated aromatic hydrocarbons (PHAHs) represent a large family of highly lipophilic and environmentally persistent substances, of which PBBs, PBDEs, and PCBs are three subfamilies that were of great concern in the present study. All three PHAHs subfamilies are notably toxic and bioaccumulative (Hardy, 2000; McDonald, 2002). Animal studies (WHO, 1994a,b) showed that the PHAHs in these three subfamilies not only were they capable of disrupting endocrine functions but could also induce carcinogenicity.

An increasing incidence of cancer (such as liver cancer, and lung cancer) has been found around the e-waste disassembly sites during the last decades. However, there is still no report on body burdens of PHAHs among these local cancer patients in the disassembly sites. Accordingly, the present study had its focus on assessing body burden (or internal exposure) of local cancer patients to the 62 PHAHs that were deemed likely present in e-waste. Human tissue samples can reflect steady-state concentrations of lipophilic chemicals, which is considered as a suitable indicator for the study of environmental or occupational exposure to many chemical pollutants including PBDEs and PCBs (Meironyté Guvenius *et al.*, 2001; Petreas *et al.*, 2004; Johnson-Restrepo *et al.*, 2005; Naert *et al.*, 2006; Covaci *et al.*, 2008). Therefore, tissue samples (kidney, liver, and lung) were collected for

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assessing body burdens of PHAHs. This exploration represented the first of its kind in reporting extensively the recent levels of PBBs, PBDEs, and PCBs in tissues of local cancer patients. Information obtained from this type of exploration may also be useful for subsequent evaluation of the health risks at issue.

2. Materials and methods

2.1. Sample collection and storage

After obtained consent from participants according to procedures approved by the Ethics Committees of Research Center for Eco-Environmental Sciences, Chinese Academy of Sciences, kidney ($n=19$), liver ($n=55$), and lung ($n=7$) tissue samples (each of ~10–50 g) were collected from these surgical patients who were newly diagnosed for cancer from April 2007 to January 2008 at the First Hospital of Wenling and the Second Hospital of Wenling, with each sample placed into a separate chemically-cleaned glass bottle, and labeled with a unique code and the proper sampling date. The mean age of the individuals was 65 years ranging from 32 to 94 years. All samples were transported to the analytical laboratory as soon as possible in ice boxes and continued to be stored in the dark at $-20\text{ }^{\circ}\text{C}$ until analysis.

2.2. Materials and chemicals

The following standards were obtained from the Cambridge Isotope Laboratory (USA): 12 native PBDE congeners; 7 $^{13}\text{C}_{12}$ -labeled PBDEs (PBDE15, 28, 47, 99, 153, 154, and 183); 23 PBB congeners; 27 PCB congeners; and surrogate standards pentachloro-nitrobenzene (PCNB), 2,4,5,6-tetrachloro-*m*-xylene (TMX), and PCB209. All solvents used (hexane, acetone, and methylene chloride) were of pesticide grade (Promochem, Germany). Silica gel (100–200 mesh) was purchased from Promochem (Germany). Aluminum foil was rinsed with acetone and dried at ambient temperature prior to use. Sodium sulfate (granular, anhydrous) was pre-cleaned with methylene chloride and purified by heating at $450\text{ }^{\circ}\text{C}$ for 8 h in a shallow metallic enamel tray. Cellulose extraction thimbles of 33 mm i.d. and 94 mm in length were from Schleicher & Schuell (Germany); these thimbles were pre-cleaned by Soxhlet extraction with *n*-hexane: acetone (3:1, v/v) for 4 h before use. Glassware was soaked, cleaned with chromic solution, rinsed thoroughly with distilled water and acetone, and finally heated in a baking oven (Heraeus, Germany) at temperatures programmed from $40\text{ }^{\circ}\text{C}$ to $420\text{ }^{\circ}\text{C}$ at a rate of $15\text{ }^{\circ}\text{C min}^{-1}$ for 16 h.

2.3. Sample preparation and clean-up

The tissue samples were first individually pulverized by a stainless-steel machine, then, were all freeze-dried. About 2–5 g of these dried tissue residues from each sample was introduced into a pre-cleaned thimble and Soxhlet extracted for 24 h using 180 mL *n*-hexane/acetone (3:1, v/v) solution. For this preparation process, TMX, PCNB, and PCB209 were added to each sample as surrogate congener standards. The extract from each sample was then concentrated to about 1 mL by rotary evaporation (550 mbar, $60\text{ }^{\circ}\text{C}$). After gravimetric lipid determination, the concentrated extracts were further cleaned individually by a multilayer silica gel column containing: 2 g of anhydrous sodium sulfate; 8 g of silver nitrate (AgNO_3) silica (10%, AgNO_3 w/w); 2 g of deactivated silica (3.3% organic-free reagent water w/w); 15 g of acidic silica (44% conc. sulphuric acid w/w); 1 g of deactivated silica (3.3% organic-free reagent water w/w); and 2 g of anhydrous sodium sulfate. The silica gel column was wrapped in aluminum foil throughout clean up, to protect samples from debromination induced from UV-light, and pre-eluted with 80 mL of hexane prior to adding to the extract. The first

fraction eluted with *n*-hexane (100 mL) was used to concentrate the PCB congeners, with the second fraction (eluted with 10% methylene chloride in 80 mL *n*-hexane) intended for collection of the PBB and PBDE congeners (US EPA, 1996). The eluants were concentrated separately to about 1 mL, again by rotary evaporation. The solvent of each sample was evaporated to dryness by gentle nitrogen stream at $25\text{ }^{\circ}\text{C}$ and redissolved in 200 μL hexane.

2.4. Chemical analysis

The chemical analysis was performed using an Agilent 5975 GC–MS system equipped with a capillary DB-5MS column (5% phenyl/95% methyl silicone, 30 m, 0.25 mm i.d., and 0.25 μm film thickness, from J&W Scientific, Folsom, California, USA). The column oven temperature was programmed from $90\text{ }^{\circ}\text{C}$ (initial time, 1 min) to $250\text{ }^{\circ}\text{C}$ at a rate of $4\text{ }^{\circ}\text{C min}^{-1}$, then from $250\text{ }^{\circ}\text{C}$ to $300\text{ }^{\circ}\text{C}$ at a rate of $25\text{ }^{\circ}\text{C min}^{-1}$, and held for 5 min. The GC injector temperature was maintained at $260\text{ }^{\circ}\text{C}$, with the temperatures of the MS ion source and of the transfer line being kept at $230\text{ }^{\circ}\text{C}$ and $300\text{ }^{\circ}\text{C}$, respectively. The carrier gas was helium at a constant flow rate of 1.5 mL min^{-1} . The mass spectrometer was operated in the electron impact (EI) ionization mode with an electron energy of 70 eV. Samples (1 μL) were injected in the splitless mode with a solvent delay set at 4 min. The molecular ions ($[\text{M}]^+$ or $[\text{M}+2]^+$) and the fragment ions resulting from the loss of X_2 (i.e., $[\text{M}-\text{X}_2+2]^+$ or $[\text{M}-\text{X}_2+4]^+$, where X = chlorine or bromine) were selected as the precursor ions for mass spectrometric analysis. Quantitative analyses of PBB209 and PBDE209 were performed on the Agilent 5975 GC–MS equipped with a DB-5MS (5% phenyl/95% methyl silicone, 15 m, 0.25 mm i.d., and 0.1 μm film thickness, from J&W Scientific, Folsom, California, USA), at temperatures programmed from $90\text{ }^{\circ}\text{C}$ (initial time, 1 min) to $250\text{ }^{\circ}\text{C}$ at a rate of $10\text{ }^{\circ}\text{C min}^{-1}$, then from $250\text{ }^{\circ}\text{C}$ to $300\text{ }^{\circ}\text{C}$ at $15\text{ }^{\circ}\text{C min}^{-1}$, and finally held for 8 min. Mass spectrometer condition was performed by EI (70 eV) and selected ion monitoring of high abundance (m/z 943 and m/z 799 for PBB209 and PBDE209, respectively).

2.5. Quality assurance/quality control

For every batch of 10 samples, a solvent blank and a procedural blank were added to ensure that the samples and the analysis process were free of contamination. The detection limits (LOD) of the targeted compounds were defined as 3 times the signal to noise (S/N) ratio, ranged from 0.08 to 0.24 ng g^{-1} lipid for PBB congeners, from 0.08 to 0.32 ng g^{-1} lipid for PBDE congeners, from 0.02 to 0.12 ng g^{-1} lipid for PCB congeners, and from 0.80 to 1 ng g^{-1} lipid for PBB209 and PBDE209. Spike recoveries for $^{13}\text{C}_{12}$ -labeled PBDEs (at 10 ng) ranged from 75.2 to 96.5%; and those for TMX, PCNB, and PCB209 ranged from 70.4 to 92.5%, 81.6 to 107.4%, and 90.8 to 112.6%, respectively. Triplicate analysis of six diluted standard solutions (1.0, 5.0, 10.0, 25.0, 50.0, and 100.0 ng mL^{-1}) was performed for each selected standard mixture. Multi-level calibration curves were constructed for the quantification; and good to excellent linearity ($r^2>0.99$) was achieved.

2.6. Data analysis

A value of half LOD was given to the samples in which the contents of PBBs, PBDEs or PCBs were not detectable. Descriptive statistics (mean, range, etc.) were computed to characterize the concentrations of PBBs, PBDEs, and PCBs in the samples. All statistical analyses were performed for congeners for which more than 50% of the samples were above the LOD, using the Statistical Package for the Social Sciences (SPSS for Windows ver. 11.5) where applicable. The (statistical) term mean used throughout this paper referred to *geometric* mean. The levels of PBB209 and PBDE209, though measured and considered duly, were not included in calculating the total concentrations of PBBs and PBDEs. Nonparametric methods (Mann–

Table 1Statistical results of PBBs (in ng g⁻¹ lipid) in human tissue samples collected from the e-waste disassembly sites in Zhejiang, China.^a

Pollutant	Kidney (n = 19)				Liver (n = 55)				Lung (n = 7)			
	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %
PBB1	N.A.	N.A.	0.08–3.58	15.8	N.A.	N.A.	0.08–34.64	12.7	N.A.	N.A.	0.08–10.47	14.3
PBB2	54.49	62.73	17.28–284.60	100	44.73	46.46	4.12–301.39	100	27.51	32.00	12.65–56.38	100
PBB3	N.D.	N.D.	N.D.	0	N.A.	N.A.	0.08–47.25	1.8	N.A.	N.A.	0.08–11.20	14.3
PBB10	0.58	0.44	0.08–29.72	73.7	1.00	0.89	0.08–34.17	100	1.29	0.91	0.16–39.24	100
PBB4	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBB9	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBB7	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBB15	11.28	25.79	0.10–140.77	78.9	9.39	24.00	0.10–71.93	81.8	12.97	25.44	0.10–71.24	85.7
PBB30	2.40	7.72	0.10–119.23	63.2	3.34	7.61	0.10–81.71	70.9	9.08	14.55	0.10–58.29	85.7
PBB18	N.A.	N.A.	0.10–40.56	36.8	1.44	4.61	0.10–38.61	56.4	1.78	7.27	0.10–59.05	57.1
PBB29	N.A.	N.A.	0.12–146.15	36.8	2.40	9.59	0.12–79.04	56.4	2.93	15.93	0.12–69.71	57.1
PBB26	N.A.	N.A.	0.12–29.43	36.8	N.A.	N.A.	0.12–51.64	45.5	N.A.	N.A.	0.12–26.00	14.3
PBB31	N.D.	N.D.	N.D.	0	N.A.	N.A.	0.12–18.43	1.8	N.D.	N.D.	N.D.	0
PBB53	N.A.	N.A.	0.15–40.00	31.6	N.A.	N.A.	0.15–45.97	30.9	N.A.	N.A.	0.15–13.94	14.3
PBB38	N.A.	N.A.	0.15–64.72	15.8	N.A.	N.A.	0.15–69.20	18.2	N.A.	N.A.	0.15–17.84	14.3
PBB52	N.A.	N.A.	0.15–218.46	31.6	N.A.	N.A.	0.15–48.66	21.8	N.A.	N.A.	0.15–68.19	14.3
PBB49	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBB103	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBB80	3.66	32.04	0.18–164.62	52.6	5.27	19.28	0.18–91.84	63.6	9.24	33.02	0.18–97.00	71.4
PBB101	N.A.	N.A.	0.24–7.73	5.3	N.A.	N.A.	0.24–9.33	5.5	N.A.	N.A.	0.32–33.45	14.3
PBB155	N.A.	N.A.	0.32–33.85	15.8	N.A.	N.A.	0.32–33.91	14.5	N.A.	N.A.	0.32–88.76	28.6
PBB153	9.97	29.87	0.32–183.85	52.6	15.62	23.09	0.32–97.55	87.3	20.15	37.96	0.32–81.52	85.7
PBBs	185.81	193.87	38.86–1137.69	100	180.59	193.17	41.90–558.57	100	191.84	149.78	102.40–676.57	100
PBB209	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0

^aPBBs = polybrominated biphenyls; a value of half the limit of detection (LOD) was given for samples in which PBB residues were not detected; all statistical analyses were performed for congeners for which more than 50% of the samples were above the LOD; G.M.: geometric mean; N.A.: not available; N.D.: not detected; >LOD%: percent detected.

Whitney U test) was used to determine differences in PHAHs levels among the three types of tissue samples, all statistical significance was set at $\alpha = 0.05$.

3. Results

3.1. Levels of PBBs measured in the samples

The levels of the 23 PBB congeners measured in the kidney, liver and lung tissue samples are summarized in Table 1. These data showed that there was a comparable PBBs level among these collected kidney (185.8 ng g⁻¹ lipid), liver (180.6 ng g⁻¹ lipid) and lung tissue samples (191.8 ng g⁻¹ lipid) ($P > 0.05$). Low-brominated PBBs (including PBB2, 10, 15, and 30) were found as the most predominant pollutants, accounting for >59% of the total PBBs observed; they were detected in more than 50% of these tissue samples. In addition, hexa-brominated congener PBB153 was also found to be one of the predominant pollutants, which accounted for >12% of the total PBBs observed. The

PBB209 levels in all tissue samples were below LOD (<0.80 ng g⁻¹ lipid).

3.2. Levels of PBDEs measured in the samples

The levels of the 12 PBDE congeners measured in the three types of tissue samples are summarized in Table 2, which shows that there was a comparable PBDEs level among these collected kidney (182.3 ng g⁻¹ lipid), liver (174.1 ng g⁻¹ lipid) and lung tissue samples (174.2 ng g⁻¹ lipid) ($P > 0.05$). The data in this table also showed that PBDE47 and 28 were the most predominant congeners, accounting for >17% and >10% of the total PBDEs observed in the collected samples, respectively. For PBDE209, its highest levels observed in kidney, liver, and lung was separately 72,964.5, 61,624.7, and 38,887.6 ng g⁻¹ lipid, which were far higher than those of the other 11 PBDE congeners combined in the corresponding samples; and PBDE209 were detected in more than 70% of the collected samples.

Table 2Statistical results of PBDEs (in ng g⁻¹ lipid) in human tissue samples collected from the e-waste disassembly sites in Zhejiang, China.^a

Pollutant	Kidney (n = 19)				Liver (n = 55)				Lung (n = 7)			
	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %
PBDE3	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBDE15	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PBDE17	4.92	17.21	0.10–75.40	68.4	5.14	15.47	0.10–94.57	69.1	N.A.	N.A.	0.10–45.94	42.9
PBDE28	29.64	48.39	0.20–80.00	94.7	17.44	35.23	0.20–102.04	85.5	38.50	36.96	12.80–82.67	100
PBDE47	32.36	53.56	0.20–80.56	94.7	36.51	50.83	0.20–108.16	98.2	19.73	38.69	0.20–73.75	85.7
PBDE66	N.A.	N.A.	0.20–60.67	5.3	N.D.	N.D.	0.20–91.94	30.9	N.D.	N.D.	N.D.	0
PBDE100	1.79	3.12	0.25–91.67	63.2	3.37	4.97	0.25–82.03	81.8	3.48	6.57	0.25–106.67	57.1
PBDE99	9.23	16.42	0.25–26.24	89.5	3.89	8.65	0.25–25.46	72.7	4.45	12.33	0.25–24.35	71.4
PBDE154	4.45	17.02	0.30–238.46	52.6	5.85	17.39	0.30–124.08	60	13.40	39.48	0.30–117.33	71.4
PBDE153	4.35	15.80	0.30–101.94	52.6	5.36	19.13	0.30–111.92	58.2	3.31	6.75	0.30–17.80	71.4
PBDE183	2.30	4.46	0.32–81.61	63.2	5.33	9.17	0.32–20.93	89.1	3.22	6.77	0.32–12.05	71.4
ΣPBDEs	182.33	186.09	56.49–410.61	100	174.10	192.61	47.69–541.10	100	174.23	171.96	64.42–333.70	100
PBDE209	113.85	191.27	1.00–72964.52	73.7	64.16	118.10	1.00–61624.65	74.5	105.07	270.00	1.00–38887.60	71.4

^aPBDEs = polybrominated diphenyl ethers; a value of half the limit of detection (LOD) was given for samples in which PBDE residues were not detected; all statistical analyses were performed for congeners for which more than 50% of the samples were above the LOD; G.M.: geometric mean; N.A.: not available; N.D.: not detected; >LOD%: percent detected.

Table 3Statistical results of PCBs (in ng g⁻¹ lipid) in human tissue samples collected from the e-waste disassembly sites in Zhejiang, China.^a

Pollutant	Kidney (n = 19)				Liver (n = 55)				Lung (n = 7)			
	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %	G.M.	Median	Range	>LOD, %
PCB8	9.29	9.24	2.61–37.22	100	9.79	8.67	2.05–284.42	100	7.23	8.76	1.73–23.00	100
PCB18	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PCB28	7.45	9.55	0.04–35.38	94.7	8.21	10.64	0.04–79.72	96.4	9.08	10.08	3.29–17.02	100
PCB52	9.55	11.67	3.33–36.92	100	7.22	8.10	0.04–46.57	96.4	6.68	9.87	1.21–12.25	100
PCB44	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PCB66	16.11	19.53	0.06–69.61	94.7	10.66	20.46	0.06–156.94	85.5	12.57	15.27	5.28–23.24	100
PCB101	8.78	14.82	0.07–104.48	89.5	11.61	15.24	0.07–111.07	90.9	1.94	7.71	0.07–13.50	71.4
PCB153	25.18	39.08	0.08–224.42	89.5	53.50	52.43	9.59–389.74	100	9.31	21.30	0.08–32.00	85.7
PCB138	41.57	53.98	0.08–223.92	94.7	66.87	65.12	12.35–240.36	100	32.58	36.95	12.90–45.50	100
PCB187	4.69	8.03	0.08–19.39	84.2	4.30	10.67	0.08–36.60	80	2.00	4.59	0.08–15.24	71.4
PCB128	2.14	9.33	0.06–53.61	63.2	1.19	5.10	0.06–45.00	56.4	5.59	12.80	0.06–17.45	85.7
PCB180	31.32	38.06	0.08–115.83	94.7	49.52	53.19	10.10–303.55	100	4.48	21.69	0.08–33.00	71.4
PCB170	18.25	23.25	0.08–56.96	94.7	21.80	25.85	0.08–131.25	96.4	8.37	18.24	0.08–31.25	85.7
PCB195	13.73	18.72	0.12–31.39	94.7	7.63	16.39	0.12–45.31	85.5	8.14	17.37	0.12–28.19	85.5
PCB206	13.41	20.22	0.12–31.03	94.7	11.04	18.46	0.12–42.45	92.7	18.38	16.73	10.74–36.95	92.7
PCB77	11.20	22.37	0.06–36.94	89.5	7.64	16.52	0.06–71.92	83.6	4.25	20.00	0.06–38.86	71.4
PCB81	N.A.	N.A.	0.06–41.32	10.5	N.A.	N.A.	0.06–88.57	10.9	N.D.	N.D.	N.D.	0
PCB105	1.91	13.68	0.07–75.47	57.9	8.81	18.60	0.07–79.24	85.5	N.A.	N.A.	0.07–16.00	42.9
PCB114	6.15	20.43	0.07–70.06	78.9	6.66	18.67	0.07–73.72	80	N.A.	N.A.	0.07–13.94	14.3
PCB118	N.A.	N.A.	0.08–25.23	36.8	N.A.	N.A.	0.08–27.42	23.6	N.A.	N.A.	0.08–16.00	28.6
PCB123	21.59	34.02	0.08–307.85	89.5	22.80	43.45	0.08–199.45	87.3	9.15	24.00	0.08–29.44	85.7
PCB126	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0	N.D.	N.D.	N.D.	0
PCB156	N.A.	N.A.	0.08–44.44	31.6	N.A.	N.A.	0.08–595.71	47.3	N.D.	N.D.	N.D.	0
PCB157	N.A.	N.A.	0.08–11.33	5.3	N.A.	N.A.	0.08–13.93	7.3	N.D.	N.D.	N.D.	0
PCB167	N.A.	N.A.	0.08–10.43	31.6	N.A.	N.A.	0.08–17.55	34.5	N.A.	N.A.	0.08–9.75	28.6
PCB169	N.A.	N.A.	0.08–0.83	5.3	N.A.	N.A.	0.08–3.92	3.6	N.D.	N.D.	N.D.	0
PCB189	2.26	7.11	0.12–45.96	57.9	N.A.	N.A.	0.12–184.68	47.3	6.12	18.08	0.12–180.22	71.4
ΣPCBs	399.35	382.15	86.92–1403.92	100	455.13	460.00	89.19–1742.57	100	257.91	304.64	104.85–373.25	100
ΣTEQs ^b	13.13 × 10 ⁻³	18.98 × 10 ⁻³	(0.01–100) × 10 ⁻³	100	16.81 × 10 ⁻³	21.09 × 10 ⁻³	(0.01–328.30) × 10 ⁻³	100	7.57 × 10 ⁻³	8.22 × 10 ⁻³	(2.23–18.00) × 10 ⁻³	100

^aCBs = polychlorinated biphenyls; a value of half the limit of detection (LOD) was given for samples in which PCB residues were not detected; all statistical analyses were performed for congeners for which more than 50% of the samples were above the LOD; G.M.: geometric mean; N.A.: not available; N.D.: not detected; >LOD%: percent detected; ^bTEQs = toxic equivalence quotients (or toxic equivalents), which each were calculated using the World Health Organization method and toxic equivalence factors (Van den Berg et al., 2006) in an effort to weight the toxicity as well as the potency of dioxin (2,3,7,8-TCDD)-like PCB compounds (i.e., those last 12 PCBs listed in this table) in relation to that of TCDD.

3.3. Levels of PCBs measured in the samples

As summarized in Table 3, among the three subfamilies of PHAHs (excluding PBDE209), PCBs had the highest concentrations in these tissue samples collected from these e-waste disassembly sites, with levels ranging from 257.9 to 455.1 ng g⁻¹ lipid. PCB138 was the most predominant congener, detected in more than 85% of the collected samples. The level ΣTEQ_{PCBs} (the sum of the toxic equivalents) observed in kidney, liver, and lung tissue samples was 13.1, 16.8, and 7.6 pg g⁻¹ lipid, respectively.

4. Discussion

The PHAHs in the three subfamilies (PBBs, PBDEs and PCBs) were all detectable in the collected tissue samples at high levels, which showed that these PHAHs had entered the patients' body through environment and dietary exposure. The detected levels of PHAHs were in the same order of magnitude in the three tissues, which indicated that any of the three tissues could be the suitable indicator for assessing actual body burden of PHAHs.

4.1. PBBs in human tissue

Low-brominated PBBs were the principal congeners in the e-waste residues, surface soil, and hair samples (Zhao et al., 2008). Results from the last study well supported the assertion that low-brominated PBBs (including PBB2, 10, 15, and 30) were the predominant congeners observed in the human tissue sample, which were seen to have accounted for as much as 59% of the total PBBs. Some investigations indicated that the high-brominated PBBs may metabolize to their low-brominated congeners in body. For example, fish (*juvenile Salmo salar*),

exposed in laboratory studies to FireMaster^(R) BP-6 in water, contained several mono to penta-bromobiphenyls that were not present in BP-6 (Zitko, 1977; WHO, 1994a; Stapleton et al., 2004).

The commercial production of PBBs began in the 1970s. Approximately 6 million kg of PBBs were produced in the United States from 1970 to 1976, hexabromobiphenyl constituted about 5.4 million kg (88%) of this total (Neufeld et al., 1977). The congener PBB153 was the principal component in commercial hexabromobiphenyl, which accounts for 60–80% of the total weight (POPRC, 2007a). Hexabromobiphenyl was widely used as a fire retardant in three main commercial products: acrylonitrile–butadiene–styrene (ABS) thermoplastics for constructing business machine housings and in industrial (e.g. motor housing), and electrical (e. g. radio and TV parts) products; as a fire retardant in coatings and lacquers; and in polyurethane foam for auto upholstery (WHO, 1994a; POPRC, 2007a). Most in the production of plastic products with an estimated use life of 5–10 years (Neufeld et al., 1977), since the cessation of production, all of these products, such as TV cabinet and business machine housings, must have been disposed of in the e-waste disassembly sites or incineration (Neufeld et al., 1977; US ATSDR, 2004). Therefore, PBB153 were deemed likely present in humans living around the e-waste disassembly sites. The results of this study showed that PBB153 was also a predominant congener, which accounted for 12% the total PBBs observed. Obviously, the content was far lower than 60–80%, which is the percent of PBB153 in this commercial production of PBBs. Large part of PBBs was produced and used in the 1970s (WHO, 1994a; POPRC, 2007a). The metabolism rate of hexa-PBBs was faster than hexa-PCBs (WHO, 1994a), and during the 30–40 year period after these compounds were produced, they might have been metabolized to low-brominated PBBs. In addition, these factors, such as photolytic degradation, microbial degradation, and degradation by plants and

animals, can lead to PBB153 decrease. PBB209 was not be detected in the collected tissue samples, this finding is consistent with the fact that no PBB209 was observed in the sampled food in our earlier study.

4.2. PBDEs in human tissue

PBDE47 was the most predominant PBDE congener, accounting for >17% of the total PBDEs observed in the collected samples. Many researchers also found similar results in human milk and blood

samples (Meironyté Guvenius et al., 2001; Covaci et al., 2008). PBDE47 was reported to be one of the main isomers in commercial pentaBDE product, and the congener was also found to be the primary PBDE congener in the food samples in our earlier study, these facts supported well the above-mentioned finding in the study.

PBDE28 was found to be one of the most abundant PBDE congeners in air particles from another electronic waste recycling site (Guiyu) (Deng et al., 2007), and was also detected at a high level in the e-waste residues, soil and food samples collected from the disassembly sites in

Table 4

Levels of PBBs, PBDEs, and PCBs (in ng g lipid⁻¹) among human tissues in various localities in China and other countries.

Countries (or districts)	N	Sampling year	Characteristics of the study	PBBs	PBDEs	PCBs	PBDE209	References
Belgium ^a	20	2000	Adipose	–	2.2–11.7	841 (286–1802)	–	Covaci et al. (2002)
Belgium ^b	25	2003–2005	Liver	–	3.6 (1–10)	380 (90–1140)	–	Covaci et al. (2008)
Belgium ^b	25	2003–2005	Adipose	–	5.3 (1.4–13.2)	490 (70–1130)	–	Covaci et al. (2008)
Belgium ^c	53	2001–2003	Adipose	–	11.1 (1.2–57.2)	763.7 (126–2090)	–	Naert et al. (2006)
China (Shanghai) ^d	5	2001	Mammary glands	–	–	70 (27–135)	–	Nakata et al. (2002)
China (Taiwan) ^e	20	2000–2001	Human milk	–	3.9	–	0.3	Chao et al. (2007)
China (Guiyu) ^f	26	2005	Serum	–	580 (140–8500)	69 (17–180)	340	Bi et al. (2007)
China (Haojiang) ^f	21	2005	Serum	–	190 (16–490)	65 (22–140)	130	Bi et al. (2007)
China (Guangdong) ^g	20	2006	Serum	–	–	–	3436	Qu et al. (2007)
China (Wenling)	19	2008	Kidney	185.8	182.3	399.4	113.9	Present study
China (Wenling)	55	2008	Liver	180.6	174.1	455.1	64.2	
China (Wenling)	7	2008	Lung	191.8	174.2	257.9	105.1	
Denmark ^h	409	1993–1997	Case Adipose	–	–	1027.4	–	Raaschou-Nielsen et al. (2005)
Denmark ^h	409	1993–1997	Control Adipose	–	–	1924.9	–	Raaschou-Nielsen et al. (2005)
Germany ⁱ	25	–	Human milk	2	–	–	–	WHO (1994a)
Japan ^j	12	2002	Human milk	–	0.7–2.8	–	–	Ohta et al. (2002)
Japan ^k	89	2005	Human milk	–	1.6	63.9	0.6	Inoue et al. (2006)
Japan ^k	89	2005	Serum	–	2.9	37.5	0.3	Inoue et al. (2006)
Singapore ^l	16	2003–2004	Adipose	–	0.5–12.3	–	–	Li et al. (2005)
Spain ^m	20	2003	Adipose	0.4	3.9	–	–	Fernandez et al. (2007)
Sweden (Stockholm) ⁿ	5	1994	Liver	–	4.5–18	459–2085	–	Meironyté Guvenius et al. (2001)
Sweden (Stockholm) ⁿ	5	1994	Adipose	–	3.8–7.7	561–2343	–	Meironyté Guvenius et al. (2001)
Sweden ^o	21	1996–1999	Adipose	–	2.3	757	–	Hardell et al. (2007)
Sweden ^o	59	1996–1999	Adipose	–	5.1	1687	–	Hardell et al. (2007)
Sweden ^p	7	2000	Serum	–	–	–	270	Thuresson et al. (2005)
UK ^q	154	2003	Serum	–	5.6 (0.6–420)	170 (14–670)	15–240	Thomas et al. (2006)
US (Michigan) ^r	64	1975–1994	Serum (<10 y)	10,456.2	–	1,998.0	–	Sweeney et al. (2001)
US (Michigan) ^r	149	1975–1994	Serum (11–16 y)	6660.0	–	1931.4	–	Sweeney et al. (2001)
US (Michigan) ^r	347	1975–1994	Serum (17–42 y)	3429.2	–	2264.4	–	Sweeney et al. (2001)
US (Michigan) ^s	196	1985	Adipose	475	–	–	–	Miceli et al. (1985)
US ^t	9	1985–1989	Serum	8.0 (2.6–9.4)	9.6 (4.6–74)	90 (48–140)	–	Sjödin et al. (2004)
US ^t	14	1990–1994	Serum	5.3 (1.0–8.6)	48 (7.5–86)	66 (38–87)	–	Sjödin et al. (2004)
US ^t	10	1995–1999	Serum	4.7 (1.9–10)	71 (42–120)	52 (36–110)	–	Sjödin et al. (2004)
US ^t	7	2000–2002	Serum	3.3 (1.4–5.5)	61 (47–160)	35 (19–49)	–	Sjödin et al. (2004)
US (Texas) ^u	47	2002	Human milk	–	73.9 (6.2–419)	–	0.9 (ND–8.2)	Schecter et al. (2003)
US (California) ^v	21	2003	Adipose	–	91 (17–440)	404	–	Petreas et al. (2004)
US (New York) ^w	52	2003–2004	Adipose	–	399 (17–9630)	144 (18.9–816)	–	Johnson–Restrepo et al. (2005)
US (Texas) ^u	11	2007	liver	–	23.1 (4–98)	–	–	Schecter et al. (2007)

^a PBDE 28, 47, 66, 71, 75, 77, 85, 99, 100, 138, 153, and 154; PCB28, 31, 52, 74, 95, 99, 101, 105, 110, 118, 128, 138, 146, 149, 153, 156, 163, 167, 170, 171, 172, 177, 178, 180, 183, 187, 189, 190, 194, 195, 196/203, 199, 206, and 209.

^b PBDE28, 47, 99, 100, 153, 154 and 183; PCB28/31, 52, 74, 95, 99, 101, 105, 110, 118, 128, 138/163, 149, 153, 156, 170, 180, 183, 187, 194, and 196.

^c PBDE28, 47, 99, 100, 154, 153, and 183; PCBs 28, 52, 101, 118, 138, 153, and 180.

^d Kanechlor mixture (KC-300, 400, 500, and 600).

^e PBDE17, 28, 47, 66, 85, 99, 100, 138, 153, 154, 183, and 209.

^f PCB28, 52, 56, 60, 70, 74, 90, 95, 99, 101, 105, 118, 138, 149, 153, 156, 157, 158, 167, 170, 180, 183, 187, 194, 203; PBDE28, 47, 85, 99, 100, 153, 154, 183, 196, 197, 203, 206, 207, 208, and 209.

^g The highest concentration of PBDE209 was observed in serum of the electronic waste dismantling workers 3436 ng g lipid⁻¹.

^h PCB28, 52, 54, 55, 99, 101, 104, 105, 118, 128, 138, 153, 156, 170, 180, 183, 187, and 201.

ⁱ PBB103, 131, 132, 135, 147, 148, 149, 153, 154, 155, 169, 176, 178, 179, 180, 181, 184, 185, 186, 187, 188, 192, 194, 197, 198, 200, 201.

^j PBDE28, 47, 99, 100, 153, and 154.

^k PBDE 15, 28, 47, 99, 100, 153, 154, 183, 196, 197, 206, 207, and 209; PCB74, 99, 118, 138, 146, 153, 156, 163/164, 170, 180, 182/187, 194, 199, 206, and 209.

^l PBDE47, 99, 100, 153, and 154.

^m PBB18, 29, 31, 22, 38, 37, 53, 52, 49, 75, 80, 56, 77, 103, 101, 155, 154, 153, and 169; PBDE 28, 75, 71, 47, 66, 77, 100, 119, 99, 85, 154, 153, 138, and 183.

ⁿ PBDE17, 28, 47, 66, 85, 99, 100, 153, and 154; PCB28, 47, 52, 101, 105, 114, 118, 122, 123, 138, 153, 156, 157, 167, 170, 180, and 189.

^o PCB28, 74, 101, 105, 114, 118, 128, 138, 149, 153, 156, 170, 180, and 187.

^p The highest PBDE209 concentration observed in serum of the rubber workers was 270 ng g lipid⁻¹.

^q PBDE17, 28, 32, 35, 37, 47, 49, 71, 75, 77, 85, 99, 100, 119, 138, 153, 154, 166, 181, 183 and 190; PCBs 18, 22, 28, 31, 41/64, 44, 49, 52, 54, 70, 74, 87, 90/101, 95, 99, 104, 105, 110, 114, 118, 123, 138, 141, 149, 151, 153, 155, 156, 157, 158, 167, 170, 174, 180, 183, 187, 188, 189, 194, 199 and 203.

^r Aroclor 1254.

^s Hexabromobiphenyls.

^t PBB153; PBDE47, 85, 99, 100, 153, and 154; PCB153.

^u PBDE17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209.

^v PBDE47, 99, 100, 153, and 154; PCB118, 138, 153, and 180.

^w PBDE28, 30, 47, 85, 99, 100, 153, and 154; Kanechlor (KC 300, 400, 500, and 600).

our earlier study (Zhao et al., 2008). The congener thus can enter the body through three main routes of exposure: inhalation, ingestion and skin penetration. In addition, the metabolism of high brominated congeners to lower brominated ones could also contribute to the high proportion of PBDE28 in samples observed.

PBDE209 has low bioaccumulative potential, and low biologic activity (Hooper and McDonald, 2000). Additionally, PBDE209 may break down in sunlight or anaerobic degradation into the low-brominated PBDE congeners (Watanabe and Tatsukawa, 1978). PBDE209 was not thus considered to be bioavailable (Kierkegaard et al., 1999), which contrasted with the result on high concentration of PBDE209 which was found in the collected human tissue samples, especially, the highest concentration of PBDE209 was detected in kidney tissue samples ($72,964.5 \text{ ng g}^{-1} \text{ lipid}$). Bi et al. (2007) and Thuresson et al. (2005) also observed high concentrations of PBDE209 in serum from electronics dismantlers ($340 \text{ ng g}^{-1} \text{ lipid}$) and rubber workers ($300 \text{ pmol g}^{-1} \text{ lipid}$) manufacturing or handling rubber compound that was flame retarded with a technical mixture of PBDE209. These findings evidently indicated that PBDE209 was bioavailable.

Sandholm (2003) observed a short half-life ($t_{1/2} = 2.4$ days) for PBDE209 in rats dosed with this congener. Also, gray seals experimentally dosed with PBDE209 indicated a short half-life (8–13 days) of this compound (Thomas et al., 2006). Observations in five electronic dismantlers sampled before and after 30 days of vacation suggested a rather rapid elimination of PBDE209, with a median decrease of 66% during this period of time (Sjödén et al., 1999). The half-life of PBDE209 is so short, yet the compound still can be found at a high level in the human tissue samples in the present study. The finding suggested that there might be new inputs of the congener. In fact, PBDE209 is the most widely used PBDEs, which accounts for more than 70% of the total PBDE demand, with a global demand of about 56,100 tonnes in 2001 and 56,418 tonnes in 2003 (US ATSDR, 2004; POPRC, 2007b). PBDE209 is used in ABS-plastics, high impact polystyrene, epoxy resins, and rubber, and thus may appear in such e-waste products as obsolete or end-of-life printed circuit boards, cables, and television sets (POPRC, 2007b). Therefore, under ideal conditions it would be more readily released into the environment. These disassembly workers might be exposed to PBDE209 by breathing contaminated workplace air and by skin contact (WHO, 1994b). Residents who live near e-waste disassembly sites may be exposed primarily by breathing air and ingesting food. In addition, PBDE209 are still manufactured and used in China. Therefore, these facts all supported the observation that PBDE209 was the predominant congener measured in the collected human tissue samples.

4.3. PCBs in human tissue

Among the three subfamilies of PHAHs (excluding PBDE209), PCBs had the highest concentrations in the human tissue samples, this finding was consistent with highest PCB intake ($12,372.9 \text{ ng day}^{-1}$) observed in the daily dietary estimation for residents living around the e-waste disassembly sites in the earlier study. The most predominant PCB congener in these samples was PCB138, with its concentrations accounting for 10% of the total PCBs concentrations observed, similar findings for the congener were also observed in the soil, food, cord blood, and milk samples from the disassembly sites (Zhao et al., 2007a,b).

Covaci et al. (2008) found that PCBs were significantly correlated with age, but no correlation was observed between PBDEs and age. There is no correlation between levels of PBDEs (or PCBs) and age in the present study. As mentioned above, e-waste is the pollution source for the local environment, therefore, the type and amount of e-waste can directly result in different types of PHAHs pollution (e.g., the primary pollutants in obsolete transformers are PCBs), which will change the type and amount of PHAHs observed in the residents,

additionally, the distance from the e-waste disassembly sites to the living sites of residents may also change the level of exposure for residents to PHAHs, these factors influence the observed values of these PHAHs in tissues of residents, which leads to the levels of PHAHs being not only affected by age.

4.4. International comparison

To further appreciate the level of contamination in human tissue from the e-waste disassembly sites, the PHAHs levels monitored in the present study were compared to those reported recently for districts located in other Chinese provinces and other countries. As shown in Table 4, the concentrations of PBBs in kidney, liver and lung tissue samples (ranging from $181\text{--}192 \text{ ng g}^{-1} \text{ lipid}$) were ten times of those reported in the general USA population ($3\text{--}8 \text{ ng g}^{-1} \text{ lipid}$), however, the observed value were still far lower than the means (ranging from $3,429$ to $10,456.2 \text{ ng g}^{-1} \text{ lipid}$) observed in the Michigan study, in which a widespread contamination of farm products by PBBs occurred. PBDE levels observed were comparable to those reported in the general USA population, which were also significantly higher than those reported values in European population. PCBs level was in the same order of magnitude as those reported on population in European industrialized country.

This investigation is the first that reports in detail the recent levels of PBBs, PBDEs and PCBs in human tissue samples from areas near the disassembly sites for e-waste in Zhejiang Province. The data from this preliminary study show that high PHAH levels were detected in the cancer patients' tissue samples, thus suggesting that the higher cancer incidence in the e-waste disassembly sites may be related to higher burdens of PBBs, PBDEs, and PCBs. However, more in-depth investigation of such an association is needed.

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