Environment International 42 (2012) 47–52

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Levels and profiles of PCDD/Fs, PCBs in mothers' milk in Shenzhen of China: Estimation of breast-fed infants' intakes

Bo Deng [a,b], JianQing Zhang [a,*], Lishi Zhang [c], YouSheng Jiang [a], Jian Zhou [a], Daokui Fang [a], Huiming Zhang [a], HaiYan Huang [a]

[a] Department of POPs Lab, Shenzhen Center for Disease Control and Prevention, No. 21, 1st Road Tianbei, Luohu District, Shenzhen, Guangdong 518020, PR China
[b] North Sichuan Medical College, Nanchong, Sichuan, 637000, PR China
[c] West China University of Medical Sciences, Sichuan University, Chengdu, Sichuan, 610041, PR China

ARTICLE INFO

Article history:
Received 18 October 2010
Accepted 30 March 2011
Available online 30 April 2011

Keywords:
Body burden
Polychlorinated dibenzo-p-dioxins
Polychlorinated dibenzofurans
Polychlorinated biphenyls
Estimated daily intakes (EDI)
Human breast milk

ABSTRACT

Sixty breast milk samples were collected in Shenzhen, China from July to November in 2007. The samples were analyzed of the concentrations of polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs). The range of upper-bound for ∑TEQ-(PCDD/Fs + PCBs) in the samples was 4.10–35.3 pg TEQ g^{-1} lipid (median: 10.6 pg TEQ g^{-1} lipid; mean: 11.9 pg TEQ g^{-1} lipid). The levels of the measured contaminants in the breast milk had significant correlations with the length of inhabitation period in Shenzhen ($r = 0.487$, $p < 0.05$ for PCDD/Fs, $r = 0.431$, $p < 0.05$ for PCBs and $r = 0.478$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)), and the consumption rate of fish ($r = 0.366$, $p < 0.05$ for PCDD/Fs, $r = 0.486$, $p < 0.05$ for PCBs and $r = 0.416$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)), respectively. Moreover, significant positive correlations were also detected between the participant's age ($r = 0.305$, $p < 0.05$ for ∑TEQ-PCBs and $r = 0.275$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)) and the body burdens of these contaminants respectively. It is estimated that the daily intake (EDI) of the sum of PCDD/Fs and DL-PCBs by the breast-fed infants was 5.60–161 pg TEQ kg^{-1} bw per day (mean: 48.2 pg TEQ kg^{-1} bw per day; median: 42.2 pg TEQ kg^{-1} bw per day). The result showed that both the body burdens of PCDD/Fs and PCBs of the recruit population and the calculated EDI of the breast-fed infants were higher than those in the non-exposed areas in mainland China. This suggests that continuous surveillance on PCDD/Fs and PCBs levels in human milk is critical to more precisely evaluate the human health risk posed by the negative environmental impact in Shenzhen in the future.

1. Introduction

Persistent organic pollutants (POPs), such as polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs) and polychlorinated biphenyls (PCBs), are a family of lipophilic stable toxic compounds that occur widely in the environment. Humans, due to their unique position on the food chains, may be exposed to higher levels of environmental pollutants through intake of contaminated food. Because of the ubiquitous occurrence, long-range transportation, bioaccumulation and metabolic persistence, and potent toxic effects of some of the congeners (including immunological, neurological, reproductive, carcinogenic effects, endocrine-disruption effects (Bláha et al., 2006), and thyroid disorders (Wang et al., 2005; Turyk et al., 2007)), they are classified into controlled and eliminated POPs. Since 1980s, many countries have banned the usage of some types of POPs such as PCBs, and have been monitoring levels and changing trend of PCDD/Fs and PCBs in organisms and environment regularly in order to control and eliminate their pollution (Hannu et al., 1999; Fürst, 2006).

Human milk reflects the body burden of dioxin and it is the main way of transporting dioxin from mothers to infants (Malisch and van Leeuwen, 2003). Moreover, collection of human milk is convenient and noninvasive and is easily replicated. Since 1987, the World Health Organization (WHO) has coordinated four rounds of exposure studies on dioxins using human milk (Malisch and Moy, 2006). China participated in the fourth WHO coordinated global survey of POPs pollution levels in breast milk in 2007. The survey included 12 representative areas in the mainland China, but unfortunately the city of Shenzhen was not included (Li et al., 2009). Shenzhen is a special economic zone in the Pearl River Delta Area of Guangdong, China, possessing around 2000 km^2 of area in the vicinity of Hong Kong. The population is about 12 million, and the residents are mostly emigrants from the other areas of mainland China and obviously have diversified diet habits.

Although Shenzhen is near to Hong Kong SAR which participated in the 3rd round of the WHO study initiated in 2000 (Hedley et al., 2006), no data have been reported on the levels of PCDD/Fs and PCBs in human breast milk in this city. In the present study, 60 milk

* Corresponding author.
E-mail address: zhjianqing95@gmail.com (J. Zhang).

0160-4120/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.envint.2011.03.022

samples from the mothers living in Shenzhen were collected, and the levels of PCDD/Fs and PCBs in these samples were firstly examined by the isotopic dilution HRGC/HRMS method. Furthermore, the exposure risk and intake for breast-fed infants were also estimated. Thus, the major objective of this study is to investigate the body burdens of dioxins of Shenzhen maternal and carry out a health risk assessment for breast-fed infants.

2. Materials and methods

2.1. Chemicals and reagents

US EPA Method 1613 and 1668 standard solutions for determining PCDD/F and PCB congeners (CS1 to CS5, window defining and isomer specificity, labeled compound Stock solution (IS), clean up standard, internal standard spiking solution (ISS)) were purchased from Cambridge Isotope Laboratories Co. (Andover, MA, USA). Organic solvents for trace residual analysis (acetone, n-hexane, dichloromethane, ethyl acetate, benzene, methanol, and toluene) were purchased from Merck (Darmstadt, Germany).

2.2. Donor selection and sample collection

The approach for participator selection and sample collection was based on the 'Guidelines for Developing a National Protocol' of the Fourth WHO-Coordinated Survey of Human milk for Persistent Organic Pollutants in Cooperation with UNEP (WHO, 2007). Some modifications were made for the special situation of Shenzhen. The participators (n = 60) were vaginal delivery primiparas aging from 20 to 34 years old (average: 28 years) and have lived in Shenzhen non-directly POPs polluted areas for 5–28 years until the sampling (average: 10 years). Personal questionnaire data were collected, including information on birth weight and length of the infants, age of participators, length of inhabitation period in Shenzhen, residence environment record, dietary habits, and consumption of animal origin food before pregnancy, including aquatic food, meat, egg and milk. Information on smoking habit and indoor usage of DDT were also obtained for all the participators. The sampling was conducted during July–November, 2007. About 50–100 ml breast milk sample was self-collected by each participator after delivery 3 weeks to 2 months. Each sample (from one participator) was stored in one pre-washed collecting jar at −20 °C right after the samplings until chemical analyses.

The Human Ethical Committee of Shenzhen Center for Disease Control & Prevention inspected, reviewed and approved the study protocol. Each of the participants was provided informed consent form after receiving a detailed explanation of the study and its potential consequences.

2.3. Sample preparation

The samples were freeze-dried and mixed well individually. After spiking with ^{13}C-labeled internal standards, about 10 g for each sample were extracted individually by ASE system (Accelerate solvent extractor equipment, ASE300, USA) with a mixture reagent of n-hexane and dichloromethane (1:1) for 10 min × 2 times under pressure (2000 psi) and temperature (150 °C). Gravimetric lipid determination was performed after solvent evaporation. The concentrated sample extract was cleaned by a acid-modified silica column before subjecting it to carbon column fractionation by an automated system (Fluid Management Systems, Waltham, MA, USA). The fraction containing PCDD/Fs, PCBs congeners was then concentrated by vacuum evaporation and further cleaned by an alumina column. The eluate was dried by nitrogen evaporation to nearly dryness, and the residue was further reconstituted in 20μL of ^{13}C-labeled injection standard in nonane waiting for HRGC/HRMS analysis.

2.4. Sample analysis

Concentrations of seven 2,3,7,8-substituted PCDDs, ten 2,3,7,8-substituted PCDFs, and the six indicator PCB (non dioxin-like PCBs) congeners (No. 28, 52, 101, 138, 153 and 180), the coplanar PCB congeners (No. 77, 81, 126 and 169), as well as the mono-ortho PCB congeners (IUPAC No. 105, 114, 118, 123, 156, 157, 167, 189) were analyzed by a high-resolution gas chromatography/high-resolution mass spectrometry (HRGC/HRMS) (MAT95XL Thermo Finnigan, Germany) with a DB-5MS capillary column (60 m × 0.25mmi. d. × 0.25 μm). Further details of the experimental procedure were presented elsewhere (Zhang et al., 2008).

The minimum detection limit (DL) and the minimum quantification limit (QL) were determined. And each compound in the standard solution for calibration curve of the minimum concentration was quantified 5 times, and then the standard deviation (SD) was calculated. Threefold of the SD was taken as DL for the instrument (IDL) and tenfold of the SD was taken as QL for the instrument (IQL). Then, blank test carried out 5 times, each compound was quantified and then the SD was calculated. Threefold of the SD was taken as DL for the method (MDL) and tenfold of the SD was taken as QL for the method (MQL). For some congeners, system blank level was subtracted in order to get MDL, then the sample detect limit and quantification limit were calculated from MDL and MQL, respectively.

2.5. Data report and estimation PCDD/Fs and PCBs intake for breast-fed infants

Toxic equivalents (TEQ) of PCDD/F, PCBs were calculated on the basis of the Toxic Equivalency Factor (TEF) published by the World Health Organization (WHO) in 2005 (Van den Berg et al., 2006). In case the concentrations of some congeners in the samples were not detectable, the MDL (Method Detect Limit) values were used as the values of the concentrations of the congeners respectively.

Estimation of the intake of PCDD/Fs, PCBs for breast-fed infants employed the upper–bound of TEQ concentration. The calculation was based on the assumption published previously (Chan et al., 2007), that infant's daily milk consumption was 700 ml and infant weight was 5 kg. Therefore, the calculation formula was:

$$\text{PCDD / Fs and PCBs intake of infants}\left(\text{pg kg}^{-1}\cdot\text{bw}\cdot\text{day}\right) = \frac{700}{5} * F * C$$

whereas F: concentration of fat in milk, %; C: concentrate of PCDD/Fs and PCBs in milk, pg/g fat.

2.6. QA/QC

Method blank and quality control samples were included with each batch of 12 samples. Certified Reference Material was served as the quality control sample to validate the long determination process. Chicken samples, purchased from the Norwegian Institute of Public Health, were measured to confirm the laboratory performance and the method validation. The laboratory is accredited to ISO/IEC 17025 by CNACL of China (No. L2154). And our laboratory regularly and successfully participated in international interlaboratory comparison study on PCDD/Fs and PCBs in human milk, beef, butter, egg yolk and herring, organized by the Norwegian Institute of Public Health since 2005.

2.7. Statistical analysis

All data analyses were performed using SAS9.1 (SAS Institute Inc., Cary, NC). Normality test (Shapiro–Wilks test) was executed for all the continuous variables. The concentration of $\sum$TEQ-(PCDD/Fs + PCBs) was beyond normal distribution, thus, Spearman correlation analysis

was used for correlation analysis. All p-values were based on two-tailed ANOVA tests, whereas the level of $p<0.05$ was considered as statistically significant.

3. Results

3.1. Levels and profiles of PCDD/Fs and PCBs in the breast milk

The mass concentrations and upper-bound TEQ values in the human milk samples were shown in Table 1. The average fat content in the milk samples was 3.02%. The upper-bound of ∑TEQ-(PCDD/Fs + PCBs) in the samples ranged between 4.10 and 35.3 pg TEQ g^{-1}lipid (median: 10.6 pg TEQ g^{-1} lipid, mean: 11.9 pg TEQ g^{-1} lipid). Among the concentration of ∑TEQ-(PCDD/Fs + PCBs), the total upper-bound TEQ of ∑TEQ-(PCDD/Fs) was 1.72–24.6 pg TEQ g^{-1} lipid (median: 6.34 pg TEQ g^{-1} lipid; mean: 7.16 pg TEQ g^{-1} lipid), and that of DL-PCBs was 1.95–10.7 pg TEQ g^{-1} lipid (median: 4.35 pg TEQ g^{-1} lipid; mean: 4.77 pg TEQ g^{-1} lipid). The results showed that the levels for PCDD/Fs, and PCBs were largely different from sample to sample.

PCDDs and PCDFs were detected in all samples (Table 1). Particularly, OCDD, 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF, 1,2,3,4,7,8-HxCDF, 1,2,3,6,7,8-HxCDF, 1,2,3,4,6,7,8-HpCDD were found in all samples. By mass concentrations, OCDD was the predominant congener in all samples, accounting for more than 68.3% of the total PCDD/Fs, followed by 2,3,4,7,8-PeCDF (6.89%), 1,2,3,4,6,7,8- HpCDD (5.63%) and 1,2,3,4,7,8-HxCDF (2.97%). By the TEQ values, 1,2,3,7,8-PeCDD (34.6%), 2,3,4,7,8-PeCDF (30.9%) and 2,3,7,8-TCDD (13.7%) were the dominant contributors. The most toxic congener, 2,3,7,8-TCDD, was detected in all except for three samples, with the concentration ranged from 0.0370 to 3.58 pg/g^{-1} lipid.

Six indicator PCB congeners (No. 28, 52, 101, 138, 153 and 180) and 12 DL-PCBs (No. 77, 81, 126, 169, 105, 114, 118, 123, 156, 157, 167, 189) were detected in all samples (Table 1). The concentrations of 6 indicator PCBs ranged between 3.40 and 39.2 ng g^{-1} lipid (median: 13.2 ng g^{-1} lipid; mean: 14.5 ng g^{-1} lipid). By mass concentration, PCB118 was the dominate congener, followed by PCB105, PCB156 and PCB167, which contributed to 54.2%, 16.6%, 14.9% and 4.71% of the total DL-PCBs respectively. PCB153, PCB138, PCB28 and PCB180 were the major indicator PCBs, which contributed to 33.8%, 26.7%, 21.1% and 8.87% of ∑indicator PCB. By the TEQ concentrations, PCB126 was the dominant congener, followed by PCB118, PCB169 and PCB105, and they accounted for 58.4%, 17.4%, 9.87% and 5.32% of the ∑TEQ-PCBs respectively.

Regarding the contamination profiles of ∑(PCDD/Fs + PCBs), we found that PCB126, 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF, and 2,3,7,8-TCDD were the dominant congeners, accounting for 23.3%, 20.8%, 18.6% and 8.22% of ∑TEQ-(PCDD/Fs + PCBs) respectively.

3.2. Exposure level of PCDD/Fs and PCBs for the breast-fed infants

In order to evaluate the infants' exposure level, the average daily intake value of PCDD/Fs and PCBs by infants was e calculated. The mean estimated daily intake value of PCDD/Fs and PCBs of breast-fed infants in Shenzhen was 48.2 pg TEQ kg^{-1} bw per day (median: 42.2 pg TEQ kg^{-1} bw per day; range: 5.60–161 pg TEQ kg^{-1} bw per day).

4. Discussion

4.1. Levels of PCDD/Fs and PCBs in the breast milk

Table 2 showed recent reported data on levels of PCDD/Fs and PCBs in human breast milk in the other places. In the present study, we found

Table 1
The mass concentrations and TEQ of PCDD/Fs and PCBs in human breast milk samples in Shenzhen (pg g^{-1} lipid).

Congeners	Mass concentration (pg/g fat)[a]					TEQ (pg/g fat)[a]				
	Mean	Median	Min	Max	Constituent ratio (%)[b]	Mean	Median	Min	max	Constituent ratio (%)[b]
2,3,7,8-TCDF	1.47	1.41	0.264	2.80	0.0282	0.147	0.141	0.0264	0.280	1.23
1,2,3,7,8-PeCDF	1.01	0.897	0.0603	3.12	0.0194	0.0303	0.0269	0.00181	0.0937	0.254
2,3,4,7,8-PeCDF	7.38	7.26	3.10	21.96	0.142	2.21	2.18	0.931	6.59	18.6
1,2,3,4,7,8-HxCDF	3.18	2.75	0.839	8.60	0.0612	0.318	0.275	0.0839	0.860	2.67
1,2,3,6,7,8-HxCDF	2.70	2.48	0.672	7.21	0.0518	0.270	0.248	0.0672	0.721	2.26
2,3,4,6,7,8-HxCDF	1.24	1.17	0.100	3.41	0.0238	0.124	0.117	0.0100	0.341	1.04
1,2,3,7,8,9-HxCDF	0.116	0.0751	0.0290	1.09	0.00223	0.0116	0.00751	0.00290	0.109	0.0971
1,2,3,4,6,7,8-HpCDF	1.92	1.69	0.139	7.45	0.0369	0.0192	0.0169	0.00139	0.0745	0.161
1,2,3,4,7,8,9-HpCDF	0.217	0.164	0.0377	1.00	0.00418	0.00217	0.00164	0.0004	0.0100	0.0182
OCDF	0.410	0.176	0.0196	6.00	0.00787	0.000123	0.0000529	0.00001	0.00180	0.00103
2,3,7,8-TCDD	0.981	0.882	0.0370	3.58	0.0189	0.981	0.882	0.0370	3.58	8.22
1,2,3,7,8-PeCDD	2.48	2.09	0.168	10.61	0.0476	2.48	2.09	0.168	10.61	20.8
1,2,3,4,7,8-HxCDD	1.27	0.945	0.0558	4.90	0.0243	0.127	0.0945	0.00558	0.490	1.062
1,2,3,6,7,8-HxCDD	2.72	2.41	0.0978	8.32	0.0522	0.272	0.241	0.00978	0.832	2.28
1,2,3,7,8,9-HxCDD	0.869	0.627	0.0542	3.52	0.0167	0.0869	0.0627	0.00542	0.352	0.729
1,2,3,4,6,7,8-HpCDD	6.04	4.70	1.26	41.6	0.116	0.0604	0.0470	0.0126	0.416	0.506
OCDD	73.2	54.3	14	595	1.41	0.0220	0.0163	0.00430	0.179	0.184
∑PCDFs	19.6	18.2	7.36	47.4	0.377	3.14	3.02	1.34	8.70	26.3
∑PCDDs	87.6	67.4	17.0	646	1.68	4.03	3.45	0.26	15.9	33.8
∑(PCDDs + PCDFs)	107.	84.2	27.0	670	2.06	7.16	6.34	1.72	24.6	60.1
PCB 77	15.2	14.0	0.08	62.6	0.292	0.00152	0.00140	0.00000801	0.00626	0.0127
PCB 81	6.32	6.00	0.40	13.6	0.121	0.00189	0.00180	0.000121	0.00408	0.0159
PCB 105	844	795	353	2371	16.2	0.253	0.238	0.106	0.711	2.12
PCB 114	143	127	53.4	391	2.75	0.0429	0.0381	0.0160	0.117	0.360
PCB 118	2764	2474	1081	7350	53.1	0.829	0.742	0.324	2.20	6.95
PCB 123	47.2	43.2	17.8	111	0.907	0.0141	0.0130	0.00535	0.0327	0.119
PCB 126	27.8	24.9	11.9	67.4	0.534	2.78	2.49	1.19	6.74	23.3
PCB 156	759	588	164	2373	14.6	0.228	0.176	0.049	0.71	1.91
PCB 157	176	146	41.7	540	3.38	0.0527	0.0438	0.013	0.162	0.442
PCB 167	240	200	56.4	654	4.62	0.0721	0.0599	0.017	0.196	0.604
PCB 169	15.7	13.0	5.77	45.1	0.302	0.470	0.391	0.173	1.28	3.94
PCB 189	58.2	45.0	10.4	275	1.12	0.0175	0.0135	0.00313	0.0826	0.146
∑DL-PCBs	5097	4580	1964	13967	97.9	4.77	4.35	1.95	10.7	39.9
∑(PCDD/Fs + PCBs)	5204	4671	1995	14111	100	11.9	10.6	4.10	35.3	100
PCB[c] 28	3.07	2.81	5.83E−5	12.9	21.1	–	–	–	–	–
PCB[c] 52	0.386	0.263	4.46 E−5	3.59	2.66	–	–	–	–	–
PCB[c] 101	0.990	0.496	1.39 E−4	7.94	6.83	–	–	–	–	–
PCB[c] 138	3.87	3.05	1.19	12.0	26.7	–	–	–	–	–
PCB[c] 153	4.91	3.66	0.502	17.7	33.8	–	–	–	–	–
PCB[c] 180	1.29	0.883	0.155	10.1	8.87	–	–	–	–	–
∑indicate-PCBs[c]	14.5	13.2	3.40	39.2	100	–	–	–	–	–

[a] For the concentrations below MDL values, concentrations were set as MDL.
[b] Constituent ratio (%) means contributing to ∑TEQ-(PCDD/Fs + PCBs) for each PCDD/F and DL-PCB congener, to ∑ indicator PCBs for indicator PCB compound.
[c] ng/g lipid.

that the median level of PCDD/Fs in Shenzhen (6.34 pg/g lipid) was obviously higher than 12 representative areas of China (3.73 pg/g lipid) (Li et al., 2009) and some non-exposed areas in mainland China such as Hebei (3.57 pg/g lipid) (Sun et al., 2006), Yantai (3.37 pg/g lipid), Shijiazhuang (4.11 pg/g lipid) and Tianjin (5.03 pg/g lipid) (Sun et al., 2010). The level was also slightly higher than those surveyed in Shanghai (5.82 pg/g lipid in rural areas; 5.68 pg/g lipid in urban areas) (Li et al., 2009). As unexpected, human milk in Shenzhen City had the highest TEQ levels of PCDDs, PCDFs, and CoPCBs of all the mainland China listed. Compared with the data from other countries, the level was much higher than those of Brazil (3.90 pg/g lipid), Fiji (3.34 pg/g lipid), Philippines (3.94 pg/g lipid) (Malisch and van Leeuwen, 2003). Moreover, the level was similar to those detected in Australia (5.57 pg/g lipid), Bulgaria (6.14 pg/g lipid), Croatia (6.4 pg/g lipid), Hungary (6.79 pg/g lipid), New Zealand (6.86 pg/g lipid), but slightly lower than the levels detected in USA (7.18 pg/g lipid), Hong Kong (8.69 pg/g lipid) (Malisch and van Leeuwen, 2003), Turkey (7.50 pg/g lipid), (Çok et al., 2009), and Central Taiwan (7.4 pg/g lipid), (Chao et al., 2005), which were reported at the third round of the WHO coordinated exposure study. However, the median level in Shenzhen was much lower than those in Angarsk and Usolye–Sibirskoye of Russian (16.3 and 28.5 pg/g lipid) (Schecter et al., 2002), Egypt (22.3 pg/g lipid), The Netherlands(18.3 pg/g lipid), Belgian (16.9 pg/g lipid) (Malisch and van Leeuwen, 2003), Saitama and Tokyo of Japan (15.6 and 16.9 pg/g lipid), (Takekuma et al., 2004; Sun et al., 2006), Luxembourg (15.0 pg/g lipid) (Malisch and van Leeuwen, 2003), Southern Taiwan (14.7 pg/g lipid) (Hsu et al., 2007), Germany (12.5 pg/g lipid), Spain (11.6 pg/g lipid), Ukraine (10.0 pg/g lipid), and Russia (9.36 pg/g lipid), (Malisch and van Leeuwen, 2003), respectively.

DL-PCBs were a group of compounds which have similar toxicity as PCDD/Fs. In this study, we found that the median level of Σ DL-PCBs in breast milk of Shenzhen (4.35 pg/g lipid) was higher than some other places in China (1.69–2.63 pg/g lipid) (Li et al., 2009; Sun et al., 2006, 2010). The level was also higher than those reported in Australia, Brazil, Fiji, Hungary, and Philippines (Malisch and van Leeuwen, 2003), while it was similar to Bulgaria, Hong Kong SAR, Ireland, New Zealand, USA (Malisch and van Leeuwen, 2003) and Turkey (Çok et al., 2009).

It is noted that the levels of PCDD/Fs in Shenzhen were similar to those in Shanghai and Hong Kong. It may be due to the similar geographical environment and economic level of these three cities. The diet habits may cause more chances of exposure. The comparison also revealed that among these countries, different levels of industrialization might have caused the difference in the levels of these contaminants.

4.2. Profiles of PCDD/Fs and PCBs in the breast milk

Regarding the contamination profiles, the distributions or accumulation patterns of these contaminants we observed are consistent with

Table 2
Summary the recent 10 years studies on PCDD/Fs and PCBs in human breast milk from worldwide areas (pg WHO-TEQ g^{-1}lipid).

Country	Pools	Year of sampling	ΣPCDD/Fs	ΣPCBs	Reference
Australia	2	2001–2003	5.57	2.89	Malisch and van Leeuwen (2003)
Belgium	2	2001–2003	16.9	12.6	Malisch and van Leeuwen (2003)
Brazil	11	2001–2003	3.9	1.77	Malisch and van Leeuwen (2003)
Bulgaria	3	2001–2003	6.14	4.21	Malisch and van Leeuwen (2003)
China.12 representative areas*	24	2007	3.73	1.69	Li et al. (2009)
China. Hebei	41[a]	2002–2003	3.57	1.88	Malisch and van Leeuwen (2003)
China.Shenzhen[b]	60[a]	2007–2009	6.34	4.35	Present study
China.Shijiazhuang[b]	20[a]	2006–2007	4.11	2.14	Sun et al. (2010)
China. Tianjin[b]	20[a]	2006–2007	5.03	2.45	Sun et al. (2010)
China. Yantai[b]	20[a]	2006–2007	3.37	2.63	Sun et al. (2010)
Croatia	2	2001–2003	6.40	7.17	Malisch and van Leeuwen (2003)
Czech Republic	3	2001–2003	7.78	15.2	Malisch and van Leeuwen (2003)
Egypt	9	2001–2003	22.3	5.48	Malisch and van Leeuwen (2003)
Fiji	2	2001–2003	3.34	1.75	Malisch and van Leeuwen (2003)
Finland	2	2001–2003	9.44	5.85	Malisch and van Leeuwen (2003)
Germany	4	2001–2003	12.5	13.7	Malisch and van Leeuwen (2003).
Germany. Duisburg	169[a]	2000–2003	13.3	13.0	Wittsiepe et al. (2007)
Germany Bavarian*	43[a]	2005	9.91	9.92	Raab et al. (2007)
HongKong SAR	11	2001–2003	8.69	4.73	Malisch and van Leeuwen (2003)
Hungary	3	2001–2003	6.79	2.87	Malisch and van Leeuwen (2003)
Ireland	4	2001–2003	7.72	4.57	Malisch and van Leeuwen (2003)
Italy	4	2001–2003	12.7	16.3	Malisch and van Leeuwen (2003)
Japan. Saitama	299[a]	1998–2000	15.6	6.45	Takekuma et al. (2004)
Japan. Tokyo	20[a]	2002	16.9	6.48	Sun et al. (2006)
Luxembourg	2	2001–2003	15.0	13.7	Malisch and van Leeuwen (2003)
New Zealand	3	2001–2003	6.86	3.92	Malisch and van Leeuwen (2003)
Norway	2	2001–2003	7.30	8.08	Malisch and van Leeuwen (2003)
Philippines	2	2001–2003	3.94	2.38	Malisch and van Leeuwen (2003)
Romania	3	2001–2003	8.86	8.06	Malisch and van Leeuwen (2003)
Russia	7	2001–2003	9.36	13.5	Malisch and van Leeuwen (2003)
Russian. Usolye-Sibirskoye*	11[a]	1998	28.5	–	Schecter et al. (2002)
Russian. Angarsk *	7[a]	1998	16.3	10.6	Schecter et al. (2002)
Slovak Republic	4	2001–2003	9.07	12.6	Malisch and van Leeuwen (2003)
Spain	6	2001–2003	11.6	9.42	Malisch and van Leeuwen (2003).
Spain. Madrid*	11[a]	2004	7.77	3.13	Bordajandi et al. (2008)
Sweden	1	2001–2003	9.58	9.71	Malisch and van Leeuwen (2003)
Taiwan. Central *	30[a]	2000–2001	7.4	–	Chao et al. (2005)
Taiwan. Southern *	37[a]	2000–2001	14.7	–	Hsu et al. (2007)
The Netherlands	3	2000	18.3	11.6	Malisch and van Leeuwen (2003)
Turkey*	51[a]	2006–2007	7.5	3.1	Çok et al. (2009)
Ukraine	3	2001–2003	10.0	20.0	Malisch and van Leeuwen (2003)
USA	2	2001–2003	7.18	4.61	Malisch and van Leeuwen (2003)

– not detected;[a] detected as individual samples; [b] based on 2005-WHO-TEF;* mean concentration, the others were median concentration.

those reported studies previously. For example, Wittsiepe et al. (2007) reported that 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF, PCB126 were the dominate congeners of ∑TEQ-(PCDD/Fs + PCBs) in German. Focant et al. (2002) reported that 2,3,7,8-TCDD, 1,2,3,7,8-PeCDD, 2,3,4,7,8-PeCDF and PCB-126 were found accounting for more than 90% of the ∑TEQ-(PCDD/Fs + PCBs) in Belgium. Furthermore, our finding was also in consistent with that of Li et al. (2009) with the investigation conducted in 12 representative areas of China, that 2,3,4,7,8-PeCDF, 1,2,3,7,8-PeCDD, PCB126 and 2,3,7,8-TCDD accounted for 29.1%, 23.4%, 20.8% and 9.2% of ∑TEQ-(PCDD/Fs + PCBs), respectively. Overall, the comparison of the profiles suggested that the contamination sources in these non-exposure areas were similar.

4.3. Estimated daily intakes of infants

The mean estimated daily intake (EDI) of PCDD/Fs and PCBs for breast-fed infants in the present study was 48.2 pg TEQ kg^{-1} bw per day (28.9 pg TEQ kg^{-1} bw per day for PCDD/Fs and 19.3 pg TEQ kg^{-1} bw per day for PCBs respectively). PCDD/Fs contributed up to 60% to the total EDI. It shows that the EDI values of Shenzhen were 2 times higher than those of the 12 representative areas of China reported by Li et al. (2009) (mean: 23.7 pg TEQ kg^{-1} bw per day; range: 11.8–42.2 pg TEQ kg^{-1} bw per day; median: 22.6 pg TEQ kg^{-1} bw per day) for the intake of ∑TEQ-(PCDD/Fs + PCBs). It was also higher than those of infants in central Taiwan (Chao et al., 2005) and southern Taiwan (Hsu et al., 2007), which were 10.5 pg TEQ kg^{-1} bw per day for PCDD/Fs in central Taiwan and 13 pg TEQ kg^{-1} bw per day for PCDD/Fs for 12-months breast-fed infants in southern Taiwan. The value of EDI was similar to that in Spain (Bordajandi et al., 2008), which was 35.4 pg TEQ kg^{-1} bw per day for PCDD/Fs and 49.6 pg TEQ kg^{-1} bw per day for ∑ (PCDD/Fs + PCBs). In contract, the EDI of Shenzhen was much lower than those in Belgium (Focant et al., 2002) and Korea (Yang et al., 2002), which were 76 pg TEQ kg^{-1} bw per day for PCDD/Fs and 103 pg TEQ kg^{-1} bw per day for ∑TEQ-(PCDD/Fs + PCBs) in Belgian, and 60 pg TEQ kg^{-1} bw per day for PCDD/Fs in Korea. The industrial development in Belgium and Korea might have posed a negative influence on the contaminated state of these countries (Focant et al., 2002; Yang et al., 2002). The estimation of EDIs of PCDD/Fs and PCBs in the other countries employed the 1998-WHO-TEF, which is different from the evaluations in this study, which was based on the 2005-WHO-TEF. Therefore, a relevant comparison is difficult here.

4.4. Correlation analysis between PCDD/Fs, PCBs levels in breast milk and the analyzed factors

The results of the correlation analyses were presented in Table 3. The body burden of PCDD/Fs or PCBs in mother (mass concentrations and TEQ values) were significantly positively correlated with the length of inhabitation period in Shenzhen ($r = 0.487$, $p < 0.05$ for PCDD/Fs, $r = 0.431$, $p < 0.05$ for PCBs and $r = 0.478$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)), the consumption of fish ($r = 0.366$, $p < 0.05$ for PCDD/Fs, $r = 0.486$, $p < 0.05$ for PCBs and $r = 0.416$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)). The results were consistent with many other studies, such as Li, Fattore, Hedley and Uemure's recent studies (Fattore et al., 2006; Hedley et al., 2006; Li et al., 2009; Uemura et al., 2008). Furthermore, in our study, significant positive correlations were found between the mother's age and the body burden of DL-PCBs and ∑(PCDD/Fs + PCBs) ($r = 0.305$, $p < 0.05$ for ∑TEQ-PCBs and $r = 0.275$, $p < 0.05$ for ∑TEQ-(PCDD/Fs + PCBs)), while no such correlation was detected between mother's age and the ∑TEQ-PCDD/Fs ($r = 0.251$, $p > 0.05$). This result was different from other previous studies (Harden et al., 2007; Hedley et al., 2006; Chao et al., 2005), in which both of the levels of PCDD/Fs and PCBs had correlation with the mother's age. However, our result was similar to Nakatani's study (2005) from Osaka City of Japan, in which only TEQ values of CoPCBs in human milk was found to correlate with the increasing age of mothers, and there weren't correlation between PCDD/Fs and mother's age. It is well known that Shenzhen is a immigrate city, people especially young working aged people migrate here from inland city, and local residents like to eat diversified aquatic food especial fish since a large quantity fish teem in this coast city. Moreover, the PCDD/Fs and PCBs possess different source, such as PCBs derive from the past use or disposal of industrial PCB products (Sakai et al., 2001; Alcock et al., 1998), but municipal solid-waste and industrial-waste incinerators are known as the main sources of PCDD/Fs pollutants. Together with the comprehensive correlation analysis data, the length of residence year in Shenzhen and fish consumption were more pronounced than age, particularly, PCDD/Fs. Body burden accumulation in the donors may different since PCDD/Fs bioaccumulative level discrepancy of environment pollution in different inland city before they settle down Shenzhen. Thus, this point is maybe an important mixture factor in this study. Furthermore, a large scale sample and deep follow study should be conducted in the future.

Among the recent studies, significant correlations were be found between the consumption of aquatic food, meat and TEQ level in human milk which was conducted by Chinese CDC, (Li et al., 2009), but no correlations were found between TEQ level and other food concerned. In Japan, it was noted that there were also no such correlations between levels of ∑TEQ-(PCDD/Fs + PCBs) and index of consumption of meat or eggs (Takekuma et al., 2004). In the present study, no correlations were found between TEQ level and other food concerned except for fish. One reason is that the data on consumption of food were derived from the diet history prior pregnancy of the mothers. The mothers normally may change their dietary habits after they got pregnant. Therefore this may lead to an inaccurate estimation. In additional, no significant correlations were detected for the body burden of PCDD/Fs or PCBs in mother with the other parameters including infant's birth weight and length. Nevertheless, a greater sample size is always preferred in our future investigation.

Table 3
Correlation analysis between the contents of PCDD/Fs and PCBs in human milk and the analyzed factors ($n = 60$).

Correction factors	PCDD/Fs		PCBs		PCDD/Fs + PCBs	
	r	*p*	r	*p*	r	*p*
Mother's age	0.251	>0.05	0.305	<0.05	0.275	<0.05
BMI	−0.0880	>0.05	0.0749	>0.05	−0.0220	>0.05
Residence years	0.487	<0.05	0.431	<0.05	0.478	<0.05
Weight of infant	−0.0990	>0.05	0.0160	>0.05	−0.0610	>0.05
Length of infant	0.00845	>0.05	0.0248	>0.05	0.0118	>0.05
Consumption of fish and fish products	0.366	<0.05	0.486	<0.05	0.416	<0.05
Consumption of other aquatic food	0.0770	>0.05	0.209	>0.05	0.135	>0.05
Consumption of milk and dairy products	−0.0980	>0.05	−0.0840	>0.05	−0.110	>0.05
Consumption of meat	0.0893	>0.05	0.159	>0.05	0.114	>0.05
Consumption of egg	0.0149	>0.05	0.0887	>0.05	0.0378	>0.05

5. Conclusion

This was the first study investigating PCDD/Fs and PCBs levels in human breast milk in Shenzhen. The results of this study have provided a baseline level for PCDDs, PCDFs and dioxin-like PCBs in Shenzhen population. Both the TEQ body burden of the recruit population and the estimated EDI of breast-fed infants were higher than those of some non-exposure areas in mainland China reported previously. Though the exposure levels were lower than those of many developed countries, the situation in Shenzhen was more serious than some underdeveloped countries and areas. The lack of the information on the previous exposure levels of PCDD/Fs and PCBs in local residents make it is difficult to estimate the time trend of body burden of PCDD/Fs and DL-PCBs in Shenzhen. Furthermore, monitoring change in the trend of PCDD/Fs and PCBs exposure in local residents in the long run is of significance. This will greatly help local government take steps to control and eliminate these POPs in the future.

Acknowledgement

This research work was funded by the National 863 plan projects, National Public Welfare Item, Bureau of Shenzhen Science & Technology, the body burden of persistent organic pollutant and exposure risk assessment in local residents. We thank all the mothers who participated in this study and donated their milk samples. We also thank all the doctors in referred hospitals who helped us with associating with the donors. Also our sincere thanks are given to Professor Yan Liang who is in Hong Kong Baptist University for her helping in revising the manuscript.

References

Alcock RE, Behnisch PA, Jones KC, Hagenmaier H. Dioxinlike PCBs in the environment-human exposure and the significance of sources. Chemosphere 1998;37:1457–72.
Bláha L, Hilscherová K, Mazurová E, Hecker M, Jones PD, Newsted JL, et al. Alteration of steroidogenesis in H295R cells by organic sediment contaminants and relationships to other endocrine disrupting effects. Environ Int 2006;32:749–57.
Bordajandi LR, Abad E, González MJ. Occurrence of PCBs, PCDD/Fs, PBDEs and DDTs in Spanish breast milk: Enantiomeric fraction of chiral PCBs. Chemosphere 2008;70:567–75.
Chan JK, Xing GH, Xu Y, Liang Y, Chen LX, Wu SC, et al. Body loadings and health risk assessment of polychlorinated dibenzo-p-dioxins and dibenzofurans at an intensive electronic waste recycling site in China. Environ Sci Technol 2007;41:7668–74.
Chao HR, Wang SL, Su PH, Yu HY, Yu ST, Päpke O. Levels of polychlorinated dibenzo-p-dioxins and dibenzofurans in primipara breast milk from Taiwan: estimation of dioxins and furans intake for breastfed infants. Hazard Mater 2005;A121:1-10.
Çok I, Donmez MK, Uner M, Demirkaya E, Henkelmann B, Shen HQ, et al. Polychlorinated dibenzo-p-dioxins, dibenzofurans and polychlorinated biphenyls levels in human breast milk from different regions of Turkey. Chemosphere 2009;76:1563–71.
Fattore E, Fanelli R, Turrini A, di Domenico A. Current dietary exposure to polychlorodibenzo-p-dioxins, polychlorodibenzofurans, and dioxin-like polychlorobiphenyls in Italy. Mol Nutr Food Res 2006;50:915–21.
Focant JF, Pirard C, Thielen C, De Pauw E. Levels and profiles of PCDDs, PCDFs and cPCBs in Belgian breast milk. Estimation of infant intake. Chemosphere 2002;48:763–70.
Fürst P. Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk. Levels, correlations, trends and exposure through breastfeeding. Mol Nutr Food Res 2006;50:922–33.
Hannu K, Raija P, Terttu V. Levels and trends of PCDD/Fs and PCBs in human milk in Finland. Chemosphere 1999;38:311–23.
Harden FA, Toms LML, Paepke O, Ryan JJ, Müller JF. Evaluation of age, gender and regional concentration differences for dioxin-like chemicals in the Australian population. Chemosphere 2007;67:S318–24.
Hedley AJ, Wong TW, Hui LL, Malisch R, Nelson EAS. Breast milk dioxins in Hong Kong and Pearl River Delta. Environ Health Perspect 2006;114:202–8.
Hsu JF, Guo YL, Liu CH, Hu SC, Wang JN, Liao PC. A comparison of PCDD/PCDFs exposure in infants via formula milk or breast milk feeding. Chemosphere 2007;66:311–9.
Li J, Zhang L, Wu Y, Liu Y, Zhou P, Wen S, et al. A national survey of polychlorinated dioxins, furans (PCDD/Fs) and dioxin-like polychlorinated biphenyls (dl-PCBs) in human milk in China. Chemosphere 2009;75:1236–42.
Malisch R, Moy G. Fourth round of WHO-coordinated exposure studies on levels of persistent organic pollutants in human milk. Organohalogen Compd 2006;68:1627–30.
Malisch R, van Leeuwen FXR. Results of the WHO-coordinated exposure study on the levels of PCBs, PCDDs AND PCDFs in human milk. Organohalogen Compd 2003;64:140–3.
Nakatani T, Okazaki K, Ogaki S, Itano K, Fujita T, Kuroda K, et al. Polychlorinated dibenzo-p-dioxins, polychlorinated dibenzofurans, and coplanar polychlorinated biphenyls in human milk in Osaka City, Japan. Arch Environ Contam Toxicol 2005;49(1):131–40.
Raab U, Schwegler U, Preiss U, Albrecht M, Fromme H. Bavarian breast milk survey — pilot study and future developments. Int J Hyg Environ Health 2007;210:341–4.
Sakai S, Hayakawa K, Takatsuki H, Kawakami I. Dioxin-like PCBs released from waste incineration and their deposition flux. Environ Sci Technol 2001;35:3601–7.
Schecter A, Piskac AL, Grosheva EI, Matorova NI, Ryan JJ, Fürst P, et al. Levels of dioxins and dibenzofurans in breast milk of women residing in two cities in the Irkutsk region of Russian Siberia compared with American levels. Chemosphere 2002;47:157–64.
Sun SJ, Zhao JH, Liu HJ, Liu DW, Ma YX, Li L, et al. Dioxin concentration in human milk in Hebei province in China and Tokyo, Japan: potential dietary risk factors and determination of possible sources. Chemosphere 2006;62:1879–88.
Sun SJ, Zhao JH, Leng JH, Wang PY, Wang Y, Fukatsu H, et al. Levels of dioxins and polybrominated diphenyl ethers in human milk from three regions of northern China and potential dietary risk factors. Chemosphere 2010;80:1151–9.
Takekuma M, Saito K, Ogawa M, Matumoto R, Susumu K. Levels of PCDDs, PCDFs and Co-PCBs in human milk in Saitama, Japan, and epidemiological research. Chemosphere 2004;54:127–35.
Turyk ME, Anderson HA, Persky VW. Relationships of thyroid hormones with polychlorinated biphenyls, dioxins, furans, and DDE in adults. Environ Health Perspect 2007;115:1197–203.
Uemura H, Arisawa K, Hiyoshi M, Satoh H, Sumiyoshi Y, Morinaga K, et al. PCDDs/PCDFs and dioxin-like PCBs: recent body burden levels and their determinants among general inhabitants in Japan. Chemosphere 2008;73:30–7.
Van den Berg M, Birnbaum LS, Denison M, De Vito M, Farland W, Feeley M, et al. The 2005 World Health Organization reevaluation of human and mammalian toxic equivalency factors for dioxins and dioxin-like compounds. Toxicol Sci 2006;93:223–41.
Wang SL, Su PH, Jong SB, Guo YL, Chou WL, Päpke O. In utero exposure to dioxins and polychlorinated biphenyls and its relations to thyroid function and growth hormone in newborns. Environ Health Perspect 2005;113:1645–50.
WHO. Fourth WHO-coordinated survey of human milk for persistent organic pollutants in cooperation with UNEP: guidelines for developing a national protocol; 2007.
Wittsiepe J, Fürst P, Schrey P, Lemm F, Kraft M, Eberwein G, et al. PCDD/F and dioxin-like PCB in human blood and milk from German mothers. Chemosphere 2007;67:S286–94.
Yang YH, Chang YS, Kim BH, Shin DC, Ikonomou MG. Congener-distribution patterns and risk assessment of polychlorinated biphenyls, dibenzo-p-dioxins and dibenzofurans in Korean human milk. Chemosphere 2002;47:1087–95.
Zhang JQ, Jiang YS, Zhou J, Fang DK, Jiang J, Liu GH, et al. Concentrations of PCDD/PCDFs and PCBs in retail foods and an assessment of dietary intake for local population of Shenzhen in China. Environ Int 2008;34:799–803.