OCCURRENCE AND ACCUMULATION PROFILES OF ORGANOCHLORINES AND PBDES IN ADIPOSE TISSUES FROM KOREN WOMEN

Moon HB[1], Lee DH[2], Lee YS[2], Choi M[3], Choi HG[3], Kannan K[4]

[1]Hanyang University, Ansan 426-791, Republic of Korea; [2]Kyungpook National University, Daegu 700-422, Republic of Korea; [3]National Fisheries Research and Development Institute, Busan 612-902, Republic of Korea; [4]Wadsworth Center, New York State Department of Health, Albany, NY 12201-0509, USA

Introduction

Environmental contamination by persistent organic pollutants (POPs) is a global concern due to their high toxic potentials to humans and wildlife. Concentrations of organochlorines (OCs), including polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs), polychlorinated biphenyls (PCBs) and organochlorine pesticides (OCPs), are generally decreasing in the environment and in humans over the past few decades[1,2]. Human specimens such as breast milk, serum and adipose tissue have been used in biomonitoring of the extent of human exposure to POPs. Measurement of contaminants in adipose tissue can provide information on steady-state concentrations and integrated levels of accumulation over time. Although the collection of blood and breast milk samples is relatively simple, and less invasive than the collection of adipose tissues, residual levels of lipophilic contaminants in whole blood, sera or plasma may fluctuate with surges in blood lipids. Although human exposure to OCs and polybrominated diphenyl ethers (PBDEs) is of great concern, few studies have examined residue levels in blood of the Korean population[3]. Considering the rapid growth of the electronics market in Korea and the associated increase in demand for brominated flame retardants (BFRs), it is important to investigate exposure of the Korean general population to PBDEs. The objective of this study was to determine the concentrations and accumulation features of OCs and PBDEs in the adipose tissues of Korean women.

Materials and methods

The use of human tissues for this study was approved by the ethics committee of the Kyungpook National University Institutional Review Board. A total of 53 female myoma patients who were undergoing laparoscopy-assisted surgery at Kyungpook National University Hospital agreed to provide adipose tissue samples. Omental fat samples were obtained from the donors between May 2007 and May 2008. All samples were stored at –70^{o}C until analyses. Donor's age, body mass index (BMI) and extractable lipid contents were summarized in Table 1. The age of the subjects ranged from 40 to 68 years (average 47 years) and lipid content of the adipose tissue samples ranged from 63% to 83% (average 73%). Preparation and instrumental analyses of PCDD/Fs, dioxin-like PCBs (DL-PCBs) and non-dioxin-like PCBs, OCPs and PBDEs in adipose tissue samples were performed following the methods described elsewhere[4,5].

Results and discussion

Concentrations of PCDD/Fs, PCBs, OCPs and PBDEs

The concentrations of PCDD/Fs, DL-PCBs, non-DL-PCBs, OCPs and PBDEs in the adipose tissues of Korean women were log-normal distributed (Q-Q test) and were summarized in Table 1. The median residue concentrations (on a lipid weight basis) in adipose tissues were ranked in the order of mono-*ortho* PCBs (9600 pg/g) > PCDDs (90 pg/g) ≥ non-*ortho* PCBs (90 pg/g) > PCDFs (20 pg/g). The concentrations of PCDDs, PCDFs, non-*ortho* PCBs and mono-*ortho* PCBs were in the ranges of 30–530 pg/g, 6.5–50 pg/g, 20–280 pg/g and 2100–34000 pg/g, respectively. The TEQ concentrations of PCDDs, PCDFs, non-*ortho* PCBs and mono-*ortho* PCBs ranged 0.02–11 pg/g, 0.7–11 pg/g, 1.4–19 pg/g and 0.1–1.0 pg/g, respectively. The concentrations of TEQs were similar for PCDD/Fs and DL-PCBs in adipose tissue samples, and were in the range of 2–12 pg/g lipid weight. Overall, 90% of samples contained total TEQ concentrations (sum of PCDD/Fs and DL-PCBs) in the range of 6–20 pg/g lipid weight.

Among OCs, concentrations of PCBs (270 ± 140 ng/g lipid wt) and DDTs (250 ± 210 ng/g lipid wt) were the highest. The concentrations of CHLs (18 ± 14 ng/g lipid wt), HCHs (12 ± 11 ng/g lipid wt), and HCB (8.6 ± 4.7 ng/g lipid wt) were 1–3 orders of magnitude lower than the concentrations of PCBs and DDTs. This pattern is similar to that reported for fish, shellfish, squids and cetaceans from Korean coastal waters[4,5]. Although the

consumption of HCHs is higher than that of DDTs in northeast Asian countries, HCH concentrations were lower than those of DDTs in adipose tissues of Korean women. This is due to higher bioaccumulation potential of DDTs than HCHs and the rapid evaporation of HCHs to the atmosphere after its application[6]. The distribution of concentrations of HCHs in adipose tissues was different from that of other POPs, with majority of samples showing concentrations below 2 ng/g lipid wt.

Most of the adipose tissue samples contained PBDE concentrations in the range of 4–20 ng/g lipid wt. The highest PBDE concentration (149 ng/g lipid wt) was found in sample from a 50-years-old woman; this was considered as an outlier value, in subsequent statistical analysis. The concentrations of PBDEs (16 ± 20 ng/g lipid wt) in adipose tissue samples were similar to those of CHLs and HCHs, but were higher than those of HCB. Although HCB was never used as pesticide in Korea, this compound was found in adipose tissues from Korean women.

Table 1. Sample information and concentrations (ng/g lipid weight) of PCDD/Fs, DL-PCBs, PCBs, OCPs, and PBDEs in the adipose tissues of Korean women

	Mean ± SD	Median	Min – Max
Age (years)	47 ± 5.6	47	40–68
Body mass index (kg/m^2)	24 ± 3.3	23	19–34
Lipid (%)	73 ± 4.9	73	63–83
PCDD/Fs[a]	7.0 ± 4.0	6.2	1.6–22
DL-PCBs[a]	6.0 ± 3.2	5.0	1.4–20
PCBs	270 ± 140	250	40–720
DDTs	250 ± 210	190	32–940
CHLs	18 ± 14	14	1.5–77
HCHs	12 ± 11	11	1.2–35
HCB	8.6 ± 4.7	7.4	1.0–21
PBDEs	16 ± 20	11	5.3–150

[b]pg TEQ/g lipid weight; TEQ concentrations of PCDD/Fs and DL-PCBs were calculated using the WHO-2005 TEF[7].

Accumulation profiles of OCs and PBDEs

The accumulation profiles of PCDD/Fs, DL-PCBs, non-DL-PCBs, OCPs and PBDEs in adipose tissue from Korea are presented in Figure 1. The proportion of PCDDs was higher than the proportion of PCDFs in all samples. OCDD was the dominant homologue group, accounting for, on average, 56% of the total PCDD/F concentrations. The next most dominant homologue groups was penta-CDD/Fs, collectively accounting for 25% of the total PCDD/Fs. Among 2, 3, 7, 8-substituted congeners of PCDD/Fs, OCDD was the greatest contributor (> 70%) to total PCDD/F concentrations. The next major congeners in adipose tissues were 2,3,4,7,8-PeCDF, 1,2,3,4,6,7,8-HpCDD and 1,2,3,6,7,8-HxCDD, and their collective contribution was less than 20% of the total PCDD/F concentrations. PCB 118 was the highest contributor to DL-PCB concentrations in tissue samples, accounting for, on average, 52% of the total DL-PCB concentrations. The other contributors were PCBs 156, 105, 157, and 167, which collectively accounted for over 40% of the total DL-PCB concentrations.

Among OCPs, the major compound was *p,p'*-DDE, which accounted for 80% of the total OCP concentrations. The higher proportion of metabolites such as *p,p'*-DDE, than the parent compound *p,p'*-DDT, suggests historical sources from technical mixtures of DDT used in Korea. Some amounts of *p,p'*-DDT (and *o,p'*-DDT) were detected in adipose tissue samples, which may be associated with exposure through seafood consumption, which is a major DDT exposure route for the general Korean population[8]. Although α-HCH comprises majority of HCH technical mixtures, ranging from 55–80%[9], only β-HCH was detected in adipose tissue samples, due to its higher persistence compared with other HCH isomers. The major CHL compound identified in this study was *trans*-nonachlor, accounting for over 60% of the total CHL concentrations, followed by oxychlordane (30%).

Hexa- and hepta-chlorobiphenyls constituted the greatest proportions (over 75%) of the total PCB concentrations. Pentachlorobiphenyls accounted for a small portion (about 10%) of the total PCBs. For all tissue samples, the major congeners were PCBs 153, 138, 180, 187, 118 and 170, which collectively accounted for 80% of the total PCB concentrations. In our study, BDE 209 (deca-BDE) was a predominant PBDE congener, accounting for 25% ± 15% of the total PBDE concentrations. In addition, more highly brominated congeners such as octa- to nona-BDEs (BDEs 197 and 207) accounted for 35% of the total PBDE concentrations. This result is likely related to the high consumption of deca-BDE technical mixtures in Korea and its on-going release into the environment. Similar to our study, some studies showed the dominance of BDE 209 in human serum, breast milk and adipose tissues from Japan[10] and China[11].

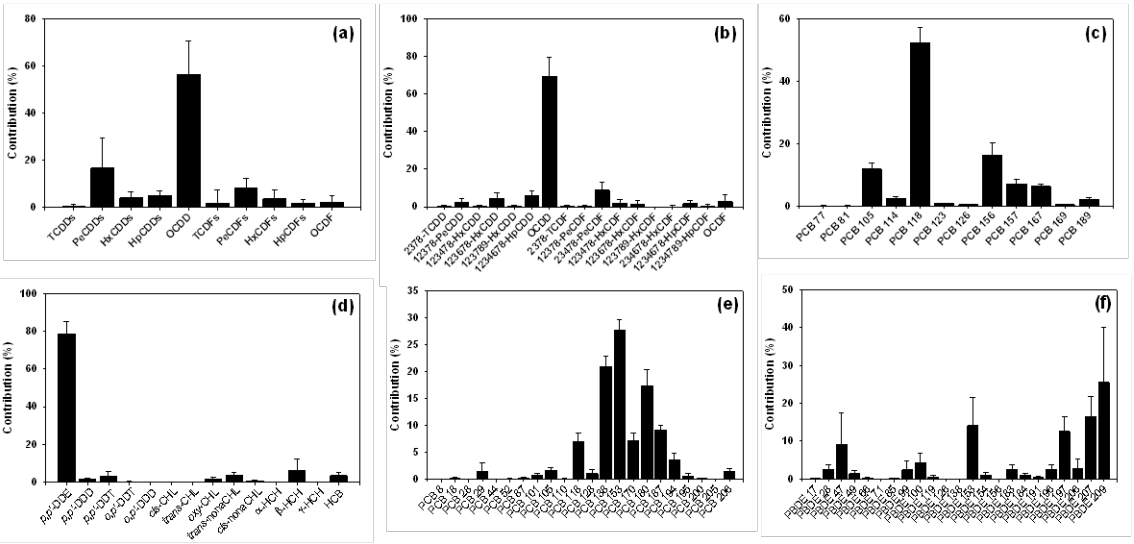

Figure 1. Accumulation profiles of (a) PCDD/F homologues, (b) 2,3,7,8-substituted PCDD/F congeners, (c) DL-PCB congeners, (d) OCP compounds, (e) non-DL-PCB congeners and (f) PBDE congeners in human adipose tissue from Korea. Data were normalized to the total concentrations of each chemical. Error bars represent standard deviations.

Correlations with demographic factors

Previous studies have reported age-dependent accumulations of some legacy POPs in human adipose tissue samples[11,13]. However, in this study no age-dependent accumulation of organochlorines such as PCDD/Fs, PCBs and OCPs were found (Table 2). Similar to OCs, PBDEs were not correlated with subject's age, which is consistent with those reported in previous studies[11,13]. In particular, higher levels of PBDEs were found in the youngest age group (40 years old), suggesting that PBDEs have an additional exposure route to humans, besides food consumption. Johnson-Restrepo and Kannan[14] showed house dust to be the major source of human exposure to PBDEs in the USA. Further studies are needed to assess the magnitude of exposure to PBDEs via dust ingestion by the Korean population.

Relationship between BMI and organohalogen contaminants in adipose tissue was investigated using Spearman's correlation analysis. There were no significant correlations between BMI and the concentrations of PCBs, OCPs, PBDEs and synthetic musk compounds (SMCs), except for HCHs (r = –0.330) and PAHs (r = –0.335). Previous studies showed positive[15] or negative[16] correlations between BMI and PCB levels. Given the range of results found in previous studies, it appears that BMI is not a sole predictor of organohalogen levels in humans.

The concentrations of PCBs, DDTs, CHLs, HCB, and PCDD/Fs (r = 0.402–0.834) were significantly correlated with each other, but not with HCHs. These correlations suggest that the contaminants share a similar exposure routes such as food ingestion and bioaccumulation in human bodies. PBDEs showed low and moderate

correlation with other POPs, indicating that their exposure routes and bioaccumulation potential are different. The concentrations of SMCs had no relationships or negatively correlated with concentrations of POPs, suggesting that musks have different exposure routes, such as dermal exposures[17].

Table 2. Spearman's correlation coefficients among the concentrations of toxic organic contaminants measured in adipose tissues of Korean women

	PCBs	DDTs	CHLs	HCHs	HCB	PBDEs	PCDD/Fs	SMCs	PAHs
DDTs	0.547***								
CHLs	0.688***	0.811***							
HCHs	0.150	–0.289	–0.214						
HCB	0.414**	0.692***	0.834***	–0.439**					
PBDEs	0.234	0.458**	0.345*	–0.133	0.296*				
PCDD/Fs	0.542***	0.425**	0.402**	0.124	0.384**	0.232			
SMCs[a]	–0.116	–0.134	–0.177	–0.045	–0.046	0.127	0.043		
PAHs[b]	0.026	–0.341*	–0.290*	–0.441**	–0.420**	–0.304*	–0.076	–0.188	
BMI	–0.052	0.137	0.230	–0.330*	0.249	0.132	–0.262	0.072	–0.335*
Age	0.259	0.204	0.341	0.083	0.262	–0.079	0.123	–0.232	0.033

* $0.01 < p < 0.05$; ** $0.01 < p < 0.001$; *** $p < 0.001$.

[a]Synthetic musk compounds (SMCs) are sum of HHCB, AHTN and musk-ketone, xylene and moskens.

[b]PAHs are sum of 16 PAHs recommended by the US EPA as priority pollutants.

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References

1. Hagmar L, Wallin E, Vessby B, Jőnsson BAG, Bergman Å, Rylander L. (2006); *Chemosphere.* 64:1507–13
2. Lignell S, Aune M, Darnerud PO, Cnattingius S, Glynn A (2009); *Environ Res*. 109:760–7
3. Kang J-H, Park H, Chang Y-S, Choi J-W. (2008); *Chemosphere*. 73:1625–31
4. Moon HB, Kannan K, Choi HG, An YR, Choi SG, Park JY, Kim ZG. (2010); *Chemosphere.* 79: 733–9
5. Park B-K, Park G-J, An Y-R, Choi H-G, Kim GB, Moon H-B. (2010); *Mar Pollut Bull*. 60:768–74
6. Loganathan BG, Kannan K. (1994); *Ambio.* 23:187–91
7. Van den Berg M, Birnbaum LS, Denison M, de Vito M, Farland W, Feeley M, Fiedler H, Hakansson H, Hanberg A, Haws L, Rose M, Safe S, Schrenk D, Tohyama C, Tritscher A, Tuomisto J, Tysklind M, Walker N, Peterson RE. (2006); *Toxicol Sci.* 93, 223−41
8. Moon H-B, Kim H-S, Choi M, Yu J, Choi H-G. (2009); *Food Chem Toxicol.* 47:1819–25
9. Willett KL, Ulrich EM, Hites RA. (1998); *Environ Sci Technol*. 32:2197–207
10. Thuresson K, Hoglund P, Hagmar L, Sjödin A, Bergman Å, Jakobsson K. (2006); *Environ Health Perspect.* 114:176–81
11. Kunisue T, Takayanagi N, Isobe T, Takahashi S, Nose M, Yamada T, Komori H, Arita N, Ueda N, Tanabe S. (2007); *Environ Int.* 33:1048–56
12. Jin J, Wang Y, Yang C, Hu J, Liu W, Cui J, Tang X. (2009); *Environ Int*. 35:1048–52
13. Johnson-Restrepo B, Kannan K, Rapaport DP, Rodan BD. (2005); *Environ Sci Technol*. 39:5177–82
14. Johnson-Restrepo B, Kannan K (2009); *Chemosphere*. 76:542–8
15. Arrebola JP, Fernazndez MF, Porta M, Rosell J, de la Ossa RM, Olea N, Martin-Olmedo P. (2010); *Environ Int.* 36:705–13
16. Fernandez MF, Kiviranta H, Molina-Molina JM, Laine O, Lopez-Espinosa MJ, Vartiainen T, Olea N. (2008); *Chemosphere*. 71:1196–205
17. Reiner JL, Kannan K. (2006); *Chemosphere*. 67:1887–96.