

# POLYBROMINATED DIPHENYL ETHERS (PBDEs) AND OTHER PERSISTENT ORGANIC POLLUTANTS IN COMPUTER USERS FROM GREECE

Olga I. Kalantzi<sup>1,2</sup>, Adrian Covaci<sup>3</sup>, Tinne Geens<sup>3</sup> & Panayotis A. Siskos<sup>2</sup>

<sup>1</sup> Department of Environment, University of the Aegean, University Hill, 81 100, Mytilene, Greece, e-mail: kalantzi@aegean.gr

<sup>2</sup> Department of Chemistry, Environmental Chemistry Laboratory, National and Kapodistrian University of Athens, Panepistimioupolis Zografou, 157 71, Athens, Greece

<sup>3</sup> Toxicological Center, University of Antwerp, Universiteitsplein 1, 2610 Wilrijk, Belgium

## Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of flame retardants, used in various consumer products such as plastics, textiles and computers to prevent flammable gas formation (WHO, 1995). PBDEs can easily leach out of these materials and volatilise into the environment, ending up in high concentrations in biota and humans. They are lipophilic, have low vapour pressures and bioaccumulate through the food chain (Haglund, 1997). PBDEs were first detected in the environment in Sweden, in 1981 by Andersson and Blomkvist. Since then they have been found in many environmental matrices including humans (de Wit, 2001). The main route of entry of these chemicals into the human body is via the food web, but occupational exposure may also occur in the workplace during handling of flame-retarded goods and inhalation of contaminated indoor air. It has been observed that computer clerks have higher PBDE levels in their blood than control groups (Sjodin et al. 1999), which indicates that PBDEs used in computers and electronics may contaminate the working environment and accumulate in workers.

Over the last two decades there have been indications of increased human PBDE concentrations, although their levels are still generally lower than those of PCBs (de Wit, 2001). This study was undertaken to investigate whether the distribution of PBDEs in human serum of computer workers is different to that of a control population, compare these levels to more “classical” persistent organic pollutants, such as PCBs and OCPs, and explore any possible correlations between lifestyle habits and PBDE levels. This is the first study to our knowledge reporting PBDE levels in human serum from an occupationally-exposed subset of the Greek population.

## Materials and methods

A total of 60 human serum samples of which 34 were males and 26 females (mean age 39 years, age range 20–69 years) were collected from workers of a large computer company in Athens (n=30) working full-time with computers, and from a control population in the Attika region with no computer use (n=30) between June and October 2007. Samples were mixed by inverting the tubes several times immediately after blood collection, and were centrifuged at 3000 rpm for 5 min, to separate the serum from the blood cells. Samples were immediately frozen at –20 °C until analysis. A questionnaire on dietary habits, computer and other electrical and electronic equipment use was completed by the participants to explore any possible correlations between diet, computer use, car use and other lifestyle habits and PBDE levels.

The method for serum analysis was slightly modified from the methods described by Covaci and

Voorspoels (2005) for the determination of PBDEs in serum, to include the determination of PCBs and OCPs. An exactly measured volume of serum (typically 3 ml for most samples) was spiked with internal standards (PCB 143,  $\epsilon$ -HCH, BDE 77, BDE 128 and  $^{13}\text{C}$ -BDE 209), diluted 1:1 with Milli Q water and mixed with formic acid. The resulting mixture was sonicated for 20 min and then extracted using solid-phase extraction cartridges (6 ml/500 mg Oasis® HLB, Waters, Milford, MA, USA). Elution was done by 10 ml of dichloromethane and the eluate was evaporated to near dryness. The eluent was reconstituted in 500  $\mu\text{l}$  hexane:dichloromethane (1:1, v/v) and subjected to clean-up on 500 mg acid silica impregnated with concentrated sulfuric acid (44%, w/w) from which the analytes were eluted with 10 ml hexane:dichloromethane (1:1, v/v). The cleaned extract was evaporated to dryness under a gentle nitrogen stream, reconstituted in 80  $\mu\text{l}$  iso-octane and transferred to an injection vial. The serum lipid content was determined from enzymatic measurements of cholesterol and triglycerides.

For the analysis of PBDEs and OCPs other than DDTs, an Agilent 6890 GC connected with an Agilent 5973 MS operated in electron capture negative ionisation (ECNI) mode was equipped with a 15 m x 0.25 mm x 0.10  $\mu\text{m}$  DB-5 capillary column (J&W Scientific, Folsom, CA, USA). For the analysis of PCBs and DDTs, an Agilent 6890 GC connected with an Agilent 5973 MS operated in electron impact ionisation (EI) mode was equipped with a 25 m x 0.22 mm x 0.25  $\mu\text{m}$  HT-8 capillary column (SGE, Zulte, Belgium).

For samples with concentrations below LOQ, we used LOQ/2 to replace non-detects during calculations. All statistical analyses were performed using SPSS 17.0.0 for Windows (SPSS Inc.).

## Results and discussion

PBDE congeners 153, 47, 99, 183, 100, 154 and 28, in order of abundance, were detected in the human serum samples. Total HBCD was also detected in 43 out of 60 samples. Total PBDE levels (sum of 7 congeners, excluding BDE-209) ranged from 1.05 to 14.0 ng/g lipid, with a geometric mean of 1.53 ng/g lipid. BDE-209 was detected only in 8 out of 60 samples, with concentrations ranging from 4.5 to 19.1 ng/g lipid, with a geometric mean of 4.5 ng/g lipid. BDE-209 had a detection limit almost 20 times higher than the rest of the PBDEs, which is why it is reported separately. BDE-153 concentrations ranged from 0.20 to 3.42 ng/g lipid, with a geometric mean of 0.51 ng/g lipid, accounting for 74% of the total PBDE concentration. The dominance of BDE-153 in PBDE congener profiles in human serum has been previously observed for Greece (Leondiadis et al. 2006) and Belgium (Roosens et al. 2009), as well as other countries, such as China (Bi et al. 2006). A summary of the PBDE data can be seen in Table 1.

Individual and  $\sum_7$  PBDE concentrations did not statistically differ between the two groups, with the exception of BDE-153 ( $p=0.024$ ) which leads to the conclusion that computer exposure by itself is not enough to cause differences in the PBDE load of an individual. There were no significant differences between males and females with regards to PBDE, PCB and OCP concentrations, with the exception of p,p'-DDE ( $p=0.015$ ) and HBCD ( $p=0.044$ ). Other factors that were explored during the present study, such as computer use (hours per day), diet and car use, did not yield any significant correlations. The duration of electrical and electronic equipment use (in hours) were significant only for BDE-99 ( $r=0.264$ ,  $p<0.05$ ). The presence of carpets at home was correlated with BDE-47 concentrations ( $p=0.044$ ).

Table 1 - Summary of the main PBDE, PCB and OCP congener concentrations in serum samples (in ng/g lipid,  $n=60$ ).

ng/g lipid	LOQ	% detection	mean	median	min	max	SD
<b>BDE-28</b>	0.20	5	0.11	0.10	0.10	0.45	0.06
<b>BDE-47</b>	0.39	43	0.67	0.20	0.20	10.0	1.37
<b>BDE-100</b>	0.22	49	0.22	0.10	0.10	1.77	0.26
<b>BDE-99</b>	0.35	26	0.25	0.20	0.15	1.51	0.18
<b>BDE-154</b>	0.17	13	0.12	0.10	0.10	0.53	0.06
<b>BDE-153</b>	0.40	74	0.57	0.51	0.20	3.42	0.49
<b>BDE-183</b>	0.30	11	0.20	0.20	0.15	0.51	0.08
<b>BDE-209</b>	9.00	13	5.82	4.50	4.50	19.1	3.67
<b>HBCD (on BDE 77)</b>	1.00	70	3.33	1.32	0.49	38.8	6.87
<b>Σ7PBDE</b>			2.15	1.53	1.05	14.0	1.96
<b>CB-74</b>	2.00	59	3.87	3.22	1.00	15.3	3.17
<b>CB-101</b>	2.00	3	1.04	1.00	1.00	2.23	0.21
<b>CB-99</b>	2.00	56	2.70	2.12	1.00	11.3	2.18
<b>CB-118</b>	1.00	92	5.94	4.43	0.50	21.5	4.97
<b>CB-105</b>	2.00	15	1.29	1.00	1.00	4.02	0.74
<b>CB-146</b>	1.00	98	4.84	3.66	0.50	16.3	3.58
<b>CB-153</b>	1.50	100	43.2	34.5	10.2	117	27.1
<b>CB-138</b>	1.50	100	24.9	19.9	6.50	71.8	15.1
<b>CB-187</b>	0.50	100	7.80	5.94	0.80	27.7	6.25
<b>CB-183</b>	0.50	100	2.97	2.34	0.49	8.28	1.94
<b>CB-156</b>	0.50	100	3.35	2.70	0.67	11.2	2.31
<b>CB-180</b>	0.50	100	31.4	25.19	7.03	101	22.1
<b>CB-170</b>	0.50	100	11.3	9.41	2.52	34.9	7.71
<b>CB-199</b>	1.50	39	2.58	0.75	0.75	18.5	3.32
<b>CB-196/203</b>	1.50	28	2.35	0.75	0.75	16.4	3.37
<b>Σ17PCB</b>			150	116	40.6	428	95.3
<b>HCB</b>	5.00	100	38.2	23.1	5.97	192	40.2
<b>Oxychlorane</b>	0.50	100	3.37	2.74	0.72	10.9	2.08
<b>Trans-nonachlor</b>	0.50	100	3.41	2.64	0.45	20.4	3.03
<b>p,p'-DDE</b>	2.00	100	379	268	53.8	1650	321
<b>p,p'-DDT</b>	2.00	100	7.89	6.32	2.41	47.7	6.49
<b>α-HCH</b>	1.00	2	0.52	0.50	0.50	1.64	0.15
<b>β-HCH</b>	2.00	100	30.6	17.8	4.15	143	32.5

\* - Σ7PBDE: BDE 28, 47, 99, 100, 153, 154 and 183.

In the control population (n=30),  $\sum_7$ PBDE concentrations ranged from 1.1 to 9.0 ng/g fat, with a geometric mean of 1.42 ng/g lipid. These concentrations for the control population are on the lower end of those that have been reported for other European countries, such as Spain (Gomara et al. 2007), United Kingdom (Thomas et al. 2003), Belgium (Roosens et al. 2009) and Romania (Dirtu et al. 2006), as well as China (Zhu et al. 2009) and the United States (Schecter et al. 2005). Age was not correlated with PBDEs, which is in accordance to what has been previously observed (Gomara et al. 2007).

All PCBs and OCPs were detected in the serum samples with the exception of PCBs 28, 52, 149 and  $\gamma$ -HCH.  $\sum_{15}$ PCB concentrations ranged from 40.6 to 428 ng/g lipid, with a geometric mean of 116 ng/g lipid. p,p'-DDE concentrations ranged from 53.8 to 1649 ng/g lipid, with a geometric mean of 268 ng/g lipid. The major PCB congeners were 153, 138, 180, 170, 187, 118, 146, 74, 156 and 99 (in respective order of abundance). CB-153 contributed an average of 29% of the total PCB concentration, CB-180 contributed 20% and CB-138 contributed 17%.

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