POLYBROMINATED DIPHENYL ETHERS (PBDEs) DECREASE IN BLOOD SERUM FROM AUSTRALIAN CHILDREN: 2006-2015

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Introduction

Around the world, polybrominated diphenyl ethers (PBDEs) have been detected in various environmental compartments that lead to direct human exposure. Despite a cessation in new usage of these flame retardants, products treated with PBDEs will remain in use for some time with the potential to provide sources to the environment and humans. Biomonitoring in Australia found unexpectedly high concentrations of PBDEs compared to "traditional" persistent organic pollutants (POPs) such as dioxins and polychlorinated biphenyls with highest concentrations in young age groups at 4 times that of adults (Toms et al. 2008). This became a cause for concern since limited data suggested the potential for adverse health effects from PBDE exposure, and young children may be considered to be a population of potentially greater susceptibility (Grandjean and Landrigan 2014).

The aim of this study was to investigate PBDE concentrations in infants and children in Australia. In addition, the results of this study will be compared to data obtained from the same age groups in 2006/07 as well as pooled data collected every two years from 2004/05 to 2014/15 to assess temporal trends.

Materials and Methods

De-identified, human blood serum samples were collected from 10 age groups (each covering 6 months and ranging from 0 to 5 years and both sexes) in South East Queensland, Australia from Sullivan Nicolaides Pathology between 2014 and 2015. 20 samples collected and pooled from each age/sex group. Ethics approval for this study was granted by The University of Queensland Medical Research Ethics Committee. For chemical analysis of BDEs -28, -47, -99, -100, -153, -154 and –183, serum was homogenized with diatomaceous earth and underwent combined extraction and in-cell clean-up modified from previous methods (Abdallah et al. 2013), followed by injection on GC/HRMS. All statistical tests were carried out using GraphPad Prism. For the purpose of averages and statistical calculations, all samples found below the method detection limit (MDL) were assigned a value of zero.

Results and Discussion

PBDEs were detected in all samples of human blood serum for children aged 0 -5 years. The BDE congener detected in the highest concentration was BDE-99 at 23.7 ng/g lipid followed by BDE-47 and BDE-153. This high value for BDE-99 could be an exception. Overall, BDE-47 was detected consistently in the highest concentrations (Table 1).

Table 1. Concentrations (ng/g lipid) of PBDEs in serum from children 0 – 4 years old

	BDE-47	BDE-99	BDE-153	ΣPBDEs
Mean ± standard deviation (ng/g lipid)	2.8 ± 2.1	2.4 ± 4.3	0.8 ± 0.7	5.9 ± 5.6
Range (ng/g lipid)	0.2 to 10.5	0.1 to 23.7	0.02 to 2.9	0.9 to 25.6

The concentrations increase slightly by age and linear regression comparing the 2006/07 and 2014/15 results show no significant difference by age between the two collection periods for BDE-47, while for BDE-153 this difference was significant ($p<0.0001$) (Figure 1). Concentrations were slightly higher in males compared to females with the mean BDE-47 and BDE-153 concentrations for males 3.22 and 0.85 ng/g lipid and for females 2.58 and 0.59 ng/g lipid, respectively.

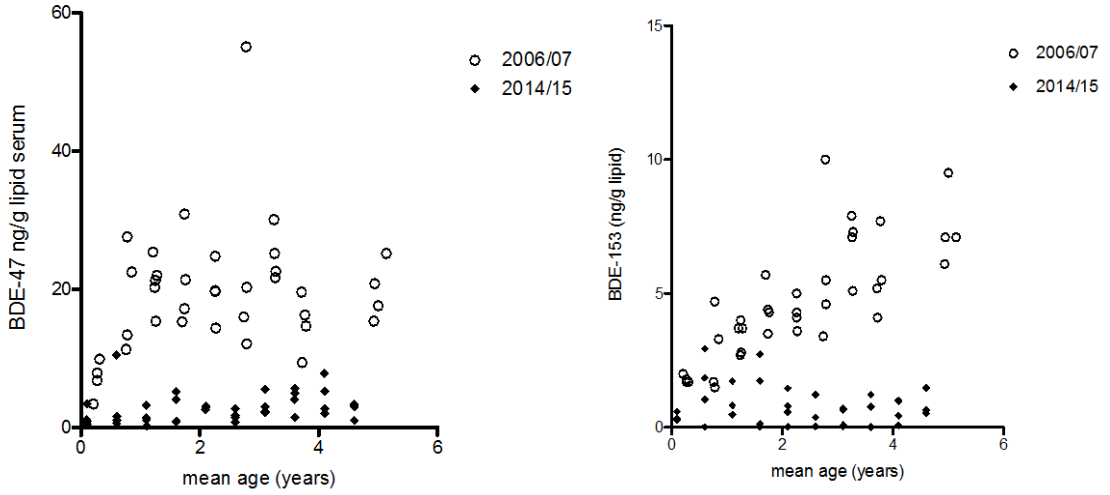

Figure 1. Concentrations of BDE-47 (left) and BDE-153 (right) (ng/g lipid) by age and year of collection.

Overall, a decrease of around 80% is seen for PBDE concentrations in children between 2006/07 to 2014/15 (Figure 2) in Australia while for adults there is a decrease of only 40% for BDE-47 but an increase in BDE-153 of 40% (years of collection 2006/07 and 2012/13, data not shown). This indicates a more rapid decrease in PBDE concentrations in children compared to adults in Australia.

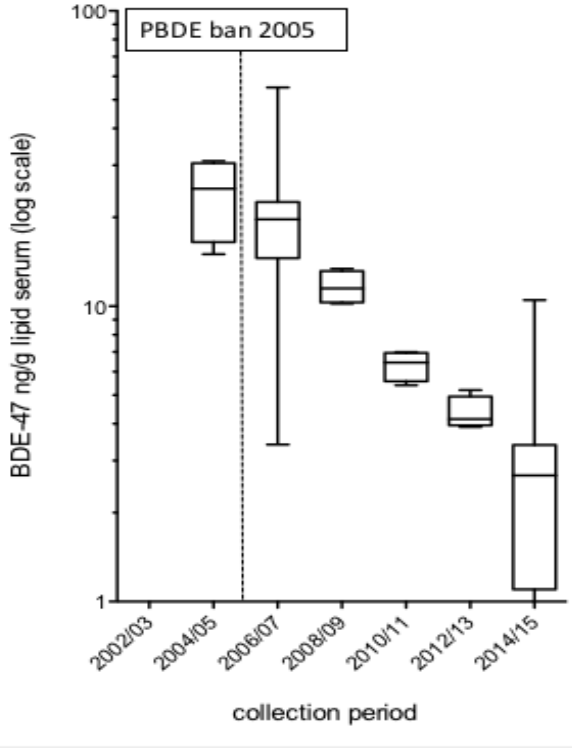

Figure 2. Concentrations of BDE-47 (ng/g lipid) for 0-4 year olds by collection period. Data from 2006/07 and 2014/15 represents 10 age groups (6 monthly from 0 to 4 years) x 2 sexes x 2 replicates; all other periods represent 1 age group (0-4 years) x 2 sexes x 2 replicates.

Human exposure to PBDEs may occur via a variety of pathways including dust contact and ingestion, inhalation, and dietary exposure (Lorber 2008). In addition, potential additional exposure pathways responsible for the elevated concentrations observed in children include placental transfer (Zhao et al. 2013), ingestion of human milk (Toms et al. 2007) and child-specific behaviours (Hoffman et al. 2016). For females of child-bearing age, BDE-47 concentrations in 15-45 year olds were on average 3.7 ng/g lipid in 2004/05 and the equivalent in 2012/13 at an average of 3.7 ng/g lipid representing no change over the collection periods. This indicates that the exposure sources of placental transfer and human milk have not declined as much as the observed concentrations in children ages 0 - 4 years. Stapleton et al. (2012) and Hoffman et al. (2016) report that child-specific activity may be a significant source of exposure to PBDEs in children.

The observed decreases in BDE-47 and BDE-153 in these children who were born post-PBDE ban (in 2005) is likely due to a reduction in exposure, possibly via child-specific behaviours, related to the banning and subsequent removal of these PBDE commercial products from the market. While some reduction in concentration was predicted, the size of the decrease in concentration of BDE 47 in this younger age group exceeded expectations. Whereas previously elevated concentrations were observed in young age groups compared to adults, this is no longer the case and we suggest that in subsequent monitoring the youngest age groups will have concentrations similar to maternal concentrations. If this pattern holds, this may be suggestive of changes in the relative sources of exposure in Australia, and that those of “traditional” POPs including placental transfer, human milk, and general exposures in the food supply, will become the major sources of exposure for infants and young children,

and that indoor environments will contribute relatively lower amounts than during peak PBDE usage times.

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