

## Human biomonitoring in Australian children

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## Human biomonitoring in Australian children: Brominated flame retardants decrease from 2006 to 2015



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### ABSTRACT

Polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCDD) were used intensively as flame retardants, worldwide. They have been detected in human serum samples and PBDEs have been found to be elevated in young children. Commercial Penta- and Octa-PBDE mixtures were banned in Australia in 2005, while HBCDD was banned worldwide in 2013. We investigated PBDE and HBCDD concentrations in serum collected from young children. We also investigated temporal trends in PBDE concentration 10 years after their Australian ban.

Surplus human blood serum samples were collected through a pathology clinic ( $n = 800$ ), in 2014/15, stratified by age (0–6, 6–12, 12–18, 18–24, 24–30, 30–36, 36–42, 42–48, 48–54 and 54–60 months) and sex and pooled for analysis of PBDEs (BDEs – 28, – 47, – 99, – 100, – 153, – 154, – 183) and HBCDD. In 2014/15, the geometric mean concentration of the sum of all PBDEs measured ( $\Sigma$ PBDEs) was 4.5 ng/g lipid (median: 4.6 ng/g lipid, range: 0.88–26 ng/g lipid). A positive association between BDE-47 concentration and age was observed ( $R = 0.41$ ,  $p = 0.008$ ), however there were no trends between other PBDE congeners or HBCDD and age. There were no significant differences between genders for PBDEs ( $t$ -test,  $p = 0.802$ ) or HBCDD ( $t$ -test,  $p = 0.740$ ). The highest concentrations observed were in pools from the females 30–36 month (26 ng/g lipid) and Males 6–12 month (21 ng/g lipid) categories. BDEs – 47 and – 99 were the predominant congeners with a combined average contribution of 75% of  $\Sigma$ PBDEs.

PBDEs showed a significant reduction in children aged 0–4 years over an eight year period. In 2014/15, the mean (range) concentration of BDE-47 is 2.8 (0.23 to 11) ng/g lipid compared to pools in 2006/07 at 19 (3–55) ng/g lipid ( $p < 0.0001$ ) and for BDE-153 is 0.73 ( $< 0.1 = -2.9$ ) ng/g lipid compared to pools in 2006/07 at 4.7 (2–10) ng/g lipid ( $p < 0.0001$ ). HBCDD concentrations were lower than PBDEs with a mean concentration of 0.45 ng/g lipid. There were no temporal trends observed for HBCDD when compared to samples collected in 2012. The dominant stereoisomer was  $\alpha$ -HBCDD (mean = 0.38 ng/g lipid) with an average contribution of 65% towards  $\Sigma$ HBCDD.

Levels of PBDEs in young Australian children have significantly decreased since the bans of commercial Penta- and Octa-BDE in 2005. There has been no observed decrease in HBCDD levels in Australian children since its ban in 2012.

### 1. Introduction

Brominated flame retardants (BFRs) such as polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCDD) have been used as a cost effective and efficient means of reducing

flammability and therefore reducing risk of harm to humans from fires. There are three commercial PBDE formulations - Penta-, Octa- and DecaBDE. The main PBDE applications include electrical and electronic equipment (EEE - such as TVs, PCs and small domestic appliances) (European Commission, 2011), soft furnishings (e.g. sofas, mattresses,

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pillows and curtains) (United Nations Environment Programme (UNEP), 2010) and in polyurethane foam (PUF) seat fillings used in automobiles (European Chemicals Bureau, 2000). The primary use of HBCDD is to flame retard expanded and extruded polystyrene (EPS/XPS) used in building insulation foam (European Chemicals Agency, 2009).

Both PBDEs and HBCDD are lipophilic and resistant to metabolism allowing them to bioaccumulate in the liver and other fatty tissues. They have long half-lives in humans of approximately 1.8–6.5 years and 0.2 years for PBDEs and HBCDD, respectively (Geyer et al., 2004), and have been associated with adverse health effects. PBDEs are thought to disrupt levels of sex hormones, including luteinising hormone and follicle stimulating hormone in men (Meeker et al., 2009), cause toxic effects in other organs including liver, kidneys and thyroid gland; neurodevelopmental deficits including inhibited foetal and infant development; and various cancers (Costa et al., 2008). Data on human health effects of HBCDD exposure is limited, however recent studies have demonstrated that HBCDD displays oestrogenic properties (Dorosh et al., 2011) and can damage dopamine neurons with potential for neurological and endocrine disruption (Genskow et al., 2015). There is also evidence for reduced birthweight and significant adverse neurodevelopment, including impaired motor skills and increased anxiety levels in rodent models (Maurice et al., 2015). Concerns over persistence and toxicity led to importation bans commercial Penta- and Octa-BDE into Australia in 2005 (Toms et al., 2009), prior to global bans of all three commercial PBDE formulations and HBCDD under the UNEP Stockholm Convention of Persistent Organic Pollutants (Stockholm Convention, 2009; Health and Environment Alliance, 2013; Chemical Watch, 2017). Penta and OctaBDEs have never been manufactured in Australia (NICNAS, 2007). These bans do not apply to PBDEs incorporated into products made in other countries and imported into Australia. Exposure to PBDEs continues with an assessment of PBDEs in microenvironments in Australia in 2015 finding sumPBDE (BDEs-28, 47, 99, 100, 153, 154, 183 and 209) concentrations in dust of 2.1 µg/g dust (He et al., 2018).

PBDEs and HBCDDs have been detected in various environmental compartments resulting in direct human exposure through food (Fernandes et al., 2004), dust (Toms et al., 2015), sediment (Drage et al., 2015) and breast milk (Toms et al., 2012). Despite a cessation in new usage of these BFRs, products treated with PBDEs and HBCDDs will remain in use for some time with the potential to provide environmental and human exposure sources. Biomonitoring in Australia revealed 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). These high concentrations raised concerns that young children may be more vulnerable to possible adverse health effects from exposure to higher concentrations of PBDEs (Grandjean and Landrigan, 2014). More recently, studies of adults in other countries have suggested that PBDE levels have reached a plateau in the US (Hurley et al., 2017; Parry et al., 2018)) while elsewhere some studies suggest a continued decrease (Ma et al., 2017; Zhang et al., 2017). Data on HBCDD in the Australian population is more limited, with only two previous studies (Toms et al., 2012; Drage et al., 2017). Both studies demonstrated high variability in HBCDD concentrations, with no obvious temporal trends. Neither study focussed on samples from children.

The aim of this study was to investigate PBDE and HBCDD concentrations in infants and children in Australia using 10 age groups under the age of 5 years to investigate specific small age group trends as first identified with pools from 2006/07 where peak PBDE concentrations occurred in children 2–5 years (Toms et al., 2009). Temporal trends were assessed by comparing the PBDE concentrations of this study to the previous 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.

## 2. Materials and methods

### 2.1. Chemicals & reagents

High purity solvents (HPLC grade), sulfuric acid (analytical grade), sodium sulfate, silica gel (high purity grade, 60 Å pore size) and Florisil (60–100 mesh particle size) were purchased from Sigma Aldrich (Germany). Hydromatrix was purchased from Agilent (Santa Clara, CA, USA). <sup>13</sup>C<sub>12</sub>-labelled standards for HBCDDs (α-, β- and γ- isomers), PBDEs (–28, –47, –99, –153, –154, –183 and –77) and <sup>13</sup>C<sub>12</sub>-TBBPA, native HBCDDs (α-, β- and γ- isomers) and PBDEs (BDEs –28, –47, –99, –100, –153, –154 and –183) were purchased from Wellington Laboratories (Guelph, ON, Canada).

### 2.2. Sample collection & preparation

Sample collection occurred in 2014 and 2015 from South East Queensland, Australia. Human blood samples were obtained in a de-identified manner from surplus pathology samples through a community pathology clinic (Sullivan Nicolaidis Pathology). The data provided was date of birth, date of collection, gender and postcode. The serum samples were pooled by gender (males and females) and from 10 age groups (0–6, 6–12, 12–18, 18–24, 24–30, 30–36, 36–42, 42–48, 48–54 and 54–60 months). The total number of samples was 800 with 20 samples in each pool and one replicate pool per strata, that is, for females 0–6 months there were two pools of 20 samples each and so on for each age group and gender. While these samples cannot be assumed to represent the entire population of Australia, this sampling methodology has been used for almost two decades to assess age, gender and temporal trends for many environmental pollutants in Australia (Toms et al., 2014; Heffernan et al., 2016; Thomas et al., 2017). There are limitations to pooling as outlined by Heffernan et al. (2014) such as an inability to detect the lowest and highest exposed in the population but the advantages include ease of logistics and trend analysis. Ethics approval for this study was granted by The University of Queensland Medical Research Ethics Committee and Queensland University of Technology.

### 2.3. Lipid analysis of serum samples

Aliquots (300 µL) of each sample were sent to Sullivan Nicolaidis Pathology for analysis of total cholesterol (TC) and triglycerides (TG). Total lipid (TL) concentration (mg/dL) was calculated using Eq. (1) (Phillips et al., 1989).

$$TL = 2.27 \cdot TC + TG + 62.3 \quad (1)$$

### 2.4. Sample extraction and clean-up

Samples underwent a combined extraction and clean-up using pressurised liquid extraction (PLE) on an ASE 350 (Dionex, Sunnyvale, CA, USA). 5 g of each serum pool was weighed into a 15 mL falcon tube. 10 µL of an internal standard mixture (containing 100 pg/µL of <sup>13</sup>C<sub>12</sub> BDEs –28, –47, –99, –100, –153, –154, –183 and <sup>13</sup>C<sub>12</sub>- α-, β- and γ-HBCDD in methanol). Samples were vortexed for 2 min and transferred into 100 mL Dionium™ ASE cells, pre-packed from the bottom upwards with 2 x glass fibre filters (GFF), 5 g silica, 2 g hydromatrix, 1 x (GFF) 12 g sulfuric acid (44%) impregnated silica, 1 x GFF, 5 g florisil, 30 g anhydrous sodium sulfate, 10 g diatomaceous earth. Cells were loaded onto the ASE 350 and extracted using hexane:DCM (3:2, v/v ratio) at 90 °C and 1500 psi. The heating time was 5 min, with a 4 min static time, purge time 120 s and flush volume 50%. Three static cycles were required to achieve maximum recovery of all target compounds. Clean extracts were transferred to round-bottomed flasks and concentrated to 1–2 mL in a rotavap (Buchi). Samples were transferred to 5 mL disposable glass tubes and concentrated to

**Table 1**  
Concentrations (ng/g lipid) of PBDEs and HBCDDs in serum from Australian children 0–4 years old in 2014/15.

Chemical	BDE 28	BDE 47	BDE 100	BDE 99	BDE 154	BDE 153	BDE 183	ΣPBDEs	α-HBCDD	β-HBCDD	γ-HBCDD	ΣHBCDD
Detection frequency (%)	50	100	30	80	7.5	75	28	N/A	85	0	75	N/A
Mean	0.22	2.8	0.22	1.9	0.11	0.73	0.18	5.9	0.38	< 0.1	0.25	0.62
Geometric mean	0.13	2.1	0.11	0.66	0.08	0.36	0.10	4.5	0.30	< 0.1	0.17	0.45
Median	0.09	2.7	< 0.1	0.85	< 0.12	0.58	< 0.15	4.61	0.32	< 0.1	0.15	0.48
Min	< 0.1	0.23	< 0.1	< 0.1	< 0.12	< 0.12	< 0.15	0.88	< 0.1	< 0.1	< 0.1	< 0.1
Max	1.4	11	2.3	24	1.2	2.9	1.1	26	1.3	< 0.1	1.4	1.90

near-dryness at 40 °C under a gentle stream of nitrogen. The sample was reconstituted in 50 µL toluene containing 1 ng of <sup>13</sup>C<sub>12</sub>-BDE-77 as a recovery standard and transferred to a 200 µL inserted autosampler vial. After analysis of PBDEs via GC/HRMS, extracts were solvent exchanged into 50 µL methanol containing 1 ng of <sup>13</sup>C<sub>12</sub>-TBBPA as a recovery standard for analysis of HBCDDs via LC-MS/MS.

### 2.5. Instrumental analysis

Samples were analysed for PBDEs (BDEs –28, –47, –99, –100, –153, –154, –183) via a Thermo 1310 gas chromatograph coupled to a DFS Magnetic Sector high resolution mass spectrometer (GC-HRMS). The HRMS was operated in electron impact-multiple ion detection (EI-MID) mode, and resolution was set to ≥10,000 (10% valley definition). Full method parameters have been published previously (Wang et al., 2017).

HBCDD (α-, β- and γ-) was measured in serum extracts using an AB/Sciex API 5500Q mass spectrometer (AB/Sciex, Concord, Ontario, Canada) coupled to a Shimadzu Nexera HPLC system (Shimadzu Corp., Kyoto, Japan). The mass spectrometer (MS) was operated in multiple reaction monitoring (MRM) mode using negative electrospray ionisation (ESI). Separation was achieved using a Kinetex XB C18, 50 × 2.0 mm 1.7 µm column (Phenomenex, Torrance CA) using a mobile phase gradient of 85% methanol, ramping up to 100% methanol over 6 min and then holding for 4 min at a flow rate of 0.3 mL/min. Full LC-MS/MS details including MRM parameters have been published previously (Drage et al., 2017).

### 2.6. Quality assurance/quality control

To avoid cross-contamination of samples during extraction and clean-up, a rinsing cycle (using the same solvent combination) was programmed before and after each different extraction. Furthermore, all pre-packed cells underwent a pre-extraction under the same parameters, prior to the loading of samples, to reduce any interferences or potential blank contamination from the in-cell components during analyses.

A blank sample consisting of 5 mL bovine serum was extracted with every batch of samples. All target analytes were below the limits of detection in blank samples. Therefore no correction for blank concentrations occurred. The method was validated by repeated analysis ( $n = 6$ ) of a pooled in-house QC sample. This consisted of pooled human serum which had been previously characterised and found to contain between < 1.0 pg/mL (BDEs –100 and –154) and 10 pg/mL (BDEs –47 and –153). Three 5 mL aliquots were spiked with 1 ng of each target analyte. The concentrations were then measured and the recoveries of target analytes calculated. Average recoveries for PBDEs ranged from 90% (BDE-153) to 116% (BDE-100). Average recoveries for HBCDDs were 91% (α-HBCDD), 90% (β-HBCDD) and 87% (γ-HBCDD). The relative standard deviation (RSD) was < 15% for all target compounds.

For all target compounds method detection limits (MDLs) were calculated based on a chromatogram signal to noise of 10:1. MDLs for PBDEs were 0.1 ng/g lipid for BDEs –28, –47, –99 and –100; 0.12 ng/g lipid for BDEs –154 and –153; and 0.15 ng/g lipid for BDE-

183. The MDL for all HBCDD isomers was 0.1 ng/g lipid.

### 2.7. Statistical analysis

All statistical tests were carried out using Microsoft Excel 2010 and Prism Graphpad. For the purpose of averages and statistical calculations, all samples found below the method detection limit (MDL) were assigned a value of MDL/√2. As the data was log-normally distributed, associations between age and contaminant concentration were assessed using a Pearson correlation on log-transformed data while significant differences between genders were tested using a *t*-test on log-transformed data. To assess temporal trends, significant differences between novel and historical datasets were tested using a Mann-Whitney *U* test. Confidence intervals were set to 95%.

BDEs –28, –100, –154 and –183 were detected in < 50% of pools and are therefore discussed briefly. However, a table of ΣPBDEs and ΣHBCDD concentrations for each individual pool is available in SI Table S1. Due to the low detection frequency of other congeners this paper will primarily discuss the individual concentrations for BDEs –47, –99 and –153 rather than the sum of all congeners.

## 3. Results and discussion

The average concentration and detection frequencies of all compounds measured in this study are presented in Table 1. PBDEs were detected in measureable concentrations in all pools ( $n = 40$ ) for children aged 0–5 years. The geometric mean concentration of the sum of all PBDEs measured (ΣPBDEs) was 4.5 ng/g lipid (median: 4.6 ng/g lipid, range: 0.88–26 ng/g lipid). BDEs –47 and –99 were the predominant congeners with a combined average contribution of 75% of ΣPBDEs. BDE-47 was detected in all samples with a median concentration of 2.7 ng/g lipid (range: 0.23–11 ng/g lipid). This was followed by BDE-99, which was detected in 80% of samples (median = 0.85 ng/g lipid, range: < 0.1–24 ng/g lipid) and BDE-153, which was detected in 75% of samples and made up 15% of ΣPBDEs (median = 0.58 ng/g lipid, range: < 0.12–2.9 ng/g lipid). The remaining average ΣPBDE content was made up of BDE-28 (4.4%), BDE-183 (2.3%) and BDE-100 (1.8%). This is consistent with congener profiles seen in, amongst others, Australia (Toms et al., 2009), USA (Stapleton et al., 2012; Eskenazi et al., 2011), Nicaragua (Athanasidou et al., 2008), Spain (Gari and Grimalt (2013), Pakistan (Ali et al., 2003) and Mexico (Pérez-Maldonado et al., 2009), and typical of exposure to products treated with the Penta-BDE commercial formulation. As the majority of PBDEs measured in this study were detected in 50% or fewer samples, only BDEs –47, –99 and, –153 are discussed from this point on (as mentioned in Section 2.7).

HBCDD was detected in 90% of samples with a geometric mean of 0.45 ng/g lipid (range: < 0.1–1.9 ng/g lipid). The dominant stereoisomer was α-HBCDD (median = 0.32 ng/g lipid, range: < 0.1–1.3 ng/g lipid) with an average contribution of 65% towards ΣHBCDD. This was followed by γ-HBCDD (median = 0.15 ng/g lipid, range: < 0.1–1.4 ng/g lipid), which contributed 35% towards ΣHBCDD. β-HBCDD was not detected in any of the 40 pools. The concentrations of HBCDD in children's serum were generally lower than those of PBDEs. This is likely to be because HBCDD is generally found in lower

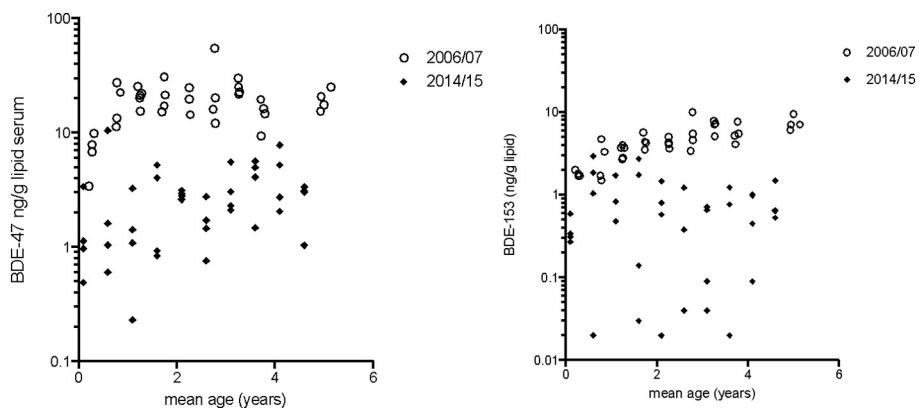


Fig. 1. Concentrations of BDE-47 (left) and BDE-153 (right) (ng/g lipid) by age and year of collection (log scale).

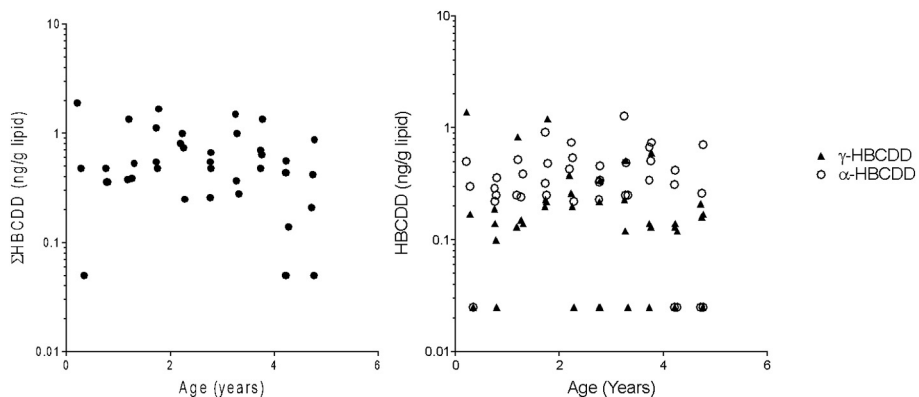


Fig. 2. Concentrations of sum HBCDD (left) and  $\alpha$  and  $\gamma$ -HBCDD (right) (log scale).

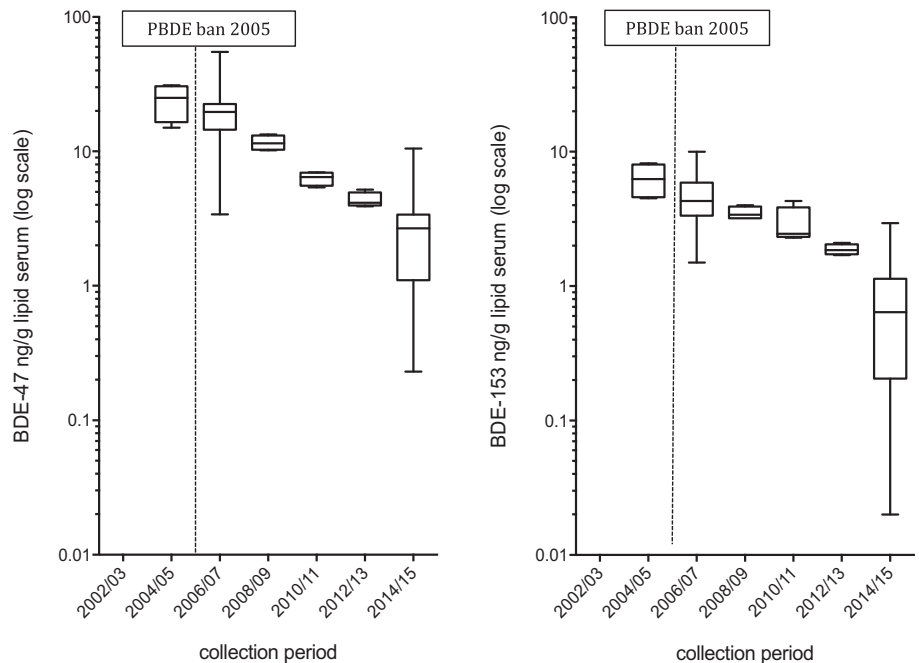


Fig. 3. Concentrations of BDE-47 and -153 (ng/g lipid) for 0–4 year olds in 2002/3 ( $n = 4$ ), 2004/5 ( $n = 4$ ), 2006/7 ( $n = 32$ ), 2008/9 ( $n = 4$ ), 2010/11 ( $n = 4$ ), 2012/13 ( $n = 4$ ) and 2014/15 ( $n = 32$ ); Box and whisker represents mean and minimum to maximum.

concentrations than PBDEs in environmental samples as it is typically added to products at 0.7–2% by weight (European Commission, 2011), while products treated with PBDEs have been treated in concentrations of up to 25% by weight of the various commercial mixtures (European

Commission, 2011; UNEP, 2010). Moreover, > 95% of HBCDD use has been application to cavity wall insulation, while PBDEs are more commonly used in electronics and furniture, and are therefore more likely to volatilise from their products through more regular use (Drage

et al., 2018).

### 3.1. Demographic trends of PBDEs and HBCDDs in Australian children

A positive association was found between BDE-47 levels and age ( $R = 0.41$ ,  $p = 0.008$ ) in serum from Australian children collected in this study (Fig. 1). However, there was no observed trend between age and concentration of BDE-99 ( $R = 0.059$ ,  $p = 0.72$ ), or BDE-153 ( $R = -0.021$ ,  $p = 0.893$ ). While not statistically significant, the total proportion of BDE-153 to total PBDE content was highest in children under 2 years ( $R = 0.288$ ,  $p = 0.074$ ). This, combined with the positive association of BDE-47 levels and age could suggest that more recently born infants are exposed to more historical PBDEs via breastmilk. This is because BDE-153 has a longer half-life in humans than other PBDEs, such as BDEs -47 and -99 (Geyer et al., 2004). Therefore a mother who has had an historical exposure to PBDEs is likely to display a higher BDE-153:ΣPBDEs ratio than one who has had a more recent exposure. This would in turn be passed on to an infant via placental transfer and breastfeeding and subsequently displayed in their serum concentrations. In contrast, BDE-47 may have been excreted more rapidly, therefore lowering mothers' body burdens and subsequently reducing placental transfer and exposure via breastmilk. In similar pools collected in 2006/7, the reverse of this trend was observed with the contribution of BDE-153 towards ΣPBDEs increasing with age of children (Toms et al., 2009).

Pools were also tested for any gender differences in PBDE levels. However there were no significant differences (*t*-test) between concentration and gender for BDE-47 ( $p = 0.42$ ), BDE-99 ( $p = 0.59$ ) or BDE-153 ( $p = 0.34$ ) with the highest concentrations observed in pools from the females 30–36 months (26 ng/g lipid) and males 6–12 months (21 ng/g lipid) categories.

The median ΣHBCDD concentration in this study (0.48 ng/g lipid) is comparable with that of 4 pooled samples collected from Australian children 0–4 year olds in 2013 at 0.35 ng/g lipid (Drage et al., 2017). No statistically significant trends were observed between ΣHBCDD and age ( $R^2 = 0.098$ ,  $p = 0.55$ , Fig. 2) or gender (*t*-test,  $p = 0.74$ ). The dominance of α-HBCDD in this study is consistent with previous studies from Australia (Toms et al., 2012; Drage et al., 2017) as well as serum from India (Devanathan et al., 2012), Sweden (Weiss et al., 2006), Canada (Rawn et al., 2014) and Japan (Kakimoto et al., 2008). Selective predominance of α-HBCDD in biota has been previously reported (Tomy et al., 2004, Law et al., 2006).

### 3.2. Temporal trends of PBDEs in serum from Australian children

For Σ<sub>4</sub>PBDEs a significant decrease of > 85% was seen for children aged 0–4 years between 2006/07 (mean = 40 ng/g lipid) and 2014/15 (mean = 5.6 ng/g lipid) in Australia (Fig. 3) (Mann-Whitney test  $p < 0.0001$ ). The *p*-values for each Mann-Whitney test conducted between 2014/15 and previous sampling campaigns are presented for BDEs -47 and -153 in the supporting information (Tables S2 and S3). These data demonstrate that PBDE levels in Australian children have undergone a dramatic year-on-year decrease consistently since their bans in Australia. This differs to the patterns observed in adults where a less prominent decrease of approximately 55% was observed for BDE-47 (mean decrease from 8.3 ng/g lipid (2006/7) to 3.6 ng/g lipid (2012/13) all pools from aged > 16 years) and a lower decrease in BDE-153 by approximately 22% (mean of > 16 years 3.6 and 2.8 ng/g lipid for 2006/07 and 2012/13, respectively) (Toms et al., 2018). This change may be related to decrease or removal of a source of exposure. Previous studies have indicated differences in adult and child exposure with regards to dust ingestion, mouthing/child-specific behaviours such as eating with fingers, and breast milk consumption (Toms et al., 2015; Heffernan et al., 2016; Jones-Otazo et al., 2005; Lorber, 2008; Stapleton et al., 2005; Harrad et al., 2008). Adult concentrations have decreased only slightly over the 10 year period. This difference between adults

and children could also be related to growth and therefore a faster rate of dilution in children compared to adults. PBDE concentrations in females of child-bearing age have not changed over this same time period (Toms et al., 2018), however, if major external sources of PBDEs are being removed from microenvironments (i.e. replaced with “PBDE-free products”, then these initial exposures via breastfeeding and placental transfer are likely to be quickly diluted as a child grows rapidly in the first few years of life.

With the apparent reduction of exposure to PBDEs for children potentially via indoor dust and child-specific behaviours, it is likely that traditional sources, i.e. placental transfer (Zhao et al., 2013), breast milk (Toms et al., 2007) and food (Domingo, 2012) will provide greater contribution to future paediatric PBDE exposure.

## 4. Conclusions

There has been a significant reduction in PBDE concentration in Australian children over the decade following the bans of commercial Penta-BDE and Octa-BDE in 2005. HBCDD concentrations were substantially lower than PBDEs but continued monitoring is required to assess temporal trends. Previous observations that PBDE concentrations are much higher in young children than in adults are no longer apparent. This decrease in PBDE concentrations appears to be rapid compared to adult PBDE concentrations over the same period. This is likely to be due to a reduction in the number of PBDE-containing products in indoor microenvironments as they have been disposed and replaced with “PBDE-free” alternatives. Subsequent assessments of the youngest age groups are predicted to show concentrations similar to maternal concentrations. If this pattern continues in future monitoring programmes in Australia, it would suggest a change in the relative sources of exposure. This means that similar to “traditional” POPs, placental transfer, human milk, and general exposures in the food supply, will become the major sources of PBDE exposure for infants and young children, and that indoor environments will make a lower contribution to overall concentrations.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.envint.2018.11.044>.

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