Environment International 121 (2018) 357–364

Contents lists available at ScienceDirect

Environment International

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

Temporal trends in serum polybrominated diphenyl ether concentrations in the Australian population, 2002–2013

Leisa-Maree L. Toms[a,f,*], Andreas Sjödin[b,f], Peter Hobson[c], Fiona A. Harden[d], Lesa L. Aylward[e,f], Jochen F. Mueller[f]

[a] School of Public Health and Social Work and Institute of Health and Biomedical Innovation, Queensland University of Technology, Brisbane, Australia
[b] Centers for Disease Control and Prevention, Atlanta, USA
[c] Sullivan Nicolaides Pathology, Brisbane, Australia
[d] Hunter Industrial Medicine, Newcastle, Australia
[e] Summit Toxicology, Falls Church, VA, USA
[f] The University of Queensland, Queensland Alliance for Environmental Sciences, Australia

ARTICLE INFO

Handling Editor: Heather Stapleton

ABSTRACT

In Australia, systematic biomonitoring of persistent organic pollutants (POPs) in pooled serum samples stratified by age and sex has occurred every two years between 2002/03 and 2012/13. Multiple regression modeling on log10-transformed serum pool concentrations of BDEs 47, 99, 100 and 153 and on the sum of these (Σ_4PBDE) was conducted to examine trends by sex and time since baseline, stratified by age group. Temporal trends were age- and congener-specific, with the largest changes per year of observation in the 0–4 year old group, with β (SE) = −0.098 (0.013) for $\log_{10}$BDE47; −0.119 (0.012) for $\log_{10}$BDE99; −0.084 (0.014) for $\log_{10}$BDE100, and −0.053 (0.013) for $\log_{10}$BDE153, all $p < 0.001$. Adults over age 16 showed much smaller decreasing temporal trends for BDE47 and BDE99, no significant changes in BDE100, and, for the oldest age groups, slight increases in BDE153. As a result, Σ_4PBDE concentrations were stable over the entire time period in adults older than 16. Concentrations of each BDE in pools from females aged 31–60 were significantly lower compared to males. Relative proportions of BDE47 declined, while BDE153 accounted for a greater share of Σ_4PBDE over time. Whereas previously we saw a large elevation in the youngest age groups compared to older children and adults, this is no longer the case. This may be due to a decline in infant and toddler exposures in the indoor environment as use of PBDEs in consumer products has been phased out, suggesting temporal changes in the relative sources of exposure for young children in Australia.

1. Introduction

Polybrominated diphenyl ethers (PBDEs) are a class of brominated flame retardants (BFR) added to products to reduce flammability and rate of ignition. In 2005, the importation of raw product penta- and octa-BDE into Australia ceased (NICNAS, 2007) following the inclusion of penta-BDE and octa-BDE into the annex of the Stockholm Convention (Stockholm Convention on POPs, 2010). The commercial PBDE flame retardant mixture deca-BDE is now being considered for listing on the Stockholm Convention (Stockholm Convention on Persistent Organic Pollutants, 2013).

Biomonitoring of PBDEs using human blood serum samples from Australia began in 2002/03 and found unexpectedly high concentrations compared to "legacy" persistent organic pollutants (POPs) such as dioxins and polychlorinated biphenyls (Harden et al., 2007; Toms et al., 2008). This first study included a young age group (< 16 years) with an average age of 11 years. In 2004/05, a 0–4 years age group was also included in the biomonitoring program with an average age of 2.4 years. The concentration of BDE-47 in this youngest age group was four times that of the ≥16 years group (Toms et al., 2008), thus demonstrating an inverse relationship between age and concentration, which was in contrast to legacy POPs (Harden et al., 2007). Higher concentrations in children have also been reported elsewhere, including the USA (Fischer et al., 2006; Lunder et al., 2010; Sjodin et al., 2014; Wu et al., 2015), Norway (Thomsen et al., 2002), and Pakistan (Ali et al., 2013). This became a cause for concern since limited data suggest the potential for adverse health effects from PBDE exposure, and young children may be considered to be a population of potentially greater

* Corresponding author.
E-mail address: leisamaree.toms@qut.edu.au (L.-M.L. Toms).

https://doi.org/10.1016/j.envint.2018.09.014
Received 12 June 2018; Received in revised form 7 August 2018; Accepted 7 September 2018

susceptibility (Darnerud, 2003; Grandjean and Landrigan, 2014). Various epidemiological studies have examined potential associations between exposure to PBDEs and diabetes; neurobehavioral and developmental disorders; reproductive health effects; alteration in thyroid function; and adverse cognitive outcomes with early life exposure (Eskenazi et al., 2013; Kim et al., 2014; Chevrier et al., 2016; Jacobson et al., 2016). The higher concentrations of PBDEs compared to other POPs in Australia coupled with the elevated concentrations in children warranted continued biomonitoring to further assess these trends.

The Australian biomonitoring program relies upon collection and analysis of pooled serum samples and allows for the assessment of age, sex and importantly, temporal trends, in POP concentrations (Toms et al., 2012, 2014). While it is expected that decreased exposure will eventually result in decreased body burden, the longevity of products treated with PBDEs combined with lengthy estimated human half-lives of 1.8 years (BDE-47), 2.9 years (BDE-99), 1.6 years (BDE-100) and 6.5 years (BDE-153) (Geyer et al., 2004), makes it difficult to anticipate the speed at which concentrations in human tissue will decrease. As well, usage change over time may result in altered PBDE congener profiles in human samples.

The aim of this study is to update the monitoring of PBDEs in human blood serum from samples of the Australian population from previous reports to include pools collected in 2010/11 and 2012/13. The time period of monitoring covers a period of great interest, with the ban on these chemicals (in 2005) going into effect early in the monitoring period. This allows an assessment of the effectiveness of the intervention to eliminate use of these chemicals and in turn decrease body burden. In addition, the data allow an investigation of whether or not congener profiles differ with changes to product usage.

2. Materials and methods

2.1. Sample collection

Sample collection for our human biomonitoring program relies upon collection of pooled samples of de-identified surplus sera from community pathology laboratories stratified by age and sex, as has been reported previously (Heffernan et al., 2014, (Toms et al., 2008, 2009a, b, 2012). In brief and as per our protocol, samples for the two new collection periods (2010/11 and 2012/13) were obtained in collaboration with Sullivan Nicolaides Pathology (SNP) from de-identified surplus pathology samples collected in south-east Queensland, Australia. Samples were stratified by age and sex. Pooled samples were placed into 100 mL solvent rinsed glass bottles. Age groups were as follows: 0–4; 5–15; 16–30, 31–45, 46–60, and ≥60 years. For each pool, 100 individual samples were combined using up to 1 mL of each of the 100 samples. For younger age groups, where less volume was available, < 1 mL was used with consistent volumes for all donors into a specific pool, e.g. 0.5 mL was taken from each sample. Two pools were made for each age and sex group for each time period in the 2010/11 and 2012/13 collection periods. As previously described, greater numbers of pools were collected for selected age groups at some earlier time periods.

When this methodology was first conceived, an innovative technique to assess if bias occurred using pathology samples was undertaken where two pools of pathology samples “sick” were compared to age, sex and postcode matched insurance samples “healthy” and analysed for dioxins. The normalised difference was 10% which could be explained by normal variations in analytical reproducibility (Harden et al. 2004). Therefore, there should not be bias related to the sample collection.

For each of 2010/11 and 2012/13, there were a total of 4800 samples assembled into 24 pools. In total, there are PBDE data from 249 pools created from 17,752 individual samples from 2002/03, 2004/05, 2006/07, 2008/09, 2010/11 and 2012/13. In 2002/03, separate pools were not collected for ages 0–4; rather, the youngest age group was < 16 years, with limited numbers of donor samples from children in the youngest age group. Beginning in 2004/05, pools were collected specifically for the 0 to 4 year age group. It was not possible to determine if any one donor contributed to more than one collection period due to the use of de-identified samples. We maintain ethics approval for this study through The University of Queensland Medical Research Ethics Committee and Queensland University of Technology Ethics Committee. The analysis of pooled samples by investigators at the U.S. Centers for Disease Control and Prevention (CDC) was determined not to constitute engagement in human subject research.

2.2. Chemical analysis

The PBDE measurements were made at the U.S. Centers for Disease Control and Prevention (CDC) in Atlanta and the methodologies have been described previously (Sjodin et al. 2004; Jones et al. 2012). Briefly, a set of samples was defined as 24 unknown samples with three analytical blanks and three quality assurance/quality control (QA/QC) samples. Each set was processed using a semi-automated sample preparation method. Human sera (2 g) were weighed into test tubes and fortified with internal standards (^{13}C-labeled) using a 215 Liquid Handler (Gilson Inc., Middleton, WI). Formic acid and water were added to denature proteins and dilute the samples on the liquid handler. The target analytes were extracted into dichloromethane using the solid phase extraction (SPE) workstation (Rapid Trace®, Zymark, Hopkinton, MA) for the 2010/11 pools and by automated liquid liquid extraction (LLE) for the 2012/13 pools. Clean up was performed on a two layered column. The top layer comprised activated silica and the bottom layer comprised silica gel/sulfuric acid (2:1 by weight). The top layer retained polar lipids such as cholesterol, while the bottom layer degraded the remaining lipids to produce an extract free of biogenic material. Samples were evaporated to 1 mL and transferred to the gas chromatograph vials, which were previously spiked with recovery standards. Samples were further evaporated to 10 µL and analysed by gas chromatography high resolution mass spectrometry. A DFS (ThermoFinnigan, Bremen, Germany) instrument was used for the analysis. The chromatographic separations were carried out on an 1310 gas chromatograph (ThermoFinnigan, Bremen, Germany) fitted with a DB5HT capillary column (15 m, 0.25 mm inner diameter, and 0.10 µm thickness). The following congeners were targeted for analysis: BDEs-17, -28, -47, -66, -85, -99, -100, -153, -154 and -183. The results are expressed as ng/g lipid. Three of the 249 pools in this analysis had non-detected concentration for one of the four main congeners. These non-detected pool concentrations were imputed using half the limit of detection (LOD), which was dependant on sample size and blanks. A summary metric (Σ_4PBDE) was calculated for each pool as the sum of BDEs-47, -99, -100 and -153.

It should be noted that pools from all collection periods were analysed at the U.S. CDC with the exception of the 2002/03 and 2004/05 pools which were analysed at Eurofins, Germany (Toms et al., 2008).

2.3. Statistical analysis

Mean and standard error of the mean of pool concentrations by analyte, age group, and sampling cycle were calculated. For each sample collection period, trends in $\log_{10}$-transformed pool concentration as a function of average age of pool contributors were evaluated using linear regression. Trends in $\log_{10}$-transformed pool concentration over years since baseline collection and by sex were examined using multiple regression (STATA IC 12.1, Stata Corp., College Station, TX). The multiple regression was stratified by age group in order to detect potential differences in time trends by age group. Contribution of each of the four major congeners (BDEs 47, 99, 100, and 153) to the sum of those four congeners was examined at different time points to evaluate potential changes in congener profiles over time.