RELATING HUMAN PBDE EXPOSURE PROFILES TO BLOOD LEVELS: A COMPARISON BETWEEN RESULTS FROM AUSTRALIA AND USA

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Introduction

Polybrominated diphenyl ethers (PBDEs) are a family of brominated flame retardants that has seen extensive commercial use in materials such as plastic and electronic equipment and textiles[1-3]. The penta- and octa-BDE commercial mixtures were voluntarily withdrawn from the US marketplace in 2004[1] and Australia ceased importation of these formulations in 2005 although there are currently no import restrictions on goods containing these PBDEs[2]. They are released to homes, offices and microenvironments such as automobiles during the use of products treated with PBDEs for flame retardation purposes. As a result, principal human background exposure routes include dust ingestion, dermal absorption, inhalation in microenvironments and dietary intake[3].

The percent contribution from each background source to cumulative exposure has been estimated in various studies[1,3,4]. Because body weight, air inhalation rates and dust ingestion rates vary by age, the daily exposure dose (DED) of PBDEs is significantly different between groups within a population such as infants, children, teenagers, and adults[4]. International differences in exposures also appear to exist with the highest levels of PBDEs in the general population consistently found in the USA. Data suggest a typical range of ~30–80 ng g^{-1} lw of total PBDEs as representative of blood in the general population of Americans[1,4-6]. In contrast, most studies from Europe, Asia, including Australia, suggest concentrations of total PBDEs in blood less than 10 ng g^{-1} lw.

Typically the DED is expressed on the basis of per kg body weight[1] and may also be used in development of pharmacokinetic (PK) models to simulate the average body burden. This allows predictions, obtained using DED data derived from levels in environmental media, to be compared with levels and profiles of PBDEs reported in human biomonitoring studies. Perhaps more importantly, the development of such models provides additional understanding of relationships between PBDE exposure and body burdens (e.g., identifying what increase in contamination of dust or food is required to influence body burdens).

In this study we collate the latest available environmental media exposure data on PBDEs in Australia to estimate DEDs for PBDEs via dietary intake, dust ingestion, dermal absorption and inhalation. The DED values are input into a simple one compartment PK model to simulate the average steady state body burden of PBDEs in the adult general Australian population. This data is compared to previously reported analogous data for the American population derived using the same model[1]. The predicted body burdens are compared with profiles of PBDEs in adult blood previously measured and reported in Australia and the USA.

Materials and methods

Exposures for the general Australian population via dietary intake were estimated using a large-scale assessment of PBDE levels in a wide variety of food items[8]. In that study estimated dietary intake of PBDEs in Australia for the general population was based on results from a total of 35 foods including meat, dairy, oils and spreads, bread and bakery products, and vegetables. Exposures via inhalation, dust ingestion and dermal absorption were estimated using data reported for 10 dwellings in Melbourne, Australia[9]. Importantly in that study indoor air concentrations for each house were determined using an active air sampling method.

A simple one-compartment lipid-based PK model that treats the whole body as a single kinetically homogenous unit previously used by US EPA[1] to predict body burdens for Americans was used for the Australian data.

With this model, the equation for the change in lipid-based PBDE concentrations over time is as follows:

$$\frac{\partial C_{BDE}}{\partial t} = \left[\frac{D_{BDE} \times ABS_{BDE}}{BL}\right] - k \times C_{BDE}$$

where C_{BDE} is the congener-specific lipid-based concentration (ng g^{-1} lw); D_{BDE} the congener-specific daily exposure dose (DED) (ng day^{-1}); ABS_{BDE} the congener-specific and route-specific absorption fraction; BL the body lipid mass (g) and k = the first-order elimination rate constant of the congener in the body (day^{-1}). The solution to this partial differential equation is:

$$C_{BDE}(t) = C_{BDE}(0) \times e^{-kt} + \left[\frac{D_{BDE} \times ABS_{BDE}}{BL}\right] \times \left[\frac{\left(1 - e^{-kt}\right)}{k}\right]$$

where $C_{BDE}(0)$ = the initial body burden at time 0 and $C_{BDE}(t)$ that at time t. Assuming a constant BL and constant DEDs, the steady state lipid concentration (i.e., when t approaches infinity) is easily calculated as:

$$C_{BDE} = \left[\frac{D_{BDE} \times ABS_{BDE}}{k \times BL}\right]$$

Apart from country-specific DED values, the same input data such as congener elimination rate constants and absorption fractions employed in the American work[1] are used here to consider the Australian data. This includes an assumed body mass 70 kg body that is 25% lipid, leading to a BL value of 17,500 g. The steady state equation is used here to estimate congener-specific PBDE lipid-based adult blood levels in the general Australian population.

Results and discussion

Comparative exposure intake data for congeners BDE-47, -99, -153 and -183 in adult general populations in the USA and Australia are shown in Figure 1. It is apparent in this comparison that the exposure profile in the USA is different to that observed in Australia. The bulk of US exposures appear to occur in the indoor environment through contact with house dust[1]. In contrast, dietary intakes dominate exposures in the general Australian population based on available data.

The difference in the USA and Australian exposure profiles is predominantly explained by the much higher levels of BDE-47, BDE-99 and BDE-153 typically reported in USA dusts compared to other countries including Australia[10]. The far higher concentrations of these congeners in the USA dusts is broadly commensurate with the the ~50-fold greater use in the USA compared to the rest of the world of the tri- through hexa-BDEs that largely comprise the penta-BDE formulation[11]. In the USA exposure intakes for the sum of congeners BDE-47, -99, -153 and -183 ($\sum_4$PBDEs) via contact with dust was estimated to be ~159 ng day^{-1} [1]. In contrast, $\sum_4$PBDE dust exposures in Australia were estimated to be ~5 ng day^{-1}. Interestingly in the current study $\sum_4$PBDEs dietary exposures in Australia were estimated at 69 ng day^{-1}, approximately double that used in the USA model[1]. Previous estimates of dietary exposure in the USA and Australia are comparatively close at ca. 50-100 ng day^{-1}. Although there is undoubtedly a high level of uncertainty associated with the ingestion rates used here and other studies, they do provide an indication of the likely range within the population. Comparisons of international exposure data suggest the range of $\sum_4$PBDEs dust exposures for the general population in the USA would be much greater than Australia[1,4,10].

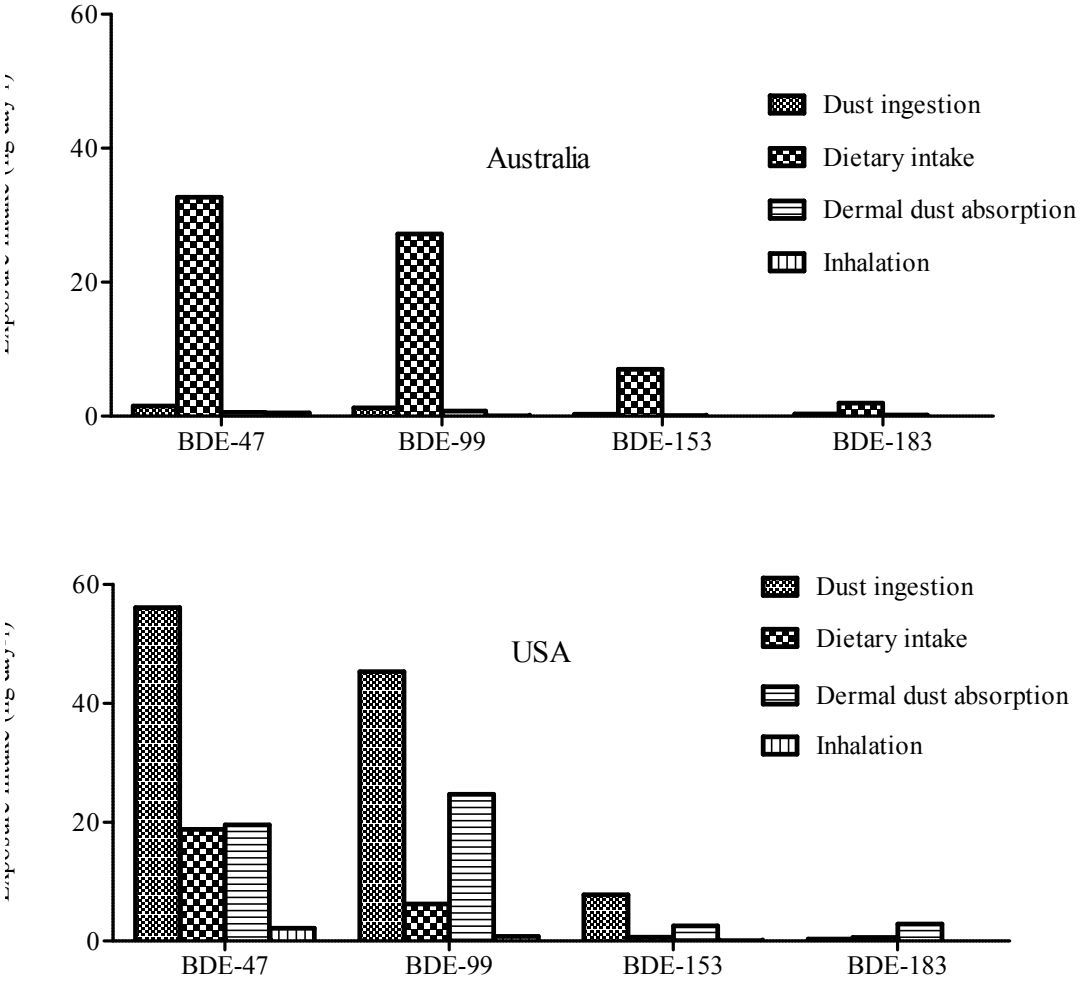

Figure 1. Comparative adult exposure intake rates (ng day^{-1}) for congeners BDE-47, -99, -153 and -183 in the Australian and American[1] general population. Exposure intake data is corrected for absorption factors.

Dietary exposure to PBDEs for the general Australian population is estimated at ~100 ng day^{-1} [8] and on its own explains the body burden in the general Australian population almost entirely. Results from this study suggest on average over 90% of $\sum_4$PBDE exposure is attributable to dietary intake. As shown in Table 1 the predicted body burden for$\sum_4$PBDEs in the adult general Australian population is 10.6 ng g^{-1} lw. In the most recent national assessment of the adult body burden in Australia the $\sum_4$PBDE level was 8.5 ng g^{-1} lw[7].

Table 1. Measured blood levels (ng g^{-1} lw) of the PBDE congeners of interest in Australia[7] compared to predicted values from a simple PK model. Also shown are analogous American data from a recent US EPA study[1].

PBDE congener	BDE-47	BDE-99	BDE-153	BDE-183	$\sum_4$PBDEs
Australia					
Measured Blood Level	4.1	1.5	2.6	0.3	8.5
Modeled Blood Level	3.2	4.8	2.6	<0.1	10.6
Average (model + measured)	3.7	3.2	2.6	0.3	9.6
SD	0.6	2.3	NA	NA	1.5
%RSD	17.4	74.1	NA	NA	15.5
USA					
Measured Blood Level	20.5	5	5.7	NA	31.2
Modeled Blood Level	8.6	12.5	3.9	<0.1	25.0
Average (model + measured)	14.6	8.8	4.8	NA	28.1
SD	8.4	5.3	1.3	NA	4.4
%RSD	57.8	60.6	26.5	NA	15.6

NA – not applicable

Also indicated in Table 1 is the error associated with the predicted and measured levels in the USA work and this study. The % relative standard deviation (%RSD) for $\sum_4$PBDE predicted and measured concentrations in both studies is ~15%. Interestingly in both studies the largest error associated with individual congener concentrations occurs with BDE-99 and may be attributed to uncertainty in the assumed half-life in humans. In the present model the half-life for BDE-99 is estimated to be 5.4 yrs, almost double that estimated for BDE-47. The reasonable match of three of the four main congeners in this simple PK exercise lends credibility to the overall approach of estimating PBDE intakes from exposure media concentrations and contact rates.

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