Australian Government

Department of the Environment and Heritage

Assessment of the concentrations of polybrominated diphenyl ether flame retardants in the Australian population: levels in blood

A consultancy funded by the Australian Government
Department of the Environment and Heritage

Prepared by Leisa Toms, Fiona Harden, Peter Hobson, Olaf Päpke, John Jake Ryan and Jochen Müller
National Research Centre for Environmental Toxicology

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1. *Assessment of concentrations of polybrominated diphenyl ether flame retardants in aquatic environments in Australia*
2. *Assessment of concentrations of polybrominated diphenyl ether flame retardants in indoor environments in Australia*
3. **Assessment of concentrations of polybrominated diphenyl ether flame retardants in the Australian population: levels in blood**

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Foreword

Polybrominated diphenyl ethers (PBDEs), a common class of brominated flame retardants, are a ubiquitous part of our built environment, and for many years have contributed to improved public safety by reducing the flammability of everyday goods.

Recently, PBDEs have come under increased international attention because of their potential to impact upon the environment and human health. Some PBDE compounds have been nominated for possible inclusion on the Stockholm Convention on Persistent Organic Pollutants, to which Australia is a Party. Work under the Stockholm Convention has demonstrated the capacity of some PBDEs to persist and accumulate in the environment and to be carried long distances. Much is unknown about the impact of PBDEs on living organisms, however recent studies show that some PBDEs can inhibit growth in colonies of plankton and algae and depress the reproduction of zooplankton. Laboratory mice and rats have also shown liver disturbances and damage to developing nervous systems as a result of exposure to PBDEs.

In 2004, the Australian Government Department of the Environment and Heritage began three studies to examine levels of PBDEs in aquatic sediments, indoor environments and human blood, as knowledge about PBDEs in Australia was very limited. The aim of these studies was to improve this knowledge base so that governments were in a better position to consider appropriate management actions.

Due to the high costs for laboratory analysis of PBDEs, the number of samples collected for each study was limited and so caution is required when interpreting the findings. Nevertheless, these studies will provide governments with an indication of how prevalent PBDEs are in the Australian population and the environment and will also contribute to international knowledge about these chemicals.

The Department of the Environment and Heritage will be working closely with other government agencies, industry and the community to investigate any further action that may be required to address PBDEs in Australia.

Department of the Environment and Heritage
November 2006

Glossary- Abbreviations

BDE	Brominated diphenyl ethers (used when specifying the congener or degree of bromination)
BFRs	Brominated flame retardants
BSEF	Bromine Science and Environmental Forum
Congener	Closely related chemicals derived from the same parent compound.
DEH	Department of the Environment and Heritage
Dioxins	Common name when referring to polychlorinated dibenzo-*p*-dioxins, polychlorinated dibenzofurans and polychlorinated biphenyls
EnTox	National Research Centre for Environmental Toxicology
EPA	Environmental Protection Agency
HRGC-HRMS	High resolution gas chromatography- high resolution mass spectrometry
IARC	International Agency for Research on Cancer
IUPAC	International Union of Pure and Applied Chemistry
NDP	National Dioxin Programme
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
ng	Nanogram 10^{-9} g
PBDE	Polybrominated diphenyl ether
PCB	polychlorinated biphenyl
pg	Picogram 10^{-12} g
pg g^{-1}	Picogram (10^{-12} g) per gram. Equal to nanogram per kilogram (ng kg^{-1}).
POP	Persistent organic pollutant
QLD	Queensland
SNP	Sullivan and Nicolaides Pathology
TBBP-A	tetrabromobisphenol A

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Project Team:
Fiona Harden (project leader), Jochen Müller, Leisa Toms, Michael Moore - National Research for Environmental Toxicology (EnTox)

Peter Hobson - Sullivan and Nicolaides Pathology

Olaf Päpke - eurofins-ERGO, Hamburg Germany.

John Jake Ryan - Health Canada.

Contributors

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Executive summary

The results of this study provided a measure of the concentrations of the brominated flame retardants (BFRs) - polybrominated diphenyl ethers (PBDEs) in pooled blood serum collected throughout Australia in 2002-03 and 2004-05. Using the following criteria, de-identified samples were collected by Sullivan and Nicolaides Pathology (SNP) from surplus stored pathology samples.

Age stratification: six age groups

- 0-4 years (2004-05 samples only)
- 5-15 years (2004-05 samples only)
- <16 years (2002-03 samples only)
- 16-30 years
- 31-45 years
- 46-60 years
- >60 years

Gender stratification

- males
- females

Regional stratification: five regions representing the regional and population distribution of Australians (2002-03 samples only)

- Southeast urban
- South urban
- Northeast urban
- West urban
- Rural region encompassing most rural regions of Australia

Using these criteria, 8132 samples from the 50 strata were collected and pooled to give 85 pools. It should be noted that the Northeast region alone was chosen for the 2004-05 samples with samples collected from six age groups and both genders. All pooled samples were analysed by eurofins-ERGO, Hamburg, Germany. An inter-laboratory comparison of 10 duplicate pools was performed by Health Canada, Ottawa, Canada.

PBDEs were detected in all strata. The concentration of ΣPBDEs ranged from 6.4 to 80 $ng.g^{-1}$ lipid. Typically BDE-47, -99, -100, -153, -207 and -209 are key components although the detectability, respective concentration and overall contribution of the latter two are more variable. Table ES.1 lists the summary statistics for ΣPBDE concentrations.

Table ES.1 ΣPBDE concentrations ($ng.g^{-1}$ lipid) by year of collection

	2002-03[a]	**2004-05[b]**
> 16 years	15 ± 5 (13)	18 ± 5 (16)
< 16 years	28 ± 8 (29)	29 ± 7 (29)
0-4 years	Na	73 ± 7 (75)

[a]all regions [b]Northeast region Na – not applicable.

An inverse relationship between age and PBDE concentration was observed and the concentrations of these chemicals could be estimated if the age of the individuals in a pool was known. The concentrations were slightly higher in males than females and were similar across all regions of Australia within each of the designated age ranges. While the results of this study did not allow temporal trend analysis, they will provide a baseline from which future monitoring of human samples will indicate whether the concentrations of PBDEs in the Australian population have reached a plateau or are in a state of flux.

The relationship between age and ΣPBDE concentration can be predicted for ages > 2.4 years using the following equation:

$$y = 28.45 * \exp^{(-0.006461x)} + 80.79 * \exp^{(-0.2030x)} - 5.53$$

where y = the predicted ΣPBDE concentration ($ng.g^{-1}$ lipid) and x = age

This is among the first studies to include a representative number of samples from a relatively young age group. When data from both the 2002-03 and 2004-05 samples were investigated an exponential decrease in the concentrations of PBDEs from the youngest age group was seen. The concentrations observed in the 0-4 years age group from the 2004-05 pools, were twice as high as the 5-15 years age group and four times higher than the > 16 years age groups. The data from this study unfortunately provide little information to allow prediction of either the peak concentration or the approximate age of the peak concentration in the body for the 0-4 years group. The elevated concentrations of PBDEs in the youngest population along with the decreasing levels by age is likely to be related to factors including: the history of exposure of the individuals in the pools (ie exposure increased from the past to the present); differences in exposure pathways (ie relatively high exposure of infants through breast milk and other pathways related to child behaviours); and the half-lives of PBDEs in humans (ie excess body burden from childhood is depleted through degradation and growth).

The exposure to PBDEs commenced in the 1970s. Hence, the oldest population received relatively low PBDE exposure. Thus the more recent contamination is subject to dilution because of a large body mass. Mazdai et al (2003) found no difference in PBDE concentrations of paired maternal and cord blood and so neonatal levels reflect maternal levels. The reasons for the higher levels in infants and young children are unclear, but Fangstrom et al (2005) suggest that exposure to persistent organic pollutants (POPs) in children is most probably via environmental sources rather than maternal transfer.

Secondly, PBDEs have half-lives that are substantially shorter in comparison to other POPs eg dioxins (Geyer et al, 2002, Sjodin et al, 2003). Hence the body reaches a steady state for PBDEs much faster and the steady state is expected to be lower relative to the exposure. This means that the effect of past elevated PBDE exposure is observable for a shorter period and current PBDE sera concentrations reflect more or less a relatively recent exposure.

Thirdly, as PBDE containing products are primarily used indoors, the concentrations of BFRs are orders of magnitude higher in indoor air compared to outdoor air (Harrad et al, 2004, Toms et al, 2006). Recent studies have suggested that besides diet, indoor air

inhalation and dust ingestion may be important routes of PBDE exposure in humans (Harrad et al, 2004, Wilford et al, 2005, Stapleton et al, 2005). Infants in particular are subjected to higher exposure to dust because they are in close contact with the floor and tend to use their mouths for sensory perception. Furthermore, PBDEs are an integral component of child specific items including bedding and particularly mattresses. Hence there is a potential for elevated exposure in infants via these pathways.

Overall, the assessment of BFRs in the Australian population demonstrated that concentrations of ΣPBDEs in Australian adults were lower than those observed for adults in North America but higher than those observed for adults in Europe and Asia. As different studies determine different PBDE congeners, comparisons with other studies are made with caution. The concentrations of ΣPBDEs in blood sera from the Australians in the youngest age group were higher than children in Norway and lower than the PBDE concentrations found in children from North America.

The results from this study indicate that a detailed assessment of the specific routes of PBDE exposure for the youngest population together with an evaluation of appropriate management options is warranted if one assumes that there is a potential risk.

1 Introduction

1.1 Background

The incorporation of brominated flame retardants (BFRs) into plastic and other materials is a cost-effective and highly efficient way to reduce flammability and therefore reduce the risk of harm caused by fires. They are incorporated into a variety of manufactured products including electronic and electrical equipment, building materials, carpet, clothing and other textiles. It is the bromine molecule that provides the flame retardant properties of the chemical. Different BFRs are used depending on the application and product requiring flame retardancy. BFRs include among others, the chemicals polybrominated diphenyl ethers (PBDEs) and tetrabromobisphenol A (TBBP-A).

There are two main types of BFR compounds: reactive and additive. Reactive flame retardants form part of the chemical makeup of the polymer and as such are bound to the polymer matrix via covalent bonds, but, some of the reactive flame retardants may not have polymerised and may be released into the environment (de Wit 2002). Additive compounds are mixed with polymers during their production and do not form chemical bonds with the polymer. As a consequence, they are able to separate or leach out of the product over time (de Wit, 2002, Alaee et al, 2003).

PBDEs (Figure 1.1) belong to the additive group of flame retardants. They are synthesised by brominating diphenyl ether in the presence of a catalyst. There are 10 hydrogen atoms in the diphenyl ether molecule and any of these can be exchanged for bromine. Therefore, there are 209 possible PBDE congeners. These are numbered according to the position of the bromine atoms on the ring using the same IUPAC system as that used for numbering polychlorinated biphenyls (PCBs) (see Table 1.1). TBBP-A is mostly used as a reactive flame retardant with limited use as an additive flame retardant (Alaee et al, 2003).

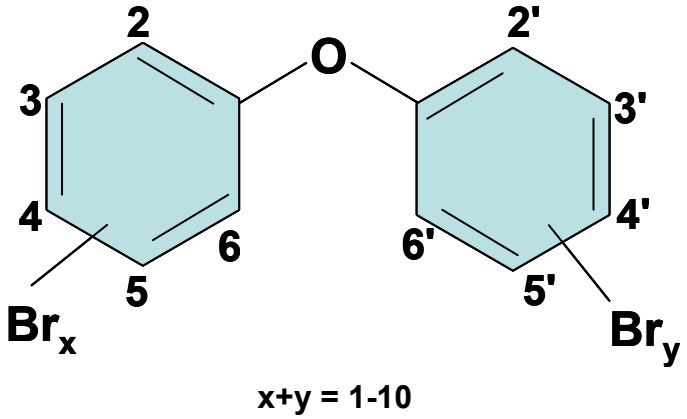


Figure 1.1 The structure of polybrominated diphenyl ethers (PBDEs).

Table 1.1 List of congeners analysed

BDE Congener	Abbreviation
2-Monobrominated diphenyl ether	BDE-1
3-Monobrominated diphenyl ether	BDE-2
4-Monobrominated diphenyl ether	BDE-3
2,4-Dibrominated diphenyl ether	BDE-7
2,6-Dibrominated diphenyl ether	BDE-10
3,4'-Dibrominated diphenyl ether	BDE-13
4,4'-Dibrominated diphenyl ether	BDE-15
2,2',4-Tribrominated diphenyl ether	BDE-17
2,3',4-Tribrominated diphenyl ether	BDE-25
2,4,4'-Tribrominated diphenyl ether	BDE-28
3,3',4-Tribrominated diphenyl ether	BDE-35
2,2',4,4'-Tetrabrominated diphenyl ether	BDE-47
2,2',4,5'-Tetrabrominated diphenyl ether	BDE-49
2,3',4,4'-Tetrabrominated diphenyl ether	BDE-66
2,3',4',6-Tetrabrominated diphenyl ether	BDE-71
2,4,4',6-Tetrabrominated diphenyl ether	BDE-75
3,3',4,4'-Tetrabrominated diphenyl ether	BDE-77
2,2',3,4,4'-Pentabrominated diphenyl ether	BDE-85
2,2',4,4',5-Pentabrominated diphenyl ether	BDE-99
2,2',4,4',6-Pentabrominated diphenyl ether	BDE-100
2,3,4,5,6-Pentabrominated diphenyl ether	BDE-116
2,3',4,4',6-Pentabrominated diphenyl ether	BDE-119
3,3',4,4',5-Pentabrominated diphenyl ether	BDE-126
2,2',3,4,4',5'-Hexabrominated diphenyl ether	BDE-138
2,2'3,4,4',6'-Hexabrominated diphenyl ether	BDE-140
2,2',4,4',5,5'-Hexabrominated diphenyl ether	BDE-153
2,2',4,4',5,6'-Hexabrominated diphenyl ether	BDE-154
2,2',4,4',6,6'-Hexabrominated diphenyl ether	BDE-155
2,3,3',4,4',5-Hexabrominated diphenyl ether	BDE-156
2,2',3,4,4',5,6-Heptabrominated diphenyl ether	BDE-181
2,2',3,4,4',5',6-Heptabrominated diphenyl ether	BDE-183
2,2,3,3',4,4',6,6'-Octabrominated diphenyl ether	BDE-197
2,2',3,4,4',5,5',6-Octabrominated diphenyl ether	BDE-203
2,2,3,3',4,4',5,6,6-Nonabrominated diphenyl ether	BDE-207
Decabromodiphenyl ether	BDE-209

PBDEs have been used in three major commercial products: penta-BDE, octa-BDE and deca-BDE. The penta-BDE product mainly consists of the tetra, penta and hexa-BDEs including BDE -47, -99, -100, -153 and -154; the octa-BDE product consists of hexa, hepta, octa and nona-BDEs including BDE -153, -154, -183, -196, -197, -206 and -207; and the deca-BDE product consists primarily of BDE-209. Both penta and octa-BDE formulations contain the hexa-BDEs -153 and -154. The penta-BDE product is used

mainly in flexible polyurethane foam for mattresses and cushioning; octa-BDE is used in the plastics industry in computer casings and monitors; and deca-BDE is used in high impact polystyrenes and other materials used in electronic and electrical appliances, the automotive industry, construction and building applications as well as textiles (Department of Health and Human Services, 2004). TBBP-A is used, for example, in epoxy resins for printed wiring boards (BSEF, 2005).

PBDEs are imported into Australia in raw chemical form and also already incorporated into manufactured products. In 2003-04, it was estimated that 180 tonnes of deca-BDE product, 20 tonnes of penta-BDE product and less than 10 tonnes of octa-BDE product were imported in raw chemical form into Australia. A decrease in the use of approximately 90% of octa-BDE and approximately 70% of penta-BDE was seen in 2003-2004 compared to 1998-1999 (NICNAS, 2005). The amount of BFRs imported into Australia in manufactured products remains unknown. There are currently no restrictions on the use of PBDEs in Australia although since the end of 2005 the penta- and octa-BDE products are no longer sold, coinciding with the worldwide cessation of penta and octa-BDE product manufacture (NICNAS, 2005). The composition of PBDE commercial products is listed in Table 1.2.

Table 1.2 General composition of PBDE based commercial products (de Wit 2002)

	Congener (%)						
Commercial Product	**Tetra-BDEs**	**Penta-BDEs**	**Hexa-BDEs**	**Hepta-BDEs**	**Octa-BDEs**	**Nona-BDEs**	**Deca-BDEs**
PeBDE	24-38	50-60	4-8				
OcBDE			10-12	44	31-35	10-11	<1
DeBDE						<3	97-98

As with other organohalogen compounds, PBDEs are both lipophilic, (log K_{OW} 4-10) and resistant to degradation and hence are termed persistent. As a result, they accumulate in the environment with a tendency to bioaccumulation (ie accumulation in biota including humans). PBDEs have been detected in various environmental and biological matrices including sediments, marine mammals, fish, bird eggs, human milk, sera and adipose tissue (eg Darnerud et al, 2001, Hites 2004, de Wit 2002). In contrast to other persistent organohalogens, such as dioxin-like compounds, the concentrations of PBDEs in some humans and marine mammal populations are reported to be increasing (Noren and Meironyte, 2000; Hites 2004). Notably for both North America and Europe a plateau with a potential start towards a decrease has been observed (Sjödin et al, 2004; Schecter et al, 2005). The suggested routes of exposure to PBDEs are: ingestion via food, mainly fatty fish, meat, dairy products and human milk; indoor air inhalation; indoor dust ingestion; and-or dermal absorption, particularly in occupationally exposed cohorts (Harrad et al, 2004; Wilford et al, 2005). From the limited data, the half-lives of PBDEs in humans are estimated to range from years (BDE-47) to months (BDE-183) to days (BDE-209) (Sjodin et al, 2003, Geyer et al, 2004). The half-life for TBBP-A is estimated to be two days (Sjodin et al, 2003). These half-lives are considerably shorter than those of dioxins at approximately eight years (Geyer et al, 2002).

Notably, when compared to other organic pollutants such as PCBs and dioxin-like chemicals, a much greater individual variation has been reported in the concentrations of PBDEs in humans (Schecter, 2003). Hence, it is possible that the exposure pathways and potential for bioaccumulation of PBDEs are different to those of other organic

pollutants. The reasons for this are yet to be elucidated. Further research is needed to determine the routes of exposure and subsequent metabolic processing of PBDEs in humans.

Low-level exposure to PBDEs probably occurs in all humans. However, assessment of health risks associated with this type of exposure is complicated and difficult to characterise (McDonald, 2002). No definitive health effects related to the use of PBDEs have so far been reported in humans (Department of Health and Human Services, 2004). Animal studies indicate that commercial products containing deca-BDE mixtures are generally less toxic than the products containing lower brominated PBDEs. Potential risks associated with exposure in animals include liver and thyroid effects, subtle behavioural changes and preliminary findings suggest that PBDEs may impair the immune system.

For most PBDEs, animal studies of carcinogenic effects are not available. Based on the limited evidence of carcinogenicity in animals as well as a lack of human data, deca-BDE has been classified in EPA (US Environmental Protection Agency) Group C (possible human carcinogen) and IARC Group 3 (not classifiable as to its carcinogenicity to humans). Nona, octa, hexa, penta, tetra and tri-BDEs are classified as EPA Group D (not classifiable as to human carcinogenicity) due to no human data and no or inadequate animal data (Department of Health and Human Services, 2004).

In Australia, BFRs or in particular, PBDEs were first determined in blood sera (Harden et al, 2004) and human milk pools (Harden et al, 2005). The results indicated that the concentrations were lower than those observed in North America but higher than in Europe and Asia. No data were available from children or older people in the population and sampling previously focused on females. Hence, the present study was developed to investigate the concentrations of BFRs in the Australian population assessing regional, age and gender differences.

1.2 Objectives

The overall objective of this project was to increase knowledge about BFRs in the Australian population by determining BFR concentrations in human blood sera.

The specific aims of this study were to:

- assess age differences in the concentration of BFRs in human blood sera, with age groups from infants up to the elderly
- assess gender differences in the concentration of BFRs in human blood sera
- assess regional differences in the concentration of BFRs in human blood sera across geographical locations of Australia
- make preliminary investigations of temporal trends by comparing results from samples collected in 2002-03 with samples collected in 2004-05
- assess BFR congener profiles to investigate human exposure to specific congeners and in turn commercial products *and*
- compare analytical results with Australian and international data.

1.3 Scope

To achieve the objectives of this project, the work was carried out in the following stages:

Stage 1 – Sample collection

The majority of blood sera samples used in this study were archived samples that were collected in 2002-03 as part of the Australian Government Department of the Environment and Heritage's National Dioxins Programme (NDP). Additional samples were collected for this study in 2004-05. To allow direct comparison with the 2002-03 samples, the same methodology and pathology company were used for the collection of the 2004-05 blood sera samples. Blood specimens were stratified, collected and pooled based on the following criteria:

- a range of age groups (<16 (0-4, 5-15), 16-30, 31-45, 46-60, >60 years)
- gender
- subjects from rural and metropolitan areas based on the regional groupings:
    - Northeast
    - Southeast
    - South
    - West
    - Rural

Stage 2 – Sample analysis

Analysis of samples was undertaken at eurofins-ERGO Research, Germany for the 35 PBDE congeners listed in Table 1.1. Another BFR chemical, TBBP-A was determined in all samples. In addition, 10 pooled samples were sent to Health Canada for inter-laboratory PBDE analysis.

Stage 3 – Collation and analysis of the data

Raw data were examined to assess PBDE congener profiles; to determine any age, gender or regional differences; to compare 2002-03 and 2004-05 samples; and to compare the results with Australian and international data.

Stage 4 – Report preparation and presentation

The results of the study of BFRs in Australian blood sera are described in this final report.

2. Project design

2.1 Sampling design

The project was designed on the basis of a request from the Australian Government Department of the Environment and Heritage (DEH) to assess the background concentrations of BFRs in the blood sera of the Australian population. Pools of human blood sera collected for the National Dioxins Programme (NDP) in 2002-03 were used as well as additional pools collected in 2004-05. The NDP was designed to assess the levels of dioxins and dioxin-like substances in the Australian environment, food and population. Further details of this programme and the results can be found at www.deh.gov.au-settlements-chemicals-dioxins-index.html.

In this study all samples were stratified according to age, gender and geographical region. To achieve Australia-wide collection of the samples, the study collaborated with Sullivan and Nicolaides Pathology (SNP), a nationwide pathology laboratory based in Brisbane.

Throughout this report blood serum specimens are referred to as samples. The archived NDP samples are referred to as 2002-03 samples and those obtained specifically for this study are referred to as 2004-05 samples. For both sample sets, de-identified serum samples were obtained by SNP from surplus stored sera that had been collected as part of their routine testing procedures for both the 2002-03 and the 2004-05 samples. The 2002-03 samples were collected between 27 November 2002 and 15 April 2003. The 2004-05 samples were collected between 20 April 2004 and 26 August 2005.

Taking into consideration the required timeframe and budget for this project, the Northeast region alone was chosen for the 2004-05 samples. This simplified the sample collection as EnTox and SNP are both located in the Northeast region. An additional age group was added to the 2004-05 samples with blood obtained from children 0-4 years. The 5-15 years age group from the 2004-05 samples replaced the < 16 years age group from the 2002-03 samples. To ensure comparability, the same methodology and procedures were used to collect the samples from both time periods. Samples were obtained according to the stratification criteria outlined below:

Age stratification

- 0-4 years (2004-05 samples only)
- 5-15 years (2004-05 samples only)
- <16 years (2002-03 samples only)
- 16-30 years
- 31-45 years
- 46-60 years
- > 60 years

Gender stratification

- Female
- Male

Regional stratification

- Four urban regions:
  - Northeast (NE) including Brisbane, Tweed and Gold Coast and major population centres in Queensland (NE only for 2004-05 samples)
  - Southeast (SE) including Sydney, Canberra, Wollongong, Newcastle and other major population centres from New South Wales
  - South (S) including Melbourne, Adelaide, Hobart and other major population centres from Victoria
  - West (W) including Perth and other major population centres in Western Australia.
- One rural region (R):
  - Including rural areas from all states and the Northern Territory. Rural areas were defined as those postcodes outside metropolitan or major regional centres.

In order to obtain the samples from the specified regions, SNP was supplied with a list of postcodes that fell under those regions. This list was compiled using the Official Australian Postcode Map, which was visibly searched and postcodes were allocated as Northeast, South, Southeast, West or Rural. Figure 2.1 depicts the regions of Australia from which samples were obtained.

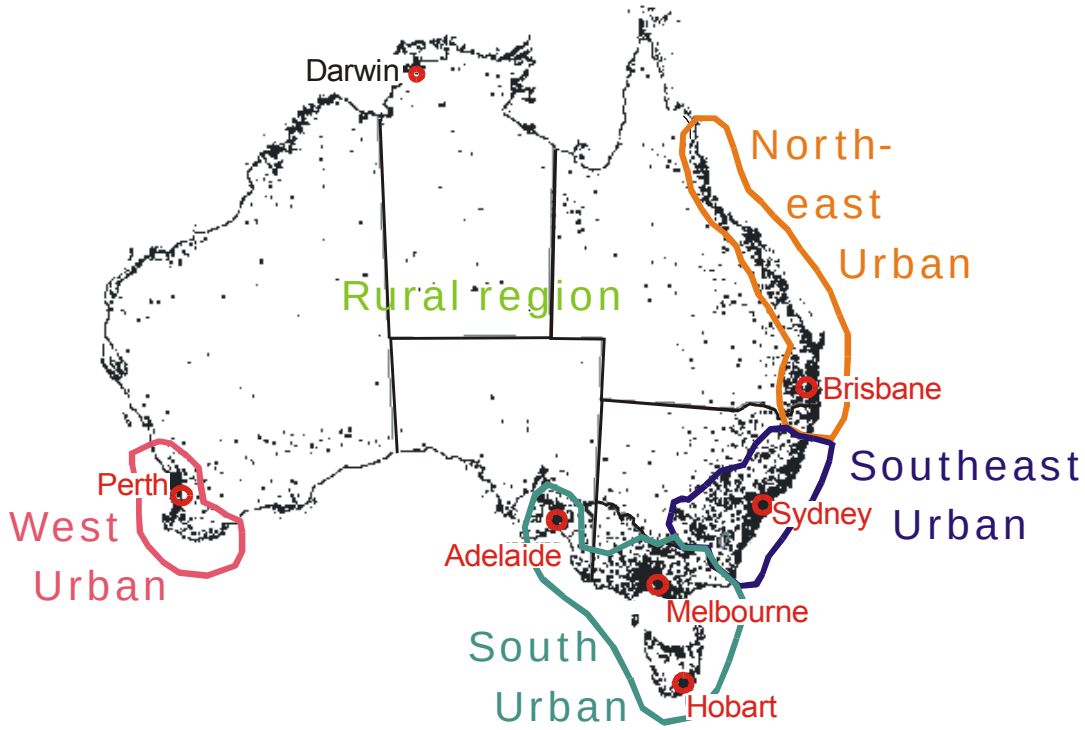


Figure 2.1 Sampling regions of four major population areas and the rural areas of Australia.

The population density of each region is indicated in black, each dot equals 1000 persons (Australian Bureau of Statistics, 2005).

It should be noted that because de-identified samples were used, it was impossible to determine the length of residence in a particular area. In addition, it was impossible to

determine if sample donors were Australian citizens or not. Despite this, the entire group of pools is referred to as a representative group of the Australian population.

For both the 2002-03 and 2004-05 sample collection, it was aimed to collect 200 samples for each stratum. These samples would be divided into two pools of 100 samples each. Stored sera were collected within each stratum until a maximum of 200 samples were achieved or the sample list was exhausted, for each stratum. In some regions particularly in the under 16 years age groups (2002-03 samples), sample collection was more difficult and as a result it was not possible to collect 200 samples in all strata. For this study, 8132 individual samples were used in 85 pools. Table 2.3 shows the number of individual samples per pool and stratum by year of collection

For each sample listed under the five regions, EnTox was provided with the date of birth, postcode, collection date and a laboratory identification number (linked to the sample, not the de-identified donor). This sample list was examined for errors in the date of birth, the collection date and the postcodes.

All dates of birth were examined to ensure that the year was a sensible four digit number. For the date of birth, some samples had the specific day, month and year of birth whereas others had only the year listed. For this reason, for the 2002-03 samples, it was decided to use only the year of birth to determine the age of the sample donor at the time of collection with no consideration given to actual age at date of collection. For the 2004-05 samples, the full date of birth and the full date of collection were used to calculate the age of the donor. The age of the person on the day of collection was used to determine the age for pooling. Hence, in Table 2.1 which shows the year of birth included in each age range, there is an overlap in the year of births for the 2004-05 samples.

Table 2.1 Year of birth included in each age range for 2002-03 and 2004-05 samples.

Age groups	Birth year	
	2002-03 samples	2004-05 samples
0-4 years	not applicable	1999-2005
< 16 years (5-15 years -2004-05)	1987-2002 inclusive	1989-2000
16-30 years	1972-1986 inclusive	1974-1989
31-45 years	1957-1971 inclusive	1960-1974
46-60 years	1942-1956 inclusive	1945-1960
> 60 years	1941 or earlier	1944 or earlier

Postcodes were examined for two reasons; firstly, to ensure that the postcode in the sample list for a certain region actually fell in that region and secondly, to ensure that the postcode was an actual Australian postcode. This was carried out using the Australia Post website Postcode Search and the Official Australian Postcode Map.

Any samples that were identified as having a postcode that was non-existent or a date of birth that was incorrect were removed from the original sample list. Where possible such a sample was replaced with another sample.

2.1.1 Pooling of samples

Once the definitive list of samples was obtained, SNP retrieved the samples for each stratum. Each stratum was divided into two pools, with 100 samples in each pool. The 200 samples were divided into two randomly, by alternately placing samples into Pool 1 and Pool 2. The 200 samples were not placed in any order by way of date of birth, postcode or collection date prior to random allocation to Pool 1 or 2. This assessment of BFRs in the Australian population used one pool per stratum with additional pools used as replicates for selected strata as indicated in Table 2.2.

The volume of blood sera for adult samples was 1ml of each of the 100 samples. This was placed into 100ml solvent rinsed Schott bottles. For samples in the 0-4, 5-15 and <16 years old groups, the stored volume may have been less than 1 ml and so for these samples, the entire sample volume (1 ml or less) was pooled.

Table 2.2 Number of pools analysed by age, gender and region.

	0-4 yrs		< 16 yrs		16-30 yrs		31-45 yrs		46-60 yrs		> 60 yrs	
Gender →	M	F	M	F	M	F	M	F	M	F	M	F
Region ↓												
NE-NDP			2	2	2	2	1	1	2	2	2	2
NE- new	2	2	2	2	2	2	2	2	2	2	2	2
SE-NDP			1	1	2	1	1	1	1	1	1	1
S - NDP			1	0	2	1	1	1	1	1	1	1
W - NDP			1	1	2	1	1	1	1	1	1	1
Rural – NDP			1	1	2	1	1	1	1	1	1	1

It should be noted that one pool was erroneously pooled to consist of a male and a female sample of the same age but from two different regions and therefore this sample was excluded from analysis. This pool was intended to be the sample for 2002-03, female, South, <16 years. This resulted in 85 pools used in total as opposed to the intended 86 pools.

2.2 Ethics

The ethics approval for this study was based on the approval given for the NDP blood study submitted to the University of Queensland Medical Research Ethics Committee and approved on 20 September 2002. An amendment was approved on 17 March 2005 to include the 0-4 years as a separate age group. The project was allocated Clearance Number 2002000656. A copy of the Ethics approval and amendment is given in Appendix A.

2.3 Sample storage and shipping

Prior to shipping, samples were stored at -30° Celsius in an alarmed freezer at EnTox. Samples were air freighted on dry ice to either eurofins-ERGO in Hamburg, Germany or Health Canada, Ottawa, Canada. Samples were received by both laboratories frozen and in good condition. The 2002-03 samples were archived at -30° Celsius in a freezer at EnTox and-or at eurofins-ERGO. All samples were in good condition when defrosted for analysis.

2.4 Sample collection

The number of samples collected in each stratum is shown in Table 2.3.

Table 2.3 Numbers of samples pooled for each stratum.

MALES	Pool	0-4 years	<16 years	16-30 years	31-45 years	46-60 years	> 60 years	TOTAL
Northeast	**1**	-	100	100	-	100	100	400
	2	-	100	100	100	100	100	500
Northeast 04-05	**1**	100	100	100	100	100	100	600
	2	100	100	100	100	100	100	600
Southeast	**1**	-	-	100	-	-	-	100
	2	-	68	100	100	99	100	467
South	**1**	-		100	-	-	-	100
	2	-	66	100	98	100	99	463
West	**1**	-	-	61	-	-	-	61
	2	-	28	61	100	100	100	389
Rural	**1**	-	-	98	-	-	-	98
	2	-	77	98	100	98	101	474
TOTAL		**200**	**639**	**1118**	**698**	**797**	**800**	**4252**

FEMALES	Pool	0-4 years	<16 years	16-30 years	31-45 years	46-60 years	> 60 years	
Northeast	**1**	-	100	100	-	100	100	400
	2	-	100	100	99	100	100	499
Northeast 04-05	**1**	99	99	99	100	100	100	597
	2	101	101	104	100	100	100	606
Southeast	**1**	-	-	-	-	-	-	-
	2	-	73	100	100	99	100	472
South	**1**	-	-	-	-	-	-	
	2	-	-	100	100	100	100	400
West	**1**	-	-	-	-	-	-	-
	2	-	24	100	100	100	100	424
Rural	**1**	-	-	-	-	-	-	-
	2	-	83	99	100	100	100	482
TOTAL		**200**	**580**	**802**	**699**	**799**	**800**	**3880**
TOTAL (male and female)		**400**	**1219**	**1920**	**1397**	**1596**	**1600**	**8132**

2.4.1 Age of participants

The average age and the age range for each of the pooled samples is given in Table 2.4. Note that for the 2002-03 Northeast females aged 46-60 years, a record of individual samples in each of the two pools was not available. For this reason, the calculation of the average age and range was made from all possible samples in the two pools combined.

Human error during the sample pooling process meant that some samples were incorrectly pooled. These were as follows:

For the Northeast region 2002-03 samples

- males 31-45 (pool 2), included one sample aged 49 years
- males >60 years (pool 2) included one sample aged 55 years
- females <16 (pool 2) included one sample aged 18 years
- females 16-30 years (pool 2) included one sample aged 54 years
- females 31-45 (pool 1), included two samples aged 51 years
- females 46-60 (pool 2) included one sample aged 16 years.

For the Northeast region 2004-05 samples

- males aged 0-4 years (pool 1) included two samples where the date of birth was after the collection date
- males 31-45 years (pool 1) included one sample aged 11 years
- males 46-60 years (pool 1) included three samples aged 45 years
- males 46-60 years (pool 2) included one sample aged 37 years
- females 0-4 years (pool 1) included one sample aged 5 years
- females 16-30 years (pool 2) included one sample aged 15 years
- females 46-60 years (pool 2) included one sample aged 44 years.

For the Southeast region

- males aged 16-30 (pool 2) included one sample aged 38 years
- females aged 16-30 years (pool 2), included one sample aged 55 years.

For the South region

- all samples were correctly pooled.

For the West region

- all samples were correctly pooled.

For the Rural region

- males <16 (pool 2) included one sample aged 49 years
- males 16-30 years (pool 1) included one sample aged 63 and one 67 years
- males 16-30 (pool 2) included one sample aged 47 and one 59 years
- males 31-45 years (pool 2) included one sample aged 49 and one 51 years
- females < 16 years (pool 2) included one sample aged 62 years.

Despite the pooling errors, the average age for both pools in all strata were remarkably similar and clearly within the accepted age range designated for that group. Based on the mean age of the donors, the 2004-05 samples for the 5-15 years age group are compared to the 2002-03 samples for the < 16 years age group.

Table 2.4 Age of donors in each pool.

Values indicate the average ages with age range given in parenthesis.

Males	Pool	0-4 years	<16 (5-15) years	16-30 years	31-45 years	46-60 years	> 60 years
Northeast	**1**	N-A	10 (1-16)	25 (16-31)	N-A	55 (47-61)	75 (62-95)
	2	N-A	11 (1-16)	25 (17-33)	40 (32-46)	55 (47-61)	75 (62-91)
Northeast 2004-05	**1**	1.9 (0-4)	10 (5-15)	19 (15-29)	38 (11-45)	52 (45-60)	71 (60-90)
	2	2.8 (0-4)	11 (5-15)	22 (16-30)	36 (30-44)	53 (37-60)	72 (60-96)
Southeast	**1**	N-A	N-A	25 (17-31)	N-A	N-A	N-A
	2	N-A	11 (1-16)	25 (17-31)	40 (32-46)	55 (47-61)	73 (62-91)
South	**1**	N-A	N-A	26 (16-31)	N-A	N-A	N-A
	2	N-A	9 (0-16)	26 (18-31)	39 (32-46)	55 (47-61)	75 (62-95)
West	**1**	N-A	N-A	26 (17-31)	N-A	N-A	N-A
	2	N-A	12 (2-16)	24 (16-31)	39 (32-46)	55 (47-61)	73 (62-102)
Rural	**1**	N-A	N-A	25 (14-31)	N-A	N-A	N-A
	2	N-A	11 (0-16)	25 (14-33)	40 (29-46)	55 (47-62)	73 (62-91)

Females		0-4 years	<16 (5-15) years	16-30 years	31-45 years	46-60 years	> 60 years
Northeast	**1**	N-A	12 (1-16)	26 (17-33)	N-A	54 (47-61)*	76 (62-95)
	2	N-A	12 (1-16)	27 (17-31)	38 (30-46)		75 (62-93)
Northeast 2004-05	**1**	2.1 (0-5)	12 (5-15)	21 (16-30)	38 (30-45)	53 (45-61)	73 (60-100)
	2	2.7 (0-4)	11 (5-15)	18 (15-30)	37 (30-44)	54 (44-60)	73 (60-98)
Southeast	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	12 (2-16)	26 (17-31)	40 (32-47)	54 (47-61)	74 (62-93)
South	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	N-A	27 (17-31)	39 (32-46)	55 (47-61)	76 (62-95)
West	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	13 (2-16)	25 (17-31)	38 (32-46)	54 (47-61)	76 (62-95)
Rural	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	13 (1-16)	25 (17-31)	40 (32-46)	54 (45-61)	75 (60-91)

N-A – not analysed.

* data to calculate separate mean for each pool was not available.

3. Sample analysis

3.1 Analytical methodology

Analysis of the samples was undertaken by two laboratories. All pooled samples were analysed by eurofins-ERGO. Analysis of duplicate quality control samples (10 pools) was undertaken by Health Canada. Full details of the analytical methodologies are given in Appendix B.

The congeners targeted for analysis by eurofins-ERGO were: BDE- 1, -2, -3, -7, -10, -13, -15, -17, -25, -28, -35, -47, -49, -66, -71, -75, -77, -85, -99, -100, -116, -119, -126, -138, -140, -153, -154, -155, -156, -181, -183, -197, -203, -207 and -209.

The congeners targeted for analysis by Health Canada were: BDEs -15, -17, -28, -47, -66, -71, -85, -99, -100, -119, -126, -138, -153, -154, -183, -190 and -205.

For both laboratories the measurement was done by means of isotope dilution technique using HRGC-HRMS (high resolution gas chromatography-high resolution mass spectrometry).

The results are expressed as $ng.g^{-1}$ lipid and are reported excluding the limit of detection values. The mean concentration is expressed ± the standard deviation. The ΣPBDE value, unless specified otherwise, is the sum of the homologue groups for the mono, di, tri, tetra, penta, hexa, hepta, octa, nona and deca-BDEs. It should be noted that the number of congeners per homologue is higher than the number of single determined PBDE-congeners with the same bromination degree. For example, the total tetra-BDE value contains all 42 tetra-BDE congeners and not only the sum of the six listed congeners (BDE #47, #49, #66, #71, #75 and #77). The sum of the homologues results from a separate integration of peaks with a specific retention time which is typical for the bromination degree. Therefore the sum of individual congeners is lower than the respective sum of the homologue group.

It should be noted that four samples (2004-05, male, 0-4 years pool 2, 5-15 years pool 2, 31-45 years pool 2 and > 60 years pool 1) were re-analysed after a) disagreement between replicates and b) data appeared to be outliers in the age versus concentration plot (Figure 4.1). Subsequently EnTox asked for a reanalysis of these samples. The revised data showed that two of the four results were only about 50% of the previous result and one other one about 75%. Only the revised analytical results were used in the report. The original and revised analytical results of these samples are presented in Appendix C.

3.2 Quality Control/Quality Assurance (QC/QA)

QC/QA were undertaken including sampling replication between pools and inter-laboratory calibration. To demonstrate the precision of the analytical method, eurofins-ERGO used a human milk QC pool with a well-known concentration and spiked it with a PBDE mixture at two different levels. For BDEs-47 and -209 the spike concentrations were 2 and 20 $ng.g^{-1}$ lipid, respectively and for other PBDEs the spike

value was 0.4 and 4 ng.g^{-1} lipid, respectively. The results of the accuracy checks by the standard addition technique found the recoveries for almost all compounds spiked at both concentrations ranged from 80 to 100% which is considered good for trace chemical analysis.

In addition to the accuracy checks, for each batch of 10 pooled samples, one blank was included in the analysis. The result of this analysis was that the blank had no relevant influence on the sample concentration.

3.2.1 Inter-laboratory calibration

Health Canada analysed ten duplicate pooled samples to assess inter-laboratory calibration. These samples were the 2002-03, Northeast region, males and females, pool 2, ages < 16, 16-30, 31-45, 46-60 and > 60 years. There were 15 congeners which were analysed by both laboratories, BDE-15, -17, -28, -47, -66, -71, 85, -99, -100, -119, -126, -138, -153, -154 and -183. Full details of the analysis are available in Appendix D.

The analysis by Health Canada found the results of one of the pools, Northeast, female, 31-45 years to be much higher than the rest of the pools. Inspection of the congener profile of this pool (relative amounts of the congeners) showed that BDE-99 was higher in concentration than BDE-47. Such a profile is typical of the commercial penta-product (DE-71 etc) and atypical of biotic samples such as blood or milk. In the latter case, BDE-47 is invariably higher than BDE-99 and other congeners either due to metabolism or selective absorption. This result has been interpreted to mean that somewhere in the analytical process the sample was contaminated with the commercial product resulting in the high concentration and aberrant pattern. The source of this contamination is unknown. This result has been classified as an outlier and not included in the assessment of inter-laboratory calibration. In addition, the result from Health Canada for the pool from Northeast, male, > 60 years, could also be considered as an outlier. In this pool, BDE-47 is greater than BDE-99 but only marginally.

Box 1. Normalised differences

In this report, comparisons between replicated analysis have been made using the normalised difference. The normalised difference between two samples is mathematically defined as:

$$\text{normalised difference (\%)} = \frac{\left|\text{value a} - \text{value b}\right|}{\frac{(\text{value a} + \text{value b})}{2}} \times 100$$

The table below provides a demonstration of the normalised difference (ND) values that would result from a range of differences in sample values.

Sample A (ng g^{-1} lipid)	Sample B (ng g^{-1} lipid)	ND %
1.0	1.2	18
1.0	1.5	40
1.0	2.0	67
1.0	3.0	100
1.0	10.0	160
1.0	100.0	200

The mean normalised difference expresses the average normalised difference for all detected congeners.

The normalised difference (see Box 1) ranged from 4-97% for ΣPBDEs (-47, -85, -99, -100, -153 and -154). When the two outliers were removed the range was 4-16%. Table 3.1 lists the concentrations and normalised differences for the ΣPBDE concentrations by age and gender. For the ten pooled samples analysed by both laboratories, the mean normalised difference (MND) obtained by taking the mean of the normalised differences for congeners BDE-47, -99, -100, -153 and -154 by sample ranged from 11-99%. When the two outliers were removed, the MND ranged from 11-33%.

Normalised differences for individual congeners with low but differing concentrations from eurofins-ERGO and Health Canada can be high and these high normalised difference percentages can have the effect of exaggerating the overall mean normalised difference between the two laboratories.

Table 3.1 Normalised difference (%) for ΣPBDE concentrations
(BDE-47, -85, -99, -100, -153 and -154) of 2002-03 pools from Northeast region.

	Males			Females		
	1	2		1	2	
	Sum PBDE concentration ($ng.g^{-1}$ lipid)		Norm. Diff. (%)	Sum PBDE concentration ($ng.g^{-1}$ lipid)		Norm. Diff. (%)
<16	23	22	4	26	24	8
16-30	21	22	5	11	12	9
31-45	13	12	8	15	43	97
46-60	11	10	10	5.7	4.1	16
>60	8	14	55	5.4	4.6	15

eurofins-ERGO (1) and Health Canada (2) ΣPBDEs (sum of BDEs-47, -85, -99, -100, -153 and -154)

The results from the inter-laboratory comparison from Health Canada are not included in the summary results, only the eurofins-ERGO results for these pools are included.

3.2.2 Replication between pools

Replication between two pools within the same stratum was carried out for eight strata from the 2002-03 samples and 12 from the 2004-05 samples. Table 3.2 shows replicated pools and the ΣPBDE concentration ($ng.g^{-1}$ lipid) for each pool.

High variability in concentration between individual samples has been previously reported (Ryan et al, 2002; Schecter et al, 2003). This level of variability has not been observed with POPs such as dioxin-like chemicals (Sjödin et al, 2003). Hence, higher PBDE results for a particular pool may indicate that an individual or a small group of individuals had elevated concentrations of PBDEs rather than reflecting levels of the group as a whole. The result for each pool represents the mean of the samples that were combined to make up the pool. In this study, there was good reproducibility between the replicate pools of a given stratum.

Since the ages in the two pools were similar (see Table 2.4) and an age trend has become apparent, EnTox expected to see similar concentrations in each pool by age. The results of the replication between pools were similar indicating that pooling was uniform and consistent and it is unlikely that contamination of the samples occurred during sampling or analysis.

Table 3.2 ΣPBDE concentrations (ng.g^{-1} lipid) for replication between pools

Region	Gender	Age	0-4 yrs	< 16 yrs	16-30 yrs	31-45 yrs	46-60 yrs	> 60 yrs
NE – 2002-03	Male	Pool 1	-	26	15	-	13	6.4
		Pool 2	-	24	22	-	12	9.1
	Female	Pool1	-	34	13	-	6.7	15
		Pool 2	-	33	17	-	9.8	8.6
NE - 2004-05	Male	Pool 1	63	24	19	24	28	23
		Pool 2	80	36	28	18	17	12
	Female	Pool 1	73	22	17	15	16	9.8
		Pool 2	77	33	16	16	12	14
S - 2002-03	Male	Pool 1	-	-	15	-	-	-
		Pool 2	-	-	15	-	-	-
SE - 2002-03	Male	Pool1	-	-	32	-	-	-
		Pool 2	-	-	21	-	-	-
R - 2002-03	Male	Pool 1	-	-	22	-	-	-
		Pool 2	-	-	19	-	-	-
W - 2002-03	Male	Pool 1	-	-	22	-	-	-
		Pool 2	-	-	22	-	-	-

3.3 Statistical analysis

Statistical analysis was undertaken using XL Stat (supplementary Microsoft Excel 2000 package). Each pool represents around 100 individuals, but, the use of pooled samples resulted in small sample sizes per stratum. Statistical analysis was not undertaken on less than four pools within a stratum because there was insufficient power for a significant difference to be detected.

4. Concentration of BFRs in the serum of a representative group of the Australian population

This study provided data on PBDE concentrations in 85 pools that were obtained from 8132 individual samples of blood sera from the Australian population. The study also aimed to include the analysis of selected other BFRs - tetrabromobisphenol-A (TBBP-A) and hexabromocyclododecane (HBCD). HBCD was not determined in these samples as the laboratory did not have an established methodology for this analysis. TBBP-A was the only other BFR to be determined in these samples. However the laboratory had not yet established a routine methodology for the analysis of this chemical and to date there have been problems with the detection limit. The results for TBBP-A are reported in Appendix G. EnTox suggest some caution with the interpretation of these results and focus on the interpretation of the PBDEs in this report. The results of the PBDE analysis are discussed in the following sections.

4.1 Overall evaluation of PBDEs

PBDEs were detected in all pools of human blood sera with 24 out of 35 BDE congeners detected. Full PBDE results for all pools are provided in Appendix E. The lipid content (%) of each pool is listed in Appendix F. The relationship between PBDE concentrations and age, gender and region are discussed in Sections 4.2- 4.4.

The ΣPBDE concentrations ranged from 6.4 to 80 $ng.g^{-1}$ lipid. In 2002-03, the mean and median concentrations of ΣPBDEs for all regions for adults aged > 16 years were 15 ± 5 and 13 $ng.g^{-1}$ lipid, respectively. In 2004-05, the mean and median concentrations of ΣPBDEs for the Northeast region only for adults aged > 16 years were 18 ± 5 and 16 $ng.g^{-1}$ lipid, respectively. The mean and median in the 2002-03 samples for all regions, aged < 16 years were 28 ± 8 and 29 $ng.g^{-1}$ lipid. The mean and median in the 2004-05 samples for the Northeast region, aged 5-15 years were 29 ± 7 and 29 $ng.g^{-1}$ lipid, respectively. The mean and median ΣPBDE concentrations in the 0-4 years age group were 73 ± 7 and 75 $ng.g^{-1}$ lipid, respectively. Table 4.1 lists the mean and range of ΣPBDE concentrations for the Northeast region 2002-03 and 2004-05 samples and for the 2002-03 samples for all regions by age and gender.

Table 4.1 Mean (± standard deviation) and range of ΣPBDE concentrations

(ng.g-1 lipid) for males and females by age group. (NE= Northeast)

		All 2002-03		NE 2002-03		NE 2004-05	
Age (years)	**Gender**	**Mean**	**Range**	**Mean**	**Range**	**Mean**	**Range**
0-4	M	N-A	N-A	N-A	N-A	72 ± 12	63-80
	F	N-A	N-A	N-A	N-A	75 ± 3	73-77
5-15 (<16 for 02-03 samples)	M	29 ± 5	24-36	25 ± 1.4	24-26	30 ± 9	24-36
	F	29 ± 11.3	14-42	33± 0.7	33-34	27.5 ± 7.8	22-33
16-30	M	20 ± 5	15-32	18.5 ± 5	15-22	23.5 ± 6.4	19-28
	F	17 ± 4.5	12-25	15 ± 2.8	13-17	16.5 ± 0.7	16-17
31-45	M	19 ± 2.6	16-22	20	N-A	21 ± 4	18-24
	F	15 ± 4.2	8.9-20	16	N-A	15.5 ± 0.7	15-16
46-60	M	14 ± 4.3	9.4-22	8.3 ± 2	6.7-9.8	22.5 ± 7.8	17-28
	F	10 ± 1.8	6.7-12	12.5 ± 0.7	12-13	14 ± 2.8	12-16
>60	M	12.5 ± 6.7	6.4-25	11.8 ± 4.5	8.6-15	18 ± 8	12-23
	F	11 ± 3.6	8.1-16	7.8 ± 2	6.4-9.1	11.9 ± 3	9.8-14

N-A – not analysed.

The results of the current study confirm the preliminary findings of a 2004 Australian study of PBDEs in blood sera (Harden et al, 2004). The 2004 study also used archived samples originally obtained for the NDP study. The 10 pools analysed were the NDP Pool 1 samples of males and females, 31-45 years from all five regions of Australia. These samples were not reanalysed in the current study. Nor were the analytical results of these samples included in the summary results of the current study. The reasons for exclusion of the 2004 results were: the samples were not analysed by eurofins-ERGO which did the analysis of the current study samples and a smaller number of congeners was determined to those of the current study.

When the key congeners were compared between the 2004 study and the current study, for replicate pools, similarities were observed. The mean concentrations of BDE-47 were 4.7 ± 1.7 $ng.g^{-1}$ lipid for the 2004 study and 4.9 ± 1.8 $ng.g^{-1}$ lipid for the current study. For BDE-99, the mean concentration was 2.3 ± 0.9 $ng.g^{-1}$ lipid for the 2004 study and 2.1 ± 0.7 $ng.g^{-1}$ lipid for the current study. The sum of 7 congeners (BDE-28, -47, -99, -100, -153, -154 and -183) was compared between the two studies and was found to be 10.9 ± 3.4 $ng.g^{-1}$ lipid for the 2004 study and 11.2 ± 3.2 $ng.g^{-1}$ lipid for the current study. There was therefore good agreement between the pools studied in 2004 and the replicates analysed in 2005. However, as EnTox wanted to use the full range of congeners analysed by eurofins-ERGO in the current study, the 2004 study results were not included. The 2004 study was important as it was the first Australian study to determine PBDEs in blood sera in males and females. It was the starting point which indicated that the PBDE concentrations in the Australian population were lower than that observed in North American populations but higher than observed in European or Asian populations. Full details of this study are available in Harden et al (2004).

4.2 Evaluation of factors that affect the concentration of PBDEs in humans

The concentrations of lipophilic chemicals such as PBDEs in humans are related to the history of exposure, the specific elimination of the chemical in individuals and the dilution factors, ie body mass. Depending on the chemicals, specific factors such as age, which affects the history of exposure and dilution; gender, which can affect uptake and elimination; and the region of residence, which can affect historical and present exposure, may need to be considered when assessing the human body burden of PBDEs. Other factors may include diet, occupational exposure, specific cultural factors or pathways that can affect exposure and elimination such as smoking. In this study, age, gender and region were the only factors that were able to be included in the assessment of PBDEs.

In addition to the current assessment of BFRs in the Australian population, a study was conducted concurrently to investigate the concentrations of BFRs in indoor environments in Australia. The details of this study are discussed in Toms et al (2006). Briefly, this study collected samples from indoor air, outdoor air, dust and surfaces from homes and offices in South East Queensland. This geographical area corresponded to the Northeast region referred to in the current report. The study of indoor environments found PBDEs to be present in indoor air, outdoor air, dust and surface wipes from homes and offices. Overall, the PBDE concentrations were higher in indoor air than in outdoor air. For indoor air, the concentration of ΣPBDEs ranged from 0.5 -179 pg-m^3 for homes and 15 – 487 pg-m^3 for offices. PBDEs were detected in all dust samples and the ΣPBDE concentration ranged from 87 - 3070 ng-g dust. PBDEs were detected on 9 out of 10 surfaces sampled and the ΣPBDE concentration ranged from non detect to 23500 ng-cm^2.

4.2.1 Relationship between age and the concentration of PBDEs in a representative group of the Australian population

For the evaluation of age as a factor affecting the concentration of PBDEs in the population, the blood samples were pooled into 6 age groups: 0-4 years (2004-05 only), 5-15 years (2004-05 only), <16 years (2002-03 only), 16-30 years, 31-45 years, 46-60 years and > 60 years. The 0-4 years age group was included in the 2004-05 samples of the current study to determine if the concentrations of PBDEs in this age group were higher than in other age groups in the Australian population. Results of the 2004-05 samples 5-15 years age group and the 2002-03 samples < 16 years age group were compared as the average age of both groups was similar. The mean ages for the current study are shown in Table 2.4. The mean age of the donors for the pool, not the age grouping, was used for all age calculations. Figure 4.1 depicts PBDE concentration by age.

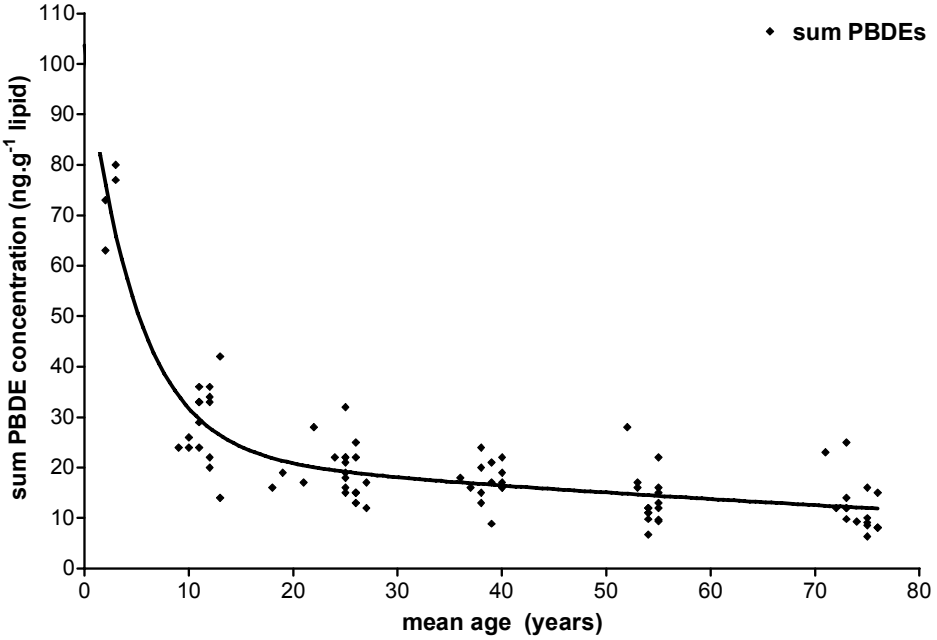


Figure 4.1 ΣPBDE concentration (ng.g^{-1} lipid) in each pool by the respective mean age (years) of donors in each pool

An inverse relationship between age and PBDE concentration was found. This relationship was observed for samples collected in 2002-03 and 2004-05 and was consistent across gender and regional strata. This study is among the first studies to include a representative number of samples from a relatively young age group. When data from all age groups were investigated, an exponential decrease in the concentrations of PBDEs from the youngest age group can be seen. This relationship can be described by the following equation:

$$y = 28.45 * \exp^{(-0.006461x)} + 80.79 * \exp^{(-0.2030x)} - 5.53$$

where y = the predicted ΣPBDE concentration (ng.g^{-1} lipid) and x = age

Examples of PBDE concentration predictions by age are listed in Table 4.2.

Table 4.2 Examples of PBDE concentration (ng.g^{-1} lipid) prediction by age (>2.4 years).

Age	ΣPBDE concentration (ng.g-1 lipid)
2.4	71.1
5	51.3
15	24.1
25	19.2
35	17.2
45	15.8
55	14.4
65	13.2
75	12.0

In this study, the mean and median ΣPBDE concentrations of the 0-4 years old groups were 73 ± 7 and 75 ng.g^{-1} lipid, respectively. The mean age of donors in this group was 2.4 years. The PBDE concentrations in the youngest groups were around a factor two higher than observed in the 5-15 years – 2004-05 samples (29 ± 7, 29) and around a factor four higher than in the > 16 years age groups - 2004-05 samples (18 ± 5, 16). When compared to the 2002-03 samples, the 0-4 years groups were around two and a half times higher than the < 16 years group and around five times higher than in the > 16 years age groups. In comparison to this steep decrease from the very young children to the older children, subsequent decreases with age are apparent but much smaller. There is no clear decrease in the concentration between the two oldest groups. Notably, the conclusion that infants have higher levels is based on only four analyses each of a pool representing 100 sub-samples. The consistently higher levels in all four pools from 0-4 year olds give reason to have confidence in these results, although more work is required to investigate PBDE concentrations in infants and young children.

There have been varying reports of the relationship between PBDE concentrations and age. Some studies have shown no variation in adult serum PBDE concentrations with age (eg Mazdai et al, 2003, Meironyte Guvenius et al, 2003) while Petreas et al (2003) and Schecter et al (2005) found results to be suggestive of an age trend in adult data but no statistically significant correlation was found. Thomsen et al (2002) suggest that no adult age trend exists because BFRs are relatively new chemicals and all age groups would have experienced similar lifetime exposure.

To the authors' knowledge, there are only four peer-reviewed studies which investigate the levels of PBDEs in infants and young children. One study from The Faroe Islands showed that in seven year olds there was no difference in PBDE concentration when compared to adult concentrations (Fangstrom et al, 2005). While in Norway, Thomsen et al (2002) found the concentration of PBDEs in a pool (n=14) of blood serum from a 0-4 years age group was 1.6-3.5 times higher than other age groups (4 to > 60 years). This was confirmed in a further study by Thomsen et al (2005) with another pool (n=20) of blood serum from a 0-4 years age group. This study found the concentration in the youngest age group to be around twice that of the older age groups and also found concentrations in the 5-14 years age group to be lower than the 0-4 years age group but higher than the pools from donors aged greater than 15 years. In addition, a family of four was studied in the US for a newspaper article and the concentrations were found to

be greatest in the 18-month-old infant followed by the five-year-old child, then the mother and father (Fischer et al, 2006).

The data from this study unfortunately provide little information that allows prediction of either the peak concentration or the approximate age of the peak concentration in the body. It cannot be assumed that the study results represent the peak concentration as the use of pooled samples means that the reported concentration of PBDEs in blood is an average of the individual donors' PBDE concentrations. Identifying the likely peak age at which the concentration is the highest may make it possible to evaluate routes of exposure at this peak age. This information could then be used to determine the factors leading to these elevated concentrations in this age group taking into consideration metabolism and other age specific factors.

The elevated concentrations of PBDEs in the youngest population along with the decreasing levels by age contrast with the relationship between age and dioxin concentrations, where the highest concentrations are observed in the oldest population. The differences between PBDEs and dioxins are likely related to differences in the history of exposure, the half-lives of the chemicals and the exposure pathways.

History of exposure: The exposure to PBDEs commenced in the 1970s and increased from then whereas exposure to dioxins commenced much earlier, peaked in the 1970s and continuously decreased in the last three decades to levels which are probably close to an order of magnitude lower than at the peak. Hence, the oldest population received large body burdens of dioxins in the earlier part of their life but relatively low PBDE exposure. Thus the more recent contamination is subject to dilution because of a large body mass. Mazdai et al (2003) found no difference in PBDE concentrations of paired maternal and cord blood and so neonatal levels reflect maternal levels.

Half-life: The second important difference between dioxins and PBDEs relates to their half-lives. PBDEs have half-lives that are substantially shorter in comparison to dioxins (Geyer et al, 2002, Sjodin et al, 2003). Hence the body reaches a steady state for PBDEs much faster and the steady state is expected to be lower relative to the exposure. This means that the effect of past elevated PBDE exposure is observable for a shorter period and current PBDE sera concentrations reflect more or less a relatively recent exposure.

Exposure pathways: The final major difference relates to the differences in exposure pathways. For POPs such as dioxins, food of animal origin is usually the main pathway. For example, with dioxins it is estimated that food and particularly those of animal origin account for > 90 % of the human body burden (Liem et al, 2000). This is in part also because the levels of these traditional POPs are usually similar or even higher in the ambient outdoor air where food is produced compared to indoor air. In addition, as PBDE containing products are primarily used indoors, the concentrations of BFRs are orders of magnitude higher in indoor compared to outdoor air (Harrad et al, 2004, Toms et al, 2006). Recent studies have suggested that besides diet, indoor air inhalation and dust ingestion may be important routes of PBDE exposure in humans (Harrad et al, 2004, Wilford et al, 2005, Stapleton et al, 2005). The presence of these chemicals in Australian air and dust suggest that air inhalation and dust ingestion are

possible routes of PBDE exposure in the Australian population. Fangstrom et al (2005) suggest that exposure to POPs in children is most probably via environmental sources in addition to maternal transfer since no association was found between PBDE congener concentrations in pregnant women and their children at seven years of age. Infants in particular are subjected to higher exposure to dust because they are in close contact with the floor and tend to use their mouths for sensory perception. Furthermore, PBDEs are an integral component of child specific items including bedding and particularly mattresses. Hence there is a potential for elevated exposure in infants via these pathways.

Therefore, even though the physico-chemical properties of PBDEs resemble those of dioxins there remain large uncertainties with respect to the exposure via food and other pathways.

The results from this study indicate that a detailed assessment of the specific routes of PBDE exposure, together with an evaluation of appropriate management options is warranted if one assumes that there is a potential risk.

4.2.2 Gender differences in the concentration of PBDEs in a representative group of the Australian population

To investigate effects of gender on PBDE concentrations, the data were combined by region as no regional differences in PBDE concentration were observed (see Section 4.2.3). Since an age difference was demonstrated (see Section 4.2.1), gender differences were investigated within the discrete age groups.

Figure 4.2 shows the ΣPBDE concentration of the 2002-03 samples by gender and age for all regions. Overall using the Mann-Whitney U-Test (two tailed) no significant difference between the PBDE concentrations in males and females was observable (ie $p=0.123$). When the results are separated by age group to assess a difference between male and female PBDE concentrations, the sample size becomes too small to carry out a statistical evaluation ($n \leq 6$). Therefore considering the relatively small difference there is insufficient power to detect a significant difference between the genders. It should however be noted that the mean concentration of PBDEs for males was consistently higher compared to females for all age groups with the exception of <16 years old group.

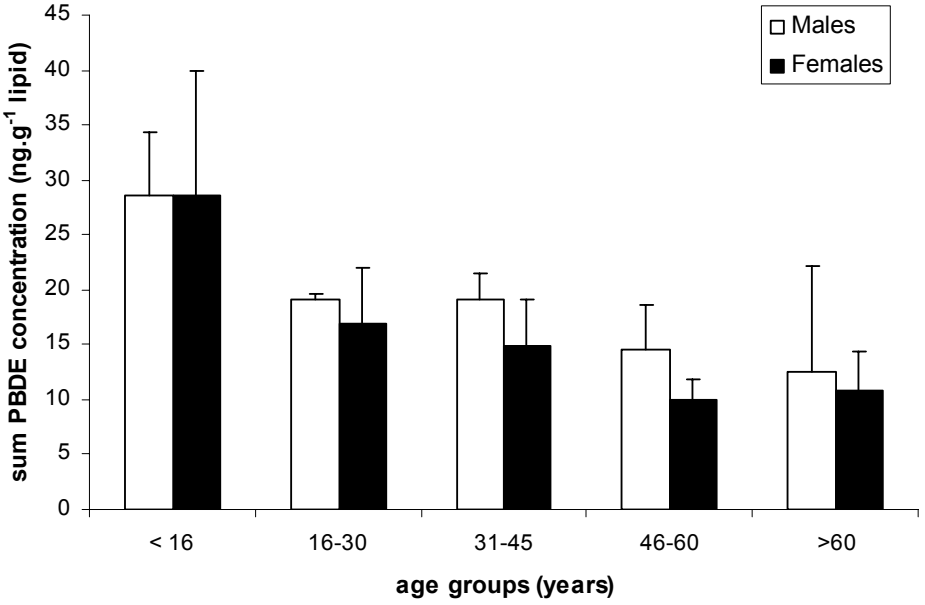


Figure 4.2 Mean (plus one standard deviation) ΣPBDE concentrations (ng.g^{-1} lipid) by gender and age for the 2002-03 samples

The 2004-05 data are depicted in Figure 4.3. Since only two pools were analysed they were both represented and no standard deviation was calculated. Similar to the data from 2002-03 the mean concentrations of PBDEs were > 25 % higher in males for the 16-30, 31-45, 46-60 and >60 years.

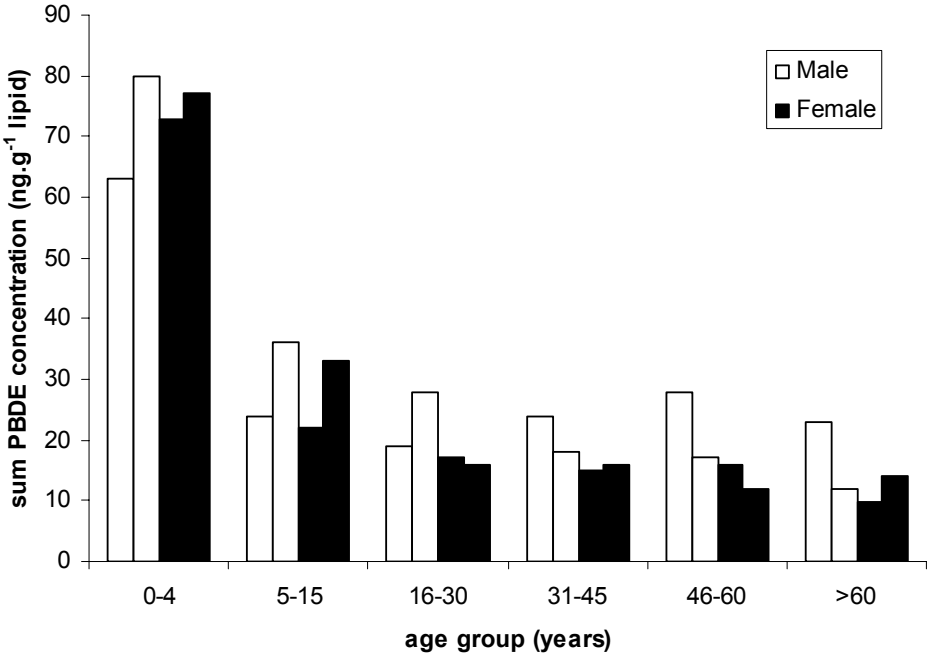


Figure 4.3 Mean ΣPBDE concentration (ng.g^{-1} lipid) by gender and age for the 2004-05.

For males higher PBDE concentrations compared to females have been reported previously although the differences were not significant (Schröter-Kermani et al, 2000,

Thomsen et al, 2002, Harden et al, 2004, Takasuga et al, 2005). For females, Schecter et al (2005) found ΣPBDEs in whole blood to be higher compared to males but again the difference was not significant. Lower PBDE concentrations in female blood may be due to reduction in body burden of persistent BFRs related to pregnancy and breast feeding (Strandman et al, 2000, Thomsen et al, 2002). However, Schecter et al (2003) showed no correlation between PBDE concentrations and number of pregnancies.

4.2.3 Regional differences in the concentration of PBDEs in a representative group of the Australian population

The sample collection in 2002-03 was carried out over five distinct regions and hence a comparison of the analytical results for these pools allowed an evaluation of regional trends in Australia. To facilitate sample collection the 2004-05 samples were collected solely from the Northeast region.

Since an age difference was demonstrated, regional differences were investigated within the discrete age groups. Therefore, statistical analysis of regional variation was not possible as each stratum was made up of only two pools, with the exception of the Northeast region which included data from four pools.

Overall the results (Figure 4.4) indicate that if any regional trends exist they are difficult to assess with data from this study with the resulting differences being far smaller than those obtained as a result of age. Similar results have been found with respect to dioxins in the Australian population (Harden et al, 2003). In contrast, differences in PBDE concentration between geographical regions of a given nation have been reported from Japan. Takasuga et al (2004) indicated that geographical differences were evident but details were not provided. Also, Koizumi et al (2005) found a difference in PBDE concentration in a particular region where a large computer factory was present.

There is no published data available on regional differences related to PBDE exposure in Australia. As the food consumed in Australia is, for the most part, derived from similar sources within the country, exposure of individuals to these compounds via diet is thought to be relatively uniform and not related to region. With regards to exposure from indoor environments a lack of information exists with the few data on PBDEs in indoor air and dust being collected from Southeast Queensland (geographical location referred to as Northeast region in this study, see Section 4.2). Notably, it is likely that the specific exposure related to PBDEs in indoor environments is likely to be more related to specific products used in a given house rather than related to regional differences.

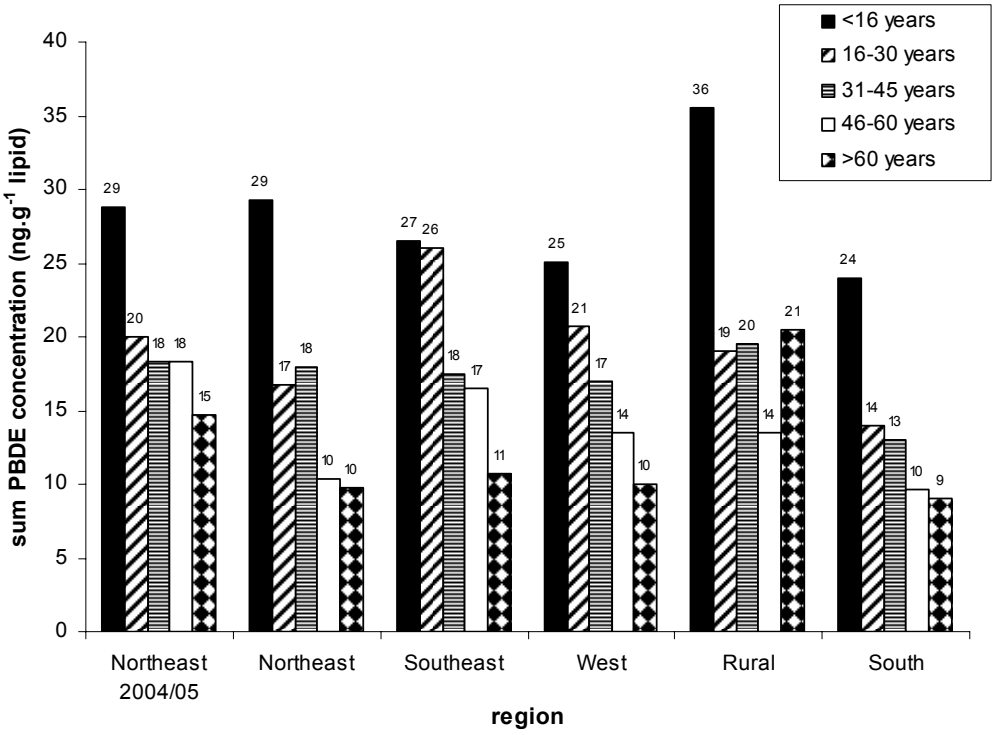


Figure 4.4 Mean ΣPBDE concentrations presented by region and age (combined gender).
The data labels represent the mean value for the strata. Northeast 2004-05 < 16 years represents the 5-15 years age group.

4.3 Temporal trends

For temporal trend analysis, the 2004-05 Northeast results were compared with the results from the blood samples collected in the same region in 2002-03. Results from the 0-4 years age group were not included as they were only available for the 2004-05 samples.

Changes in the PBDE concentrations between the two sampling periods (2002-03 and 2004-05) were evaluated for the males and females. In this study the mean concentration of PBDEs collected from males increased significantly (p=0.04, two-tailed student t-test) from the 2002-03 samples (mean 15.9 ± 6.7, median 15 ng.g^{-1} lipid) to the 2004-05 samples (5 to > 60 years) (mean 22.9 ± 6.7, median 23.5 ng.g^{-1} lipid). In contrast, no change in the concentration was observable for females (p=0.743, two-tailed Mann-Whitney's U-test) from the 2002-03 samples (mean 17.4 ± 9.9, median 15 ng.g^{-1} lipid) to the 2004-05 samples (mean 17.1 ± 6.4, median 15.8 ng.g^{-1} lipid).

Studies have shown an increase in PBDE concentration over the last decades. In Germany, Schröter-Kermani et al (2000) found the median ΣPBDE concentration (BDE-28, -47, - 66, - 85, - 99, 100, - 153 and -154) increased from 3.1 ng.g^{-1} lipid in 1985 to 4.7 ng.g^{-1} lipid in 1999. In Norway, the sum of six BDEs (BDE-28, -47, -99, -100, -153 and -154) increased from 0.4 ng g^{-1} in 1977 to 3.3 ng g^{-1} lipid in 1999 (Thomsen et al 2002). Thomsen et al (2005) further reported a stabilisation or decrease in PBDE concentrations since the late 1990s. In Japan, Koizumi et al (2005) observed a

significant increase in PBDE serum concentrations between 1980 and 1995 from 0.5 to 1.8 ng.g^{-1} lipid. In the US, Petreas et al (2003) analysed serum from the 1960s and the late 1990s and found BDE-47 ranged from non-detected in the 1960s samples to a mean of 50.6 ng.g^{-1} lipid in the late 1990s. Schecter et al (2005) found PBDE concentrations increased by over two orders of magnitude over time from 0.77 ng.g^{-1} lipid in 1973 to between 29.6 and 79.7 ng.g^{-1} lipid in 2003. Sjodin et al (2004) found concentrations of ΣPBDEs (BDE-47, -85, -99, -100, -153 and - 154) to increase over time from 9.6 ng.g^{-1} lipid in 1985-1989 to a maximum of 71 ng.g^{-1} lipid in 1995-1999. Interestingly, Sjodin et al (2004) observed that the concentrations peaked in the late 1990s observing a decrease to 61 ng.g^{-1} lipid in the 2000-2002 samples.

Due to the small time difference between collection periods (2-3 years) the results of the current study did not allow temporal trend analysis and these results should not be used for such an evaluation. However, it would appear that there is a trend towards increasing concentrations over the time period as opposed to the decrease seen in the US (Sjodin et al 2004). The results of the current study provide a baseline from which future monitoring of human samples will indicate whether the concentrations of PBDEs in the Australian population increase, decrease or plateau.

4.4 Congener profiles and contributions of PBDEs

The congener profile was calculated for each sample by dividing the concentration of each congener by the ΣPBDE concentration for that sample to give a percent of each congener to the sum. Where a congener was not detected it was considered to contribute 0% to the sum.

The PBDE congener profile was relatively similar in all pools analysed in this study with the highest contribution from BDE-47 followed by BDE-153 and -99. The average congener profile showed that in the 2002-03 samples, BDE-47 contributed around 32% to the sum followed by BDE-153 at 14% and BDE-99 at 13%, while for the 2004-05 samples, BDE-47 contributed 24%, BDE-153 12% and BDE-99 8% (Table 4.3). For the 2004-05 samples the 0-4 years group had a higher contribution from BDE-99 than -153. For the 2002-03 samples, BDE-209 was detected in 45% of samples. The detection of BDE-209 appeared not to be related to age or gender. In the 2004-05 samples, BDE-209 was detected in only one (8%) of the female samples (0-4 years) and in 50% of male samples. The detection of BDE-209 appeared not to be related to age. Overall, no differences in congener profile by gender, age or region were observed.

Table 4.3 Mean and range contribution (%) of individual congeners to the ΣPBDE concentration by year of collection.

	2002-03		2004-05	
congeners	Mean (%)	Range (%)	Mean (%)	Range (%)
47	32	18-47	24	14-37
153	14	7-23	12	7-25
99	13	n.d.-24	8	4-13
207	8	n.d.-23	11	5-20
100	8	5-16	7	4-13
209	6	n.d.-29	4	n.d.-29
197	6	2-11	5	2-8
154	2	1-3	1	1-2

n.d. – non-detect

The congener profile in the current study was similar to the PBDE profile in Australian human milk (Harden et al, 2005); the preliminary study of PBDEs in Australian blood serum (Harden et al, 2004); and studies of PBDEs in blood from various countries (eg Schecter et al, 2005, Sjodin et al, 2004, Liberda et al, 2005, Sandanger et al, 2005, Thomsen et al, 2003). In contrast to most studies from Europe and North America, a Japanese study (Takasuga et al, 2004) found a different profile where BDE-209 dominated followed by -47, -153, -183, -100 and -99. The authors postulated that the use of penta-BDE commercial product in Japan was discontinued in 1990 and replaced with the deca-BDE product thus changing exposure and resulting in a profile with low serum concentrations of BDE-47 and high concentrations of BDE-209.

For the interpretation of PBDE congener profiles it is important to understand that exposure is only one of many factors that affect the concentration of individual congeners and their ratios or contribution to the sum of all congeners. Besides exposure the body burden of individual congeners and the profile is affected by the bioavailability including resorption of a given congener, the half-life of the congener in the body and even potential formation of lower brominated congeners as a result of debromination of higher brominated congeners. In addition, analysis of pooled samples can only provide a general picture of average congener profiles where individuals may well differ due to specific exposure or even metabolism.

Despite the limitations on the use of congener profiles, Figure 4.5 shows the contribution of the congener groups to the ΣPBDE concentration by collection year for all ages, gender and regions combined. The data may suggest a slight shift in the PBDE congener profile towards higher brominated PBDEs from the first (2002-03) to the second (2004-05) collection period (the exception being deca-BDE which was only detectable in less than half of the samples probably due to its short half-life and low bioavailability). The increase towards higher brominated BDEs in the 2004-05 samples may be indicative of an increase in use of the deca-BDE commercial product and- or the decrease in production of lower brominated BDEs.

Due to the complexity of the factors resulting in the congener profile EnTox were unable to use PBDE profiles from human serum to predict the specific sources of PBDE exposure in the Australian population. Future monitoring of PBDEs in the Australian population will help to assess whether or not the phasing out of the penta and octa-BDE products results in a change in the congener profile.

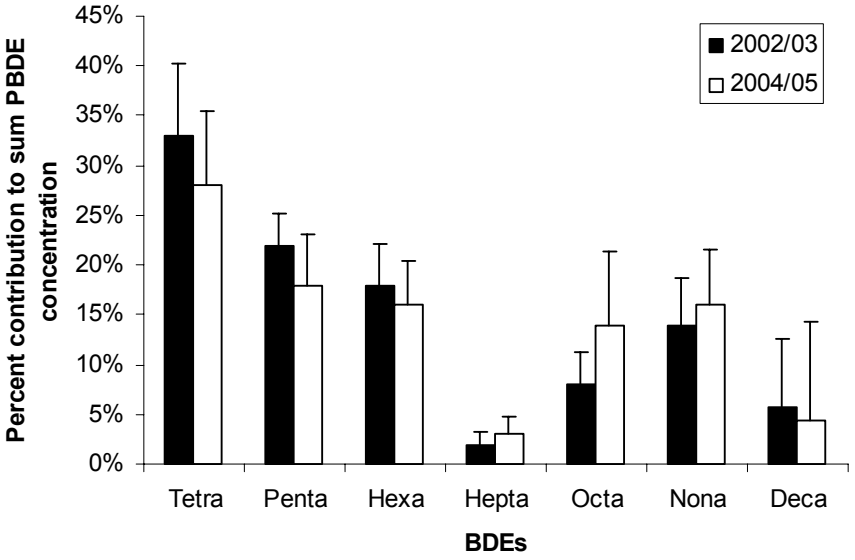


Figure 4.5 Percent contribution of congener groups to the sum PBDE concentration (gender, age and region combined) by year of collection

4.5 Comparison of Australian BFR concentrations with international data

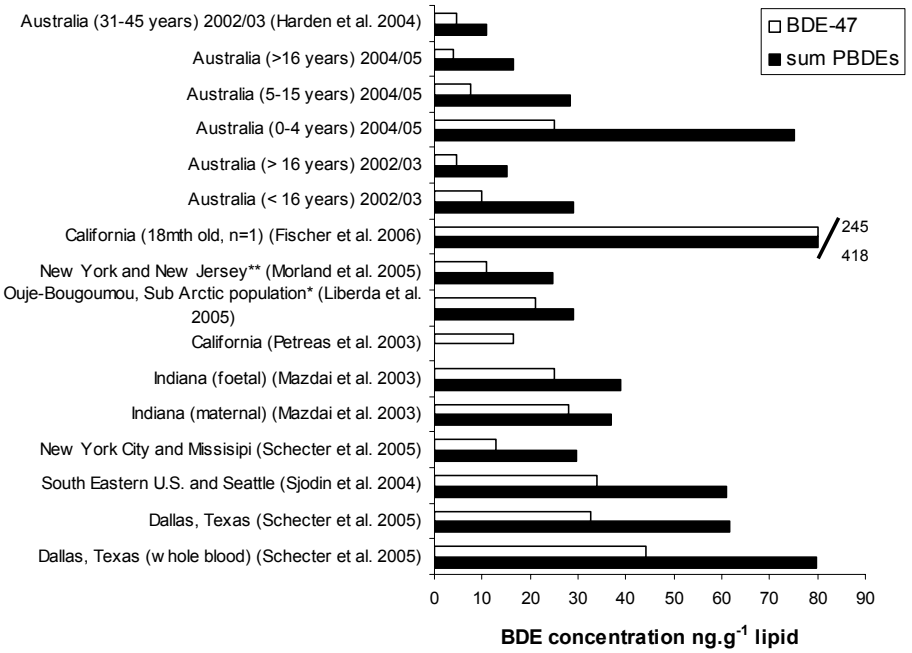
The concentrations of ΣPBDEs and BDE-47 in the Australian adult population are lower than concentrations found in sera from North America (Figure 4.6). However, they are higher than the concentrations found in populations in Asia, continental Europe and the United Kingdom (Figure 4.7). Detailed descriptions of these studies are reported in Appendix H. As occupational exposure was not a focus of the current study, international data on occupational exposure to PBDEs was not included.

There are some issues that must be considered when comparing the results of PBDE concentrations from various international studies. These are noted by LaKind and Berlin (2000) for human milk but are applicable for human sera and include:

- various sampling and analysis methodology, eg, pooled samples versus individual samples
- incomplete reporting, eg, reporting demographic information on the sample donors
- non-representative sampling, eg, use of small sample sizes *and*
- number and types of chemicals, ie different studies include analysis on difference chemicals or congeners.

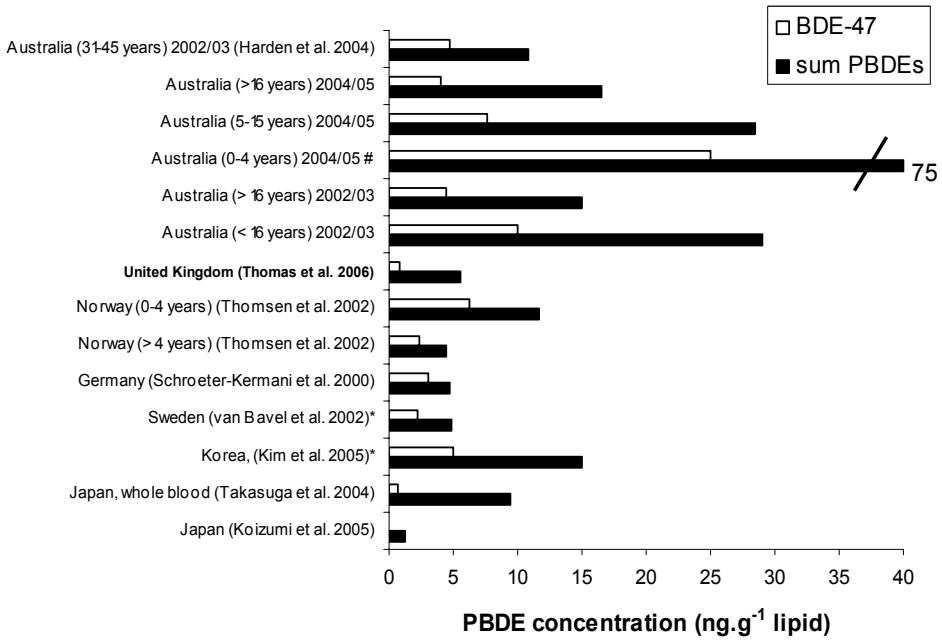
The use of opportunistic samples, often obtained for previous research studies and small sample sizes mean the results obtained may not be representative of the general population or of that specific region or country. Despite these factors, most studies suffer from the same issues and different studies from the same countries report similar values whether they are low serum concentrations in Europe or higher concentrations in North America.

Most of the studies of PBDEs in human blood serum did not compare PBDE results to dietary and-or lifestyle factors while some investigated relationships between PBDE concentrations and age and gender. The value for the median concentration of ΣPBDEs for the current study is the median across all regions of the congeners listed in Table 1.1. The congeners used to obtain ΣPBDE values in the various studies are included in the text if supplied by the authors and concentrations are reported in $ng.g^{-1}$ lipid unless specified otherwise.



*mean ΣPBDEs and BDE-47 ** mean ΣPBDEs only

Figure 4.6 Median concentrations (ng.g^{-1} lipid) of ΣPBDEs and BDE-47 from blood sera **(unless specified otherwise) from Australian and North American studies**



* mean ΣPBDEs and BDE-47 # median = 75 ng.g^{-1} lipid

Figure 4.7 Median concentration (ng.g^{-1} lipid) of ΣPBDEs and BDE-47 from blood sera (unless specified otherwise) from Australian, Asian and European studies

5. Summary of findings

The results of this study provided a measure of the concentrations of PBDEs in pooled blood sera collected throughout Australia in 2002-03 and 2004-05. In total, 8132 de-identified samples, collected by Sullivan and Nicolaides Pathology from surplus stored pathology samples, were stratified by age, gender and region.

The ΣPBDE concentrations ranged from 6.4 to 80 $ng.g^{-1}$ lipid. In 2002-03, the mean and median concentrations of ΣPBDEs for all regions for adults aged > 16 years were 15 ± 5 and 13 $ng.g^{-1}$ lipid, respectively. In 2004-05, the mean and median concentrations of ΣPBDEs for the Northeast region only for adults aged > 16 years were 18 ± 5 and 16 $ng.g^{-1}$ lipid, respectively. The mean and median concentrations in the 2002-03 samples for all regions, aged < 16 years were 28 ± 8 and 29 $ng.g^{-1}$ lipid, respectively. The mean and median concentrations in the 2004-05 samples for the Northeast region, aged 5-15 years were 29 ± 7 and 29 $ng.g^{-1}$ lipid, respectively. The mean and median ΣPBDE concentrations of the 0-4 years old groups were 73 ± 7 and 75 $ng.g^{-1}$ lipid, respectively.

An inverse relationship between age and concentration was observed and the levels of these chemicals in sample pools could be estimated if the age of individuals contributing to the pool were known. The PBDE concentrations in the youngest group (0-4 years) were around two times higher than in the next age group (5-15 years – 2004-05 samples) and around four times higher than in the > 16 years age groups (2004-05 samples). The concentrations were slightly higher in males than females although this was not significant. Concentrations were similar across all regions of Australia within each of the designated age ranges. While the results of this study did not allow temporal trend analysis, they will provide a baseline. With the availability of these data, future monitoring of human samples will allow an assessment of the effectiveness of intervention (ie. the discontinuation in use of PBDEs other than BDE- 209 and the effect their inclusion as a POP under the Stockholm Convention may have) and provide important information on continuous exposure of the population to these chemicals. Furthermore it will allow assessment of whether PBDE concentrations in the Australian population have reached a plateau or are in a state of flux.

Overall, the assessment of BFRs in the Australian population demonstrated that mean concentrations of ΣPBDEs in Australian adults were lower than those observed for adults in North America but higher than those observed for adult in Europe and Asia. The concentrations of ΣPBDEs in blood sera from the Australians in the youngest age group were higher than children in Norway and lower than the PBDE concentrations found in children from North America.

A key uncertainty with PBDEs relates to their toxicology and particularly effects related to potentially chronic exposure. Our study clearly demonstrates that children may be the most vulnerable to these chemicals. A detailed assessment of the specific routes of PBDE exposure for the youngest population together with an evaluation of appropriate management options is warranted if one assumes that there is a potential risk.

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Appendix A Ethics Approval

OFFICE OF RESEARCH AND POSTGRADUATE STUDIES

DIRECTOR
JAN MASSEY

Tel: (07) 3365 3924
Fax: (07) 3365 4455
Email: humanethics@research.uq.edu.au

THE UNIVERSITY OF QUEENSLAND

Cumbrae-Stewart Building
Research Road
Brisbane Qld 4072 Australia
Telephone (07) 3365 3560, (07) 3365 4584
International +61 7 3365 3560, +61 7 3365 4584
Facsimile (07) 3365 4455

Friday, 20 September 2002

Dr Fiona Harden
National Research Centre for Environmental Toxicology

Dear Dr Harden

Concerning:Ethical clearance for project:- ***Determination of the levels of dioxins in the Australian population by analysis of blood serum***

Clearance No: 2002000656

The Medical Research Ethics Committee has approved your project.

Please note that:-

(i) The Clearance number should be quoted on the protocol coversheet when applying to a granting agency and in any correspondence relating to ethical clearance;

(ii) Clearance will normally be for the duration of the project unless otherwise stated in the institutional clearance:

(iii) Adverse reaction to treatment by subjects, injury or any other incident affecting the welfare and/or health of subjects attributable to the research should be promptly reported to the Head of Department and the Behavioural and Social Sciences Ethical Review Committee.

(iv) Amendments to any part of the approved protocol, documents or questionnaires attached to this clearance are to be submitted to the Behavioural and Social Sciences Ethical Review Committee for approval.

Institutional Approval Form For Experiments On Humans Including Behavioural Research

Chief Investigator:	Dr Fiona Harden
Project Title:	Determination of the levels of dioxins in the Australian population by analysis of blood serum
Supervisor:	None
Co-Investigator(s)	Dr Jochen Mueller
Department(s):	National Research Centre for Environmental Toxicology
Project Number:	2002000656
Granting Agency/Degree:	Environment Australia
Duration:	12-15 months

Comments:

Name of responsible Committee:-
Medical Research Ethics Committee
This project complies with the provisions contained in the *National Statement on Ethical Conduct in Research Involving Humans* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:-
Dr Peter Nixon
Chairperson
Medical Research Ethics Committee

Date 15 [illegible] 2002 Signature [illegible]

2002000656

(v) Advisers on 'Integrity in Research'
As part of the University's commitment to the institutional statement, *Code of conduct for the Ethical Practice of Research (1990)*, and the NH&MRC's *National Statement on Ethical Conduct in Research Involving Humans (1999)*, designated positions have been appointed as advisers on integrity in research. The Chairperson of each ethics committee acts in an advisory capacity to provide confidential advice on such matters as misconduct in research, the rights and duties of postgraduate supervisors, and procedures for dealing with allegations on research misconduct within the University. The contact number for the Chairperson of each ethics committee can be obtained from the Ethics Officer.

(vi) The Committee reserves the right to visit the research site and materials at any time during the project.

(vii) It is the Committees expectation whenever possible, this work should result in publication and the Committee would require details to be submitted for our records.

Staff and students are also encouraged to contact either the Ethics Officer (3365 3924), or Chairperson on other issues concerning the conduct of experimentation/research (e.g. involvement of children, informed consent) prior to commencement of the project and throughout the course of the study.

Yours sincerely

Michael Tse
Ethics Officer

Encs.
cc: Head of School, National Research Centre for Environmental Toxicology

THE UNIVERSITY OF QUEENSLAND
AUSTRALIA

Office of Research and Postgraduate Studies

DIRECTOR
Jan Massey

Tel: (07) 3365 3924
Fax: (07) 3365 4455
Email: humanethics@research.uq.edu.au

The University of Queensland
Cumbrae-Stewart Building
Research Road
Brisbane Qld 4072 Australia
Telephone +61 7 3365 3560
Telephone +61 7 3365 4584
Facsimile +61 7 3365 4455

23 March 2005

Dr Fiona Harden
National Research Centre for Environmental Toxicology

Dear Dr Harden

Concerning:Ethical clearance for project:- ***Determination of the levels of dioxins in the Australian population by analysis of blood serum - 17/03/2005 - AMENDMENT***

Clearance No: 2002000656

The Medical Research Ethics Committee has approved your project. The Institutional Clearance Form is attached and you will need to refer to this form for any additional notes and/or special conditions.

Please note that:-

(i) The Clearance number should be quoted on the protocol coversheet when applying to a granting agency and in any correspondence relating to ethical clearance;

(ii) Clearance will normally be for the duration of the project unless otherwise stated in the institutional clearance:

(iii) Adverse reaction to treatment by subjects, injury or any other incident affecting the welfare and/or health of subjects attributable to the research should be promptly reported to the Head of School and the Ethics Committee.

(iv) Amendments to any part of the approved protocol (including change of Investigator/s), documents or questionnaires attached to this clearance must be submitted to the Ethics Committee for approval.

2002000656

(v) Advisers on 'Integrity in Research'
As part of the University's commitment to the institutional statement, *Code of conduct for the Ethical Practice of Research (1990),* and the NH&MRC's *National Statement on Ethical Conduct in Research Involving Humans (1999)*, designated positions have been appointed as advisers on integrity in research. The Chairperson of each ethics committee acts in an advisory capacity to provide confidential advice on such matters as misconduct in research, the rights and duties of postgraduate supervisors, and procedures for dealing with allegations on research misconduct within the University. The contact number for the Chairperson of each ethics committee can be obtained from the Ethics Officer.

(vi) The Committee reserves the right to visit the research site and view materials at any time, and to conduct a full audit of the project.

(vii) It is the Committee's expectation, whenever possible, this work should result in publication and the Committee would require details to be submitted for our records.

Staff and students are also encouraged to contact either the Ethics Officer (3365 3924), or Chairperson on other issues concerning the conduct of experimentation/research (e.g. involvement of children, informed consent) prior to commencement of the project and throughout the course of the study.

Yours sincerely

Per.

Michael Tse
Ethics Officer

Encs
cc: None

THE UNIVERSITY OF QUEENSLAND

Institutional Approval Form For Experiments On Humans Including Behavioural Research

Chief Investigator:	Dr Fiona Harden
Project Title:	Determination of the levels of dioxins in the Australian population by analysis of blood serum – 17/03/2005 - AMENDMENT
Supervisor:	None
Co-Investigator(s)	Dr Jochen Mueller
Department(s):	National Research Centre for Environmental Toxicology
Project Number:	2002000656
Granting Agency/Degree:	Department of Environmental and Heritage (formerly Environment Australia
Duration:	31st December 2005

Comments:

Name of responsible Committee:-
Medical Research Ethics Committee
This project complies with the provisions contained in the *National Statement on Ethical Conduct in Research Involving Humans* and complies with the regulations governing experimentation on humans.

Name of Ethics Committee representative:-
Dr Bill Vicenzino
Chairperson
Medical Research Ethics Committee

Date: 23.3.2005 Signature:

Appendix B Analytical methodology

Eurofins-ERGO, Germany

General comments for analytical procedures

Samples for the requested analytes were analysed in sequences of 10 unknown samples, 1 blank and 1 known sample (QC-pool). Quantification was performed by use of a 5 point calibration curve. For all sequences a so called 'recal' was measured in parallel.

PBDEs (polybrominated diphenyl ethers)

The single components analysed are shown in the following table. For each compound a native reference standard was available. Samples were extracted-solved by means of ultratrace solvents. Before the extraction the following internal standards (all ^{13}C-UL labelled) were added to the samples:

IUPAC-code	Internal standards (^{13}C-UL) PBDE	
3	4-	Mono-BDE
15	4,4'-	Di-BDE
28	2,4,4'-	Tri-BDE
47	2,2',4,4'-	Tetra-BDE
99	2,2',4,4',5-	Penta-BDE
153	2,2',4,4',5,5'-	Hexa-BDE
154	2,2',4,4',5,6'-	Hexa-BDE
183	2,2',3,4,4',5',6-	Hepta-BDE
197	2,2',3,3',4,4',6,6'-	Octa-BDE
207	2,2',3,3',4,4',5,6,6'-	Nona-BDE
209	2,2',3,3',4,4',5,5',6,6'-	Deca-BDE

The fat content was determined by gravimetry. For the determination of polybrominated diphenyl ethers the sample extract was taken up in n-hexane and treated by a clean-up including H_2SO_4-SiO_2. Afterwards the extract was reduced to 10 µl in a nitrogen stream. After addition of the syringe standard 2,2',3,4,4',6-Hexabromdiphenylether (Hexa-BDE 139 13C-UL labeled), the PBDEs were measured by high resolution gaschromatography and mass spectrometry.

The measurement was done by means of HRGC-HRMS (high resolution gas chromatography- high resolution mass spectrometry, VG Autospec resp. Finnigan MAT 95 XL) using a DB 5 column for gaschromatographic separation. The quantification was performed by means of internal - external standards (isotope dilution).

Due to the chemical and physical properties of PBDEs, data were reported on a lipid basis.

TBBP-A (Tetrabromobisphenol A)

The analytical methodology was as for PBDEs above, however, before the extraction the following ^{13}C-UL-labeled internal standard was added to the sample:

^{13}C-TBBP-A (^{13}C-UL-labeled)

After the spiking, the sample was extracted with appropriate solvents for ultratrace-analyses (eg nanograde) and afterwards a column clean up was performed. The measurement was done by means of high resolution gaschromatography and low resolution mass spectrometry (HRGC-LRMS) using a HP 5-MS column.

Due to the chemical and physical properties of TBBP-A data were reported on a lipid weight basis.

Health Canada

Polybrominated diphenyl ethers

General comments for analytical procedures

Each sample batch contained a laboratory blank to gauge the amount of analyte from the laboratory processing. This amount was subtracted from the total amount in the unknown sample prior to calculation of concentration. The blank value can be significant particularly for certain PBDE congeners such as 47 and 99 and, in certain cases, can also be the main determinant of the detection limit. A reference or repeat sample was also analysed in every batch to ensure the analytical process was under control and results comparable to previous work and other laboratories. Detection limits for persistent organic pollutants (POPs) in human blood depend on the sample size, its lipid content, and contribution from the laboratory blank. Typically for a 50 mL sample of human blood containing about 0.5 % lipid, the limit of detection (LOD) on a lipid basis would be about 0.5 ppb for PBDE 47. A significant response from the laboratory blank adversely affects the limit of detection (LOD).

Polybrominated diphenyl ethers

To each sample 500 pg of carbon-13 labelled surrogate standards were added to be determined using the isotope dilution internal standard method. These surrogates consisted of: eight of the most common PBDE congeners (di- up to hepta- bromo) as well as the deca- (BDE-209).

For each volume of plasma, an equal volume of ethanol and an equal volume of saturated aqueous ammonium sulfate were added followed by three volumes of hexane. The mixture was then homogenised with a mechanical homogeniser and, after separation of phases, the hexane portion was withdrawn. The extraction, mixing, and separation were repeated with 1.5 volumes of hexane.

The combined hexane extracts were filtered, washed with water to remove residual ethanol, dried over a little anhydrous sodium sulfate, and evaporated to dryness on a rotary evaporator under vacuum. The residue was weighed over a period of time (12-24 hrs) until constant weight was obtained. This weight was used to calculate the lipid content of the sample. The lipid residue containing the persistent organic pollutants (POPs) was reconstituted in about 150 mL hexane and defatted by shaking in a separatory funnel with 20 mL portions of concentrated sulfuric acid. The acid portion was withdrawn and discarded and the acid treatment was repeated up to 10 times until the acid portions were clear and pale yellow. The hexane extract was washed with water, dilute aqueous base, again with water, dried and concentrated to a small volume (circa 1-2 mL) in preparation for column purification.

Extract purification was performed using a) Acid silicate and Florisil columns: The hexane extract was added to a small silicate column containing strong acid to remove traces of interfering compounds. The elute from this column went directly onto a heat activated (150 EC) and not water deactivated Florisil column (1.5 g). Two fractions were collected: 1) about 40 mL of hexane (discarding the first 3.5 mL containing polar lipids) consisting of: a) most of the PCBs including all eight mono *ortho* congeners,

b) most of the organochlorine pesticides, and c) a few higher brominated octa- to deca-PBDE congeners, 2) 60 mL of dichloromethane containing a) dioxins, furans, and the four non *ortho* PCBs and b) the bulk of the PBDEs. Fraction 1 (most PCBs and organochlorines (OCs) and a few PBDEs) only was evaporated passively to 50 uL by weight in steps. Twenty (20) uL were taken and made up to 40 uL with recovery standard prior to injection on high resolution GC-MS. Depending on the amount of blood used and its particular content, Fraction 1 may have had to be rechromatographed on a second silica column by rejecting the first 3-4 mL of eluent containing interfering polar lipid residues and collecting the next 30 or so mL of hexane.

b) Carbon column:
fraction 2 (dioxins, furans, non *ortho* PCBs and all but a few higher brominated PBDEs) from the Florisil column was further purified on a Carbopack C carbon column. The PBDEs were not adsorbed and pass through the column with hexane (fraction 2a). The dioxins, furans and non *ortho* PCBs were sorbed from the hexane and desorbed in the forward direction with toluene (fraction 2b). Both purified extracts (F2a; F2b) were taken to dryness in steps and reconstituted in 5 or 10 uL of toluene containing recovery standards prior to MS.

The measurement was done using gas chromatography (GC)-mass spectrometry (MS)
A) The GC is a Agilent 6890 containing:
i) for fraction F2a (PBDEs) - a DB-5 MS bonded phase capillary column of 15 m length, 0.25 mm id, and 0.1 μm thickness with retention gap. Injection of 1 μL was by the on column method at 80 EC with a fast ramp to 300 EC. The GC column was programmed in steps from 80 EC up to 300 EC in a total run time of about 15 min.

B) The MS is a Micromass Auto Spec Ultima operating in the positive electron impact (EI) mode at 40 eV ionisation energy, source temperature of 250 E C, interface temperature of 250EC (270 EC for BDEs), and mass resolution (10 % valley) of 10 K. Up to 14 masses were monitored in each group of six or more groups in the selected ion mode (SIM) usually with two masses for each isotopomer plus a lock and dummy mass. Identification of each analyte was governed by its gas chromatographic retention time (within 1.2 seconds of the standard), correct amu ion ratio (within 15% of standard), and a signal to noise ratio of at least 3:1. Under these conditions the detectability for PBDE 47 was about 1 picogram.

C) i) PBDE fraction (F2a): masses monitored were the M+ (except deca- which was the M^+-2Br value) and include the di-, tri-, tetra-, penta-, hexa-, hepta-, octa-, nona-, and deca- homologues from a little over 320 amu up to about 880 amu.

D) Quantification: a standard curve was established consisting of a six to eight point concentration level of carbon 12 analytes with constant concentration of carbon 13 isotopomers. Concentrations in the sample were calculated from the standard curve using the isotope dilution internal standard method comprising relative response factors (RRFs), concentration changes, and amounts of whole weight and lipid in the unknown sample. Results were expressed in ng-kg (parts per trillion; ppt) on both a whole and lipid basis. Recoveries of the carbon-13 surrogates added at the beginning were calculated using the recovery standards themselves added just prior to GC-MS.

Appendix C Reanalysis of outliers

Table C.1 Results of reanalysis of outliers (ng.g^{-1} lipid)

		REANALYSIS		REANALYSIS
Sample code	**MA, Pool 2 Male 0 - 4 years Pool 2, 30 ml**	**MA, Pool 2 Male 0 - 4 years Pool 2, 30 ml**	**MB, Pool 2 Male 5 - 15 years Pool 2, 30 ml**	**MB, Pool 2 Male 5 - 15 years Pool 2, 30 ml**
BDE #1	n.d.(2)	n.d. (0.9)	n.d.(4)	n.d.(1)
BDE #2	n.d.(1.0)	n.d.(0.4)	n.d.(2)	n.d.(0.8)
BDE #3	n.d.(0.8)	n.d.(0.5)	n.d.(2)	n.d.(0.6)
total Mono-BDE	n.d.	n.d.	n.d.	n.d.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #13	n.d.(0.01)	n.d.(0.01)	0.023	n.d.(0.02)
BDE #15	n.d.(0.1)	n.d.(0.05)	0.27	n.d.(0.06)
total Di-BDE	n.d.	n.d.	0.30	n.d.
BDE #17	n.d.(0.02)	0.020	0.13	n.d.(0.01)
BDE #25	n.d.(0.02)	n.d.(0.01)	0.075	n.d.(0.01)
BDE #28	0.56	0.74	6.9	0.22
BDE #35	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)
total Tri-BDE	0.67	0.96	8.0	0.45
BDE #47	30	31	23	9.4
BDE #49	0.36	0.16	0.18	0.07
BDE #66	0.15	0.26	0.23	0.07
BDE #71	0.051	n.d.(0.01)	n.d.(0.06)	n.d.(0.01)
BDE #75	0.029	0.041	n.d.(0.06)	0.014
BDE #77	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)
total Tetra-BDE	34	34	25	10
BDE #85	0.31	0.87	n.d.(0.01)	0.22
BDE #99	10	9.7	4.8	3.1
BDE #100	8.7	8.5	2.9	2.7
BDE #116	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.2)	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)
total Penta-BDE	22	20	8.4	6.7
BDE #138	0.21	0.17	0.073	0.071
BDE #140	0.14	0.11	0.049	0.038
BDE #153	8.8	8.2	7.3	7.7
BDE #154	1.2	1.1	0.37	0.38
BDE #155	0.14	0.10	n.d.(0.05)	0.027
BDE #156	n.d.(0.05)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)
total Hexa-BDE	12	11	8.4	8.9
BDE #181	n.d.(0.2)	n.d.(0.07)	n.d.(0.09)	n.d.(0.1)
BDE #183	0.51	0.77	0.29	0.44
total Hepta-BDE	1.3	0.91	0.29	0.67
BDE #197	2.1	1.6	1.5	1.3
BDE #203	0.48	0.38	0.47	0.53
total Octa-BDE	5.6	2.9	2.8	1.8
BDE #207	4.8	3.9	2.9	3.7
total Nona-BDE	7.2	6.1	4.2	3.7
BDE #209	n.d.(4)	5.1	4.5 (M)	3.3
total BDE	**82**	**80**	**62**	**36**

n.d. = not detectable,
detection limit in ()

Table C.1 cont.

		REANALYSIS		REANALYSIS
Sample code	**MD, Pool 2 Male 31 - 45 years Pool 2, 30 ml**	**MD, Pool 2 Male 31 - 45 years Pool 2, 30 ml**	**MF, Pool 1 Male > 60 years Pool 1, 30 ml**	**MF, Pool 1 Male > 60 years Pool 1, 30 ml**
BDE #1	n.d.(2)	n.d.(0.8)	n.d.(8)	n.d.(0.8)
BDE #2	n.d.(2)	n.d.(0.5)	n.d.(5)	n.d.(0.5)
BDE #3	n.d.(1)	n.d.(0.4)	n.d.(4)	n.d.(0.4)
total Mono-BDE	n.d.	n.d.	n.d.	n.d.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #13	0.013	n.d.(0.01)	n.d.(0.01)	0.018
BDE #15	n.d.(0.1)	n.d.(0.06)	n.d.(0.10)	n.d.(0.04)
total Di-BDE	0.013	n.d.	n.d.	0.018
BDE #17	0.020	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #25	0.036	n.d.(0.01)	n.d.(0.03)	n.d.(0.01)
BDE #28	0.22	0.12	n.d.(0.2)	0.1
BDE #35	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.06)
total Tri-BDE	0.62	0.46	0.074	0.27
BDE #47	6.0	4.2	4.2	4.1
BDE #49	n.d.(0.01)	0.05	0.037	0.048
BDE #66	n.d.(0.02)	0.04	0.036	0.046
BDE #71	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	n.d.(0.03)
BDE #75	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)
total Tetra-BDE	6.3	4.9	4.5	4.7
BDE #85	n.d.(0.01)	0.1	0.12	0.1
BDE #99	1.6	1.2	1.3	1.2
BDE #100	1.00	1.10	1.0	0.9
BDE #116	n.d.(0.01)	n.d.(0.02)	n.d.(0.04)	n.d.(0.01)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.02)	n.d.(0.03)	0.12	n.d.(0.03)
total Penta-BDE	2.8	2.7	2.6	2.4
BDE #138	0.052	n.d.(0.05)	0.10	n.d.(0.03)
BDE #140	0.034	n.d.(0.02)	0.071	n.d.(0.02)
BDE #153	2.4	2.3	2.1	1.8
BDE #154	0.20	0.20	0.26	0.27
BDE #155	n.d.(0.02)	n.d.(0.03)	n.d.(0.05)	n.d.(0.02)
BDE #156	n.d.(0.02)	n.d.(0.04)	n.d.(0.06)	n.d.(0.03)
total Hexa-BDE	2.9	2.9	2.8	2.5
BDE #181	n.d.(0.1)	n.d.(0.1)	n.d.(0.2)	n.d.(0.1)
BDE #183	0.58	0.38	n.d.(0.3)	0.28
total Hepta-BDE	1.6	0.59	0.50	0.44
BDE #197	1.4	1.1	1.8	1.2
BDE #203	0.46	n.d.(0.5)	n.d.(0.3)	n.d.(0.2)
total Octa-BDE	2.5	1.1	5.7	1.2
BDE #207	2.9	2.7	5.0	4.5
total Nona-BDE	4.8	2.7	8.7	4.5
BDE #209	13 (M)	2.6	6.7 (M)	6.5
total BDE	**35**	**18**	**32**	**23**

n.d. = not detectable,
detection limit in ()

Appendix D Inter-laboratory calibration

Table D.1 Results (ng.g^{-1} lipid) from eurofins-ERGO (1) and Health Canada (2) for Northeast region, females and normalised difference (%) for the results of the congeners analysed by both laboratories.

Region	Northeast													
Gender	Female													
Age	< 16 years			16-30 years			31-45 years			46-60 years			> 60 years		
	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.
sample weight (g)	15.268	18.020		15.128	18.58		15.049	17.86		15.083	18.520		15.537	18.600	
lipid content (%)	0.492	0.370		0.553	0.477		0.605	0.503		0.652	0.587		0.639	0.580	
BDE #15	n.d.(0.1)	n.d. (0.011)	n.c.	n.d.(0.09)	0.1	n.c.	n.d.(0.1)	0.6	n.c.	n.d.(0.08)	n.d. (0.004)	n.c.	n.d.(0.3)	0.010	n.c.
BDE #17	n.d.(0.02)	0.044	n.c.	n.d.(0.02)	n.d. (0.06)	n.c.	0.027	n.d. (0.180)	n.c.	n.d.(0.01)	0.016	n.c.	n.d.(0.02)	n.d. (0.042)	n.c.
BDE #28	n.d.(0.2)	0.546	n.c.	n.d.(0.2)	0.4	n.c.	0.72	1.03	35.73	n.d.(0.1)	0.383	n.c.	n.d.(0.2)	0.650	n.c.
BDE #47	12.5	11.9	4.7	5.9	6.0	1.4	8.0	15.7	64.6	3.3	2.3	35.2	2.6	2.5	4.3
BDE #66	n.d. (0.1)	0.1	n.c.	n.d. (0.07)	0.2	n.c.	n.d. (0.1)	0.3	n.c.	n.d. (0.08)	0.009	n.c.	n.d. (0.04)	0.02	n.c.
BDE #71	0.0	0.1	n.c.	n.d. (0.01)	0.2	n.c.	0.0	0.6	n.c.	n.d. (0.02)	0.02	n.c.	n.d. (0.01)	0.04	n.c.
BDE #85	0.3	0.3	1.3	0.04	0.03	36.8	0.1	0.6	165.7	0.1	0.007	152.9	0.03	0.04	27.2
BDE #99	5.1	5.4	6.0	1.6	2.9	55.9	3.4	17.8	135.6	n.d. (0.8)	0.5	n.c.	0.8	0.9	6.6
BDE #100	3.2	2.5	24.9	1.3	1.2	11.6	1.5	3.5	83.0	0.8	0.5	50.5	0.6	0.4	32.1
BDE #119	n.d. (0.01)	n.d. (0.012)	n.c.	n.d. (0.01)	n.d. (0.038)	n.c.	n.d. (0.01)	n.d. (0.187)	n.c.	n.d. (0.01)	n.d. (0.008)	n.c.	n.d. (0.01)	n.d. (0.021)	n.c.
BDE #126	n.d. (0.04)	0.0	n.c.	n.d. (0.02)	n.d. (0.026)	n.c.	n.d. (0.04)	n.d. (0.071)	n.c.	n.d. (0.05)	n.d. (0.005)	n.c.	n.d. (0.02)	n.d. (0.026)	n.c.
BDE #138	n.d. (0.09)	0.0	n.c.	n.d. (0.04)	n.d. (0.111)	n.c.	n.d. (0.04)	n.d. (0.315)	n.c.	0.0	n.d. (0.015)	n.c.	n.d. (0.03)	n.d. (0.111)	n.c.
BDE #153	4.0	3.5	13.4	1.7	1.4	21.7	1.5	2.7	60.1	1.3	0.8	46.5	1.1	0.8	33.2
BDE #154	0.5	0.5	6.1	0.2	0.4	44.2	0.3	2.6	153.0	0.2	n.d. (0.013)	n.c.	0.2	n.d. (0.091)	n.c.
Sum of 47, 85, 99, 100, 153 and 154 (excl. LOD)	**26**	**24**	**8.0**	**11**	**12**	**8.7**	**15**	**43**	**96.6**	**5.7**	**4.1**	**16.0**	**5.35**	**4.6**	**15.0**
MND (BDE-47, 85, 99,100, 153, 154)			**9.4**			**28.6**			**110.0**			**71.3**			**20.7**

1 - eurofins/ERGO

2 - Health Canada

Table D.2 Results (ng.g^{-1} lipid) from eurofins-ERGO (1) and Health Canada (2) for Northeast region, males and normalised difference (%) for the results of the congeners analysed by both laboratories.

Region	Northeast													
Gender	Male													
Age	< 16 years			16-30 years			31-45 years			46-60 years			> 60 years		
	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.	1	2	Norm. diff.
sample weight (g)	15.256	18.670		15.87	17.490		15.118	18.690		15.185	18.020		15.167	18.250	
lipid content (%)	0.480	0.424		0.566	0.445		0.699	0.605		0.719	0.581		0.579	0.433	
BDE #15	n.d.(0.4)	n.d. (0.01)	n.c.	0.34	0.034	164.08	1.2	n.d. (0.009)	n.c.	n.d.(0.3)	n.d. (0.004)	n.c.	n.d.(0.3)	0.148	n.c.
BDE #17	n.d.(0.03)	0.038	n.c.	n.d.(0.02)	n.d. (0.051)	n.c.	n.d.(0.02)	0.024	n.c.	n.d.(0.01)	0.019	n.c.	n.d.(0.02)	n.d. (0.099)	n.c.
BDE #28	n.d.(0.2)	0.533	n.c.	0.25	0.807	106.13	n.d.(0.1)	0.486	n.c.	n.d.(0.1)	0.729	n.c.	n.d.(0.2)	0.597	n.c.
BDE #47	10.2	9.6	6.3	9.7	10.2	5.2	6.4	5.8	10.8	5.7	5.5	3.5	3.6	5.2	36.9
BDE #66	-0.1	0.1	n.c.	n.d. (0.1)	0.1	n.c.	0.0	0.0	n.c.	0.0	0.1	n.c.	-0.1	0.2	n.c.
BDE #71	0.0	0.1	n.c.	0.0	0.1	n.c.	0.0	0.1	n.c.	0.0	0.1	n.c.	0.0	0.1	n.c.
BDE #85	0.1	0.2	95.5	0.0	0.3	137.0	0.1	0.1	-62.1	0.0	0.1	-78.5	0.0	0.1	136.0
BDE #99	4.4	4.7	7.6	2.8	4.3	42.3	2.6	2.4	8.0	1.8	2.0	7.0	1.3	4.1	101.5
BDE #100	2.8	2.1	27.1	3.5	2.8	24.0	1.6	1.1	42.8	1.2	0.9	32.7	0.8	1.2	40.7
BDE #119	0.0	n.d. (0.026)	n.c.	0.0	n.d. (0.073)	n.c.	0.0	n.d. (0.011)	n.c.	0.0	n.d. (0.006)	n.c.	0.0	n.d. (0.067)	n.c.
BDE #126	-0.1	n.d. (0.013)	n.c.	n.d. (0.1)	n.d. (0.035)	n.c.	-0.1	n.d. (0.017)	n.c.	0.0	n.d. 90.008)	n.c.	0.0	n.d. (0.051)	n.c.
BDE #138	0.0	0.0	n.c.	n.d. (0.1)	n.d. (0.25)	n.c.	-0.1	0.0	n.c.	-0.1	n.d. (0.066)	n.c.	0.0	n.d. (0.399)	n.c.
BDE #153	5.2	5.0	4.1	4.1	3.8	8.6	2.4	2.1	15.0	1.8	1.8	0.4	1.9	1.9	1.0
BDE #154	0.4	0.4	10.4	0.4	0.8	73.8	0.3	0.2	46.8	0.3	0.1	75.3	0.2	1.1	131.8
Sum of 47, 85, 99, 100, 153 and 154 (excl. LOD)	**23**	**22**	**4.4**	**21**	**22**	**4.7**	**13**	**12**	**8.0**	**11**	**10**	**9.5**	**8**	**14**	**54.6**
MND (BDE-47, 85, 99,100, 153, 154)			25.2			48.5			30.9			32.9			74.7

1 - eurofins/ERGO

2 - Health Canada

Appendix E Concentrations of PBDEs in the serum of a representative group of the Australian population

Note: Total BDE concentrations are the sum of the total mono, di, tri, tetra, penta, hexa, hepta, octa, nona and deca-BDE groups.

Gender	Female	Female	Female	Female	Female	Female	Female	Female	Female (pool 1 and 2)
Region	Northeast (2005)	Northeast (2005)	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	west
Age	0-4	0-4	<16	<16	5-15	5-15	<16	<16	<16
Pool	1	2	1	2	1	2	2	2	1+2
BDE #1	n.d.(2)	n.d.(1)	n.d.(1)	n.d.(0.7)	n.d.(4)	n.d.(1)	n.d.(0.3)	n.d.(0.4)	n.d.(5)
BDE #2	n.d.(2)	n.d.(0.9)	n.d.(0.8)	n.d.(0.5)	n.d.(3)	n.d.(0.7)	n.d.(0.2)	n.d.(0.3)	n.d.(3)
BDE #3	n.d.(1)	n.d.(0.7)	n.d.(0.7)	n.d.(0.4)	n.d.(2)	n.d.(0.5)	n.d.(0.2)	n.d.(0.2)	n.d.(2)
total Mono-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #13	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #15	n.d.(0.08)	n.d.(0.09)	n.d.(0.1)	n.d.(0.1)	n.d.(0.08)	n.d.(0.09)	n.d.(0.1)	n.d.(0.06)	n.d.(0.09)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.03)	n.d.(0.01)	n.d.(0.02)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	0.013	n.d.(0.02)
BDE #28	0.33	0.50	0.21	n.d.(0.2)	n.d.(0.1)	0.21	n.d.(0.3)	0.66	n.d.(0.2)
BDE #35	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tri-BDE	0.47	0.66	0.21	n.d.	0.059	0.33	n.d.	0.67	n.d.
BDE #47	21	29	14	12	5.6	9.2	8.4	16	3.2
BDE #49	0.061	0.40	0.091	0.11	n.d.(0.04)	0.082	n.d.(0.07)	0.20	n.d.(0.05)
BDE #66	0.091	0.14	n.d.(0.2)	n.d.(0.1)	n.d.(0.03)	0.039	0.076	0.20	n.d.(0.03)
BDE #71	n.d.(0.06)	0.040	n.d.(0.02)	n.d.(0.02)	0.034	0.031	n.d.(0.01)	n.d.(0.01)	0.037
BDE #75	0.021	0.039	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	0.013	0.020	0.031	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.015	n.d.(0.01)
total Tetra-BDE	23	33	15	13	6.7	11	8.5	17	3.5
BDE #85	0.31	0.54	0.15	0.26	0.016	0.021	0.16	0.25	n.d.(0.02)
BDE #99	7.9	10	5.4	5.1	1.9	3.5	3.8	6.1	2.3
BDE #100	5.8	8.6	3.5	3.2	1.8	2.9	2.3	3.4	1.3
BDE #116	n.d.(0.02)	n.d.(0.02)	n.d.(0.1)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	0.14	0.18	n.d.(0.05)	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	n.d.(0.06)	n.d.(0.06)	n.d.(0.03)
total Penta-BDE	16	22	9.0	8.6	4.8	7.8	6.2	11	4.4
BDE #138	0.18	0.19	n.d.(0.1)	n.d.(0.09)	0.073	0.096	0.11	0.12	0.074
BDE #140	0.100	0.12	n.d.(0.08)	n.d.(0.08)	0.050	0.076	n.d.(0.08)	0.091	0.036
BDE #153	4.9	7.6	4.4	4.0	3.0	4.2	2.6	5.0	2.6
BDE #154	0.83	1.0	0.48	0.52	0.27	0.40	0.42	0.53	0.27
BDE #155	0.12	0.15	n.d.(0.05)	n.d.(0.09)	0.045	n.d.(0.04)	n.d.(0.06)	n.d.(0.05)	n.d.(0.04)
BDE #156	n.d.(0.06)	n.d.(0.06)	n.d.(0.03)	n.d.(0.05)	n.d.(0.06)	n.d.(0.06)	n.d.(0.03)	n.d.(0.03)	n.d.(0.05)
total Hexa-BDE	8.1	11	5.5	5.2	4.3	6.0	3.3	7.2	3.2
BDE #181	n.d.(0.02)	n.d.(0.2)	n.d.(0.09)	n.d.(0.1)	n.d.(0.3)	n.d.(0.3)	n.d.(0.1)	n.d.(0.09)	n.d.(0.2)
BDE #183	0.62	0.96	n.d.(0.2)	0.25	n.d.(0.3)	0.31	n.d.(0.3)	0.23	n.d.(0.3)
total Hepta-BDE	1.0	1.3	0.12	0.35	0.50	0.31	0.034	0.35	0.41
BDE #197	1.7	1.9	1.2	0.94	0.97	1.2	0.63	0.79	0.82
BDE #203	1.1	0.43	n.d.(0.4)	n.d.(0.4)	0.23	n.d.(0.3)	n.d.(0.3)	n.d.(0.2)	n.d.(0.2)
total Octa-BDE	4.5	3.5	1.2	0.94	1.2	2.9	0.63	1.5	0.82
BDE #207	7.0	4.1	3.3	2.1	2.1	2.9	1.6	2.1	1.7
total Nona-BDE	16	6.0	3.3	2.1	4.3	4.6	1.6	2.6	1.7
BDE #209	4.3 (M)	n.d.(4)	n.d.(7)	3.2	n.d.(3)	n.d.(3)	n.d.(2)	1.7	n.d.(2)
total BDE	**73**	**77**	**34**	**33**	**22**	**33**	**20**	**42**	**14**

Gender	Female	Female	Female	Female	Female	Female	Female	Female
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	16-30	16-30	16-30	16-30	16-30	16-30	16-30	16-30
	1	2	1	2	2	2	2	2
BDE #1	n.d.(1)	n.d.(0.5)	n.d.(1)	n.d.(1)	n.d.(0.3)	n.d.(0.7)	n.d.(0.2)	n.a.
BDE #2	n.d.(0.6)	n.d.(0.3)	n.d.(0.7)	n.d.(0.7)	n.d.(0.2)	n.d.(0.4)	n.d.(0.1)	n.a.
BDE #3	n.d.(0.5)	n.d.(0.2)	n.d.(0.5)	n.d.(0.5)	n.d.(0.1)	n.d.(0.3)	n.d.(0.1)	n.a.
total Mono-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #13	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #15	n.d.(0.1)	n.d.(0.09)	n.d.(0.08)	n.d.(0.08)	n.d.(0.06)	n.d.(0.06)	n.d.(0.04)	n.d.(0.1)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)
BDE #25	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	n.d.(0.2)	n.d.(0.2)	0.11	n.d.(0.1)	0.15	0.12	0.16	n.d.(0.1)
BDE #35	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tri-BDE	n.d.	n.d.	0.11	0.036	0.15	0.12	0.16	n.d.
BDE #47	6.1	5.9	3.9	4.6	6.9	4.6	6.3	2.8
BDE #49	0.038	0.064	n.d.(0.04)	n.d.(0.04)	0.059	0.038	0.032	n.d.(0.03)
BDE #66	n.d.(0.10)	n.d.(0.07)	n.d.(0.02)	n.d.(0.02)	0.055	0.032	n.d.(0.05)	n.d.(0.03)
BDE #71	n.d.(0.02)	n.d.(0.01)	0.022	0.026	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #75	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.015	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
total Tetra-BDE	6.2	6.0	4.5	5.3	7.5	4.9	6.3	2.9
BDE #85	0.056	0.042	0.095	n.d.(0.01)	0.073	0.063	0.13	0.078
BDE #99	1.9	1.6	1.3	1.6	3.9	2.0	2.8	1.4
BDE #100	1.2	1.3	1.1	1.1	1.8	1.1	1.4	0.86
BDE #116	n.d.(0.07)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)
BDE #119	n.d.(0.02)	n.d.(0.01)	0.010	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.05)	n.d.(0.02)	0.13	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	n.d.(0.07)	n.d.(0.03)
total Penta-BDE	3.2	3.0	3.2	3.2	6.1	3.3	4.4	2.4
BDE #138	n.d.(0.07)	n.d.(0.04)	n.d.(0.05)	n.d.(0.06)	n.d.(0.03)	0.051	n.d.(0.01)	0.042
BDE #140	n.d.(0.04)	n.d.(0.06)	0.057	0.037	0.049	0.034	0.037	n.d.(0.03)
BDE #153	1.5	1.7	1.6	1.6	2.3	1.4	1.7	1.6
BDE #154	0.17	0.23	0.17	0.19	0.41	0.23	0.27	0.20
BDE #155	n.d.(0.02)	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	0.038	n.d.(0.03)	n.d.(0.04)	n.d.(0.02)
BDE #156	n.d.(0.03)	n.d.(0.03)	n.d.(0.06)	n.d.(0.06)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)
total Hexa-BDE	1.7	2.2	2.2	2.4	2.9	1.8	2.2	2.0
BDE #181	n.d.(0.09)	n.d.(0.07)	n.d.(0.2)	n.d.(0.2)	n.d.(0.09)	n.d.(0.09)	n.d.(0.07)	n.d.(0.1)
BDE #183	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	0.24	0.37	n.d.(0.1)
total Hepta-BDE	0.12	0.086	0.64	0.35	0.057	0.32	0.51	0.11
BDE #197	0.57	1.2	1.0	0.89	0.81	0.73	1.2	0.71
BDE #203	n.d.(0.3)	n.d.(0.3)	0.23	n.d.(0.3)	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	n.d.(0.3)
total Octa-BDE	0.57	1.2	2.1	1.8	1.5	1.1	1.8	1.4
BDE #207	1.7	2.4	2.3	1.7	2.4	1.8	2.2	n.d.(3)
total Nona-BDE	1.7	2.4	3.9	3.0	3.3	2.5	2.9	n.d.
BDE #209	n.d.(6)	2.3	n.d.(3)	n.d.(3)	3.3	1.4	n.d.(2)	3.6
total BDE	**13**	**17**	**17**	**16**	**25**	**16**	**18**	**12**

Gender	Female	Female	Female	Female	Female	Female	Female
Region	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	31-45	31-45	31-45	31-45	31-45	31-45	31-45
	2	1	2	2	2	2	2
BDE #1	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(0.2)	n.d.(0.8)	n.d.(0.3)	n.a.
BDE #2	n.d.(0.7)	n.d.(1)	n.d.(1)	n.d.(0.1)	n.d.(0.5)	n.d.(0.2)	n.a.
BDE #3	n.d.(0.5)	n.d.(1)	n.d.(1)	n.d.(0.1)	n.d.(0.4)	n.d.(0.2)	n.a.
total Mono-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
BDE #13	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #15	n.d.(0.1)	n.d.(0.05)	n.d.(0.06)	n.d.(0.05)	n.d.(0.05)	n.d.(0.04)	n.d.(0.2)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	0.027	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	0.72	n.d.(0.1)	0.099	n.d.(0.1)	0.17	0.14	n.d.(0.1)
BDE #35	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)
total Tri-BDE	0.74	0.049	0.16	0.040	0.17	0.14	n.d.
BDE #47	8.0	2.9	3.4	3.1	4.3	4.4	2.3
BDE #49	0.13	0.035	0.058	0.028	0.053	0.043	n.d.(0.03)
BDE #66	n.d.(0.1)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	0.020	n.d.(0.05)	n.d.(0.02)
BDE #71	n.d.(0.02)	0.020	0.025	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #75	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #77	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tetra-BDE	8.2	3.7	4.0	3.7	4.6	4.4	2.4
BDE #85	0.058	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	0.070	0.076	0.027
BDE #99	3.4	1.1	1.1	1.3	1.5	1.6	1.3
BDE #100	1.5	1.00	0.94	0.94	1.1	0.92	0.72
BDE #116	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.04)	n.d.(0.03)	n.d.(0.05)	n.d.(0.04)	n.d.(0.07)	n.d.(0.06)	n.d.(0.03)
total Penta-BDE	4.9	2.6	2.5	2.5	2.8	2.6	2.1
BDE #138	n.d.(0.04)	n.d.(0.04)	n.d.(0.05)	n.d.(0.03)	0.044	n.d.(0.03)	0.048
BDE #140	n.d.(0.04)	n.d.(0.03)	n.d.(0.04)	0.036	0.045	0.028	n.d.(0.04)
BDE #153	1.5	2.0	1.7	1.6	1.6	1.2	1.7
BDE #154	0.34	0.19	0.18	0.25	0.25	0.21	0.21
BDE #155	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	n.d.(0.03)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)
BDE #156	n.d.(0.01)	n.d.(0.05)	n.d.(0.07)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)
total Hexa-BDE	2.0	2.9	2.4	2.2	2.1	1.7	2.2
BDE #181	n.d.(0.06)	n.d.(0.2)	n.d.(0.2)	n.d.(0.08)	n.d.(0.09)	n.d.(0.1)	n.d.(0.1)
BDE #183	0.20	n.d.(0.2)	n.d.(0.2)	0.88	0.23	0.22	0.26
total Hepta-BDE	0.30	0.56	0.38	1.00	0.32	0.34	0.34
BDE #197	0.81	0.99	0.66	1.3	0.75	0.89	0.87
BDE #203	n.d.(0.3)	n.d.(0.3)	n.d.(0.3)	0.50	n.d.(0.2)	n.d.(0.2)	n.d.(0.3)
total Octa-BDE	0.81	2.2	3.7	2.3	1.3	1.4	1.8
BDE #207	1.4	2.0	1.5	2.5	1.7	1.9	n.d.(2)
total Nona-BDE	1.4	3.3	2.6	3.2	2.4	2.3	n.d.
BDE #209	1.6	n.d.(3)	n.d.(3)	1.5	3.1	n.d.(2)	n.d.(4)
total BDE	**20**	**15**	**16**	**16**	**17**	**13**	**8.9**

Gender	Female	Female	Female	Female	Female	Female	Female	Female
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	46-60	46-60	46-60	46-60	46-60	46-60	46-60	46-60
	1	2	1	2	2	2	2	2
BDE #1	n.d.(0.4)	n.d.(1)	n.d.(2)	n.d.(2)	n.d.(0.3)	n.d.(0.7)	n.d.(0.2)	n.a.
BDE #2	n.d.(0.3)	n.d.(0.6)	n.d.(1)	n.d.(1.0)	n.d.(0.2)	n.d.(0.4)	n.d.(0.1)	n.a.
BDE #3	n.d.(0.2)	n.d.(0.5)	n.d.(1)	n.d.(0.8)	n.d.(0.1)	n.d.(0.3)	n.d.(0.09)	n.a.
total Mono-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #13	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #15	n.d.(0.09)	n.d.(0.08)	n.d.(0.1)	n.d.(0.07)	n.d.(0.05)	n.d.(0.05)	n.d.(0.04)	n.d.(0.2)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	n.d.(0.1)	n.d.(0.1)	0.33	0.091	n.d.(0.1)	0.12	0.13	0.14
BDE #35	n.d.(0.04)	n.d.(0.10)	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)	n.d.(0.05)	n.d.(0.02)	n.d.(0.01)
total Tri-BDE	n.d.	n.d.	0.38	0.091	n.d.	0.12	0.13	0.14
BDE #47	3.0	3.3	4.2	2.7	2.7	2.9	3.2	3.4
BDE #49	0.029	0.051	n.d.(0.04)	n.d.(0.03)	0.037	0.043	0.022	0.048
BDE #66	n.d.(0.06)	n.d.(0.08)	n.d.(0.02)	n.d.(0.02)	0.020	0.025	n.d.(0.02)	0.031
BDE #71	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	0.023	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #75	n.d.(0.01)	n.d.(0.02)	0.021	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tetra-BDE	3.0	3.3	4.7	3.2	3.1	3.3	3.3	3.5
BDE #85	n.d.(0.02)	0.052	0.19	0.047	n.d.(0.02)	0.043	0.043	0.064
BDE #99	n.d.(0.8)	n.d.(0.8)	1.1	0.92	1.0	1.2	1.2	1.6
BDE #100	0.65	0.76	1.5	0.79	0.79	0.68	0.71	0.85
BDE #116	n.d.(0.08)	n.d.(0.03)	n.d.(0.03)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.07)	n.d.(0.05)	n.d.(0.05)	n.d.(0.03)	n.d.(0.04)	n.d.(0.06)	n.d.(0.01)	n.d.(0.04)
total Penta-BDE	0.65	0.82	2.9	2.0	1.8	2.0	1.9	2.6
BDE #138	n.d.(0.06)	n.d.(0.04)	n.d.(0.06)	n.d.(0.03)	n.d.(0.04)	n.d.(0.05)	n.d.(0.03)	0.046
BDE #140	n.d.(0.03)	n.d.(0.04)	n.d.(0.04)	n.d.(0.02)	0.056	n.d.(0.05)	0.024	n.d.(0.04)
BDE #153	1.0	1.3	2.4	1.3	1.4	1.2	1.2	1.9
BDE #154	0.13	0.21	0.24	0.18	0.24	0.21	0.20	0.24
BDE #155	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)
BDE #156	n.d.(0.03)	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.03)
total Hexa-BDE	1.1	1.6	3.0	1.9	2.0	1.4	1.7	2.4
BDE #181	n.d.(0.06)	n.d.(0.1)	n.d.(0.1)	n.d.(0.3)	n.d.(0.09)	n.d.(0.1)	n.d.(0.07)	n.d.(0.1)
BDE #183	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	n.d.(0.1)	0.23	0.26	0.16
total Hepta-BDE	0.085	0.064	0.00	0.95	0.072	0.31	0.33	0.22
BDE #197	0.55	0.57	0.70	0.66	0.57	0.61	0.69	0.76
BDE #203	n.d.(0.2)	n.d.(0.4)	n.d.(0.3)	n.d.(0.1)	n.d.(0.1)	n.d.(0.3)	n.d.(0.1)	n.d.(0.3)
total Octa-BDE	0.55	0.57	2.1	2.9	0.97	0.88	1.1	0.76
BDE #207	1.3	1.2	1.8	1.3	1.4	1.5	1.6	n.d.(3)
total Nona-BDE	1.3	1.2	2.6	1.3	1.7	2.6	2.3	n.d.
BDE #209	n.d.(4)	2.2	n.d.(3)	n.d.(3)	1.5	1.1	n.d.(1)	n.d.(6)
total BDE	**6.7**	**9.8**	**16**	**12**	**11**	**12**	**11**	**9.7**

Gender	Female	Female	Female	Female	Female	Female	Female	Female
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	>60	>60	>60	>60	>60	>60	>60	>60
	1	2	1	2	2	2	2	2
BDE #1	n.d.(0.4)	n.a.	n.d.(0.6)	n.d.(0.5)	n.d.(0.3)	n.a.	n.d.(0.2)	n.a.
BDE #2	n.d.(0.2)	n.a.	n.d.(0.4)	n.d.(0.3)	n.d.(0.2)	n.a.	n.d.(0.09)	n.a.
BDE #3	n.d.(0.2)	n.a.	n.d.(0.3)	n.d.(0.2)	n.d.(0.1)	n.a.	n.d.(0.07)	n.a.
total Mono-BDE	n.d.	n.a.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.01)	n.d.(0.08)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.05)
BDE #10	n.d.(0.01)	n.d.(0.06)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.06)
BDE #13	n.d.(0.01)	n.d.(0.05)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)
BDE #15	n.d.(0.1)	n.d.(0.3)	n.d.(0.04)	n.d.(0.04)	n.d.(0.07)	n.d.(0.05)	n.d.(0.04)	n.d.(0.2)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
BDE #28	0.26	n.d.(0.2)	n.d.(0.08)	n.d.(0.09)	n.d.(0.2)	0.26	0.078	0.12
BDE #35	n.d.(0.1)	n.d.(0.1)	n.d.(0.01)	n.d.(0.01)	n.d.(0.08)	n.d.(0.06)	n.d.(0.06)	n.d.(0.03)
total Tri-BDE	0.26	n.d.	0.14	0.12	n.d.	0.26	0.078	0.12
BDE #47	6.7	2.6	1.8	2.4	2.0	5.6	2.7	2.4
BDE #49	0.13	0.049	n.d.(0.03)	0.052	n.d.(0.03)	0.073	0.039	0.030
BDE #66	n.d.(0.09)	n.d.(0.04)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)	0.034	n.d.(0.04)	n.d.(0.06)
BDE #71	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	0.018	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.05)
BDE #75	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	0.011	n.d.(0.01)	0.013	n.d.(0.01)	n.d.(0.03)
BDE #77	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
total Tetra-BDE	6.8	2.6	2.1	2.8	2.3	6.2	2.8	2.6
BDE #85	0.038	0.033	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)	0.11	0.049	0.045
BDE #99	1.6	0.80	0.55	0.78	0.76	2.1	0.99	1.6
BDE #100	1.1	0.60	0.56	0.66	0.65	1.2	0.61	0.68
BDE #116	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.05)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
BDE #126	n.d.(0.05)	n.d.(0.02)	n.d.(0.03)	n.d.(0.04)	n.d.(0.04)	n.d.(0.1)	n.d.(0.02)	n.d.(0.1)
total Penta-BDE	2.7	1.4	1.4	1.8	1.4	3.6	1.7	2.4
BDE #138	n.d.(0.04)	n.d.(0.03)	n.d.(0.04)	n.d.(0.05)	n.d.(0.03)	0.052	n.d.(0.02)	0.083
BDE #140	n.d.(0.04)	0.046	0.034	n.d.(0.03)	0.025	0.027	0.021	n.d.(0.06)
BDE #153	1.4	1.1	1.1	1.2	1.2	1.6	1.1	1.6
BDE #154	0.28	0.17	0.21	0.19	0.20	0.35	0.25	0.25
BDE #155	n.d.(0.03)	0.017	n.d.(0.04)	n.d.(0.04)	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)	n.d.(0.06)
BDE #156	n.d.(0.02)	n.d.(0.01)	n.d.(0.05)	n.d.(0.06)	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)	n.d.(0.09)
total Hexa-BDE	1.9	1.4	1.7	2.0	1.5	2.3	1.6	1.9
BDE #181	n.d.(0.08)	n.d.(0.06)	n.d.(0.2)	n.d.(0.3)	n.d.(0.06)	n.d.(0.09)	n.d.(0.06)	n.d.(0.4)
BDE #183	n.d.(0.2)	0.29	n.d.(0.2)	n.d.(0.2)	n.d.(0.2)	0.19	0.24	0.29
total Hepta-BDE	0.070	0.33	0.37	0.88	0.075	0.21	0.32	0.29
BDE #197	0.45	0.95	0.54	0.69	0.49	0.57	0.53	0.63
BDE #203	n.d.(0.3)	n.d.(0.2)	n.d.(0.2)	n.d.(0.3)	n.d.(0.1)	n.d.(0.2)	n.d.(0.2)	n.d.(0.9)
total Octa-BDE	0.45	0.95	1.9	4.6	0.83	0.57	0.53	0.71
BDE #207	1.1	1.5	1.1	1.1	1.1	1.5	1.2	n.d.(9)
total Nona-BDE	1.1	1.9	2.2	2.2	1.7	1.5	1.2	n.d.
BDE #209	1.6	n.d.(2)	n.d.(2)	n.d.(2)	1.4	1.2	n.d.(1)	n.a.
total BDE	**15**	**8.6**	**9.8**	**14**	**9.3**	**16**	**8.1**	**8.1**

Gender	Male	Male	Male	Male	Male	Male	Male	Male	Male (pool 1 and 2)	Male
Region	Northeast (2005)	Northeast (2005)	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	west	South
Age	0-4	0-4	<16	<16	5-15	5-15	<16	<16	<16	<16
	1	2	1	2	1	2	2	2	1+2	1+2
BDE #1	n.d.(0.9)	n.d. (0.9)	n.a.	n.a.	n.d.(0.9)	n.d.(1)	n.d.(0.8)	n.d.(7)	n.d.(1)	n.a.
BDE #2	n.d.(0.6)	n.d.(0.4)	n.a.	n.a.	n.d.(0.5)	n.d.(0.8)	n.d.(0.5)	n.d.(4)	n.d.(0.7)	n.a.
BDE #3	n.d.(0.5)	n.d.(0.5)	n.a.	n.a.	n.d.(0.4)	n.d.(0.6)	n.d.(0.4)	n.d.(4)	n.d.(0.5)	n.a.
total Mono-BDE	n.d.	n.d.	n.a.	n.a.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.01)	n.d.(0.01)	n.d.(0.5)	n.d.(0.1)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.08)
BDE #10	n.d.(0.01)	n.d.(0.01)	n.d.(0.8)	n.d.(0.2)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.1)
BDE #13	n.d.(0.01)	n.d.(0.01)	n.d.(0.5)	n.d.(0.1)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.06)
BDE #15	n.d.(0.09)	n.d.(0.05)	n.d.(0.4)	n.d.(0.2)	n.d.(0.1)	n.d.(0.06)	n.d.(0.09)	n.d.(0.07)	n.d.(0.1)	n.d.(0.3)
total Di-BDE	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	0.020	n.d.(0.03)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.05)
BDE #25	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.04)
BDE #28	0.29	0.74	n.d.(0.2)	n.d.(0.2)	n.d.(0.1)	0.22	n.d.(0.3)	0.14	n.d.(0.3)	n.d.(0.3)
BDE #35	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)
total Tri-BDE	0.39	0.96	n.d.	n.d.	0.072	0.45	n.d.	0.14	0.064	n.d.
BDE #47	15	31	10	11	6.0	9.4	10	9.4	8.6	8.4
BDE #49	0.15	0.16	0.063	0.067	0.031	0.07	n.d.(0.07)	0.059	0.068	n.d.(0.08)
BDE #66	0.072	0.26	n.d.(0.08)	n.d.(0.09)	0.030	0.07	n.d.(0.05)	0.059	0.057	0.075
BDE #71	0.17	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)	0.020	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.054	n.d.(0.07)
BDE #75	0.036	0.041	n.d.(0.02)	0.019	0.013	0.014	n.d.(0.02)	0.016	n.d.(0.01)	n.d.(0.05)
BDE #77	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)
total Tetra-BDE	17	34	10	11	6.4	10	11	10	9.4	8.6
BDE #85	0.38	0.87	0.072	0.15	0.025	0.22	0.20	0.24	n.d.(0.2)	0.25
BDE #99	6.0	9.7	4.4	4.4	2.2	3.1	4.7	4.2	4.5	5.6
BDE #100	4.5	8.5	2.8	3.1	1.8	2.7	2.9	2.3	2.4	2.4
BDE #116	n.d.(0.02)	n.d.(0.01)	n.d.(0.2)	n.d.(0.2)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.08)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)
BDE #126	n.d.(0.03)	n.d.(0.02)	n.d.(0.06)	n.d.(0.08)	n.d.(0.03)	n.d.(0.02)	n.d.(0.05)	n.d.(0.07)	n.d.(0.03)	n.d.(0.2)
total Penta-BDE	12	20	7.2	7.6	4.5	6.7	7.8	7.4	7.6	8.6
BDE #138	0.12	0.17	n.d.(0.04)	n.d.(0.07)	0.061	0.071	0.082	0.090	0.074	0.12
BDE #140	0.084	0.11	n.d.(0.06)	0.046	n.d.(0.04)	0.038	0.059	0.071	0.056	n.d.(0.08)
BDE #153	4.5	8.2	5.2	5.2	6.1	7.7	5.1	3.9	4.6	4.5
BDE #154	0.65	1.1	0.42	0.38	0.29	0.38	0.48	0.45	0.55	0.59
BDE #155	0.075	0.10	0.054	0.040	n.d.(0.04)	0.027	0.072	0.047	n.d.(0.06)	n.d.(0.09)
BDE #156	n.d.(0.04)	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.03)	n.d.(0.04)	n.d.(0.04)	n.d.(0.04)	n.d.(0.1)
total Hexa-BDE	6.2	11	5.7	5.7	6.9	8.9	6.5	5.3	5.3	5.3
BDE #181	n.d.(0.3)	n.d.(0.07)	n.d.(0.09)	n.d.(0.1)	n.d.(0.08)	n.d.(0.1)	n.d.(0.1)	n.d.(0.1)	n.d.(0.2)	n.d.(0.6)
BDE #183	0.67	0.77	0.21	0.17	n.d.(0.3)	0.44	n.d.(0.3)	0.18	n.d.(0.4)	n.d.(0.3)
total Hepta-BDE	1.5	0.91	0.28	0.28	n.d.	0.67	0.14	0.28	1.0	0
BDE #197	1.9	1.6	0.73	0.95	1.2	1.3	1.0	0.86	1.7	1.0
BDE #203	0.49	0.38	n.d.(0.3)	n.d.(0.2)	0.42	0.53	n.d.(0.3)	n.d.(0.2)	n.d.(0.3)	n.d.(1)
total Octa-BDE	4.6	2.9	0.73	0.95	2.5	1.8	1.9	1.6	5.0	1.0
BDE #207	5.8	3.9	n.d.(3)	n.d.(2)	2.3	3.7	2.1	2.4	4.4	n.d.(7)
total Nona-BDE	8.8	6.1	n.d.	n.d.	3.5	3.7	3.4	2.4	4.4	n.d.
BDE #209	12 (M)	5.1	n.d.(8)	n.d.(4)	n.d.(4)	3.3	2.2	1.4	3.0 (M)	n.a.
total BDE	**63**	**80**	**24**	**26**	**24**	**36**	**33**	**29**	**36**	**24**

Gender	Male	Male	Male	Male	Male	Male	Male	Male	Male	Male	Male
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Southeast	Rural	Rural	West	West	South
Age	16-30	16-30	16-30	16-30	16-30	16-30	16-30	16-30	16-30	16-30	16-30
	1	2	1	2	1	2	1	2	1	2	1
BDE #1	n.a.	n.a.	n.d.(1)	n.d.(1)	n.d.(0.4)	n.d.(0.5)	n.a.	n.d.(2)	n.d.(0.3)	n.d.(0.3)	n.a.
BDE #2	n.a.	n.a.	n.d.(0.9)	n.d.(0.6)	n.d.(0.2)	n.d.(0.3)	n.a.	n.d.(1.0)	n.d.(0.2)	n.d.(0.2)	n.a.
BDE #3	n.a.	n.a.	n.d.(0.6)	n.d.(0.5)	n.d.(0.2)	n.d.(0.2)	n.a.	n.d.(0.8)	n.d.(0.1)	n.d.(0.1)	n.a.
total Mono-BDE	n.a.	n.a.	n.d.	n.d.	n.d.	n.d.	n.a.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.a.	n.d.(0.4)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #10	n.a.	n.d.(0.7)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #13	n.a.	n.d.(0.5)	0.015	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #15	n.a.	0.34	n.d.(0.1)	n.d.(0.08)	n.d.(0.07)	n.d.(0.06)	n.d.(0.05)	n.d.(0.06)	n.d.(0.05)	n.d.(0.05)	n.d.(0.1)
total Di-BDE	n.a.	0.34	0.015	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)
BDE #25	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	0.20	0.25	n.d.(0.1)	0.13	0.36	n.d.(0.1)	0.15	0.19	0.13	0.15	0.12
BDE #35	n.d.(0.05)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tri-BDE	0.20	0.25	0.10	0.18	0.36	n.d.	0.15	0.19	0.13	0.15	0.12
BDE #47	5.7	9.7	4.1	5.3	12	5.8	6.4	4.9	5.0	6.0	5.1
BDE #49	0.038	0.039	n.d.(0.03)	0.058	0.10	0.036	0.038	0.037	0.032	0.037	n.d.(0.03)
BDE #66	n.d.(0.1)	n.d.(0.1)	0.041	0.025	0.083	0.037	0.019	n.d.(0.02)	n.d.(0.05)	n.d.(0.05)	0.042
BDE #71	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)	0.026	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #75	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.013	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #77	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
total Tetra-BDE	5.7	9.7	4.4	6.3	12	6.3	6.6	5.1	5.0	6.0	5.6
BDE #85	0.044	0.049	n.d.(0.04)	0.039	0.17	0.056	0.13	0.082	0.082	0.100	0.041
BDE #99	2.1	2.8	1.4	1.9	4.7	2.3	3.0	2.1	2.1	2.4	2.5
BDE #100	1.4	3.5	1.2	1.6	2.4	1.6	1.6	1.2	1.1	1.7	1.2
BDE #116	n.d.(0.2)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.05)
BDE #119	n.d.(0.07)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.05)	n.d.(0.06)	n.d.(0.04)	n.d.(0.02)	n.d.(0.04)	n.d.(0.05)	n.d.(0.09)	n.d.(0.04)	n.d.(0.06)	n.d.(0.1)	n.d.(0.07)
total Penta-BDE	3.5	6.4	2.8	4.1	7.6	3.9	4.9	3.7	3.4	4.2	4.5
BDE #138	n.d.(0.1)	n.d.(0.09)	0.050	n.d.(0.06)	0.078	0.046	0.080	0.041	n.d.(0.04)	n.d.(0.05)	0.034
BDE #140	n.d.(0.09)	n.d.(0.06)	0.029	0.058	0.060	0.043	0.051	0.037	0.045	0.042	n.d.(0.07)
BDE #153	3.4	4.1	3.1	3.5	3.2	3.0	3.5	2.8	2.6	2.8	3.1
BDE #154	0.22	0.38	0.23	0.26	0.46	0.31	0.36	0.27	0.25	0.28	0.26
BDE #155	0.028	0.042	n.d.(0.04)	0.036	n.d.(0.05)	0.037	n.d.(0.04)	n.d.(0.03)	n.d.(0.03)	n.d.(0.03)	n.d.(0.03)
BDE #156	n.d.(0.09)	n.d.(0.03)	n.d.(0.04)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.02)	n.d.(0.04)
total Hexa-BDE	3.7	4.5	3.7	4.4	4.6	3.8	5.2	3.6	3.2	3.3	3.4
BDE #181	n.d.(0.09)	n.d.(0.09)	n.d.(0.1)	n.d.(0.5)	n.d.(0.06)	n.d.(0.06)	n.d.(0.08)	n.d.(0.2)	n.d.(0.09)	n.d.(0.07)	n.d.(0.1)
BDE #183	0.38	0.22	0.28	n.d.(0.2)	0.20	n.d.(0.2)	0.20	0.29	0.45	0.26	0.22
total Hepta-BDE	0.52	0.28	0.79	0.64	0.32	0.16	0.30	0.45	0.61	0.48	0.35
BDE #197	1.1	0.98	1.3	1.4	1.1	1.3	0.91	1.0	1.5	1.2	1.4
BDE #203	n.d.(0.3)	n.d.(0.3)	0.46	0.50	0.27	0.30	n.d.(0.4)	n.d.(0.1)	n.d.(0.2)	n.d.(0.3)	n.d.(0.5)
total Octa-BDE	1.1	0.98	3.8	3.5	1.9	2.3	1.7	1.6	2.3	2.2	1.4
BDE #207	n.d.(2)	n.d.(2)	2.4	2.0	1.9	2.0	1.6	1.7	2.7	2.5	n.d.(3)
total Nona-BDE	n.d.	n.d.	3.8	3.0	3.0	2.8	1.6	2.3	3.3	3.3	n.d.
BDE #209	n.d.(7)	n.a.	n.d.(4)	5.6 (M)	1.9	1.7	1.4	1.5	3.7	2.2	n.d.(15)
total BDE	**15**	**22**	**19**	**28**	**32**	**21**	**22**	**19**	**22**	**22**	**15**

Gender	Male	Male	Male	Male	Male	Male	Male
Region	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	31-45	31-45	31-45	31-45	31-45	31-45	31-45
	2	1	2	2	2	2	2
BDE #1	n.a.	n.d.(0.7)	n.d.(0.8)	n.d.(0.2)	n.d.(0.8)	n.d.(0.4)	n.a.
BDE #2	n.a.	n.d.(0.4)	n.d.(0.5)	n.d.(0.09)	n.d.(0.5)	n.d.(0.2)	n.a.
BDE #3	n.a.	n.d.(0.3)	n.d.(0.4)	n.d.(0.08)	n.d.(0.4)	n.d.(0.2)	n.a.
total Mono-BDE	n.a.	n.d.	n.d.	n.d.	n.d.	n.d.	n.a.
BDE #7	n.d.(0.2)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #10	n.d.(0.2)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #13	n.d.(0.1)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #15	1.2	n.d.(0.06)	n.d.(0.06)	n.d.(0.05)	n.d.(0.04)	n.d.(0.05)	n.d.(0.1)
total Di-BDE	1.2	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	n.d.(0.1)	0.13	0.12	n.d.(0.1)	0.27	0.23	0.12
BDE #35	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.1)	n.d.(0.02)	n.d.(0.03)	n.d.(0.01)
total Tri-BDE	n.d.	0.22	0.46	n.d.	0.27	0.23	0.12
BDE #47	6.4	4.4	4.2	4.3	5.9	6.8	3.9
BDE #49	0.049	0.10	0.05	0.046	0.059	0.048	0.031
BDE #66	n.d.(0.05)	n.d.(0.02)	0.04	0.029	0.023	n.d.(0.06)	0.033
BDE #71	n.d.(0.01)	0.079	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #75	n.d.(0.02)	0.14	n.d.(0.01)	n.d.(0.01)	0.012	n.d.(0.01)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
total Tetra-BDE	6.5	8.2	4.9	4.7	6.2	6.8	4.1
BDE #85	0.064	0.020	0.1	0.038	0.092	0.12	0.042
BDE #99	2.6	2.0	1.2	2.5	2.6	2.6	1.8
BDE #100	1.6	1.3	1.10	1.6	1.4	1.8	1.1
BDE #116	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)
BDE #119	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.06)	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)	n.d.(0.05)	n.d.(0.04)	n.d.(0.04)
total Penta-BDE	4.3	3.8	2.7	4.3	4.3	4.5	3.2
BDE #138	n.d.(0.05)	n.d.(0.05)	n.d.(0.05)	0.081	0.049	n.d.(0.05)	0.067
BDE #140	n.d.(0.07)	0.024	n.d.(0.02)	0.078	0.049	0.055	n.d.(0.05)
BDE #153	2.4	2.5	2.3	2.5	2.4	2.8	2.9
BDE #154	0.29	0.25	0.20	0.44	0.37	0.37	0.27
BDE #155	0.029	0.028	n.d.(0.03)	0.032	n.d.(0.03)	n.d.(0.04)	n.d.(0.04)
BDE #156	n.d.(0.03)	n.d.(0.02)	n.d.(0.04)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)
total Hexa-BDE	2.7	3.3	2.9	3.8	3.4	3.7	3.5
BDE #181	n.d.(0.1)	n.d.(0.2)	n.d.(0.1)	n.d.(0.1)	n.d.(0.06)	n.d.(0.07)	n.d.(0.09)
BDE #183	0.30	0.23	0.38	0.21	0.80	0.33	0.28
total Hepta-BDE	0.39	0.76	0.59	0.34	0.91	0.47	0.47
BDE #197	0.85	1.0	1.1	0.94	1.3	1.1	1.3
BDE #203	n.d.(0.3)	0.37	n.d.(0.5)	0.31	n.d.(0.2)	n.d.(0.2)	n.d.(0.3)
total Octa-BDE	0.85	4.3	1.1	2.0	2.0	1.9	2.3
BDE #207	n.d.(1)	1.8	2.7	1.8	3.3	2.3	2.4
total Nona-BDE	n.d.	3.4	2.7	2.6	3.3	3.5	2.4
BDE #209	n.a.	n.d.(3)	2.6	1.3	2.0	n.d.(2)	1.4
total BDE	**16**	**24**	**18**	**19**	**22**	**21**	**17**

Gender	Male	Male	Male	Male	Male	Male	Male	Male
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	46-60	46-60	46-60	46-60	46-60	46-60	46-60	46-60
	1	2	1	2	2	2	2	2
BDE #1	n.a.	n.a.	n.d.(3)	n.d.(4)	n.d.(0.3)	n.d.(0.2)	n.d.(0.3)	n.d.(0.8)
BDE #2	n.a.	n.a.	n.d.(2)	n.d.(3)	n.d.(0.2)	n.d.(0.1)	n.d.(0.2)	n.d.(0.5)
BDE #3	n.a.	n.a.	n.d.(2)	n.d.(2)	n.d.(0.1)	n.d.(0.09)	n.d.(0.1)	n.d.(0.4)
total Mono-BDE	n.a.	n.a.	n.d.	n.d.	n.d.	n.d.	n.d.	n.d.
BDE #7	n.d.(0.4)	n.d.(0.04)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #10	n.d.(0.4)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #13	n.d.(0.3)	n.d.(0.02)	n.d.(0.01)	0.018	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #15	0.44	n.d.(0.3)	n.d.(0.07)	n.d.(0.1)	n.d.(0.05)	n.d.(0.04)	n.d.(0.04)	n.d.(0.08)
total Di-BDE	0.44	n.d.	n.d.	0.018	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #25	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.024	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	0.18	n.d.(0.1)	n.d.(0.1)	0.23	0.15	0.19	0.21	n.d.(0.1)
BDE #35	n.d.(0.04)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.04)	n.d.(0.08)	n.d.(0.03)	n.d.(0.02)
total Tri-BDE	0.18	n.d.	0.059	0.58	0.15	0.19	0.21	n.d.
BDE #47	5.4	5.7	3.7	4.3	5.2	4.1	4.6	3.4
BDE #49	0.046	0.046	0.084	0.050	0.048	0.045	0.056	0.035
BDE #66	n.d.(0.05)	n.d.(0.04)	0.020	0.084	0.033	n.d.(0.05)	n.d.(0.05)	n.d.(0.04)
BDE #71	n.d.(0.02)	n.d.(0.03)	0.031	0.027	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #75	n.d.(0.03)	n.d.(0.01)	0.011	0.020	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tetra-BDE	5.4	5.8	4.4	4.8	5.7	4.2	4.7	3.4
BDE #85	0.030	0.043	n.d.(0.01)	n.d.(0.01)	0.10	0.060	0.063	0.044
BDE #99	1.9	1.8	1.2	1.4	2.1	1.4	1.6	1.3
BDE #100	1.0	1.2	1.1	1.0	1.5	0.99	1.2	0.93
BDE #116	n.d.(0.2)	n.d.(0.07)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.1)
BDE #119	n.d.(0.03)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)
BDE #126	n.d.(0.07)	n.d.(0.03)	0.024	n.d.(0.03)	n.d.(0.03)	n.d.(0.05)	n.d.(0.04)	n.d.(0.10)
total Penta-BDE	2.9	3.1	2.8	2.6	3.9	2.4	2.8	2.2
BDE #138	n.d.(0.05)	n.d.(0.06)	0.030	0.041	0.060	n.d.(0.03)	n.d.(0.04)	n.d.(0.09)
BDE #140	n.d.(0.03)	n.d.(0.05)	0.030	0.022	0.043	0.035	0.037	n.d.(0.05)
BDE #153	2.2	1.8	2.2	2.2	3.0	1.9	1.9	2.1
BDE #154	0.24	0.27	0.29	0.23	0.34	0.26	0.35	0.25
BDE #155	0.027	0.030	0.029	n.d.(0.02)	0.039	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)
BDE #156	n.d.(0.03)	n.d.(0.02)	n.d.(0.04)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)
total Hexa-BDE	2.5	2.1	3.2	2.7	4.1	2.7	2.7	2.3
BDE #181	n.d.(0.09)	n.d.(0.06)	n.d.(0.2)	n.d.(0.06)	n.d.(0.06)	n.d.(0.06)	n.d.(0.06)	n.d.(0.1)
BDE #183	0.19	0.28	0.29	0.51	0.22	0.33	0.38	0.33
total Hepta-BDE	0.26	0.34	0.97	0.51	0.35	0.47	0.52	0.47
BDE #197	0.86	0.85	1.8	1.3	1.1	1.0	1.3	1.0
BDE #203	n.d.(0.3)	n.d.(0.2)	n.d.(0.6)	0.28	0.26	n.d.(0.2)	n.d.(0.3)	n.d.(0.5)
total Octa-BDE	0.86	0.85	6.8	3.4	1.8	1.7	1.8	1.0
BDE #207	n.d.(3)	n.d.(2)	4.1	2.0	2.3	2.1	2.4	n.d.(2)
total Nona-BDE	n.d.	n.d.	6.9	2.7	3.2	3.0	3.4	n.d.
BDE #209	n.d.(6)	n.d.(4)	2.6 (M)	n.d.(3)	2.7	n.d.(1)	n.d.(1)	n.a.
total BDE	**13**	**12**	**28**	**17**	**22**	**15**	**16**	**9.4**

Gender	Male	Male	Male	Male	Male	Male	Male	Male
Region	Northeast	Northeast	Northeast (2005)	Northeast (2005)	Southeast	Rural	West	South
Age	>60	>60	>60	>60	>60	>60	>60	>60
	1	2	1	2	2	2	2	2
BDE #1	n.a.	n.a.	n.d.(0.8)	n.d.(4)	n.d.(3)	n.d.(0.2)	n.a.	n.d.(0.9)
BDE #2	n.a.	n.a.	n.d.(0.5)	n.d.(2)	n.d.(2)	n.d.(0.1)	n.a.	n.d.(0.5)
BDE #3	n.a.	n.a.	n.d.(0.4)	n.d.(2)	n.d.(2)	n.d.(0.1)	n.a.	n.d.(0.4)
total Mono-BDE	n.a.	n.a.	n.d.	n.d.	n.d.	n.d.	n.a.	n.d.
BDE #7	n.d.(0.1)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #10	n.d.(0.2)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)
BDE #13	n.d.(0.2)	n.d.(0.02)	0.018	0.010	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #15	n.d.(0.3)	n.d.(0.3)	n.d.(0.04)	n.d.(0.1)	n.d.(0.05)	n.d.(0.04)	n.d.(0.07)	n.d.(0.09)
total Di-BDE	n.d.	n.d.	0.018	0.010	n.d.	n.d.	n.d.	n.d.
BDE #17	n.d.(0.02)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)
BDE #25	n.d.(0.01)	n.d.(0.03)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #28	n.d.(0.2)	n.d.(0.2)	0.1	0.12	0.094	0.41	0.080	n.d.(0.2)
BDE #35	n.d.(0.07)	n.d.(0.08)	n.d.(0.06)	n.d.(0.01)	n.d.(0.05)	n.d.(0.08)	n.d.(0.02)	n.d.(0.02)
total Tri-BDE	n.d.	n.d.	0.27	0.50	0.094	0.41	0.080	n.d.
BDE #47	2.3	3.6	4.1	3.1	2.7	11	2.2	2.0
BDE #49	0.026	0.049	0.048	0.049	0.036	0.15	n.d.(0.03)	0.032
BDE #66	n.d.(0.06)	n.d.(0.06)	0.046	0.034	n.d.(0.02)	n.d.(0.1)	n.d.(0.02)	n.d.(0.05)
BDE #71	n.d.(0.01)	n.d.(0.01)	n.d.(0.03)	0.030	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #75	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	0.017	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)
BDE #77	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
total Tetra-BDE	2.3	3.7	4.7	3.5	2.9	11	2.4	2.0
BDE #85	0.018	0.027	0.1	0.024	0.041	0.23	0.032	n.d.(0.03)
BDE #99	0.98	1.3	1.2	0.93	1.1	3.9	1.1	n.d.(1)
BDE #100	0.61	0.81	0.9	0.78	0.78	2.3	0.73	0.62
BDE #116	n.d.(0.07)	n.d.(0.05)	n.d.(0.01)	n.d.(0.01)	n.d.(0.02)	n.d.(0.01)	n.d.(0.02)	n.d.(0.04)
BDE #119	n.d.(0.02)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)	n.d.(0.01)
BDE #126	n.d.(0.05)	n.d.(0.05)	n.d.(0.03)	n.d.(0.02)	n.d.(0.05)	n.d.(0.02)	n.d.(0.07)	n.d.(0.04)
total Penta-BDE	1.6	2.2	2.4	1.7	2.0	6.5	1.9	0.62
BDE #138	n.d.(0.08)	n.d.(0.05)	n.d.(0.03)	0.024	0.039	n.d.(0.08)	0.029	n.d.(0.07)
BDE #140	n.d.(0.07)	n.d.(0.05)	n.d.(0.02)	0.012	0.063	0.044	0.097	n.d.(0.04)
BDE #153	1.3	1.9	1.8	1.9	1.6	2.7	1.6	1.9
BDE #154	0.20	0.23	0.27	0.21	0.30	0.52	0.25	0.21
BDE #155	0.039	0.027	n.d.(0.02)	n.d.(0.02)	n.d.(0.03)	n.d.(0.06)	n.d.(0.03)	n.d.(0.03)
BDE #156	n.d.(0.05)	n.d.(0.03)	n.d.(0.03)	n.d.(0.02)	n.d.(0.03)	n.d.(0.02)	n.d.(0.03)	n.d.(0.03)
total Hexa-BDE	1.6	2.2	2.5	2.4	2.3	3.8	2.2	2.3
BDE #181	n.d.(0.10)	n.d.(0.09)	n.d.(0.1)	n.d.(0.08)	n.d.(0.1)	n.d.(0.05)	n.d.(0.1)	n.d.(0.06)
BDE #183	0.20	0.27	0.28	n.d.(0.2)	0.23	0.19	0.22	n.d.(0.2)
total Hepta-BDE	0.22	0.32	0.44	n.d.	0.35	0.30	0.36	0.10
BDE #197	0.73	0.79	1.2	0.58	0.63	0.80	1.3	0.85
BDE #203	n.d.(0.4)	n.d.(0.3)	n.d.(0.2)	n.d.(0.1)	n.d.(0.2)	n.d.(0.3)	n.d.(3)	n.d.(0.2)
total Octa-BDE	0.73	0.79	1.2	2.0	0.90	1.5	1.3	0.85
BDE #207	n.d.(2)	n.d.(2)	4.5	1.3	1.7	1.6	1.6	2.3
total Nona-BDE	n.d.	n.d.	4.5	2.0	2.1	2.3	2.2	2.3
BDE #209	n.d.(3)	n.a.	6.5	n.d.(3)	1.1	n.d.(1)	1.7	2.0
total BDE	**6.4**	**9.1**	**23**	**12**	**12**	**25**	**12**	**10**

Appendix F Lipid content

Table F.1 Lipid content of blood serum samples (%).

MALES	Pool	0-4 years	<16 years	16-30 years	31-45 years	46-60 years	> 60 years
Northeast	**1**	-	0.486	0.618	-	0.785	0.656
	2	-	0.480	0.566	0.699	0.719	0.579
Northeast 04-05	**1**	0.462	0.451	0.457	0.600	0.637	0.526
	2	0.410	0.483	0.526	0.613	0.613	0.568
Southeast	**1**	-	-	0.520	-	-	-
	2	-	0.495	0.537	0.677	0.651	0.563
South	**1**	-		0.611	-	-	-
	2	-	0.446	0.589	0.620	0.659	0.535
West	**1**	-	-	0.520	-	-	-
	2	-	0.406	0.472	0.559	0.647	0.551
Rural	**1**	-	-	0.548	-	-	-
	2	-	0.485	0.498	0.675	0.724	0.591
Mean		**0.436**	**0.467**	**0.539**	**0.635**	**0.679**	**0.571**

FEMALES	Pool	0-4 years	<16 years	16-30 years	31-45 years	46-60 years	> 60 years
Northeast	**1**	-	0.488	0.571	-	0.665	0.652
	2	-	0.492	0.553	0.605	0.652	0.639
Northeast 04-05	**1**	0.465	0.442	0.482	0.517	0.578	0.627
	2	0.423	0.490	0.490	0.546	0.604	0.612
Southeast	**1**	-	-	-	-	-	-
	2	-	0.525	0.600	0.666	0.712	0.673
South	**1**	-	-	-	-	-	-
	2	-	-	0.550	0.597	0.632	0.644
West	**1**	-	-	-	-	-	-
	2	-	0.503	0.572	0.580	0.706	0.633
Rural	**1**	-	-	-	-	-	-
	2	-	0.499	0.542	0.556	0.659	0.566
Mean		**0.444**	**0.491**	**0.545**	**0.581**	**0.651**	**0.631**
Mean (male and female)		**0.440**	**0.478**	**0.541**	**0.608**	**0.665**	**0.601**

Appendix G TBBP-A

TBBP-A was detected in 59 out of 85 pools. The concentration ranged from non-detect to 0.07 ng.g^{-1} lipid weight. Table E.1 lists the results of the TBBP-A analysis.

Eurofins-ERGO are continuing work on the methodology for TBBP-A analysis and therefore results should be considered as preliminary and interpreted with caution. The blood samples were analysed in two batches for TBBP-A. TBBP-A was detected at low levels in the first batch and was not detected in any of the samples analysed in the second batch. The first batch included most of the 2002-03 samples while the second batch included all 2004-05 and two 2002-03 samples (West, <16 years, male and female). Since there may have been analytical problems it should not be assumed that the concentration of TBBP-A in Australian serum has decreased from 2002-03 to 2004-05. To be certain of a temporal trend in TBBP-A, further analysis of samples would be required once the laboratory has verified their analytical methodology.

Table F.2 Concentration of TBBP-A (ng.g^{-1} lipid)

Males	Pool	0-4 years	<16 years	16-30 years	31-45 years	46-60 years	> 60 years
Northeast	**1**	N-A	0.03	0.03	N-A	0.03	0.03
	2	N-A	0.03	0.03	0.03	0.03	0.03
Northeast 2004-05	**1**	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)
	2	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)	n.d.(0.07)
Southeast	**1**	N-A	N-A	0.05	N-A	N-A	N-A
	2	N-A	0.06	0.04	0.05	0.05	0.05
South	**1**	N-A	N-A	0.04	N-A	N-A	N-A
	2	N-A	0.04	0.05	0.04	0.02	0.03
West	**1**	N-A	N-A	0.04	N-A	N-A	N-A
	2	N-A	n.d. (0.07)	0.04	0.04	0.04	0.05
Rural	**1**	N-A	N-A	0.04	N-A	N-A	N-A
	2	N-A	0.04	0.05	0.06	0.03	0.03
Females	**Pool**	**0-4 years**	**<16 years**	**16-30 years**	**31-45 years**	**46-60 years**	**> 60 years**
Northeast	**1**	N-A	0.02	0.02	N-A	0.03	0.03
	2	N-A	0.03	0.03	0.02	0.03	0.02
Northeast 2004-05	**1**	n.d.(0.09)	n.d.(0.09)	n.d.(0.09)	n.d.(0.09)	n.d.(0.07)	n.d.(0.09)
	2	n.d.(0.09)	n.d.(0.09)	n.d.(0.09)	n.d.(0.09)	n.d.(0.07)	n.d.(0.09)
Southeast	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	0.06	0.05	0.04	0.04	0.05
South	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	N-A	0.04	0.07	0.05	0.04
West	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	n.d. (0.07)	0.03	0.03	0.04	0.02
Rural	**1**	N-A	N-A	N-A	N-A	N-A	N-A
	2	N-A	0.04	0.05	0.04	0.04	0.05

International TBBP-A comparison

There is limited data on TBBP-A in human blood serum. The TBBP-A concentrations found in the current study are lower than that reported from Norway and similar to that reported from Sweden.

In Norway, Thomsen et al (2002) investigated temporal trends and the role of age and gender on BFRs in human serum. Serum from 40 to 50 year old men collected at six time periods during 1977 and 1999 was pooled into six samples. These people were patients in county hospitals and disease or reason for hospitalisation were not considered when using serum for analysis. Eight groups of differing age and gender samples in 1998 were pooled into eight samples. TBBP-A was observed in the pools from 1986, 1990, 1995 and 1999 and ranged from 0.42 to 0.65 $ng.g^{-1}$ lipid. TBBP-A was detected in all age groups and ranged from 0.34 to 0.71 $ng.g^{-1}$ lipid. The highest concentration was found in the 0-4 years group at 0.65 $ng.g^{-1}$ lipid weight. Thomsen et al (2005) continued their 2002 study and analysed pools consisting of serum from about 20 persons made from different age and gender groups. In the serum pools from persons of different age, the mean TBBP-A concentration was 0.15 $ng.g^{-1}$ lipid. From the samples separated by age group, the TBBP-A concentration ranged from non-detect to 2 $ng.g^{-1}$ lipid.

De Wit (2002) reported on Klasson-Wehler (1997) that TBBP-A was found in the low $ng.g^{-1}$ lipid in a Swedish study of human blood. The exact concentrations were not reported.

Appendix H International data

The following provides additional details of the studies used in Figures 4.6 and 4.7. Included here are examples of studies of PBDEs in human blood from North America, Continental Europe, United Kingdom, Asia and South America. No data were found from Africa. As occupational exposure was not a focus of the current study international data on occupational exposure to PBDEs is not included here.

North America

Fischer et al (2006) determined the concentration of PBDEs in blood serum from a family of four living in California in 2004. Concentrations of BDE-47 varied from 32 $ng.g^{-1}$ lipid in the father to 60, 137 and 245 $ng.g^{-1}$ lipid in the mother, child and toddler, respectively. The ΣPBDE concentration (BDEs -47, -99, -100, -153 and -154) were 64, 106, 247 and 418 for the father, mother, child and toddler, respectively. Based on the small sample size these results should be treated with caution.

Schecter et al (2005) determined the concentration of PBDEs in human whole blood and serum from samples collected in 2003. These samples consisted of 29 individuals from Mississippi, 10 individuals from New York City, and one pool of serum (n=100) and one pool of whole blood (n=100) both obtained as anonymous samples from Dallas, Texas. The sum PBDE concentration (BDE-17, -28, -47, -66, -77, -85, -99, -100, -138, -153, -154, -183 and -209) for the 2003 pooled whole blood and blood serum were 79.7 and 61.8 $ng.g^{-1}$ lipid, respectively. For the 39 individual whole blood samples the mean and median ΣPBDE concentrations were 52.6 and 29 $ng.g^{-1}$ lipid, respectively. The congener profile was similar with BDE-47 the dominant congener. The authors investigated the differences in gender and age using the individual samples. The mean concentration of ΣPBDEs was 35.9 and 74.1 $ng.g^{-1}$ for individual samples of males and females, respectively. This gender difference was not statistically significant. The authors also found no significant correlation between PBDE concentrations in blood and age of the donor although results are suggestive of a decrease of PBDE levels with age. BDE-209 was detected at low concentrations in the pooled serum, whole blood and individual samples ranging from not assessable to 2.7 $ng.g^{-1}$ lipid. The concentration of BDE-47 ranged from 12.8 $ng.g^{-1}$ lipid in the individual whole blood (median) to 44.2 $ng.g^{-1}$ lipid in the whole blood pool.

Petreas et al (2003) investigated the concentrations of PBDEs from 50 women who were Laotian immigrants living in California, USA. Serum samples were obtained between 1997 and 1999 from women aged 19-40 years. Individual samples were analysed but due to a small sample quantity only BDE-47 could be measured above the blank and only in 24 of the 50 samples. The concentration of BDE-47 in serum ranged from <10 $ng.g^{-1}$ lipid to 511 $ng.g^{-1}$ lipid with a mean and median of 50.6 and 16.5 $ng.g^{-1}$ lipid, respectively.

Sjodin et al (2004) reports a retrospective time-trend of PBDEs in human serum. 40 pools of serum were collected in the south eastern US from 1985 to 2002 and from Seattle, Washington from 1999 to 2002. The authors found serum concentrations of

ΣPBDEs (BDE-47, -85, -99, -100, -153 and -154) to be a maximum of 71 $ng.g^{-1}$ in 1995-99 then 61 $ng.g^{-1}$ lipid in the 2000-02 samples. The serum pools from the southeastern US were made up of 40 to 200 samples while from Seattle the pools contained 6 to 8 samples. Geographical differences and their possible impact on time trends were not able to be investigated as anonymous samples were used, nor were gender or individual differences investigated due to the use of pooled samples. For all years, the ΣPBDE concentration was dominated by BDE-47. In the pools from 1995-99, BDE-47 was followed by BDE-99, 100 and 153, this changed slightly in 2000-02 where BDE-47 was followed by -99, 153 and 100.

Mazdai et al (2003) determined the concentration of PBDEs as well as age, race, smoking habits and occupational exposure from pregnant women, 18 years and older who presented to two hospitals in Indiana during August-December 2001. 12 paired samples of maternal and cord blood were obtained. The concentration (ΣPBDEs including BDE-47, -99, -100, -153, -154 and -183) in maternal sera ranged from 15 to 580 $ng.g^{-1}$ lipid (median 37 $ng.g^{-1}$ lipid) with the concentration in the foetal sera ranging from 14 to 460 $ng.g^{-1}$ lipid (median 39 $ng.g^{-1}$ lipid). The median concentration of BDE-47 was 28 and 25 $ng.g^{-1}$ lipid for the maternal and foetal blood, respectively. Maternal blood was obtained prior to delivery and foetal blood was obtained from the umbilical cord vein by syringe after delivery. Both the maternal and foetal samples were dominated by BDE-47 followed by BDE-99. BDE-209 not analysed in this study. The authors report that the maternal and cord blood PBDE concentrations were highly correlated (r-squared = 0.986) and there was not a statistically significant difference between maternal and foetal blood. Likewise, PBDE concentrations did not vary according to age or BMI, nor was there any relationship between infant birth weight or any clinical parameters and PBDE concentrations.

Liberda et al (2005) determined the concentration of PBDEs in plasma from First Nations women aged 18 to 40 years living in the Ouje-Bougoumou community, a sub-artic population. Individual samples were analysed and the ΣPBDE concentration (BDE-28, -47, -85, -99, -100, -153, -154 and -183) was 28.99 $ng.g^{-1}$ lipid. The mean concentration of BDE-47 was 21 $ng.g^{-1}$ lipid. The congener profile was dominated by BDE-47 followed by BDE-153 and BDE-28.

Continental Europe

In Sweden van Bavel (2002) studied a cohort of 220 people from Sweden. This specific cohort included mother and son pairs of a non-cancer group and a group of which the sons were diagnosed with testicular cancer. The mean ΣPBDEs (BDE-47, -99 and -153) was 4.9 $ng.g^{-1}$ lipid, excluding outliers. The congener profile was dominated by BDE-47 followed by BDE-153, then BDE-99. There were 10 outliers with high concentrations of PBDEs, one exceeding 1000 $ng.g^{-1}$ lipid. The congener profile was slightly different for these samples with dominance by BDE-47, followed by BDE-99 and then BDE-153. High concentrations were found in both the non-cancer and cancer groups with two mother-son pairs identified as having high concentrations with the mother having higher concentrations than the son.

In Germany, Schröter-Kermani et al (2000) determined the concentrations of PBDEs in 1999 in human whole blood from 10 males and 10 females, aged 20-30 years. BDE-47 occurred at the highest level, followed by BDE-153, BDE-99 and BDE-100. The authors found concentrations of PBDEs in females to be approximately 20% lower compared to males. The median ΣPBDE concentration (including BDE-28, -47, -66, -85, -99, -100, -153 and -154) was 4.7 $ng.g^{-1}$ lipid.

In Norway, Thomsen et al (2002) investigated temporal trends and the role of age and gender on PBDE concentration in human serum. Serum from 40 to 50-year-old men collected at six time periods during 1977 and 1999 was pooled into six samples by year of collection. These people were patients in county hospitals and disease or reason for hospitalisation were not considered when using serum for analysis. Eight groups of differing age and gender samples in 1998 were pooled into eight samples by age. BDEs investigated. The authors note that the sum of the six BDEs (BDE-28, -47, -99, -100, -153, -154) increased from 0.44 ng g^{-1} in 1977 to 3.3 ng g^{-1} lipid in 1999. The serum concentrations from the different age groups were relatively similar, except for the age group 0-4 years, which had 1.6-3.5 times higher concentrations than the other age groups. Women older than 25 years had lower concentrations of PBDEs and the body burden appears to be independent of age, except for infants 0-4 years. The authors suggest this is due to different age groups experiencing a similar lifetime exposure because the chemicals are relatively new. As in other studies BDE-47 dominated the congener profile.

Asia

In Japan, Koizumi et al (2005) investigated the level of four PBDE congeners (BDE-47, -99, -100 and -153) in blood from 40 females in 1980 and 40 females in 1995. The 1980 and 1995 samples were not obtained from the same individuals, but from individuals living in the same community. The authors reported that ΣPBDE concentrations increased significantly during the 15 years from 0.5 (3.5) to 1.8 (3.7) $ng.g^{-1}$ lipid ($p<0.05$). The 1980 samples were collected between 1977 and 1981 while the 1995 samples were collected between 1991 and 1997. 98% of participants were multiparous and most were farmers and-or farmers' wives with less exposure to contaminants living in rural areas, no occupational exposure and lived within the community their entire lives. Eight sites were sampled to represent the country geographically. One region had much higher concentrations of PBDEs than the other regions and also showed a 20-fold increase from 1980 to 1995. It has been hypothesised by the authors that this may be due to the huge notebook-type computer manufacturing factory in this particular community since the late 1980s. BDE-209 was not analysed. For the 1980 samples, BDE-99 was the dominant congener while in the 1995 samples, BDE-47 was the dominant congener.

Also from Japan, Takasuga et al (2004) investigated the concentration of PBDE in human whole blood from 11 husband and wife pairs. The concentration of PBDEs was higher in males than in females, although females had higher concentrations of BDE-209. The median concentrations of BDE-47 and ΣPBDEs were 0.74 and 9.5 $ng.g^{-1}$ lipid, respectively. Individual variation in homologue pattern was observed between families and between couples. Therefore exposure scenario in between husband and

wife seemed slightly different. BDE-209 was the dominant congener followed by -47, -153, -183, -100 and -99.

In Korea, Kim et al (2005) determined the levels of PBDEs in the general population compared to workers. Samples were collected in 2001 from 22 residents (10 males and 12 females) living in areas near municipal waste incinerators. The general population was aged between 21-63 years and had lived within 5km of two incinerators for at least five years. The general population residents did not have occupational exposure to PBDEs. The ΣPBDE concentration including BDE-28, -47, -99, -100, -153, -154 and -183 was 15.1 $ng.g^{-1}$ lipid. The dominant congener was BDE-47.

United Kingdom

Thomas et al (2006) reported the concentration of PBDEs in blood serum from 154 people at 13 UK locations in 2003. The median concentration of ΣPBDEs (-35, -37, -47, -49, -71, -75, -77, -85, -99, -100, -119, -138, -153, -154, -166, -181, -183, -190, and -209) was 5.6 $ng.g^{-1}$ lipid. The median concentration of BDE-153 was higher than that of BDE-47 (1.7 vs 0.82 $ng.g^{-1}$ lipid). The concentration of BDE-209 ranged from <15 to 240 $ng.g^{-1}$ lipid.