FOOD STANDARDS
Australia New Zealand
Te Mana Kounga Kai – Ahitereiria me Aotearoa

POLYBROMINATED DIPHENYL ETHERS (PBDE) IN FOOD IN AUSTRALIA

STUDY OF CONCENTRATIONS IN FOODS IN AUSTRALIA INCLUDING DIETARY EXPOSURE ASSESSMENT AND RISK CHARACTERISATION

EXECUTIVE SUMMARY

Polybrominated diphenyl ethers (PBDEs) are anthropogenic chemicals that are added to a wide variety of consumer/commercial products in order to improve their fire resistance. PBDEs are found in the form of a number of closely related chemicals, known as congeners. PBDEs have come under increased scrutiny because of their potential to impact upon human health and the environment. Food Standards Australia New Zealand (FSANZ) commissioned an analytical survey of PBDEs in a limited range of Australian foods, which taken together with data on the concentration of PBDEs in breast milk in a separate study commissioned by the Commonwealth Department of the Environment and Water Resources, have been used in a population dietary exposure assessment and health risk appraisal.

A total of 35 foods were analysed including meat, dairy, oils and spreads, bread and bakery products, and vegetables to cover as broad a spectrum of the diet as possible. The majority of the congener measurements analysed reported 'non-detects', i.e. the congeners were not found. Furthermore, the following foods had no detectable PBDE congeners: tap water, full fat milk, low fat milk, canola oil and iodised table salt. The highest levels of PBDEs were detected in boiled eggs, grilled pork chops, bacon and cream, with infant foods containing relatively low levels. The concentrations and types of PBDEs detected in Australian food appear to be reasonably similar to those reported in other areas of the world. Australian breast milk has been found to contain PBDEs at levels below those reported for North America but higher than those in Europe and Japan. To estimate dietary exposure arising from 'non detects', lower, middle and upper bound mean concentrations were determined for each food. The lower bound assumes there are actually no PBDEs present for the 'non-detect' results, while the middle and upper bounds assume either that PBDEs may be present at half of the limit of quantification (LOQ) of the assay or at the LOQ, respectively. It is recognized that the upper and middle bound estimates are likely to be conservative since they will be profoundly influenced by any low sensitivity assay with a high LOQs.

Dietary exposure assessments were undertaken for the following population groups: 3 month old breast fed infants; 9 month old infants receiving half of their energy requirements from either breast milk or infant formula, and half from solid foods; 2-5 year old males and females; 6-12 year old males and females; 13-18 year old males and females; 19 years and above males and females. Dietary exposure of the general population to PBDEs in food is low. The main contributors to dietary exposure across the majority of the population groups assessed were bread, eggs, vegetables and meat, except for fully breast fed infants.

In the absence of sufficient data to establish a tolerable weekly or monthly intake for PBDEs, the margin of exposure (MOE) was used to determine the health risk of dietary PBDE exposure in different population groups. The MOEs for the majority of the population groups assessed were around or above 10,000. The population groups with comparatively high dietary exposures included 3 month old fully breast fed infants (MOE = 2000) and 9 month old breast fed infants (MOE ~3000). However, as these dietary exposures were still over 1,000-fold below any adverse effect dose observed in laboratory animals they are unlikely to constitute a risk to infant health. In addition, the significant benefits of breast feeding have been reported by many national Health Authorities and the World Health Organization.

On the basis of the available data and taking into account all the inherent uncertainties and limitations it can be concluded that the Australian public health risk arising from dietary exposure to PBDEs in food is unlikely to be of public health and safety significance.

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ABBREVIATIONS

ATDS	Australian Total Diet Survey
BDE	Brominated diphenyl ethers (used when specifying the congener or degree of bromination)
DIAMOND	Dietary Modelling of Nutritional Data (FSANZ computer software program), based on food consumption data from the 1995 NNS
EnTox	National Research Centre for Environmental Toxicology
FAO	Food and Agriculture Organization
FSANZ	Food Standard Australia New Zealand
fw	Fresh weight (the term *wet weight* is equivalent)
lw	Lipid weight
IUPAC	International Union of Pure and Applied Chemistry.
JECFA	Joint FAO/WHO Expert Committee on Food Additives
LOD	Limit of Detection
LOR	Limit of Reporting
LOQ	Limit of Quantification
MOE	Margin Of Exposure
mg/kg	Milligrams per kilogram
mg/kg bw	Milligrams per kilogram of body weight
NSS	National Nutrition Survey
ng	Nanogram (10^{-9} g)
NHMRC	National Health and Medical Research Council
NICNAS	National Industrial Chemicals Notification and Assessment
PBDE	Polybrominated diphenyl ether (used to describe all PBDEs when not necessarily specifying which congener or degree of bromination)
pg	Picogram (10^{-12} g)
pg/g	Picogram (10^{-12} g) per gram. Equal to nanogram per kilogram (ng/kg)
TMI	Tolerable Weekly Intake
TWI	Tolerable Monthly Intake
WHO	World Health Organization
ww	Wet weight

Note: Definitions for some of these abbreviations can be found in Appendix 1.

1. BACKGROUND

Polybrominated diphenyl ethers (PBDEs) are anthropogenic chemicals that are added to a wide variety of consumer/commercial products (e.g. high-impact polystyrene, flexible polyurethane foam, textile coating (not clothing), wire and cable insulation, electrical/electronic connectors and other interior parts) in order to improve their fire resistance. It is believed that the addition of PBDEs to consumer products have significantly contributed to a reduction in the loss of human life and property through fire.

PBDEs have come under increased scrutiny because of their potential to impact upon the environment and human health. Some PBDEs have been nominated for possible inclusion on the Stockholm Convention on Persistent Organic Pollutants, to which Australia is a Party. In Australia, relevant Australian Government agencies, such as the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) and the Department of Environment and Water Resources have been undertaking monitoring programs to determine whether PBDEs are present in humans, the environment and food.

As a part of these monitoring programs and their reporting, Food Standards Australia New Zealand (FSANZ) has undertaken a dietary exposure assessment to estimate the potential exposure of Australians to PBDEs from food. A survey of PBDEs in a range of foods, which are representative of the total diet, was the first step in conducting a dietary exposure assessment. This dietary exposure assessment was used to characterise the risk associated with PBDE residues in food.

1.1 Polybrominated diphenyl ethers

PBDEs have the general chemical formula $C_{12}H_{(9-0)}Br_{(1-10)}$ where the sum of hydrogen and bromine atoms must equal to 10. Although it is theoretically possible to have 209 possible hydrogen/bromine atom combinations (or congeners) divided into 10 homolog groups (i.e. mono to decabromodiphenyl ethers) many are chemically unstable. There are three major commercial PBDE products with an average of five (pentabromodiphenyl ether), eight (octabromodiphenyl ether) or ten (decabromodiphenyl ether) bromine atoms. They do not solely contain penta-, octa- and deca-brominated congeners, respectively, as suggested by the names. These names refer to an average number of bromine atoms per molecule. For example, PentaBDE contains mostly congeners in the tetrabrominated (BDE-47), pentabrominated (BDE-99, BDE-100) and hexabrominated (BDE-153, BDE-154) range. OctaBDE commonly contains hexabrominated (BDE-153, BDE-154), heptabrominated (BDE-183), octabrominated (BDE-196, BDE-197, BDE-203), nonabrominated (BDE-206, BDE-207) and decabrominated (BDE-209) congeners (see Table 1). While there is some variability in congener composition in the main commercial products they are all characterised by having poor water solubility (<1 microgram/mL) but good solubility in oils and fats. PBDEs are added to a wide variety of consumer/commercial products (e.g. high-impact polystyrene, flexible polyurethane foam, textile coating (not clothing), wire and cable insulation, electrical/electronic connectors and other interior parts) in order to improve their fire resistance.

Table 1: General composition of commercial PBDE flame retardants and substitution pattern of selected congeners

PBDE	
Mixture	***Congener composition (% of total)***
PentaBDE	24–38% tetraBDEs, 50–60% pentaBDEs, 4–8% hexaBDEs
OctaBDE	10–12% hexaBDEs, 44% heptaBDEs, 31–35% octaBDEs, 10–11% nonaBDEs, < 1% decaBDEs
DecaBDE	< 3% nonaBDEs, 97–98% decaBDE
Individual congeners	***Substitution pattern***
BDE-47	2,2',4,4'-tetraBDE
BDE-99	2,2',4,4',5-pentaBDE
BDE-153	2,2',4,4',5,5'-hexaBDE
BDE-209	2,2',3,3',4,4',5,5',6,6'-decaBDE

Source: JECFA (2006)

Additive flame retardants, such as the PBDEs, are mixed with the polymer material being treated, but are not chemically linked to the polymer matrix (as is the case with reactive flame retardants). This means there is potential for the chemical to slowly leach out of the treated plastic product over its functional lifetime. The environmental release of PBDEs can therefore occur not only during their production, but also through the use and/or disposal of the products that contain PBDEs (Ohta *et al*, 2002).

Commercial production of PBDEs began in the 1970s and their use has increased over time due to stricter fire control measures in many countries and greater use of plastic materials and synthetic fibres (ASTDR, 2004). PBDEs are now considered to be ubiquitous in the environment, having been detected from the Arctic to the Antarctic. PBDEs have been detected in sediments, marine mammals, fish, bird eggs and human milk, serum and adipose tissue (Darnerud *et al*, 2001).

1.2 Routes of exposure

A number of studies from around the world, including Australia and New Zealand, indicate that human exposure to PBDEs is widespread; although at this stage the body burden is considered to be low (Toms *et al*, 2006). Some longitudinal studies have shown that the levels of PBDEs in the environment and human tissue have increased over time (Jones-Otazo *et al*, 2005). For example, in Sweden, levels in breast milk increased 60-fold between 1972 and 1997 (from 0.07 to 4.02 ng/g, respectively[Meironyte et al., 1999]), and in Canada levels in breast milk increased over 200 fold from samples collected in 1982 (mean 0.2 ng/g) and 2001- 2002 (mean 42.5 ng/g) (Health Canada, 2006). However, more recent studies in Sweden have demonstrated a reduction in levels of PBDE in breast milk (2.14 ng/g). This is thought to be a result of a decline in the use of products containing PBDE (Guvenius et al., 2003).

Although the presence of PBDEs in the blood lipid fraction indicates exposure, it is currently not possible to determine the major route of that exposure. Based on the evidence provided in this report it is unlikely to be from the diet. House dust appears to be a major source but there appears to be little correlation with the year of house construction, type of flooring (i.e. hardwood versus carpet) or the number of television sets or personal computers in the home (Jones-Otazo *et al.,* 2005; Stapleton *et al*, 2005). A recent review of studies relating to the US population, concluded dietary exposure did not explain the current body burdens and exposure to house dust was estimated to account for 82% of the overall estimated intake (Lorber, 2007).

1.3 PBDEs in food and breast milk

In 2004, the Australian Government Department of the Environment and Water Resources (formerly the Department of the Environment and Heritage) contracted the University of Queensland's National Research Centre for Environmental Toxicology (EnTox) to measure PBDEs in breast milk samples which had been collected in 2002/2003 (Harden *et al*, 2005). The results of this analysis indicated that on a worldwide basis, the levels of PBDEs detected in Australian breast milk were higher than those levels observed in Europe and Japan, but lower than those observed in North America (Harden *et al*, 2005). However, it should be noted that much of the data from Japan is based on very low sample numbers. In some cases, it is the result of only a single analysis. The levels reported for North America are likely to be related to their high utilisation of products and articles containing flame retardants, particularly the high relative usage of pentaBDE in North America (Bromine Science and Environmental Forum, 2000).

Around the world, PBDEs have been detected in dairy products, meat, fish, fish oils, shellfish, eggs, vegetables and vegetable oils (Bocio *et al*, 2003; Knutsen *et al*, 2005).

2. SURVEY OF PBDEs IN AUSTRALIAN FOOD

The analysis of PBDEs in selected foods was performed in accordance with quality assurance procedures and the results were forwarded to FSANZ in order to calculate the estimated dietary exposures. The analytical methodologies used are reported in Appendix 2.

2.1 Foods included in the survey

The 35 foods (see Appendix 3 for full list) selected for PBDEs analysis were selected from the range that had already been sampled for the 22nd Australian Total Diet Study (ATDS). Foods were selected for analysis if they were thought to potentially contain higher concentrations of PBDEs, based on overseas studies, or if they could contribute significantly in the diet due to higher consumption. While it was recognised that the range of foods selected was relatively small due to budget constraints it was considered that it was adequate to provide an estimate of overall dietary exposure.

To best represent the food consumed all the foods analysed in this study were prepared to a 'table ready' state, in accordance with the usual Total Diet Study methods (see http://www.foodstandards.gov.au/monitoringandsurveillance/australiantotaldiets1914.cfm). For example; the chicken breast, beef sausage, potatoes and lamb chops were cooked. Appendix 3 provides the list of foods indicating how they were prepared.

The samples analysed for PBDEs were selected at random from the composite samples collected by the Australian States and Territories for the 22nd ATDS. Each composite sample analysed comprised three primary samples ('purchases') from a particular Australian State or Territory. Appendix 3 summarises the derivation of the samples.

2.2 PBDE sample analysis

The analysis included 26 individual congeners (including tri-, tetra-, penta-, hexa-, hepta-, octa- nona-, and deca–brominated diphenyl ethers) and these are summarised in Table 2. These congeners were chosen as they were commonly found in food and the environment and also had

validated methods of analysis. The samples were analysed on a fresh weight basis and concentrations reported in picograms per gram. If more than one composite sample for a food was analysed for PBDEs then the mean concentration (pg/g, fresh weight) of the samples was reported for that food.

Table 2: Individual congeners analysed

Individual congeners analysed	Abbreviation (IUPAC No.)	Congener classification
2,2',4-triBDE	BDE 17	Tribrominated diphenyl ether
2,4,4'-triBDE	BDE 28	
2',3,4-triBDE	BDE 33	
2,2',4,4'-tetraBDE	BDE 47	Tetrabrominated diphenyl ether
2,2'4,5'-tetraBDE	BDE 49	
2,3',4,4'-tetraBDE	BDE 66	
2,3',4',6-tetraBDE	BDE 71	
3,3'4,4'-tetraBDE	BDE 77	
2,2',3,4,4'-pentaBDE	BDE 85	Pentabrominated diphenyl ether
2,2',4,4',5-pentaBDE	BDE 99	
2,2',4,4',6-pentaBDE	BDE 100	
2,3',4,4',6-pentaBDE	BDE 119	
3,3',4,4',5-pentaBDE	BDE 126	
2,2',3,4,4',5'-hexaBDE	BDE 138	Hexabrominated diphenyl ether
2,2'4,4',5,5'-hexaBDE	BDE 153	
2,2',4,4',5,6'-hexaBDE	BDE 154	
2,3,3',4,4',5-hexaBDE	BDE 156	
2,3,4,4',5,6-hexaBDE	BDE 166	
2,2',3,4,4',5',6'-heptaBDE	BDE 183	Heptabrominated diphenyl ether
2,2'3,4,4',6,6'-heptaBDE	BDE 184	
2,3,3',4,4',5',6-heptaBDE	BDE 191	
2,2',3,3',4,4',5,6-octaBDE	BDE 196	Octabrominated diphenyl ether
2,2'3,3'4,4',6,6'octaBDE	BDE 197	
2,2',3,3',4,4',5,5',6-nonaBDE	BDE 206	Nonabrominated diphenyl ether
2,2',3,3',4,4',5,6,6'-nonaBDE	BDE 207	
2,2',3,3',4,4',5,5',6,6'-decaBDE	BDE 209	Decabrominated diphenyl ether

2.3 Analytical method detection limits

The Limit of Quantification (LOQ) is the lowest concentration of a chemical that can be detected and quantified, using the specified laboratory method and equipment, with an acceptable degree of certainty. In contrast the Limit of Detection (LOD) is the lowest concentration of a chemical that can be qualitatively detected using a specified laboratory method and/or item of laboratory equipment (i.e. its presence can be detected but not accurately quantified). In this analysis of PBDEs the LOQ is equal to the LOD and this limit varies for each congener in each sample analysed. There is a lower degree of certainty where the results are reported as being less than the LOQ and the relative uncertainty increases further where the LOQ is higher. Appendix 2 describes the analytical method used in the study.

2.3.1 Lower, middle and upper bound concentrations

A human dietary exposure assessment needs to take into consideration whether the sampling was representative of all foods and whether the detection assay was sufficiently sensitive. Another important aspect is the procedure used to estimate the contribution of congeners that are not actually detected or quantified. Such 'non-detect values' need to be considered because it may mean that either the compound was actually not present or that the assay method used was not sufficiently sensitive to quantify it. When PBDE congeners are not detected there are three commonly used approaches to incorporate such estimates into the exposure assessment. One is to assume that the congener is not actually present and assign a zero value. This estimate is commonly known as the *lower bound*. The other two procedures involve assuming either that the congener may be present at ½ of the LOQ of the assay or at the LOQ. These two estimates are referred to as the *middle* and *upper* bound estimates respectively. The upper bound estimate is likely to be a gross overestimate of the likely true value since the assumption that all congener concentrations reported as being < LOQ are actually at the LOQ is highly conservative. The extent of over-estimation decreases with increasing sensitivity of the analytical methodology. It should be noted that if the lower bound and upper bound totals are far apart (as is often the case, particularly when assay methodology is not particularly sensitive), the middle bound estimate will not be necessarily any closer to the 'true' exposure than is either of the other two estimates. Lower, middle and upper bound concentrations are listed in the report however only the lower and upper bound values for each PBDE congener are presented in Appendix 5.

3. DIETARY MODELLING

Dietary modelling is a tool used to estimate exposures to food chemicals from the diet as part of the risk assessment process. Dietary modelling uses analytical results for individual foods in combination with food consumption data to calculate estimates of dietary exposure that can be compared to established reference health standards. Food regulators have used dietary modelling techniques internationally for a many years as part of the risk assessment process to determine if dietary exposure to specific food chemicals represents an unacceptable risk to public health and safety. The comparison of dietary exposure estimates to these health standards is crucial in identifying whether the estimated dietary exposure to food chemicals could potentially result in an unacceptable health risk to any population sub-group.

3.1 Food consumption data

The dietary exposure assessment was conducted using food consumption data from the 1995 National Nutrition Survey (NNS) that surveyed 13 858 Australians aged 2 years and above using a 24-hour food recall methodology.

The foods reported as being consumed in the 1995 NNS were matched (or mapped) to the 35 foods analysed (refer to Appendix 6). This process assigns the levels of PBDEs detected in the survey foods to the appropriate food consumption data to estimate dietary exposure to PBDEs. Given that it is impractical to analyse all foods in the food supply, a single food (for example, carrots) may be assumed to represent a whole group of foods (for example, all vegetables). Recipes are used for mixed foods to assign their ingredients to the appropriate survey food (for example, the proportion of potato in Shepherd's Pie). The mapping process may result in the estimated dietary exposures being overestimated as it is assumed that the analytical level of PBDEs in an analysed food, is representative of all foods in that group. Where significant uncertainties in the data exist, conservative assumptions are generally used to ensure that the dietary exposure assessment does not underestimate exposure.

3.1.2 Population groups assessed

The population groups assessed were aged:

- 3 months;
- 9 months;
- 2-5 years;
- 6-12 years;
- 13-18 years; and
- 19 years and above.

These age groups were selected as they represent specific life stages such as infants (3 and 9 months), toddlers (2-5 years), school children (6-12 years), teenagers (13-18 years) and adults (19 years and above). Males and females were assessed separately for all age groups except for infants aged 3 months and 9 months.

3.2 Dietary exposure calculations

DIAMOND (Dietary Modelling of Nutritional Data) is a computer program developed by FSANZ to computerise dietary exposure assessment calculations. The dietary exposure to PBDEs was calculated for each individual in the NNS using his or her individual food records from the dietary survey. The DIAMOND program multiplies the specified concentration of PBDEs by the amount of food that an individual consumed in order to estimate the exposure to PBDEs from each food. Once this has been completed for all of the foods specified to contain PBDEs, the total amount of PBDEs consumed from all foods is summed for each individual. Population statistics (mean and high percentile exposures) are then derived from the individuals' ranked exposures. This process is repeated based on lower, middle and upper bound PBDE concentrations.

Where estimated dietary exposures are expressed per kilogram of body weight, each individual's total dietary exposure is divided by their own body weight, the results ranked, and population statistics derived. A small number of NNS respondents did not provide a body weight. These respondents are not included in calculations of estimated dietary exposures that are expressed per kilogram of body weight. The food consumption patterns of the minor number of respondents who did not provide a weight are generally consistent with those that did and therefore their non inclusion in the distribution of estimated dietary exposures on a body weight basis is not considered to be of significance. A summary of the mean body weights for each age/gender group assessed can be found in Appendix 7.

3.2.1 Assumptions in the dietary exposure assessment

The aim of the dietary exposure assessment was to make as realistic an estimate of dietary exposure to PBDEs as possible. However, where significant uncertainties in the data existed, conservative assumptions were generally used to ensure that the dietary exposure assessment did not underestimate exposure.

In the dietary exposure assessment the following broad assumptions were made:

- all NNS foods that were mapped to an analysed food contain PBDEs at the specified concentration for the analysed food;
- where an individual NNS food was not mapped to an analysed food, it contains a zero concentration of PBDEs (e.g. fruit);

- where a food has a specified PBDE concentration, this concentration was carried over to mixed foods where the food has been used as an ingredient e.g. milk in a sauce or custard; and
- consumption of foods as recorded in the NNS represent current food consumption patterns.

In the mapping of food consumption data to the foods for which survey data were available, the following assumptions were made:

- all nuts have the same PBDE content (including peanut butter);
- fruit bread and cheese-, bacon- or ham- topped bread and rolls do not contain significantly different PBDE from other bread, therefore they have been considered to be equivalent to white bread;
- all brown and multigrain bread are equivalent to white bread; and
- all vegetables are equivalent to either potato or carrots. The higher concentration determined for carrots was used to represent all vegetables, other than potatoes, as this better reflected levels found in vegetables in other studies (Ohta *et al*, 2002) and yielded a more conservative estimate of exposure.

3.2.2 Food contribution calculations

The percentage contribution each food makes to total estimated dietary exposures was calculated by dividing the sum of all consumers' exposures from one food group by the sum of all consumers' exposures from all foods containing PBDEs assessed, and multiplying this by 100. Lower bound results were used to calculate the percentage contribution each food group makes to total estimated exposures, as this provides the best indication of the food groups most likely to contribute to dietary exposure as it only includes foods containing levels of PBDEs at or above the LOQ.

3.3 Limitations associated with the food consumption data

Conducting dietary modelling based on 1995 NNS food consumption data provides the best estimate of actual consumption of a food and the resulting estimated exposure to a food chemical. However, it should be noted that limitations exist within the NNS data. These limitations relate to the age of the data and the changes in eating patterns that may have occurred since the data were collected. Generally, consumption of staple foods such as fruit, vegetables, meat, dairy products and cereal products, which make up the majority of most people's diet, is unlikely to have changed markedly (Cook *et al.*, 2001a; Cook *et al*., 2001b). However, there is an increasing level of uncertainty associated with the consumption of other foods where these may have changed in consumption since 1995, or where new foods on the market were not available in 1995. Trends such as the increasing move towards eating leaner cuts of meat would be reflected in this study through the analytical results as the samples are purchased to best represent foods consumed at the time of the ATDS survey and analysed in their ready to eat state. In the dietary exposure assessment the total amount of meat consumed in 1995 is assumed to be the total amount of 'leaner' meat consumed in the present day.

A limitation of estimating dietary exposure over a period of time is that only 24-hour dietary survey data were available, and these tend to overestimate habitual food consumption amounts for high consumers. Therefore, predicted high percentile exposures are likely to be higher than actual high percentile exposures over a lifetime. For commonly consumed foods such as bread, milk and meat, which are generally consumed on a daily basis by the majority of Australians, a 24-hour recall provides a relatively accurate estimate of daily consumption amounts over a

longer period of time. For occasionally consumed foods, the predicted daily consumption based on 24-hour dietary survey data is not representative of longer-term daily consumption.

3.4 Infant diets

Estimated dietary exposures to PBDEs were calculated for infants at two different ages. Firstly for infants aged 3 months who are solely breast fed and secondly for infants 9 months of age who consume a diet consisting of milk (either breast milk or infant formula) and solid foods. The estimates of exposure for infants were based on model diets. The aim of the infant dietary exposure assessment was to make as realistic an estimate of dietary exposure to PBDEs as possible. However, where significant uncertainties in the data existed, conservative assumptions were generally used to ensure that the dietary exposure assessment did not underestimate exposure.

In order to assess the dietary exposure to PBDEs for infants at 3 months of age that are fully breast fed, the quantity of breast milk consumed needed to be determined and mean body weight. Reilly *et al.* (2005) found that infants at 3 months of age from presumably well-nourished populations, including Australia, consumed a mean amount of 796 grams of breast milk per day (weighted for sample size). The median (50^{th} percentile) weight for a female infant at 3 months is 6.4 kg (WHO, 1983). Girl's weights were used for the 3 month old diet because they tend to be lighter than boys at 3 months but consume similar quantities of breast milk and so the body burden for a bio-accumulative compound will be correspondingly greater. Therefore, using these figures, a fully breast fed infant at 3 months of age consumes 124 g breast milk/kg bw/day. The PBDEs concentrations for breast milk used for calculating PBDE exposure were taken from Harden *et al.* (2005).

An assessment of dietary exposure for infants aged 9 months of age was also undertaken to determine dietary exposure to PBDEs when a combination of solid foods and either breast milk or infant formula were being consumed. As there were no food consumption data available from the 1995 NNS on children under two years, a diet was constructed. The patterns of consumption of a two year old child from the NNS were scaled down and used to determine the solid portion of the 9 month old's diet, with the exclusion of some foods that are inappropriate for infants.

Details of the constructed diet for infants at 9 months are available in Appendix 4. In this constructed diet, milk was assumed to provide half of the infant's energy requirements (Hitchcock et al., 1986). The infant's energy requirement was calculated from the recommended energy intake (FAO, 2004) for a 9 month old boy at the 50^{th} percentile for weight (WHO, 1983), which gives an energy requirement of 3082 kJ/day. Boys' weights were used because at 9 months boys tend to have higher energy and food requirements relative to body weight and so at this age the body burden for a bio-accumulative compound will be correspondingly greater for boys. Therefore, based on the energy value for infant formula of 274kJ/100g, a 9 month old infant was assumed to consume 562 g of formula per day. Infant formula was the sole source of the milk in the constructed infant diet for formula fed infants.

In order to determine the dietary exposure to PBDEs for breast fed infants at 9 months, this same constructed diet was used and all milk consumption was assumed to be breast milk. All other assumptions relating to the construction of the diet remained the same. As for infants at 3 months, the PBDE concentrations for breast milk used for calculating PBDE exposure for infants at 9 months were taken from Harden *et al.* (2005). As milk was assumed to only provide 50% of the energy requirements of an infant at 9 months, PBDE concentrations for all other foods, making up the other 50% of energy requirements, were derived from the analytical survey as described above.

For breast fed infants, the following assumptions were made:

- milk was the only food consumed for infants at 3 months;
- the PBDE concentrations in breast milk were the same for all infants, irrespective of the number of breast fed siblings;
- PBDE concentrates in the lipid portion of breast milk and the mean fat content of Australian breast milk is 3.7% (Harden *et al*., 2005);
- one gram of breast milk is equal to one millilitre of breast milk; and
- breast milk provides the same energy per gram as infant formula.

4. FOOD SURVEY ANALYTICAL RESULTS

4.1 PBDE concentrations in foods

A summary of the lower, middle and upper bound summed concentrations levels of PBDEs in foods are shown in Table 3 and Figures 1-4. As food samples were randomly collected nationally and composited prior to analysis, it is likely that only limited conclusions on the possible sources of contamination may be drawn from an examination of individual sample congener profiles. However, raw analytical data showing congener profiles for each food analysed has been presented in Appendix 5.

There were 35 different foods and a total of 39 samples analysed (which included duplicate samples of; full fat, low fat milk, pork chops and beef sausage), with each analysis examining 26 congeners, which gave rise to 1014 congener data points. Of these 1014 congener measurements, the majority (69%) reported non-detects, with a total of 310 congener data points (31%) with quantified values or "detections" with values ≥LOQ. Based on the lower bound mean, the following foods had no detectable PBDE congeners: tap water, full fat milk, low fat milk, canola oil and iodised table salt. In the cases of tap water and canola oil the LOQs were the highest in the assay (3,120 pg/g and 1,022 pg/g respectively) creating a large difference between the lower and upper bounds.

Table 3: Upper, middle and lower bound fresh weight levels of PBDEs in foods (pg/g)

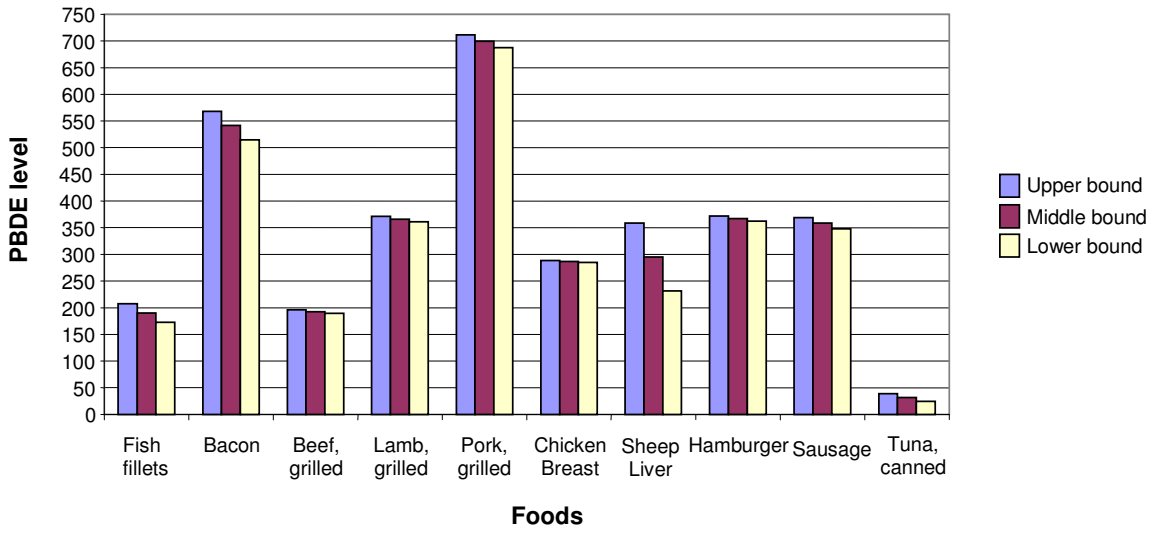
Foods	PBDEs (pg/g fresh weight)[3]		
	Upper bound mean	Middle bound mean	Lower bound mean
Bacon	568.3	541.3	514.3
Beef steak, rib/ribeye/sirloin, grilled	196.1	193.0	189.8
Bread, white	225.4	209.9	194.4
Butter, regular	357.8	273.4	189.1
Canola oil	1022.0	511.0	0
Carrots, cooked	60.1	58.8	57.6
Cheese, cheddar, full fat	265.7	193.5	121.3
Chicken breast	288.9	287.1	285.3
Chocolate, milk	377.2	343.1	309.0
Coconut, desiccated	364.6	304.1	243.6
Cream, pure (not thickened)	512.48	490.7	469.0
Eggs, boiled	954.6	932.1	909.7
Fish fillets	208.0	190.3	172.6
Hamburger[1]	372.3	367.1	361.8
Ice Cream, full fat, vanilla	133.8	118.9	104.0
Infant cereal, mixed	91.8	91.4	90.9
Infant dessert, dairy based	75.3	74.1	73.0
Infant dessert, fruit	73.8	63.0	52.3
Infant dinner	101.7	94.3	86.8
Infant formula, prepared	100.6	94.5	88.3
Lamb chops, loin, grilled	371.2	366.1	360.9
Margarine/ Margarine spread, poly-unsaturated	383.5	273.8	164.1
Milk, full fat[2]	73.9	36.9	0
Milk, modified, low fat[2]	59.8	29.9	0
Peanut butter	232.4	178.9	125.4
Pizza[1]	409.3	366.8	324.3
Pork chops, grilled[2]	711.3	699.4	687.5
Potato crisps	374.8	337.6	300.4
Potato, cooked	39.1	20.4	1.7
Salt, table, non-iodised	68.0	34.0	0
Sausage, beef[2]	369.0	358.5	348.0
Sheep liver	358.7	295.2	231.7
Tuna, canned in brine	39.2	31.9	24.5
Water, tap	3120.0	1560.0	0
Yoghurt, fruit, full fat	95.8	89.7	83.6

Explanatory Note: 1 = Hamburger and pizza were sourced from major fast-food outlets
2 = Values are averaged from two sample results
3 = The middle and upper bounds include values where congeners were not detected and therefore conservative. The derivation of the upper, middle and lower bound concentrations is outlined in Section 2.3.1.

4.1.1 Meat and meat products

Figure 1 shows that for meat and meat products, pork chops followed by bacon and lamb had the highest levels of PBDEs. Chicken, lamb and beef had the least variation between the lower and upper bounds, while the fish, bacon and sheep liver had the most variation between the lower and upper bound. This variation is a result of the number of <LOQ results for congeners in the foods.

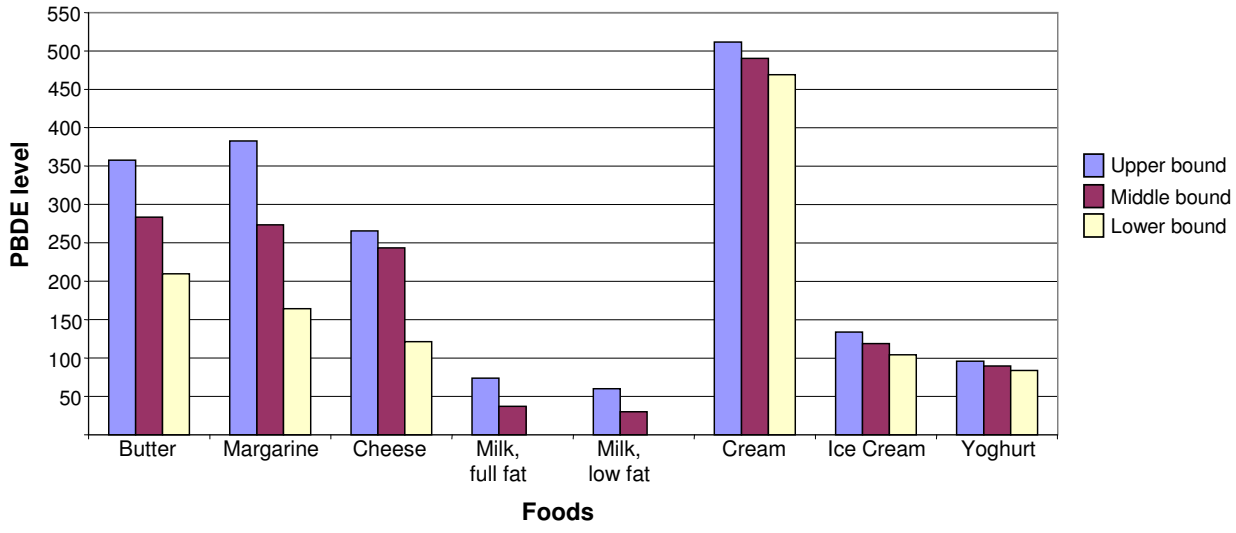
Figure 1: Levels of PBDEs (pg/g fw) in meat and meat products



4.1.2 Dairy products

The highest levels of PBDEs for dairy products were found in cream, followed by both butter and margarine (Figure 2). Again there was a high level of variation between the lower and upper bound for all dairy products, resulting from the high number of <LOQ results for congeners.

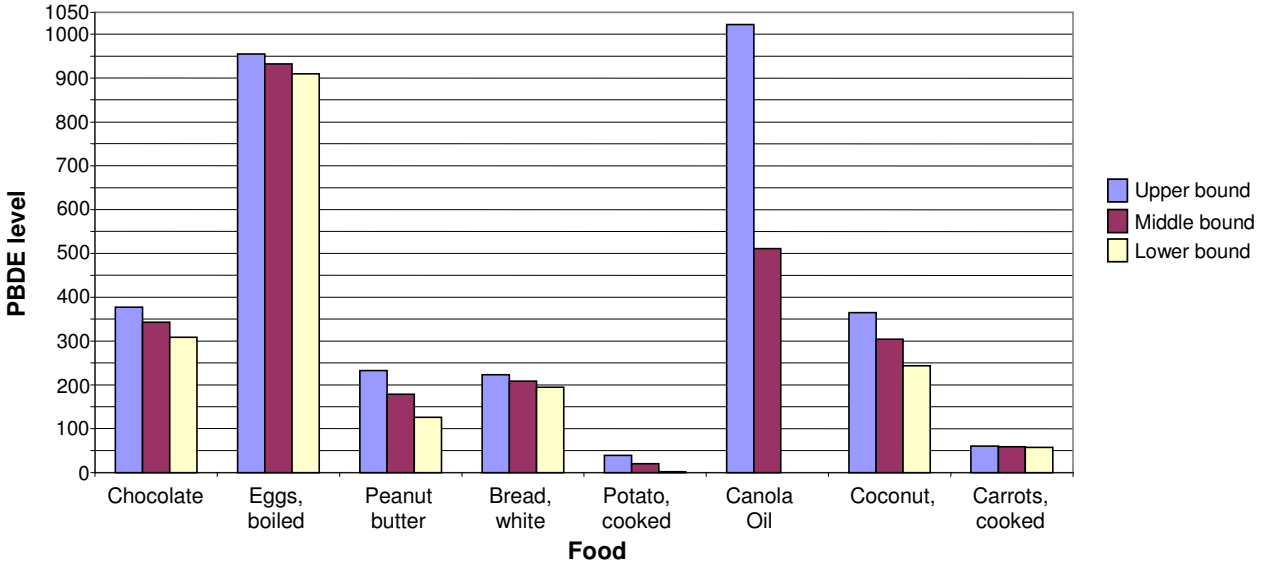
Figure 2: Levels of PBDEs (pg/g fw) in dairy products



4.1.3 Other foods

Of all foods tested, the highest quantified levels of PBDEs were measured in eggs, with little variation between the lower and upper bounds (Figure 3). Canola oil had the second highest (tap water was highest) upper bound concentration, however, this was due to the high LOQ for this sample and no PBDE congeners were actually quantified in canola oil.

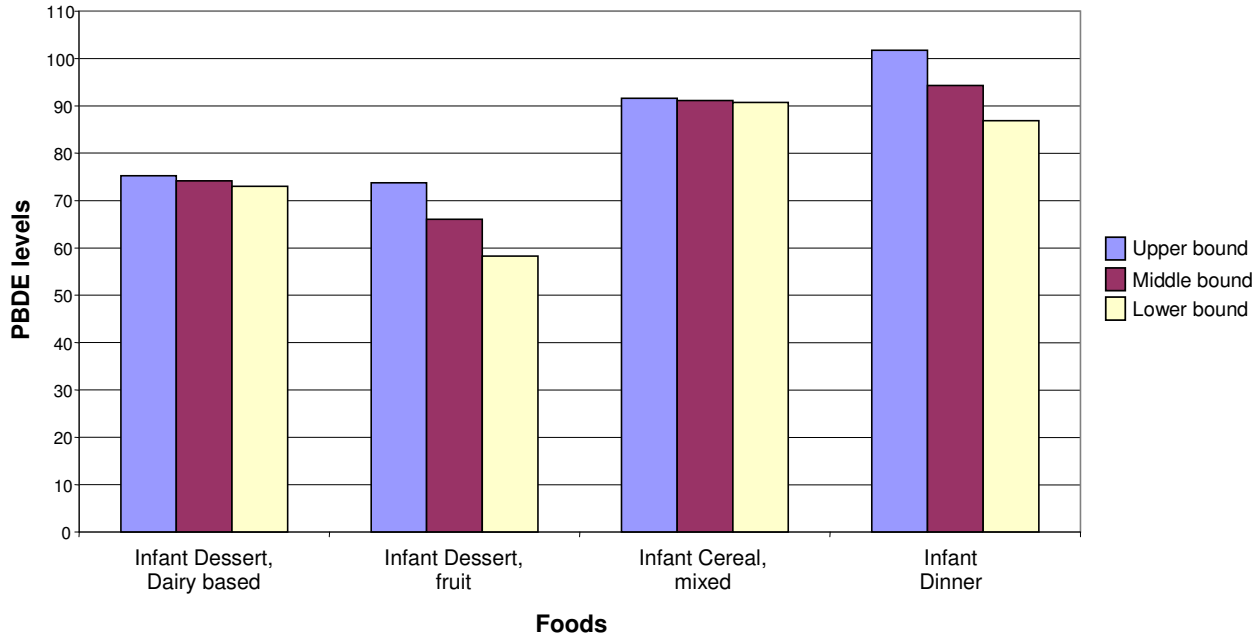
Figure 3: Levels of PBDEs (pg/g fw) in other foods



4.1.4 Infant foods

Infant foods had relatively low quantified levels (52-90 pg/g at the lower bound) in comparison to levels detected in other foods (Table 3). Figure 4 shows that dairy based infant dessert and infant cereal had little variation between lower, middle and upper bounds while fruit based infant dessert and infant dinner had higher variation across the bounds.

Figure 4: Levels of PBDEs (pg/g fw) in infant foods



4.2 PBDE Congener profiles

Full details of the PBDE congener profiles for each food for fresh weight analysis and lipid weight analysis from this study can be found in Appendix 5. In the literature BDE 47, 99, 100, 153 and 154 are the most abundant in organisms and are generally the most commonly reported (Harrad *et al.,* 2004). Appendix 5 shows the sum of all congeners across all foods. Tetra and penta-BDEs appear to be the predominant congeners in Australian food.

4.2.1 Meat and meat products

BDE 47 (tetra-BDE), BDE 100 (penta-BDE) and BDE 209 (deca-BDE) were the dominant congeners in meat products. Beef, bacon, lamb and sheep liver had highest levels of BDE 47 (65, 160, 160 and 110 pg/g respectively) while pork had higher levels of BDE 99 (280, 260 pg/g). Interestingly the profile of beef sausage and hamburger (beef) differed from that of beef steak as BDE 209 was highest (110 and 120 pg/g) in sausages, while BDE 99 was highest in hamburgers (110 pg/g). Chicken also had high levels of BDE 209 (120 pg/g).

4.2.2 Fish

Two types of fish were analysed in this study, fish fillets and canned tuna, both of which had BDE 47(tetra-BDE) at 79 and 15 pg/g as the dominant congener.

4.2.3 Dairy products

In cheese, butter, cream and ice cream BDE 47 (tetra-BDE) followed by BDE 99 (penta-BDE) had the highest levels. Full fat and reduced fat milk had similar profiles however there was a large range between lower and upper bound values. In yoghurt, BDE 209 predominated at 42 pg/g.

4.2.4 Other foods

The profile for polyunsaturated margarine was similar to that of butter with BDE 47 (tetra-BDE) at 95 pg/g being highest followed by BDE 99 (penta-BDE) at 76 pg/g. The dominant congener in bread was BDE 47 at 73 pg/g. For boiled eggs and pizza BDE 99 was the dominant congener (360 and 140 pg/g respectively). In potato crisps and peanut butter BDE 47 was dominant (140 and 85 pg/g respectively) while in chocolate, BDE 209 (deca-BDE) was dominant at 290 pg/g.

4.2.5 Fruit and vegetables

The dominant congener for cooked carrots was BDE 47 (tetra-BDE) at 22 pg/g, for cooked potato all the congener levels were quite low with BDE 183 (penta-BDE) dominating at 0.53 pg/g. Desiccated coconut had a different profile to all other foods as BDE 28 and 33 (tri-BDE) were dominant at 130 pg/g.

4.2.6 Infant foods

All of the infant foods had similar congener profiles with BDE 47 (tetra-BDE) being the highest for all products.

4.3 Comparison of PBDE concentrations in foods from other countries

A comparison of PBDE concentrations in food from different countries is difficult as there are many differences in foods sampled, analytical methodologies used and the calculation and reporting of individual congeners. However, Table 4 gives some indication of the measured concentrations of PBDEs in selected Australian foods compared with those from other parts of the world. In addition to these data some countries have focused on particular food items. For example, Japan measured PBDE concentrations in a variety of fish species (17.7-1720 pg/g fresh weight). While levels of PBDE in Japanese fish were relatively high other foods recorded relatively low concentrations. Japan also analysed beef (16.2 pg/g), pork (63.6 pg/g) and chicken (6.25 pg/g) meat as well as potato (47.6 pg/g), spinach (134 pg/g) and carrot (38.4 pg/g) (Ohta et al., 2002). While Australian foods appear to have PBDE levels which are reasonably similar to those reported in other areas of the world caution needs to be exercised in making direct comparisons. Differences in reported concentrations may be due to variation in:

- analytical methods used;
- Limit of Quantification or Reporting;
- treatment of non detections;
- congeners included in the analysis and summed;
- averaging of concentration data across foods; and/or
- fresh/wet weight vs lipid weight analysis and conversion factors.

Table 4: Comparison of multinational PBDE mean values for specific foods in pg/g lipid weight (lw), wet weight (ww) or fresh weight (fw)*

Food	Mean concentration of PBDEs (pg/g)				
	Norway (lw)	**Australia (fw)** ND=1/2 LOQ	**Canada (ww)**	**USA (ww)**	**Spain (ww)** ND=1/2LOR
Butter	227.0	273.4	264.5	412.0	N/A
Margarine	3079.0	273.8	4.4	0.9	155.0
Cheese[(1)]	114.0	193.5	58.9	679.0	N/A
Milk, whole	282.0	36.9	3.39	N/A	24.0
Milk, formula	N/A	N/A	0.3	30.3	N/A
Ice cream, full fat	257.0	118.9	18.35	149.0	N/A
Yoghurt[(2)]	N/A	89.7	8.47	31.5	N/A
Eggs	3852.0	932.1	79.6	73.7	64.0
Fresh Fish	(Salmon) 2390.0	190.3	1313.4	1660.7	553.5
Canned Fish	N/A	(Tuna) 31.9	36.3	N/A	260.0
Bacon[(3)]	N/A	541.3	169.2[(3)]	52.0	N/A
Beef Steak	105.0	193.0	46.2	N/A	42.0
Lamb	(Mutton) 592.0	366.1	39.6	N/A	31.0
Pork	537.0	699.4	40.8	41.0	172.0
Chicken Breast	932.0	287.1	N/A	283.0	10.0
Sausages[(4)]	495.0	358.5	163.2	1353.0	N/A

* Data taken from Knutsen et al., 2005; Health Canada, 2004, Schecter et al., 2004; Bocio et al., 2003. Australian data from this study.

Explanatory Notes: 1 = Cheese in Australia was cheddar only; Canada and USA also included cottage and processed varieties; 2 = Australian sample was full fat with fruit, Canada undefined, USA low fat; 3 = For Canada "cured pork" is assumed to be bacon; 4 = Includes wieners, pork sausages & sausages; Direct comparisons are not possible as some countries use lipid weight and others use fresh (wet) weight.

5. DIETARY EXPOSURE

5.1 Estimated dietary exposures to PBDEs

5.1.1 Estimated dietary exposures for infants

In this assessment 3 month old infants are assumed to have a diet consisting entirely of breast milk. As described in section 3.4, a 3 month old, fully breast fed infant consumes 796 g breast milk per day (or 124 g on a per kilogram bodyweight basis using a mean bodyweight of 6.4 kg) (Reilly *et al.,* 2005). Analysis of PBDEs in breast milk by Harden *et al*. (2005) determined a mean concentration of 11 ng/g lipid, with a range of 6-18 ng/g lipid. Given that the mean fat content of Australian breast milk is 3.7% (Harden *et al*., 2005), a 3 month old, fully breast fed infant would be exposed to 324 ng PBDEs per day [3.7% (mean fat content) x 796 g (daily consumption of breast milk) x 11 ng/g lipid (mean concentration of PBDEs)] (Table 5). No lower, middle or upper bound estimates of exposure were determined for breast milk fed infants because the data of Harden *et al.* (2005) were composite samples taken from 17 regions and all samples contained some PBDEs.

Estimated dietary exposures for the 9 month old, where the milk component of the diet was breast milk, were slightly lower at the mean (between 247 and 302 ng/day lower bound to upper bound) compared to the 3 month old breast fed infant. The 95th percentile exposures were between 616 and 756 ng/day. For the 9 month old diet where formula was included, the estimated dietary exposures at the mean and 95th percentile were lower than the mean exposures for the breast fed 9 month old; between 67 and 130 ng/day at the mean and between 168 and 325 ng/day at the 95th percentile. This difference can be attributed to the higher concentration of PBDEs in breast milk (407 pg/g) compared to infant formula (94.5 pg/g middle bound). Calculations of the estimated dietary exposure for 9 month olds can be found in Appendix 9.

5.1.2 Estimated dietary exposures for population groups aged 2 years and above

For the dietary exposure assessments conducted using the 1995 NNS, 99.6% or more of the respondents in each age/gender group assessed were consumers of PBDEs. This results in estimated exposures for all respondents or consumers only of PBDEs being virtually identical. Therefore, results reported below are for consumers only of PBDEs. Appendix 7 shows the number of respondents and the number of consumers of PBDEs in each age/gender group assessed (excluding infants).

Table 5 summarises the mean and 95th percentile exposures of each age/gender group assessed expressed in nanograms per day. Appendix 7 provides a summary of mean food consumption data for consumers only of each survey food derived from the 1995 NNS using DIAMOND. Depending on gender and concentration used (lower, middle or upper bound) the estimated mean dietary exposures to PBDEs for 2-5 year olds range between 60 and 2885 ng/day and 95th percentile exposures range between 128 and 6875 ng/day. For 6-12 year olds estimated exposures range between 87 and 3742 ng/day at the mean and 178 and 7440 ng/day at the 95th percentile. For 13-18 year olds estimated exposures range between 96 and 5588 ng/day at the mean and 189 and 13446 ng/day at the 95th percentile. For the 19 years and above age group estimated exposures range between 90 and 3982 ng/day at the mean and 188 and 10795 ng/day at the 95th percentile.

Table 5: Estimated dietary exposure to PBDEs for each population group assessed

Age	Gender	PBDE[1] concentration Type[2]	Mean dietary exposure (nanogram/day)[3]	95th percentile dietary exposure (nanogram/day)
3 months Breast fed infant[4]	Both combined	Lower Bound	324	NA
9 months Formula fed infant	Both combined	Upper Bound	130	325
		Middle Bound	99	247
		Lower Bound	67	168
9 months Breast fed infant	Both combined	Upper Bound	302	756
		Middle Bound	274	686
		Lower Bound	247	616
2-5 years	Male	Upper Bound	2885	6875
		Middle Bound	1474	3468
		Lower Bound	63	128
	Female	Upper Bound	2553	5526
		Middle Bound	1306	2798
		Lower Bound	60	128
6-12 years	Male	Upper Bound	3742	7440
		Middle Bound	1920	3789
		Lower Bound	100	202
	Female	Upper Bound	3443	7103
		Middle Bound	1765	3601
		Lower Bound	87	178
13-18 years	Male	Upper Bound	5588	13446
		Middle Bound	2865	6816
		Lower Bound	142	269
	Female	Upper Bound	4247	8671
		Middle Bound	2171	4420
		Lower Bound	96	189
19 years and above	Male	Upper Bound	3982	10795
		Middle Bound	2059	5456
		Lower Bound	136	287
	Female	Upper Bound	3531	8423
		Middle Bound	1810	4260
		Lower Bound	90	188

Explanatory notes: Figures are rounded to the nearest whole number;
1 = sum of all PBDE congeners;
2 = Lower Bound – assumes results reported as being below the LOQ are zero, Upper Bound – assumes results reported as being below the LOQ are at the LOQ, Middle Bound – assumes results reported as being below the LOQ are 50% LOQ;
3 = estimated dietary exposures are based on food consumption data from the 1995 NNS, with the exception of 3 month old infants, which was based on data from Reilly *et al.,* (2005) and Harden *et al.,* (2005); NA = not assessed.
4 = PBDE exposure estimates for breast fed infants were based on concentration data obtained from a separate study described in Harden *et al.* (2005).

5.2 Major contributing foods to total estimated dietary exposures

The major contributors (≥5%) to total PBDE dietary exposures are shown in Figures 5-14. Lower bound results were used to calculate the percentage contribution each food group makes to total estimated exposures, as this provides the best indication of the food groups most likely to contribute to dietary exposure as it only includes foods containing levels of PBDEs at or above the LOQ. It should be noted that the percent contribution of each food group is based on total dietary PBDE exposure for all consumers of PBDEs in the population groups assessed. Therefore the total PBDE exposures differ for each population group. The major contributors are shown for all population groups assessed, apart from infants 3 months of age who only consume breast milk. A full list of all the food groups and their contributions can be found in Appendix 8.

5.2.1 Infants aged 9 months

The major contributors to PBDE exposure for 9 month old infants were calculated based on the theoretical diet.

For 9 month old infants who consume infant formula, the major contributors were infant formula (74%) and bread (6%). Of all other foods in the infant diet, most contributed 1% or less to total PBDE exposure, except for eggs (3%), bacon (2%), vegetables (2%) and pizza (2%). Figure 5 shows the major contributors to PBDE exposure for 9 month old infants who consume infant formula.

For 9 month old infants who consume breast milk, the major contributor was breast milk (93%). All other foods combined contributed 7% of total PBDE exposure, with each food contributing less than 1%, except bread (2%). Figure 6 shows the major contributors to PBDE exposure for 9 month old infants who consume breast milk.

Figure 5: Major contributing foods to PBDE exposure in infants aged 9 months who consume infant formula

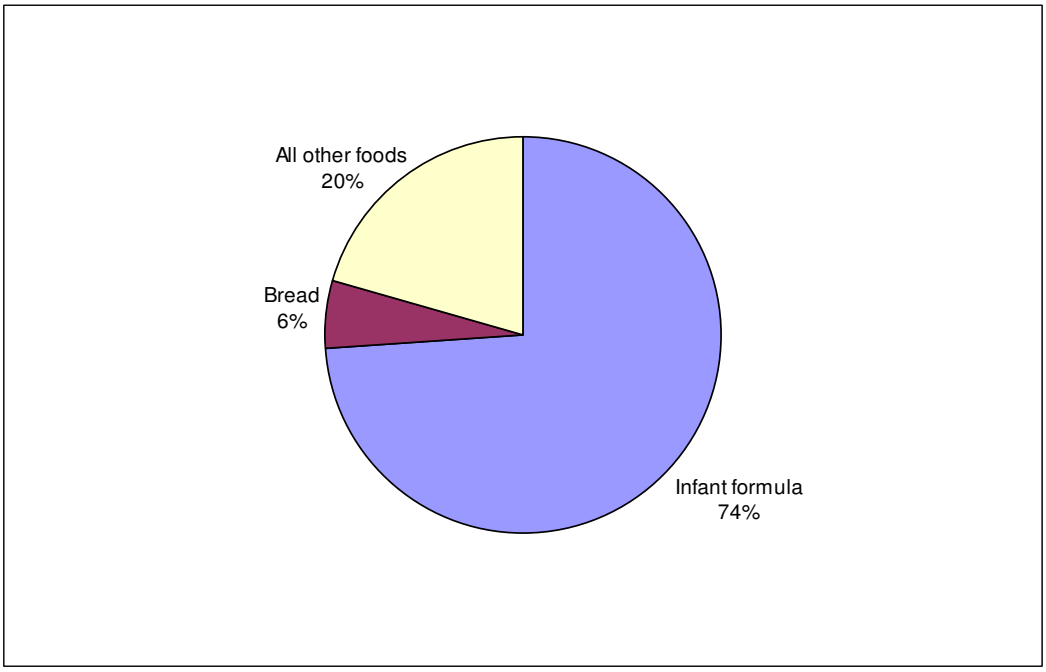
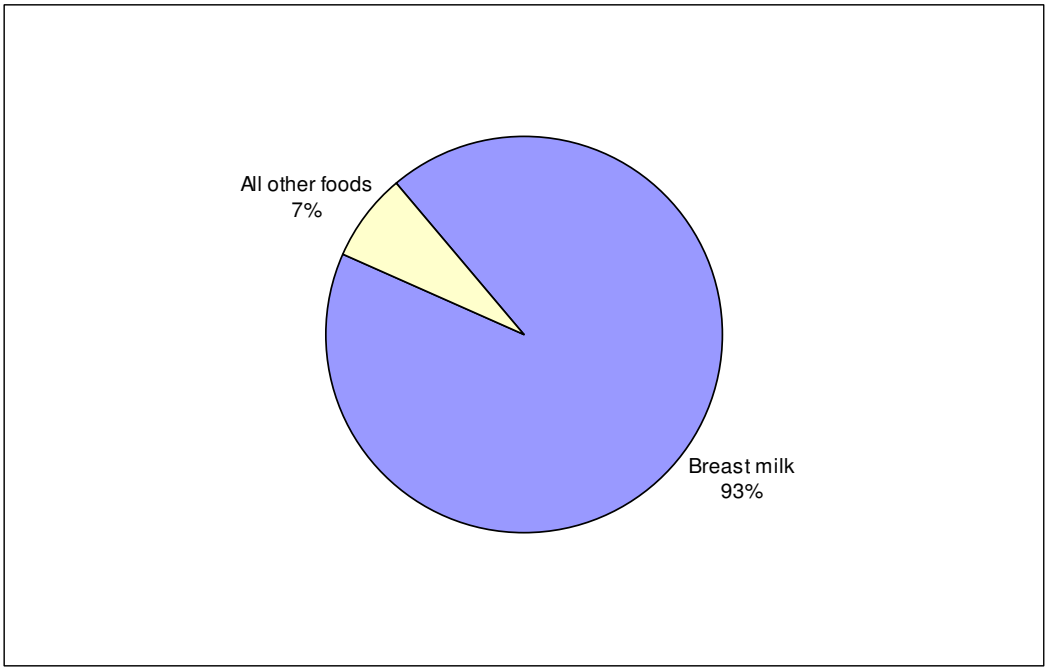


Figure 6: Major contributing foods to PBDE exposure in infants aged 9 months who consume breast milk



5.2.2 Children aged 2-5 years

Figures 7 and 8 show the foods which were major contributors to total PBDE exposure in males and females aged 2-5 years respectively. For 2-5 year old boys these included bread (24%), eggs (11%), bacon (7%), sausages (6%), chicken (6%), vegetables (5%), beef (5%) and pizza (5%). For 2-5 year old females these were similar and included bread (23%), eggs (14%), sausages (7%), chicken (6%), vegetables (6%), pizza (6%) and bacon (6%).

Figure 7: Major contributing foods to total estimated PBDE dietary exposure for males aged 2-5 years

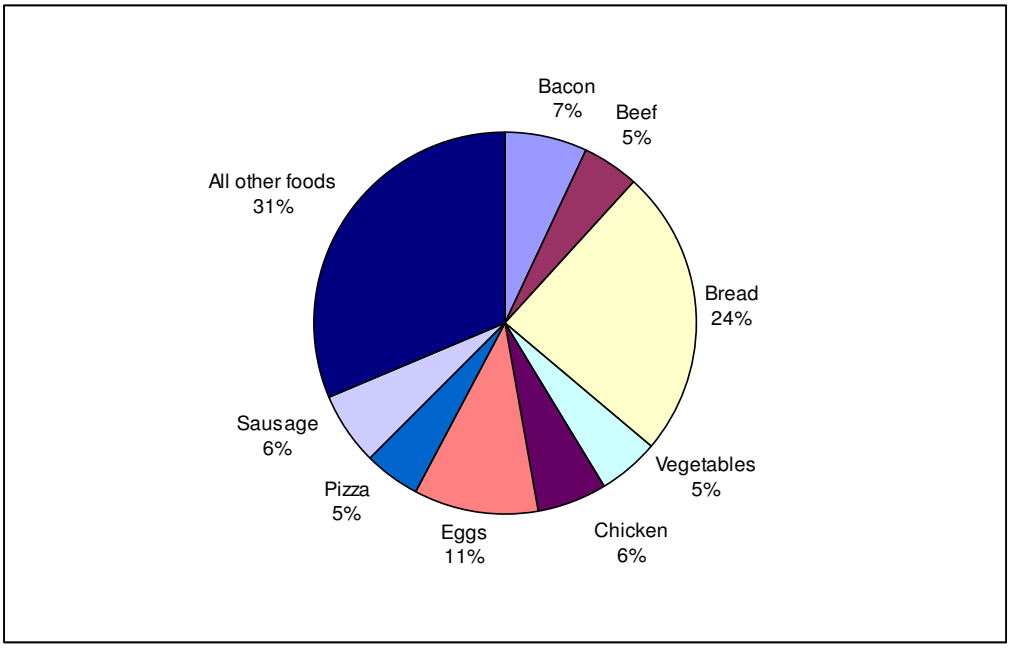
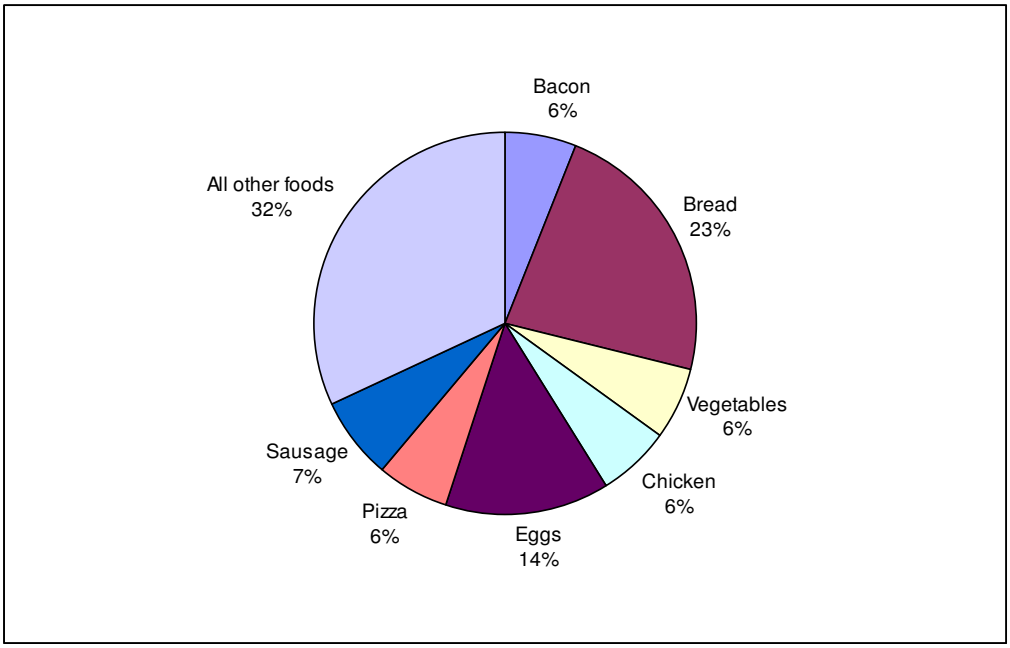


Figure 8: Major contributing foods to total estimated PBDE dietary exposure for females aged 2-5 years



5.2.3 Children aged 6-12 years

Figures 9 and 10 show the foods which were major contributors to total PBDE exposure in males and females aged 6-12 years respectively. For boys aged 6-12 years the major contributors were bread (21%), eggs (13%), pizza (9%), sausages (6%), chicken (6%), vegetables (5%), ice cream (5%), bacon (5%) and beef (5%).The major contributors for females were similar and included bread (20%), eggs (12%), vegetables (7%), pizza (7%), chicken (6%), sausages (6%) and ice cream (5%).

Figure 9: Major contributing foods to total estimated PBDE dietary exposure for males aged 6-12 years

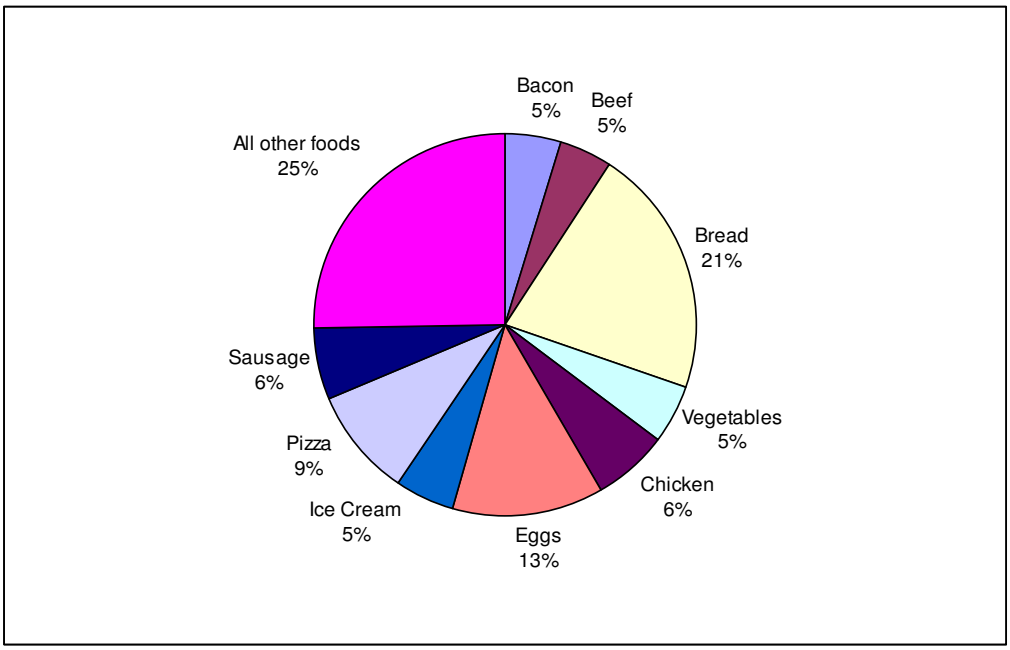
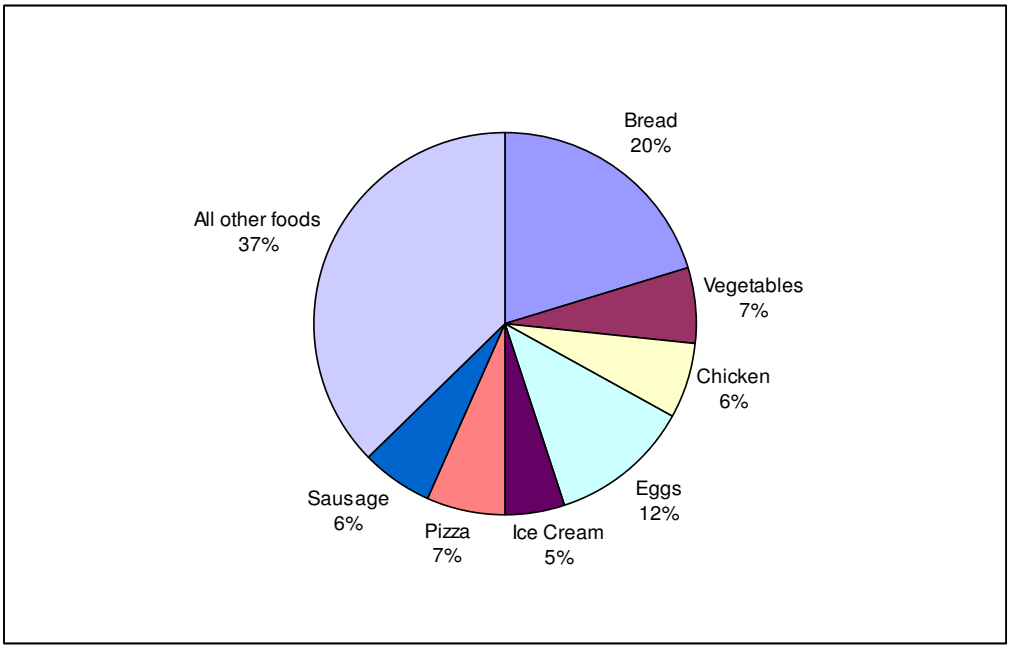


Figure 10: Major contributing foods to total estimated PBDE dietary exposure for females aged 6-12 years



5.2.4 Adolescents aged 13-18 years

Figures 11 and 12 show the foods which were major contributors to total PBDE exposure in males and females aged 13-18 years respectively. The major contributors for males were bread (18%), eggs (11%), pizza (10%), chicken (8%), sausages (6%), vegetables (6%), bacon (6%), beef (6%), hamburgers (5%) and ice cream (5%). For females 13-18 years the major contributing foods included bread (19%), eggs (10%), chicken (9%) vegetables (7%), pizza (7%) and beef (6%).

Figure 11: Major contributing foods to total estimated PBDE dietary exposure for males aged 13-18 years

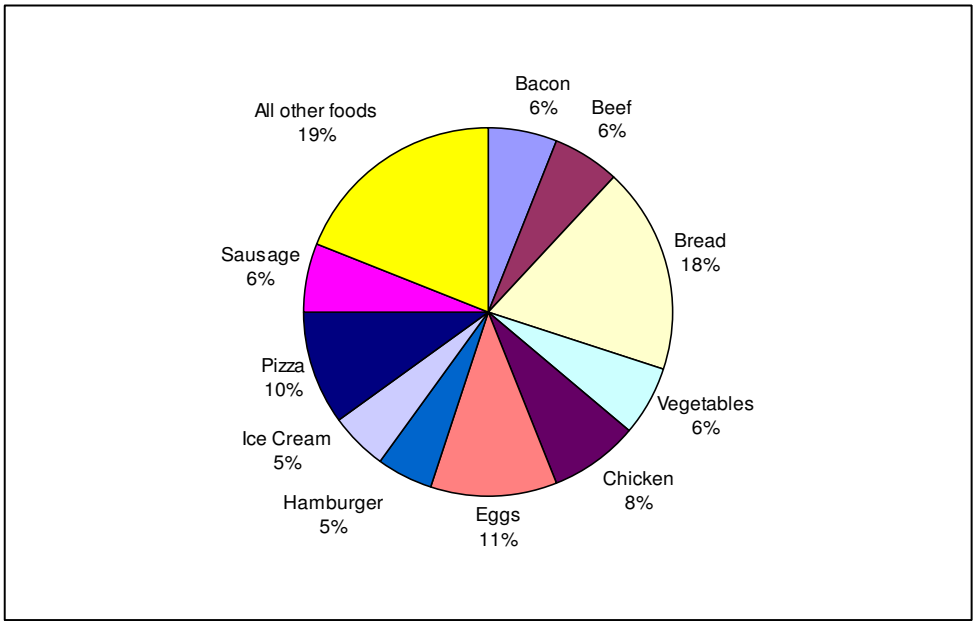
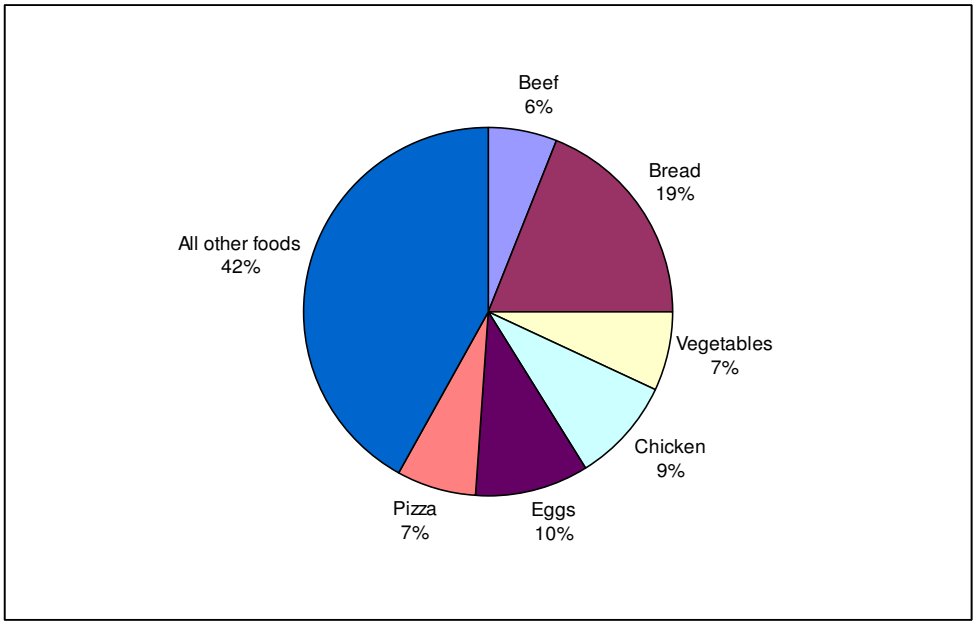


Figure 12: Major contributing foods to total estimated PBDE dietary exposure for females aged 13-18 years



5.2.5 Adults aged 19 years and above

Major contributing foods for PBDE exposure in the adult population were also similar to children and adolescents as shown in Figures 13 and 14. The major contributors for males 19 years and above were bread (18%), eggs (11%), vegetables (9%), chicken (8%), beef (8%), bacon (7%), pizza (7%), and pork (5%). For females 19 years and above the major contributing foods were similar and included bread (19%), eggs (13%), vegetables (12%), chicken (9%), beef (6%), bacon (5%) and pizza (5%).

Figure 13: Major contributing foods to total estimated PBDE dietary exposure for males aged 19+ years

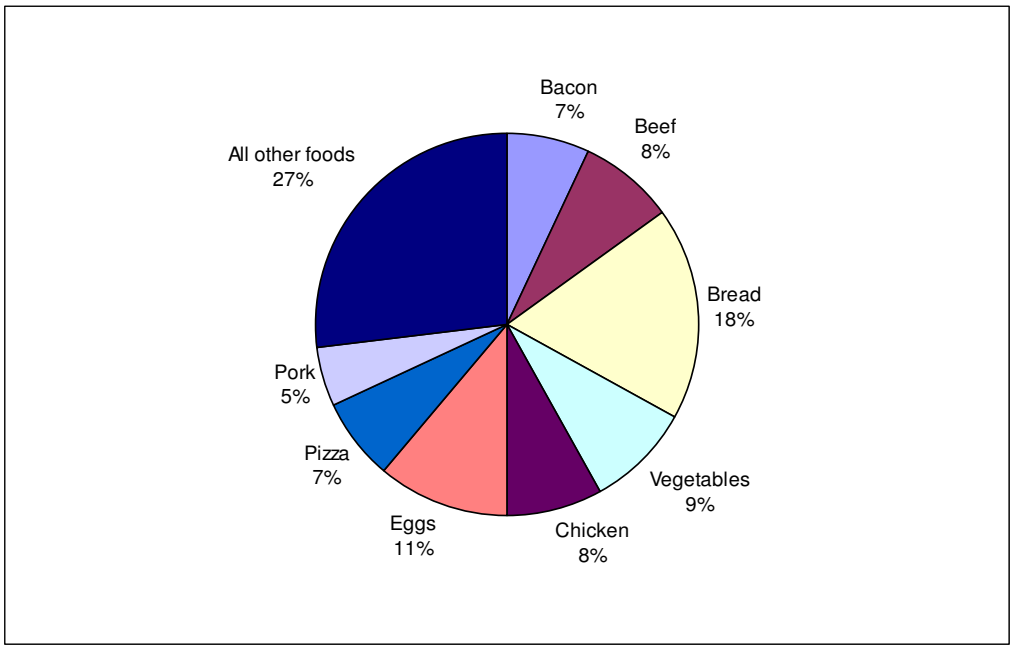
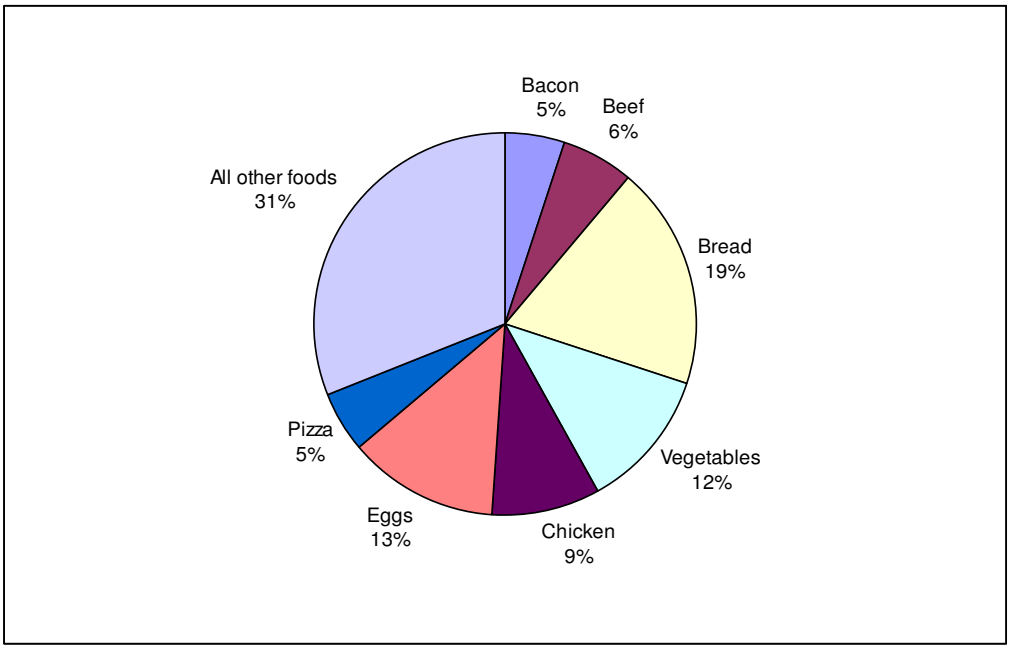


Figure 14: Major contributing foods to total estimated PBDE dietary exposure for females aged 19+ years



6. RISK CHARACTERISATION

In characterising the risk associated with PBDE exposure through food, it is necessary to consider the nature of the adverse health effects associated with exposure, the timeframe in which these effects are observed, whether there is a threshold dose for these effects, the level of exposure for sensitive subpopulations, and the limitations and uncertainties inherent in the available data.

6.1 Adverse effects and tolerable intake of PBDE

The nature of the adverse effects associated with PBDE exposure has been described in several national and international reviews. In Australia, NICNAS has reviewed the available toxicological data and identified an unacceptably low margin of exposure (MOE) in women of child bearing age when considering all sources of exposure, which includes food, inhalation and house dust. As a consequence, NICNAS has taken regulatory action against the more toxic PBDE mixtures by implementing an interim suspension on the importation and manufacture of penta-BDE flame retardants (NICNAS, 2007) and removal of octaBDE from the Australian Inventory of Chemical Substances (AICS).

In 2005 the Joint FAO/WHO Expert Committee on Food Additives (JECFA) reviewed the available toxicity data for polybrominated diphenyl ethers at its 64th meeting in Rome (JECFA, 2006). JEFCA serves as the scientific advisory body to the Codex Alimentarius Commission (CAC) on matters relating to food additives, contaminants, and residues of veterinary drugs in food. The CAC sets international food standards aimed at protecting the health of consumers and facilitating international trade in foods.

JECFA noted that 'the acute toxicity of mixtures of PBDEs is low in rodents. Generally, even at the highest doses (several g/kg bw), there are no observable effects in standard tests for acute toxicity after exposure to DecaBDE and OctaBDE, although certain effects (increased mortality, behavioural symptoms and changes in gross pathology) are seen after exposure to PentaBDE at similar high doses. Induction of enzymes, changes in levels of hormones and neurobehavioural effects are observed after bolus administration of mixtures of PBDEs (PentaBDE and OctaBDE), and of specific congeners at considerably lower doses. In short-term studies of toxicity, the main effects of mixtures of PBDEs were seen in the liver, kidney and thyroid of both sexes. Enlargement of the liver is a common finding, which may be connected to increased activity of microsomal enzymes in the liver. Histological changes occur in liver (enlargement, 'round bodies', vacuolization, necrosis), kidney (hyaline degenerative cytoplasmic changes) and thyroid (hyperplasia). In short-term studies, effects on thyroid hormone, vitamin A homeostasis and microsomal enzymes were observed at doses of 1–10 mg/kg bw per day'.

The JECFA meeting concluded that due to the paucity of reliable toxicity data it was not possible to establish a common mechanism of action for the observed perturbations in thyroid homeostasis and that an interpretation of the reported neurotoxicity findings could not be made due to the preliminary nature of the findings. Furthermore, 'The available data are inadequate to establish a common mechanism of action that would allow a single congener to be used as a surrogate for total exposure or, alternatively, as the basis for establishing toxic equivalency factors'. JECFA also noted that for related bioaccumulative persistent contaminants such as polychlorinated biphenyls and dioxins (WHO, 2002, JECFA, 2002), the appropriate dose-metric for interspecies comparison of risk was a measure of the accumulating potency of PBDEs ('body burden' approach). For the majority of PBDEs studied, however, the data from experimental animals or concentrations in human tissues were considered insufficient to allow a comparison with an external dose. While it would be normal practice to establish a provisional tolerable

weekly or monthly intake for bio-accumulative contaminants such as the PBDEs, the incompleteness of the available database for each congener led JECFA to conclude that 'The limited toxicity data suggests that for the more toxic [less brominated] PBDE congeners [eg. BDE-47 and BDE-99] adverse effects would be unlikely to occur at doses of less than approximately 100 µg/kg bw per day'. Following an independent review of the available toxicological data FSANZ concurs with the JECFA conclusions. This threshold dose (100 µg/kg bw/day) has been used as the basis for determining the magnitude of the MOE for all PBDEs.

6.2 Estimated dietary exposure to PBDEs expressed as a margin of exposure for population groups aged 2 years and above

As an initial step in characterising the risk associated with PBDE exposure from food, the estimated dietary exposures for various population groups were compared to the threshold dose of 0.1 mg/kg bw/day (see Table 6). The MOE is calculated by dividing the dose at which adverse effects were observed in laboratory animal studies by the estimated exposure to PBDEs from food. The lower the MOE, the greater is the public health risk.

As can be seen in Table 6, the choice of considering the lower, middle or upper bound estimate of dietary exposure has a profound effect on the magnitude of the MOE. The MOEs for the subgroup with the highest exposure, namely 2-5 year old males, are 14,000, 500 and 300 for 95th (results for female age 2-5 were similar) percentile exposures at the lower, middle and upper bounds, respectively. The very large difference in the MOE between the lower bound, which includes only measured values, and the middle or upper bounds, which includes default values for foods that were below the LOQ, warrants some comment. If there are many food types in which no PBDEs are detected then the effect on the apparent MOE, and therefore the level of concern, will be exaggerated. In the case of tap water this exaggeration was profound in this study. Tap water was the highest consumed food and while no congeners were actually detected in the sample the LOQ was also the highest with the resultant middle and upper bound dietary exposure estimates being substantially influenced by this single sample. Furthermore, only 31% of all congener data points reported a quantifiable measure in food. It is intuitive that the absence of any detectable PBDEs should not result in an unacceptable MOE (i.e. < 1,000). It seems reasonable therefore to conclude that the likely true MOE for 2-5 year old males will be between the middle and lower bound. However given the overrepresentation of non-detects in this survey, the MOE is more likely to be closer to the lower bound of 14,000 than the middle bound of 500. The MOE is therefore estimated to be around 10,000 based on 95th percentile exposures. The MOE will therefore be in excess of 10,000 for all other age/gender groups.

Table 6: MOEs for PBDE in various population groups aged 2 years and above

Age/Gender	Concentration Type[1]	Mean dietary exposure (ng/kg bw/day)	95th percentile dietary exposure (ng/kg bw/day)	MOE[2] (mean exposure)	MOE (95th percentile exposure)
2-5 years Male	Upper Bound	162	389	600	300
	Middle Bound	83	198	1200	500
	Lower Bound	4	7	28000	14000
2-5 years Female	Upper Bound	149	327	700	300
	Middle Bound	76	165	1300	600
	Lower Bound	3	7	29000	14000
6-12 years Male	Upper Bound	118	242	800	400
	Middle Bound	61	122	1700	800
	Lower Bound	3	7	32000	15000
6-12 years Female	Upper Bound	104	228	1000	400
	Middle Bound	53	115	1900	900
	Lower Bound	3	5	39000	20000
13-18 years Male	Upper Bound	89	199	1100	500
	Middle Bound	45	100	2200	1000
	Lower Bound	2	5	44000	21000
13-18 years Female	Upper Bound	74	153	1400	700
	Middle Bound	38	78	2700	1300
	Lower Bound	2	3	59000	29000
19+ years Male	Upper Bound	49	132	2000	800
	Middle Bound	25	67	4000	1500
	Lower Bound	2	4	59000	27000
19+ years Female	Upper Bound	54	131	1900	800
	Middle Bound	27	67	3600	1500
	Lower Bound	1	3	73000	34000

Explanatory Notes: Estimated dietary exposures in ng/kg bw/day are rounded to the nearest whole number. MOE figures are rounded to one significant figure for MOEs up to 1000 and to two significant figures where MOEs were above 1000;
1 = Lower Bound – zero value assigned to all results below the LOQ (non-detections).
Middle Bound – 50% LOQ value assigned to all results below the LOQ (non-detections).
Upper Bound – The LOQ assigned to all results below the LOQ (non-detections).
The middle and upper bounds include values where congeners were not detected and therefore conservative.
2 = threshold dose (0.1 mg/kg bw/day) ÷ mean dietary exposure (ng/kg bw/day).

6.3 Estimated dietary exposure of infants to PBDEs expressed as a margin of exposure

Table 7 gives the estimated mean dietary exposures for 3 month old, fully breast fed infants and 9 month old infants receiving 50% of their energy requirements from breast milk or infant formula, and 50% from solid foods. As discussed in Section 3.4, a theoretical diet was utilised for 9 month old infants because there are no data available from the NNS on children under 2 years old. For breast fed infants, calculations were based on the data of Harden *et al.,* (2005), which investigated the current levels of PBDE in the breast milk of Australian mothers, and the data of Reilly *et al.*, (2005), which determined the daily average consumption of breast milk. Using the results of these analyses and the measured concentrations of PBDE in infant formula, the MOE for PBDE in breast fed and non breast fed infants can be estimated.

Table 7: MOEs for PBDE for 3 and 9 month old infants

Population group	Mean Body Weight (kg)	Concentration Type[3]	Mean dietary exposure[4] (ng/kg bw/day)	95th percentile dietary exposure (ng/kg bw/day)	MOE[5] (mean exposure)	MOE (95th percentile exposure)
3 month breast fed infant	6.4[1]	Lower Bound	51	NA	2000	NA
9 month breast fed infant	9.2[2]	Upper Bound	35	87	3000	1200
		Middle Bound	32	80	3400	1300
		Lower Bound	29	72	3700	1500
9 month formula-fed infant	9.2[2]	Upper Bound	14	35	7000	2800
		Middle Bound	10	27	9300	3700
		Lower Bound	7	18	14000	5500

Explanatory Notes: Estimated dietary exposures in ng/kg bw/day are rounded to the nearest whole number. MOE figures are rounded to one significant figure for MOEs up to 1000 and to two significant figures where MOEs were above 1000;
1 = Female bodyweight WHO (1983);
2 = Male bodyweight WHO (1983);
3 = Lower Bound – zero value assigned to all results below the LOQ (non-detections).
Middle Bound – 50% LOQ value assigned to all results below the LOQ (non-detections).
Upper Bound – The LOQ assigned to all results below the LOQ (non-detections).
The middle and upper bounds include values where congeners were not detected and therefore conservative.
4 = mean dietary exposure (ng/day) (see Table 5) ÷ mean bodyweight;
5 = threshold dose (0.1 mg/kg bw/day) ÷ mean dietary exposure (ng/kg bw/day); NA = not assessed.

For formula fed 9 month old infants, the MOEs were approaching or exceeding 10,000 for lower and middle bound dietary exposure estimates. Even at the 95th percentile dietary exposure the MOEs were 5500 and 3700 at the lower and middle bound respectively, and are sufficiently high to be of no concern.

The level of exposure to PBDEs in 3- and 9 month old breast fed infants were approximately 4-fold higher than formula fed infants but still lower than the upper bound exposure of 2-5 year old males. Nine month old infants would be expected to have higher dietary exposure to PBDEs on a body weight basis compared to children aged 2-5 years due to their high food consumption relative to body weight. However, in the current study, the estimated dietary exposure (ng/kg bw/day) for 2-5 year olds was higher than for infants at the middle and upper bounds. This finding may be attributable to methodological differences in estimating dietary exposure of 9 month olds compared with other population groups. In particular, the theoretical diet constructed for infants does not use individual dietary records to calculate consumption and dietary exposure estimates. Based on the conclusion in Section 6.2 that the true likely MOEs for 2-5 year old males will be close to 10,000, the exposure estimates for 3 and 9 month old breast fed infants are unlikely to be of concern, particularly as they are three orders of magnitude above the estimated threshold dose (Tables 6 & 7).

While 3 month old breast fed infants qualitatively have the highest exposure to PBDEs, it is difficult to quantify the incremental increase in dietary exposure relative to any other population group because the estimation is based on a different dataset. In addition, 3 month old breast fed infants are unique in that they receive their entire dietary exposure through a single food source. The concentration of PBDE congeners in breast milk were based on the analysis of 157 samples, which were pooled into 17 different geographical regions (Harden *et al.,* 2005). All pooled

samples contained PBDEs and on this basis all 157 samples were assumed to have contained PBDEs. This is obviously different to all other foods, where some foods contained no PBDE congeners. In addition for other foods, of the 1014 congener measurements made, the majority (69.4%) reported non-detects. However, given that PBDEs are fat soluble, bioaccumulative compounds, it is reasonable to assume that they would be present in the majority, but not necessarily all, individual breast milk samples included in the analysis, although this would need to be confirmed.

While the MOE for 3 month old breast fed infants was the highest of the infant population groups studied, it is important to note that all babies are exposed to PBDEs even if they are not breast fed. Of particular importance is that the relatively high dietary exposure of 3 month old breast fed infants is transient in nature and the contribution of this food diminishes with growth and rapid dietary changes. In addition, the concentration of PBDEs detected in pooled Australia breast milk samples (11 ng/g lipid) is lower than the concentrations detected in North America (74 ng/g; Schecter *et al.*, 2003) but higher than in European countries (Sweden 1.84 ng/g; UK 6.6 ng/g) and Japan (0.93 ng/g) (summarised in Harden *et al.,* 2005). Calculations performed by JECFA (2006) indicated that the dietary exposure to PBDEs for breast fed infants in North America is 120 ng/kg bw/day, which is at least two-fold higher than the current Australian estimated dietary exposure.

Additionally, the dietary exposure of Australian 3 month old, breast fed infants would decrease over time in light of the implementation of the interim suspension on the import and manufacture of penta-BDE and removal of octa-BDE from AICS by NICNAS.

Due to the inherent uncertainties and conservative nature of the current assessment, the findings should not in any way be taken to suggest any change to the current advice of the World Health Organization (WHO) or the National Health and Medical Research Council (NHMRC) in Australia that breast milk is the best food for babies. In particular, the NHMRC recommends exclusive breast feeding to 6 months of age because it confers protection against infection and chronic diseases and leads to improved cognitive development.

On the basis of all of the above considerations the dietary exposure of 3 month old infants to PBDEs in breast milk does not constitute an appreciable risk to their health.

7. CONCLUSIONS

The data presented in this report represent the most comprehensive analysis of PBDE levels in Australian foods yet undertaken and form the basis of an analysis of the dietary exposure of the Australian population to PBDEs. The dietary exposure assessment has been used in conjunction with the available information on the hazard characterisation of PBDEs to assess the human health risk associated with exposure to PBDEs in food. It should be noted that the concentrations of the 26 different congeners have not been weighted to take account of differences in their relative toxicity. This is due to the absence of toxic equivalency factors. As a result the MOEs presented are lower (ie. more conservative) than would otherwise be the case.

It is re-assuring that of the 1014 congener measurements made, across the 39 individual samples (comprising 35 different foods) analysed, the majority (69%) reported non-detects. Furthermore, the following foods had no detectable PBDE congeners: tap water, full fat milk, low fat milk, canola oil and iodised table salt. Utilising these analytical data in combination with appropriate food consumption data enabled a dietary exposure assessment to be undertaken for various population groups, including infants. These calculations indicated that dietary exposure of the

general population to PBDEs in food is low, with the MOEs for the majority of population groups at or above 1,000 and therefore not of concern. The relatively low MOE of 600 calculated for 2-5 year old males based on the mean dietary exposure using upper bound PBDE concentrations was discounted due to the overly conservative nature of the estimate, particularly as the majority of samples contained no detectable PBDEs. Other population groups with relatively high dietary exposures included 3- and 9 month old breast fed infants. However, as these dietary exposures are still over 1,000-fold below any adverse effect dose observed in laboratory animals. On the basis of the available data and taking into account all the inherent uncertainties and limitations it can be concluded that the Australian public health risk arising from dietary exposure to PBDEs is unlikely to be of public health and safety significance.

Given that NICNAS has taken action against the more toxic PBDEs by implementing an interim suspension on the import and manufacture of penta-BDE and removing octa-BDE from AICS, and their use is already being phased out internationally, it is reasonable to expect the low levels found in food to diminish in the future.

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APPENDIX 1: DEFINITIONS AND GLOSSARY OF TERMS

Congeners
Closely related chemicals derived from the same parent compound.

fw (Fresh weight)
The amount of a food chemical which is present in a given weight of the while food as it is actually eaten. Fresh weight concentrations are used, combined with dietary survey data, to estimate dietary exposure.

Limit of Detection (LOD)
The LOD is the lowest concentration of a chemical that can be qualitatively detected using a specified laboratory method and/or item of laboratory equipment (i.e. its presence can be detected but not quantified). For the purposes of this study, analytical results reported as being less than the LOD were assumed to be zero.

Limit of Quantification (LOQ)
The LOQ is the lowest concentration of a chemical that can be detected and quantified, with an acceptable degree of certainty, using a specified laboratory method and/or item of laboratory equipment.

Limit of Reporting (LOR)
The LOR is the lowest concentration level that the laboratory reports analytical results. For the purposes of this report, the LOD was chosen as the basis for the LOR (i.e. the LOR is equivalent to the LOD).

Lower bound
An estimate of dietary exposure assuming analytical results reported as being below the LOQ are assumed to be equal to zero.

Mapping
The process that assigns the levels of substances detected in survey foods to the appropriate food consumption data to estimate dietary exposure to the substance. Given that a survey cannot analyse all foods in the food supply, a single survey food may be assumed to represent a whole group of foods with appropriate adjustment factors for concentration.

Middle bound
An estimate of dietary exposure assuming analytical results reported as being below the LOQ are equal to 50 % of the reported LOQ when used to calculate the sum of PBDEs.

Margin of Exposure (MOE)
The ratio of the No Observable Adverse Effect Level (NOAEL) to the estimated exposure dose.

Toxic Equivalency Factors (TEFs)
Are toxicity potency factors that are used by the World Health Organization (WHO) and by scientists and regulators globally as a consistent method to evaluate the toxicities of highly variable mixtures of dioxin and dioxin like compounds.

Upper bound
An estimate of dietary exposure assuming analytical results reported as being less than the LOQ are equal to the LOQ.

APPENDIX 2: METHODS OF ANALYSIS AND QUALITY ASSURANCE

Samples were composited to produce a homogenous sample. A rendered or extractable portion of fat was removed and spiked with a range of isotopically labelled surrogate standards. Clean up was affected by partitioning with sulphuric acid and then distilled water. Further purifications were performed using column chromatography on acid and base modified silica gels and basic alumina. After clean-up, the extract was concentrated to near dryness. Immediately prior to injection, internal standards were added to each extract, and aliquot of the extract was injected into the gas chromatograph. The analytes were separated by the gas chromatograph and detected by a high-resolution (≥10,000) mass spectrometer. The quality of the analysis was assured through reproducible calibration and testing of the extraction, cleanup, and Gas Chromatography/Mass Spectrometer systems.

The detection limit levels used in this method are highly sensitive and are usually dependent on the level of interferences rather than instrumental limitations. The concentrations in foods were quantifiable in the picograms per gram (parts per trillion) range. The analytical methodology for the determination of PBDEs were based on the Draft USEPA Method 1614 August 2003.

Table A 1: Limits of detection for PBDEs

Sample Type	**Test**	**Limit of Detection (units)**
Food (high lipid content >4% fat) Meat fat, vegetable oils, butter, cheese, milk, eggs, fish, marine mammals	PBDEs	50-70 pg/g (ppt)
	Sample size: A minimum of 25 g extractable fat or 250 g fresh weight is required.	
Solids Biosolids, dust, soil and sediments	PBDEs	100-200 pg/g (ppt)
	Sample size: A minimum of 25 g wet weight is required	

APPENDIX 3: DERIVATION OF FOODS ANALYSED FOR PBDES FROM 22ND ATDS SAMPLING

Table A 2: Food samples analysed for PBDEs from samples collected for the 22nd Australian Total Diet Study by Australian States & Territories

FOOD ANALYSED	22nd ATDS		PBDEs	
	No. States/Territories sampled in 22nd ATDS	Total no. primary samples ('purchases') for all States/Territories in the 22nd ATDS[ℓ]	No primary samples ('purchases') analysed for PBDEs[†]	No analyses (composites) analysed for PBDEs[‡]
Bacon, cooked	3	18	3	1
Beef steak, rib/ribeye/sirloin, grilled	5	30	3	1
Bread, white	5	30	3	1
Butter, regular	3	18	3	1
Carrot cooked	5	30	3	1
Cheese, cheddar, full fat	5	30	3	1
Chicken, breast, fillet, cooked	3	18	3	1
Chocolate, milk	3	18	3	1
Coconut, desiccated	3	18	3	1
Cream, pure (not thickened)	3	18	3	1
Eggs, boiled	5	30	3	1
Fish fillets, cooked	5	30	3	1
Hamburger, cooked	5	30	3	1
Ice Cream, full fat, vanilla	3	18	3	1
Infant Cereal, mixed	3	18	3	1
Infant Dessert, dairy based	3	18	3	1
Infant Dessert, fruit	3	18	3	1
Infant Dinner, meat, chicken or fish	3	18	3	1
Infant Formula, powder, cow's milk based	3	18	3	1
Lamb Chops, loin, grilled	5	30	3	1
Liver, sheep, cooked	5	30	3	1
Margarine/ Spread, Polyunsaturated	3	18	3	1
Milk, full fat	5	30	6	2
Milk, modified, low fat	5	30	6	2
Oil, canola	3	18	3	1
Peanut butter	3	18	3	1
Pizza, meat & vegetable, cooked	3	18	3	1
Pork Chops, grilled	3	18	6	2
Potato crisps	3	18	3	1
Potatoes cooked	5	30	3	1
Salt, table, non-iodised	3	18	3	1
Sausage, beef, cooked	5	30	6	2
Tuna, canned in brine	3	18	3	1
Water, Tap	8	48	3	1
Yoghurt, fruit, full fat	3	18	3	1

[ℓ] In the 22nd ATDS each time a State or Territory collected samples they were required to collect six individual purchases which were prepared into two composite samples comprising three individual purchases in each composite sample.

[†] A single composite sample was randomly selected from the State/Territory composite sample collected for the 22nd ATDS. Each single composite sample analysed for PBDEs comprised 3 primary samples ('purchases').

[‡] Of the two composite samples collected by the States and Territories for the 22nd ATDS the sample(s) analysed for PBDEs were selected at random.

APPENDIX 4: CONSTRUCTION OF THE INFANT DIET – FURTHER DETAILS

Assumptions and limitations of the infant diets

Certain foods consumed by 2 year old children, such as nuts, alcohol and tea, were removed from the infant diet since they are inappropriate for infants (NHMRC, 2003). Fluid milk was also excluded from the infant diet as it was assumed that milk was consumed either in the form of infant formula or breast milk. Consumption of breakfast cereals was assumed to be in the form of either infant cereal or single grain breakfast cereals, excluding bran-based cereals.

As the infant diet estimates a theoretical mean dietary exposure only the 95th percentile dietary exposure to PBDEs was also estimated using the internationally accepted formula shown below (WHO, 1985).

$$95^{th} \text{ percentile exposure} = \text{mean exposure} \times 2.5$$

For 9 month old infants, both models assumed that 50 % of the infant's energy intake comes from breast milk or infant formula. The amount of milk/breast milk consumed in order to meet the energy level was calculated and used for both models.

In 2004, the Australian Government Department of the Environment and Water Resources (formerly the Department of the Environment and Heritage) commissioned a study to determine the levels of PBDEs in the Australian population from breast milk. The mean level of 11 ng/g lipid weight was used in the infant diet to estimate dietary exposure through breast milk (Harden *et al*., 2005). This was converted to a fresh weight concentration of 407 pg/g, based on an average lipid content of breast milk of 3.7% (Harden *et al*., 2005), which was used for the lower, middle and upper bound dietary exposure estimate.

Assigning PBDE concentrations

The food groups and mapping may be slightly different for the infant diet based on the model diets from the 22nd ATDS compared to the exposure estimates for people aged 2 years and above based on DIAMOND and new mapping constructed for the PBDE exposure assessments. However, for the infant diets, mapping followed the same principles and food groups. For example, the concentration for carrots was assigned to all vegetables other than potatoes and fruit was assigned a zero concentration.

APPENDIX 5: PBDE CONGENER CONCENTRATIONS IN FOODS ANALYSED

Table A 3: PBDE concentrations in specific foods (fresh weight pg/g)

	Food												
PBDE Congener	Bacon	Beef steak, grilled	Bread, white	Butter, regular	Canola Oil	Carrots, cooked	Cheese, cheddar, full fat	Chicken Breast	Chocolate, milk	Coconut, desiccated	Cream, pure (not thickened)	Eggs, boiled	Fish fillets
BDE 17	<5	0.45	0.43	<0.6	<2	0.26	<0.3	0.44	<0.2	1.2	0.68	0.25	0.59
BDE 28 + BDE 33	<20	21	<9	<20	<40	15	<10	13	<7	130	58	8.5	<20
LB Σ tri	0	21.4	0.43	0	0	15.26	0	13.44	0	131.2	58.68	8.75	0.59
UB Σ tri	25	21.4	9.43	20.6	42	15.26	10.3	13.44	7.2	131.2	58.68	8.75	20.59
BDE 47	160	65	73	95	<200	22	54	40	<20	110	200	170	79
BDE 49	<1	1.3	1.7	<2	<10	0.41	<0.7	0.8	<0.4	1.3	1.8	1.9	5.9
BDE 66	<1	0.75	1.3	<2	<10	0.18	<3	<0.8	<0.4	<3	<1	<2	1.7
BDE 71	<1	<0.1	<1	<2	<10	<0.04	<1	<0.4	<2	<1	<0.5	<0.8	<0.1
BDE 77	<1	<0.1	<1	<0.6	<10	<0.05	<0.7	<0.4	<0.4	<1	<0.5	<0.4	<0.2
LB Σ tetra	160	67.05	76	95	0	22.59	54	40.8	0	111.3	201.8	171.9	86.6
UB Σ tetra	164	67.07	76	101.6	240	22.68	59.4	42.4	23.2	116.3	203.8	175.1	86.9
BDE 85	5	1.7	2.9	2.1	<10	0.26	<1	1.4	<0.4	1.1	4.2	5.9	<0.5
BDE 99	150	51	65	76	<300	9.4	50	48	<20	<30	160	360	24
BDE 100	30	11	14	<8	<50	2	8.4	10	<4	<7	16	100	16
BDE 119	<3	<0.1	<2	<2	<10	<0.06	<2	<0.4	<0.4	<3	<0.5	<6	<0.5
BDE 126	<3	<0.4	<2	<2	<10	<0.06	<1	<0.4	<0.4	<3	<0.5	<2	<0.5
LB Σ penta	185	63.7	81.9	78.1	0	11.66	58.4	59.4	0	1.1	180.2	465.9	40
UB Σ penta	191	64.2	85.9	90.1	380	11.78	62.4	60.2	25.2	44.1	181.2	473.9	41.5
BDE 138 + BDE 166	<2	<1	<1	<0.8	<10	<0.06	<0.7	0.64	<0.4	<3	<1	<20	<0.4
BDE 153	22	5.4	7.5	10	<20	0.75	6	8.4	<2	<3	17	99	4.3
BDE 154	12	3.6	4.5	5.1	<50	0.64	2.9	5.6	<1	<3	10	63	9.3
BDE 156	<1	<0.1	<1	<0.7	<10	<0.04	<1	<0.4	<0.4	<3	<0.5	<0.7	<0.2
LB Σ hexa	34	9	12	15.1	0	1.39	8.9	14.64	0	0	27	162	13.6
UB Σ hexa	37	10.1	14	16.6	90	1.49	10.6	15.04	3.8	12	28.5	182.7	14.2
BDE 183	7	2	2	0.87	<10	0.11	<2	6	0.82	<3	1.3	4.2	<1
BDE 184	<5	<0.5	<2	<2	<10	<0.1	<2	<0.4	<0.4	<3	<0.5	<1	<1
BDE 191	<5	<0.5	<2	<2	<10	<0.1	<2	<0.4	<0.4	<3	<0.5	<1	<1
LB Σ hepta	7	2	2	0.87	0	0.11	0	6	0.82	0	1.3	4.2	0

PBDE Congener	Food												
	Bacon	Beef steak, grilled	Bread, white	Butter, regular	Canola Oil	Carrots, cooked	Cheese, cheddar, full fat	Chicken Breast	Chocolate, milk	Coconut, desiccated	Cream, pure (not thickened)	Eggs, boiled	Fish fillets
UB Σ hepta	17	3	6	4.87	30	0.31	6	6.8	1.62	9	2.3	6.2	3
BDE 196	<1	<0.5	<5	<20	<10	<1	<10	3	<4	<3	<2	<6	<5
BDE 197	3.3	0.63	<5	<20	<10	<1	<20	4.4	<4	<3	<2	<5	<5
LB Σ octa	3.3	0.63	0	0	0	0	0	7.4	0	0	0	0	0
UB Σ octa	4.3	1.13	10	40	20	2	30	7.4	8	6	4	11	10
BDE 206	<5	<1	1.5	<6	<10	0.19	<10	7.6	9.3	<3	<2	4	1.6
BDE 207	15	<2	1.6	<8	<10	0.46	<7	16	8.9	<3	<2	8.9	2.2
LB Σ nona	15	0	3.1	0	0	0.65	0	23.6	18.2	0	0	12.9	3.8
UB Σ nona	20	3	3.1	14	20	0.65	17	23.6	18.2	6	4	12.9	3.8
BDE 209	110	26	19	<70	<200	5.9	<70	120	290	<40	<30	84	28
LB Σ deca	110	26	19	0	0	5.9	0	120	290	0	0	84	28
UB Σ deca	110	26	19	70	200	5.9	70	120	290	40	30	84	28
LB Σ PBDE	514.3	189.83	194.43	189.07	0	57.56	121.3	285.28	309.02	243.6	468.98	909.65	172.59
UB Σ PBDE	568.3	196.13	225.43	357.77	1022	60.07	265.7	288.88	377.22	364.6	512.48	954.55	207.99

Table A 3: PBDE concentrations in specific foods (fresh weight pg/g)

PBDE Congener	Food												
	Hamburger	Ice Cream, full fat, vanilla	Infant Cereal, mixed	Infant Dessert, dairy based	Infant Dessert, fruit	Infant Dinner	Infant Formula, prepared	Lamb Chops, loin, grilled	Margarine	Milk, full fat	Milk, full fat (duplicate)	Milk, modified, low fat	Milk, modified, low fat (duplicate)
BDE 17	0.43	0.12	0.21	0.22	0.35	0.64	0.17	2.3	<1	<0.06	<0.1	<0.08	<0.08
BDE 28 + BDE 33	18	<10	15	7.8	18	27	15	100	<20	<6	<6	<8	<7
LB Σ tri	18.43	0.12	15.21	8.02	18.35	27.64	15.17	102.3	0	0	0	0	0
UB Σ tri	18.43	10.12	15.21	8.02	18.35	27.64	15.17	102.3	21	6.06	6.1	8.08	7.08
BDE 47	100	49	29	25	33	34	41	160	74	<20	<30	<20	<20
BDE 49	0.8	0.4	0.44	0.33	0.57	0.85	0.54	3.9	<1	<0.1	<0.3	<0.2	<0.2
BDE 66	<0.6	0.41	0.27	0.23	0.37	0.46	0.48	1.4	<1	<0.6	<0.3	<0.2	<0.2
BDE 71	<0.4	<0.3	<0.07	<0.05	<0.06	<0.2	<0.2	0.52	<1	<0.3	<0.2	<0.08	<0.08
BDE 77	<0.4	<0.2	<0.05	<0.05	<0.03	<0.2	<0.03	<0.08	<0.7	<0.06	<0.06	<0.02	<0.02
LB Σ tetra	100.8	49.81	29.71	25.56	33.94	35.31	42.02	165.8	74	0	0	0	0
UB Σ tetra	102.2	50.31	29.83	25.66	34.03	35.71	42.25	165.9	77.7	21.06	30.86	20.5	20.5
BDE 85	1.5	1	0.86	0.55	<0.2	0.42	0.51	<4	1.8	<1	<0.6	<0.4	<0.4
BDE 99	110	39	19	19	<10	16	22	41	68	<30	<20	<20	<20
BDE 100	11	6.9	3.9	3.4	<2	3.5	4.1	14	11	<3	<3	<2	<2
BDE 119	<4	<0.1	<0.3	<0.2	<0.2	<0.2	<0.1	<0.3	<2	<0.03	<0.03	<0.08	<0.08
BDE 126	<1	<0.5	<0.1	<0.2	<0.1	<0.2	<0.07	<0.4	<1	<0.03	<0.03	<0.08	<0.08
LB Σ penta	122.5	46.9	23.76	22.95	0	19.92	26.61	55	80.8	0	0	0	0
UB Σ penta	127.5	47.5	24.16	23.35	12.5	20.32	26.78	59.7	83.8	34.06	23.66	22.56	22.56
BDE 138 + BDE 166	<0.6	<0.2	0.19	<0.5	<0.1	0.16	<0.1	0.45	<2	<0.3	<0.3	<0.08	<0.08
BDE 153	21	4.6	1.7	1.5	<0.6	2.4	1.5	6.1	5.2	<3	<2	<0.8	<0.8
BDE 154	9.7	2.6	1.4	1.3	<0.4	1.4	1.2	2.7	4.1	<2	<1	<0.8	<0.8
BDE 156	<0.1	<0.5	<0.02	<0.1	<0.1	<0.2	<0.03	<0.1	<0.7	<0.3	<0.06	<0.08	<0.08
LB Σ hexa	30.7	7.2	3.1	2.8	0	3.96	2.7	9.25	9.3	0	0	0	0
UB Σ hexa	31.4	7.9	3.12	3.4	1.2	4.16	2.83	9.35	12	5.6	3.36	1.76	1.76
BDE 183	2.1	<0.5	0.75	<0.2	<0.2	<0.3	0.44	3.5	<4	<0.3	<0.3	<0.2	<0.3
BDE 184	<1	<0.5	<0.2	<0.1	<0.1	<0.2	<0.2	<0.7	<4	<0.2	<0.2	<0.08	<0.08
BDE 191	<0.8	<0.5	<0.2	<0.1	<0.1	<0.2	<0.2	<0.7	<4	<0.2	<0.2	<0.08	<0.08
LB Σ hepta	2.1	0	0.75	0	0	0	0.44	3.5	0	0	0	0	0
UB Σ hepta	3.9	1.5	1.15	0.4	0.4	0.7	0.84	4.9	12	0.7	0.7	0.36	0.46
BDE 196	<0.6	<3	0.35	<0.1	<0.4	<0.6	<0.7	0.66	<30	<0.3	<0.3	<0.2	<0.3

PBDE Congener	Food												
	Hamburger	Ice Cream, full fat, vanilla	Infant Cereal, mixed	Infant Dessert, dairy based	Infant Dessert, fruit	Infant Dinner	Infant Formula, prepared	Lamb Chops, loin, grilled	Margarine	Milk, full fat	Milk, full fat (duplicate)	Milk, modified, low fat	Milk, modified, low fat (duplicate)
BDE 197	<1	<4	0.56	<0.2	<0.3	<0.6	<0.7	1.4	<30	<0.3	<0.3	<0.2	<0.4
LB Σ octa	0	0	0.91	0	0	0	0	2.06	0	0	0	0	0
UB Σ octa	1.6	7	0.91	0.3	0.7	1.2	1.4	2.06	60	0.6	0.6	0.4	0.7
BDE 206	4.6	<1	0.55	<0.5	<0.3	<1	0.71	<1	<7	<0.6	<0.6	<0.3	<0.5
BDE 207	5.7	<0.5	1.7	0.65	<0.3	<1	0.65	<3	<10	<0.6	<0.6	<0.5	<0.6
LB Σ nona	10.3	0	2.25	0.65	0	0	1.36	0	0	0	0	0	0
UB Σ nona	10.3	1.5	2.25	1.15	0.6	2	1.36	4	17	1.2	1.2	0.8	1.1
BDE 209	77	<8	15	13	<6	<10	<10	23	<100	<6	<6	<5	<6
LB Σ deca	77	0	15	13	0	0	0	23	0	0	0	0	0
UB Σ deca	77	8	15	13	6	10	10	23	100	6	6	5	6
LB Σ PBDE	361.83	104.03	90.88	72.98	52.29	86.83	88.3	360.93	164.1	0	0	0	0
UB Σ PBDE	372.33	133.83	91.82	75.28	73.78	101.73	100.63	371.21	383.5	75.28	72.48	59.46	60.16

Table A 3: PBDE concentrations in specific foods (fresh weight pg/g)

PBDE Congener	Peanut butter	Pizza	Pork Chops, grilled	Pork Chops, grilled (duplicate)	Potato crisps	Potato, cooked	Salt, table, non-iodised	Sausage, beef	Sausage, beef (duplicate)	Sheep liver	Tuna, canned in brine	Water, tap**	Yoghurt, fruit, full fat
BDE 17	0.8	0.37	0.47	0.31	2	<0.09	<0.1	0.47	0.35	1.5	0.11	<20	<0.07
BDE 28 + BDE 33	38	<6	<10	<10	100	<6	<10	<10	<10	67	<4	<300	<7
LB Σ tri	38.8	0.37	0.47	0.31	102	0	0	0.47	0.35	68.5	0.11	0	0
UB Σ tri	38.8	6.37	10.5	10.31	102	6.09	10.1	10.47	10.35	68.5	4.11	320	7.07
BDE 47	85	110	230	220	140	<10	<20	98	90	110	15	<700	21
BDE 49	1.6	0.91	<0.8	<0.7	2.6	0.19	<0.1	1.4	1.2	2.9	1.5	<20	0.19
BDE 66	<2	0.68	<1	<1	1.1	<0.1	<0.1	1.3	1.4	<5	0.61	<20	0.14
BDE 71	<0.5	<0.5	<0.9	<0.9	<1	<0.1	<0.1	<1	<1	<3	<0.03	<20	<0.07
BDE 77	<0.5	<0.5	<0.3	<0.3	<0.4	<0.1	<0.1	<1	<1	<3	<0.02	<20	<0.04
LB Σ tetra	86.6	111.59	230	220	143.7	0.19	0	100.7	92.6	112.9	17.11	0	21.33
UB Σ tetra	89.6	112.59	233	222.9	145.1	10.49	20.4	102.7	94.6	123.9	17.16	780	21.44
BDE 85	<1	4.9	10	9.5	<2	0.3	<0.6	2.2	1.8	<3	<0.1	<60	0.49
BDE 99	<30	140	280	260	<40	<9	<20	88	80	43	<6	<500	16
BDE 100	<5	15	54	36	<4	<2	<3	11	8.6	<6	1.9	<100	<2
BDE 119	<1	<1	<1	<5	<4	<0.5	<0.1	<1	<1	<6	0.22	<50	<0.1
BDE 126	<0.5	<1	<0.9	<1	<1	<0.5	<0.1	<1	<1	<6	<0.1	<30	<0.07
LB Σ penta	0	159.9	344	305.5	0	0.3	0	101.2	90.4	43	2.12	0	16.49
UB Σ penta	37.5	162.9	345.9	311.5	51	12.3	23.8	103.2	92.4	64	8.32	740	18.66
BDE 138 + BDE 166	<1	1.9	5.2	4.7	<2	0.12	<0.2	0.49	0.49	<3	<0.1	<20	<0.1
BDE 153	<2	24	48	50	5.3	<1	<1	9	8.6	4.4	1.8	<40	1.6
BDE 154	<2	13	27	26	<2	<1	<1	5.7	5.5	2.9	2.3	<20	1.1
BDE 156	<0.5	<1	<0.1	<0.4	<2	<0.5	<0.1	<1	<1	<3	<0.1	<30	<0.04
LB Σ hexa	0	38.9	80.2	80.7	5.3	0.12	0	15.19	14.59	7.3	4.1	0	2.7
UB Σ hexa	5.5	39.9	80.3	81.1	11.3	2.62	2.3	16.19	15.49	13.3	4.3	110	2.84
BDE 183	<2	4.9	25	26	3.4	0.53	<0.1	1.2	1.3	<3	1.1	<50	<0.2
BDE 184	<2	<1	<0.3	<0.5	<2	<0.5	<0.1	<1	<1	<3	<0.2	<70	<0.2
BDE 191	<2	<1	<0.5	<1	<4	<0.5	<0.1	<1	<1	<3	<0.1	<50	<0.2
LB Σ hepta	0	4.9	25	26	3.4	0.53	0	1.2	1.3	0	1.1	0	0
UB Σ hepta	6	6.9	25.8	27.5	9.4	1.53	0.3	3.2	3.3	9	1.4	170	0.6
BDE 196	<5	<1	2.5	2.7	<2	0.11	<0.6	<2	<2	<8	<0.7	<60	<0.4
BDE 197	<10	<2	7.6	8.3	<2	0.12	<0.5	<2	<2	<10	<0.8	<50	<0.7

PBDE Congener	Peanut butter	Pizza	Pork Chops, grilled	Pork Chops, grilled (duplicate)	Potato crisps	Potato, cooked	Salt, table, non-iodised	Sausage, beef	Sausage, beef (duplicate)	Sheep liver	Tuna, canned in brine	Water, tap**	Yoghurt, fruit, full fat
LB Σ octa	0	0	10.1	11	0	0.23	0	0	0	0	0	0	0
UB Σ octa	15	3	10.1	11	4	0.23	1.1	4	4	18	1.5	110	1.1
BDE 206	<5	<10	<1	1.5	<2	0.3	<1	10	11	<6	<0.2	<20	1.1
BDE 207	<5	8.6	3.6	4.6	<4	<0.5	<1	14	13	<6	<0.2	<70	<1
LB Σ nona	0	8.6	3.6	6.1	0	0.3	0	24	24	0	0	0	1.1
UB Σ nona	10	18.6	4.6	6.1	6	0.8	2	24	24	12	0.4	90	2.1
BDE 209	<30	<60	<10	32	46	<5	<8	110	120	<50	<2	<800	42
LB Σ deca	0	0	0	32	46	0	0	110	120	0	0	0	42
UB Σ deca	30	60	10	32	46	5	8	110	120	50	2	800	42
LB Σ PBDE	125.4	324.26	693.37	681.61	300.4	1.67	0	352.76	343.24	231.7	24.54	0	83.62
UB Σ PBDE	232.4	409.26	720.17	702.41	374.8	39.06	68	373.76	364.24	358.7	39.19	3120	95.81

**Water was measured in pg/L
LB Σ = Lower bound sum
UB Σ = Upper bound sum

Table A 4: Lower and Upper bound sums of all congeners across all foods analysed for PBDEs

Bound	Congener	Total PBDE congeners by classification across foods analysed (fw pg/g)
Lower	Tri	666.37
Upper	Tri	1252.08
Lower	Tetra	2780.71
Upper	Tetra	4008.84
Lower	Penta	2686.71
Upper	Penta	4211.41
Lower	Hexa	615.74
Upper	Hexa	909.01
Lower	Hepta	94.52
Upper	Hepta	389.04
Lower	Nona	159.51
Upper	Nona	391.31
Lower	Octa	35.63
Upper	Octa	414.33
Lower	Deca	1160.90
Upper	Deca	2692.90
Total Lower	**BDE all foods**	**8200.09**
Total Upper	**BDE all foods**	**14268.92**

APPENDIX 6: FOODS ANALYSED AND CORRESPONDING NATIONAL NUTRITION SURVEY FOODS

Table A 5: Mapping of foods analysed

Food Category	Foods analysed	NNS foods represented
Dairy products	Milk, full fat	All fluid milks whole
		Full fat flavoured milks
	Milk, modified, low fat	All fluid milk, reduced or low fat
		Reduced or low fat flavoured milks
	Cheese, cheddar, full fat	Ripened cheeses
		Unripened cheeses
		Processed cheeses
		Whey cheese
	Cream, pure (not thickened)	All cream (whipped, thickened , sour)
	Yoghurt, fruit, full fat	Yoghurt (plain, flavoured, frozen, full fat, skim, fromage frais)
Ice cream	Ice Cream, full fat, vanilla	Ice cream (regular, skim, flavoured, tub or stick)
		Ice confection
		Thickshakes
Edible oils and oil emulsions	Butter, regular	Butter (regular, flavoured, reduced salt, salt free)
	Margarine or Margarine Spread, Polyunsaturated	Margarine polyunsaturated
		Margarine monounsaturated
		Margarine spreads
		Commercial fats
		Not specified fats
	Oil, canola	All oils (vegetable, seed, nut) including single source or blended
Vegetables	Carrots, cooked	All vegetables (raw, cooked , canned, juices) except potatoes
	Potatoes, cooked	Potato - boiled, baked, canned, mashed
		Potato - battered, crumbed, pattied
		Salad potato
		Sweet potato
	Coconut, desiccated	All coconut (dry, fresh, milk, cream, canned)
Chocolate	Chocolate, milk	Chocolate, milk (bars, filled, coated)
		Chocolate, carbohydrate-modified
Breads and Bakery Products	Bread, white	All "regular breads and rolls", including wholemeal or whole grain breads and products
		English style muffins, "crumpets", "flat breads", "sweet breads and buns", 'tortilla and corn bread".
		Doughnuts
Meat and Meat Products	Bacon	All "bacon"
		Ham
		All salamis, cabanossi

Food Category	Foods analysed	NNS foods represented
Meat and Meat Products	Beef steak, rib/ribeye/sirloin, grilled	All "beef steak", "beef brisket", "beef silverside", "beef patty (meat only)", "minced meats"
		All beef "corned", "smoked", "deli sliced", "cooked"
		All "veal", "kangaroo", "rabbit", "venison"
	Lamb Chops, loin, grilled	Lamb or mutton "all chops", "minced", "smoked", "deli sliced"
	Pork Chops, grilled	Pork "all chops", "minced", "smoked", "deli sliced"
	Chicken, breast, fillet	All chicken "raw", "cooked", "smoked", "deli sliced"
		All "duck", "quail", "emu" and "turkey"
	Liver, sheep	All liver and internal organs
	Sausage, beef	All "sausages" and sausage patties
		All plain "frankfurts, and saveloys"
Fish and Fish products	Fish fillets	All fish (cooked, uncooked, smoked)
		All crustacea and molluscs (cooked, uncooked, smoked)
	Tuna, canned in brine	Canned Tuna or sardine in brine/ water/ oil
Eggs and Egg products	Eggs, boiled	All eggs cooked or uncooked, scrambled omelettes
Salt	Salt, table, non-iodised	Salt
Foods for infants	Infant Cereal, mixed	Infant Cereal, mixed
	Infant Dessert, dairy based	Infant Dessert, dairy based
	Infant Dessert, fruit	Infant Dessert, fruit
	Infant Dinner, containing meat, chicken or fish	Infant Dinner, containing meat, chicken or fish
	Infant Formula, powder, cow's milk based	Infant Formula, powder, cow's milk based
Water	Water, Tap	Water, tap and bottled
		Mineral water
		Soda water
		Fruit drinks
		Soft drinks
		Cordials
Mixed Foods and snacks	Pizza, meat & vegetable-containing	All "pizza" and pies (vegetable, seafood, meat)
	Hamburger	All hamburger and meat patties
	Potato crisps	Potato (plain, flavoured, restructured), corn chips pretzels, bhuja/snack mixes
	Peanut butter	All nuts

Note: Other mixed foods contain foods analysed and listed above (e.g. crumbed fish contains fish fillets). The proportion of the ingredients in these mixed foods, as determined by standard recipes in DIAMOND, were given the concentration of PBDE assigned to that food.

APPENDIX 7: SUMMARY DATA FROM THE DIETARY EXPOSURE ASSESSMENTS INCLUDING FOOD CONSUMPTION DATA, RESPONDENT AND CONSUMER NUMBERS AND BODY WEIGHTS

Table A 6: Mean consumption for consumers only of each food analysed for each age-gender group in grams per day, derived from the 1995 NNS and DIAMOND

Food	Male 2-5 years	Female 2-5 years	Male 6-12 years	Female 6-12 years	Male 13-18 years	Female 13-18 years	Male 19+ years	Female 19+ years
Bacon	36	29	39	33	55	33	53	37
Beef	63	56	92	87	145	108	151	98
Bread	86	78	118	101	147	112	144	102
Butter	6	6	10	8	15	9	18	12
Carrots, cooked	81	80	122	124	176	151	232	204
Cheese, cheddar, full fat	27	27	36	28	46	35	41	32
Chicken	66	61	91	84	140	102	134	97
Chocolate	24	22	32	33	53	44	46	40
Coconut, desiccated	7	6	11	7	28	5	31	21
Cream	9	11	16	14	23	27	30	24
Eggs, boiled	12	14	20	17	26	20	30	22
Fish fillets	59	39	82	60	135	84	122	98
Hamburger	97	95	158	126	194	157	209	159
Ice Cream	82	71	141	121	207	125	123	85
Infant Cereal, mixed	NC	NC	NC	NC	NC	NC	8	100
Infant Dessert, dairy based	66	100	NC	68	NC	NC	58	58
Infant Dessert, fruit	34	92	20	138	NC	NC	115	13
Infant Dinner	110	NC	NC	NC	NC	NC	NC	33
Infant Formula, powder	103	NC	NC	NC	NC	310	NC	152
Lamb Chops, loin, grilled	59	63	81	84	117	84	133	87
Sheep Liver	4	6	29	57	102	67	64	39
Margarine/ Margarine Spread, Poly-unsaturated	13	11	19	16	25	17	24	16
Milk, full fat	402	342	343	261	417	242	238	169
Milk, modified, low fat	310	300	373	291	422	323	276	216
Canola Oil	3	3	6	5	9	7	10	7
Peanut butter	7	7	10	10	12	13	18	13
Pizza	114	99	180	143	224	152	244	169
Pork	49	47	65	83	130	73	116	83
Potato crisps	34	31	33	34	50	38	49	38
Potato, cooked	122	106	173	168	258	176	196	145
Salt, table, non-iodised	1	<1	2	<1	2	2	1	1
Sausage	63	59	86	76	106	79	118	87
Tuna, canned in brine	55	30	73	81	241	87	91	71
Water, tap	915	800	1160	1073	1744	1352	1308	1137
Yoghurt	111	107	123	138	197	161	144	140

NC = Not consumed

Table A 7: Mean body weights in kilograms for each age-gender category assessed

Age group	Source	Mean Body Weight (kg)	
		Males	Females
3 months	Female bodyweight WHO (1983)	6.4	
9 months	Male bodyweight WHO (1983)	9.2	
2-5 years	1995 NNS	18	17
6-12 years	1995 NNS	33	35
13-18 years	1995 NNS	65	59
19 years and above	1995 NNS	82	68

Note: For populations aged 2 years and above, individual body weight was used in the calculations

Table A 8: Number of respondents and consumers of PBDEs per age gender group assessed from the 1995 NNS

Age group	Number of respondents		Number of consumers of PBDEs (% of all respondents)		
	Males	Females	Scenario	Males	Females
2-5 years	380	413	LB	380 (100)	413 (100)
			MB	380 (100)	413 (100)
			UB	380 (100)	413 (100)
6-12 years	664	622	LB	663 (99.8)	621 (99.8)
			MB	664 (100)	622 (100)
			UB	664 (100)	622 (100)
13-18 years	491	437	LB	491 (100)	436 (99.8)
			MB	491 (100)	437 (100)
			UB	491 (100)	437 (100)
19 years and above	5081	5770	LB	5076 (99.9)	5749 (99.6)
			MB	5080 (99.98)	5770 (100)
			UB	5080 (99.98)	5770 (100)

Note to Table A 8: In some cases there are fewer consumers of PBDEs for the lower bound scenario. This is because of the way DIAMOND counts consumers. Even if a respondent consumed a food that was analysed for PBDEs, if the lower bound concentration assigned to that food was a zero, a respondent does not get counted as a consumer of PBDEs for that food.

APPENDIX 8: PERCENT CONTRIBUTION OF FOODS TO PBDE DIETARY EXPOSURE

Table A 9: Contribution of each food for each age/gender group assessed (excluding infants) using the Lower bound concentration.

Food	Male 2-5 years	Female 2-5 years	Male 6-12 years	Female 6-12 years	Male 13-18 years	Female 13-18 years	Male 19+ years	Female 19+ years
Bacon	7	6	5	4	6	3	7	5
Beef	5	4	5	4	6	6	8	6
Bread	24	23	21	20	18	19	18	19
Butter	<1	<1	<1	<1	<1	<1	<1	<1
Vegetables (Carrots)	5	6	5	7	6	7	9	12
Cheese	2	2	2	2	2	2	2	2
Chicken	6	6	6	6	8	9	8	9
Chocolate	3	3	3	4	3	4	1	2
Coconut, desiccated	<1	<1	<1	<1	<1	<1	<1	<1
Cream	2	3	2	3	2	3	3	3
Eggs, boiled	11	14	13	12	11	10	11	13
Fish fillets	1	1	1	1	1	1	2	2
Hamburger	3	2	3	3	5	4	3	2
Ice Cream	4	3	5	5	5	4	2	1
Infant Cereal, mixed	NC	NC	NC	NC	NC	NC	<1	<1
Infant Dessert, dairy based	<1	<1	NC	<1	NC	NC	<1	<1
Infant Dessert, fruit	<1	<1	<1	<1	NC	NC	<1	<1
Infant Dinner	<1	NC	NC	NC	NC	NC	NC	<1
Infant Formula, powder	<1	NC	NC	NC	NC	0	NC	<1
Lamb Chops	3	2	2	3	2	3	4	4
Sheep Liver	<1	<1	<1	<1	<1	<1	<1	<1
Margarine/ Margarine Spread	3	3	3	3	2	2	2	2
Milk, full fat	0	0	0	0	0	0	0	0
Milk, modified, low fat	0	0	0	0	0	0	0	0
Canola Oil	0	0	0	0	0	0	0	0
Peanut butter	1	1	1	1	<1	1	1	1
Pizza	5	6	9	7	10	7	7	5
Pork	2	2	3	4	4	4	5	4
Potato crisps	4	3	3	4	3	3	1	1
Potato	<1	<1	<1	<1	<1	<1	<1	<1
Salt, table, non-iodised	0	0	0	0	0	0	0	0
Sausage	6	7	6	6	6	4	4	3
Tuna, canned in brine	<1	<1	<1	<1	<1	<1	<1	<1
Water, tap	0	0	0	0	0	0	0	0
Yoghurt	4	3	1	2	1	2	1	2

NC = this food had a PBDE concentration however it was not consumed and therefore did not make a contribution to PBDE dietary exposure.

0 = this food was consumed however there was a lower bound concentration of zero for this food and therefore did not make a contribution to PBDE dietary exposure.

APPENDIX 9: INFANT DIET ESTIMATED DIETARY EXPOSURES AND PERCENT CONTRIBUTION OF FOODS

Table A 10: Infant diet calculations including infant formula and breast milk

Food	PBDE concentration (pg/g)			Consumption (g/day)	Estimated dietary exposure (pg/day)			Percent contribution	
	Lower Bound	Middle Bound	Upper Bound		Lower Bound	Middle Bound	Upper Bound	Formula fed	Breast milk fed
Bacon	514.3	541.3	568.3	2.0	1022	1076	1130	2	<1
Beef	189.8	193	196.1	1.2	220	224	228	<1	<1
Bread	194.4	209.9	225.4	20.1	3905	4217	4528	6	2
Breast milk[1]	407	407	407	562.4	228897	228897	228897	NC	93
Butter	189.1	273.4	357.8	0.4	73	106	139	<1	<1
Carrots, cooked	57.6	58.8	60.1	20.5	1182	1206	1233	2	<1
Cheese, cheddar, full fat	121.3	193.5	265.7	3.9	473	755	1036	1	<1
Chicken	285.3	287.1	288.9	3.5	984	991	997	1	<1
Chocolate	309	343.1	377.2	2.1	656	728	800	1	<1
Coconut, desiccated	243.6	304.1	364.6	0.5	131	163	195	<1	<1
Cream	469	490.7	512.48	0.9	442	462	483	1	<1
Eggs, boiled	909.7	932.1	954.6	2.4	2147	2200	2253	3	1
Fish fillets	172.6	190.3	208.0	1.0	166	183	201	<1	<1
Hamburger	361.8	367.1	372.3	0.0	12	12	12	<1	<1
Ice Cream	104	118.9	133.8	5.8	599	685	771	1	<1
Infant Cereal, mixed	90.9	91.4	91.8	7.5	685	689	692	1	<1
Infant Dessert, dairy based	73	74.1	75.3	2.3	169	172	175	<1	<1
Infant Dessert, fruit	52.3	63	73.8	2.6	138	167	195	<1	<1
Infant Dinner	86.8	94.3	101.7	1.9	166	180	194	<1	<1
Infant Formula	88.3	94.5	100.6	562.4	49660	53147	56577	74	NC
Lamb Chops, loin, grilled	360.9	366.1	371.2	0.7	242	245	249	<1	<1
Sheep Liver	231.7	295.2	358.7	0.0	4	5	7	<1	<1
Margarine/ Margarine Spread, Poly-unsat.	164.1	273.8	383.5	1.5	243	406	568	<1	<1
Milk, full fat	0	36.9	73.9	0.0	0	0	0	NC	NC
Milk, modified, low fat	0	29.9	59.8	0.0	0	0	0	NC	NC
Canola Oil	0.0	511.0	1022.0	0.3	0	169	339	0	0
Peanut butter	125.4	178.9	232.4	0.0	0	0	0	NC	NC
Pizza	324.3	366.8	409.3	3.8	1218	1378	1537	2	<1
Pork	687.5	699.4	711.3	0.4	297	302	307	0	<1
Potato crisps	300.4	337.6	374.8	2.3	703	790	877	1	<1
Potato, cooked	1.7	20.4	39.1	12.8	22	260	499	<1	<1
Salt, table, non-iodised	0	34	68	0.0	0	0	0	NC	NC
Sausage	348	358.5	369	2.5	868	894	920	1	<1
Tuna, canned in brine	24.5	31.9	39.2	0.3	9	11	14	<1	<1
Water, tap	0	1560	3120	16.6	0	25938	51876	0	0
Yoghurt	83.6	89.7	95.8	10.4	872	936	999	1	<1
Total breast fed (pg/day)					**246545**	**274446**	**302348**		
Total breast fed (ng/day)					**247**	**274**	**302**		
Total formula fed (pg/day)					**67308**	**98696**	**130029**		
Total formula fed (ng/day)					**67**	**99**	**130**		

NC = this food had a PBDE concentration however it was not consumed and therefore did not make a contribution to PBDE dietary exposure.

0 = this food was consumed however there was a lower bound concentration of zero for this food and therefore did not make a contribution to PBDE dietary exposure.

[1] PBDE concentration data for breast milk was obtained from a separate study described in Harden *et al.* (2005).