

TOX/2014/27

## **COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT**

### **First draft statement on the potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet**

#### **Introduction**

1. The Committee on Toxicity (COT) has been asked to consider aspects related to the toxicity of chemicals in the infant diet, in support of a review by the Scientific Advisory Committee on Nutrition (SACN) of Government recommendations on complementary and young child feeding. Members concluded that brominated flame retardants (BFRs) should be considered as part of that body of work. The polybrominated diphenyl ethers (PBDEs) are a group of BFRs comprising 209 structurally-related congeners. A scoping paper (TOX/2014/19) was presented to Members in May 2014.

2. Annex A contains a first draft COT statement summarising the available information, taking into account the discussion of the scoping paper presented in May 2014 and a subsequent literature search.

#### **Questions on which the views of the Committee are sought**

3. Members are invited to comment on the structure and content of the first draft statement, and to advise on conclusions to be incorporated into a further draft.

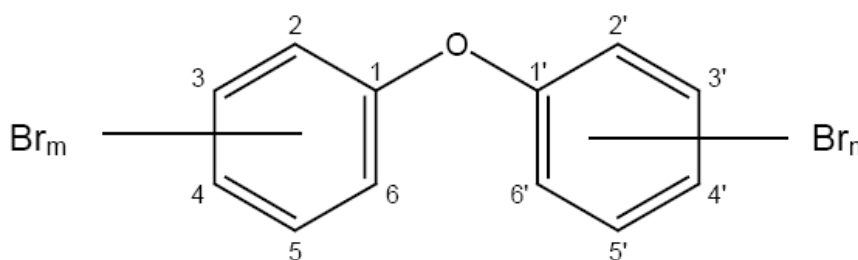
**Secretariat**  
**August 2014**

## COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

### First draft statement on the potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet

#### Background

1. The Scientific Advisory Committee on Nutrition (SACN) is undertaking a review of scientific evidence that bears on the Government's dietary recommendations for infants and young children. The review will identify new evidence that has emerged since the Government's current recommendations were formulated, and will appraise that evidence to determine whether the advice should be revised. The recommendations cover diet from birth to age five years, but will be considered in two stages, focussing first on infants aged 0 – 12 months, and then on advice for children aged 1 to 5 years. SACN is examining the nutritional basis of the advice, and has asked that evidence on possible adverse effects of diet should be considered by other advisory committees with relevant expertise. SACN asked COT to review the risks of toxicity from chemicals in the infant diet.
2. This statement gives an overview of the potential risks from polybrominated diphenyl ethers (PBDEs) in the infant diet. PBDEs are brominated flame retardants (BFRs), which are used in the manufacture of a range of products to increase their fire-related safety. None of Government's current dietary recommendations for infants and young children relates to PBDEs.
3. PBDEs comprise two phenyl rings linked by an ether group and bromine atoms substituting different combinations of ring hydrogens. Their generic structure is represented within Figure 1. The ten homologues and their 209 isomeric congeners within the PBDE category are listed in Table 1.



Where (m) plus (n) equal between 1 and 10 bromine atoms

Figure 1: Generic structure of the PBDEs

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table 1: PBDE homologues and congeners (from EFSA, 2011)

Homologues	Chemical formula	Molecular mass	Isomeric congeners	Number of congeners
monoBDEs	C <sub>12</sub> H <sub>9</sub> BrO	249.1	BDE-1 to BDE-3	3
diBDEs	C <sub>12</sub> H <sub>8</sub> Br <sub>2</sub> O	328.0	BDE-4 to BDE-15	12
triBDEs	C <sub>12</sub> H <sub>7</sub> Br <sub>3</sub> O	406.9	BDE-16 to BDE-39	24
tetraBDEs	C <sub>12</sub> H <sub>6</sub> Br <sub>4</sub> O	485.8	BDE-40 to BDE-81	42
pentaBDEs	C <sub>12</sub> H <sub>5</sub> Br <sub>5</sub> O	564.7	BDE-82 to BDE-127	46
hexaBDEs	C <sub>12</sub> H <sub>4</sub> Br <sub>6</sub> O	643.6	BDE-128 to BDE-169	42
heptaBDEs	C <sub>12</sub> H <sub>3</sub> Br <sub>7</sub> O	722.5	BDE-170 to BDE-193	24
octaBDEs	C <sub>12</sub> H <sub>2</sub> Br <sub>8</sub> O	801.4	BDE-194 to BDE-205	12
nonaBDEs	C <sub>12</sub> HBr <sub>9</sub> O	880.3	BDE-206 to BDE-208	3
decaBDE	C <sub>12</sub> Br <sub>10</sub> O	959.2	BDE-209	1

4. Technical mixtures of PBDEs have been widely used as additive flame retardants in polymers and textiles, construction materials, furniture, and electrical equipment. Because PBDEs are not chemically bound to the polymers they can leach into the environment and enter the food chain. Table 2 lists the 8 congeners that were present in the largest amounts in commercial technical mixtures of pentaBDE, octaBDE and decaBDE (EFSA, 2011). International agreements on bans and regulations for production and use of technical mixtures of PBDEs have been introduced since 2004, leading to declining levels in the environment (EFSA, 2011). However, there are still some uses of commercial decaBDE.

Table 2: Predominant congeners in commercial technical mixtures of PBDEs (EFSA, 2011)

Congener	Bromine substitution	CAS number
BDE-28	2,2',4-triBDE	41318-75-6
BDE-47	2,2',4,4'-tetraBDE	5436-43-1
BDE-99	2,2',4,4',5-pentaBDE	60348-60-9
BDE-100	2,2',4,4',6-pentaBDE	189084-64-8
BDE-153	2,2',4,4',5,5'-hexaBDE	68631-49-2
BDE-154	2,2',4,4',5,6'-hexaBDE	207122-15-4
BDE-183	2,2',3,4,4',5',6-heptaBDE	207122-16-5
BDE-209	2,2',3,3',4,4',5,5',6,6'-decaBDE	1163-19-5

5. PBDE congeners are susceptible to photolysis, reductive debromination and radical reactions in the environment. The chemical stability of the PBDE congeners varies with their individual structure but in general congeners with up to three bromine substituents and those with nine and ten bromine substituents are more susceptible to abiotic transformation.

6. In 2004, the COT published a statement on PBDE residues in fish from two rivers in England<sup>1</sup>. Subsequently in 2006, the COT published a risk assessment for PBDE residues in a broader range of fish and shellfish<sup>2</sup>. The EFSA Panel on Contaminants in the Food Chain issued a comprehensive opinion on PBDEs in 2011 (EFSA, 2011). This statement draws on information from the EFSA review and more recent literature. Literature searches were conducted from January 2011 to July 2014 using the term “PBDE” together with the terms “neurodevelopment”, “neurotoxicity”, “infant” and “neonatal”, focussing on studies of relevant to infants.

## Evaluations by COT and EFSA

### COT

7. In its 2004 Statement, the COT noted that toxicity studies were mainly conducted on commercial mixtures of PBDEs, the composition of which was unclear and likely to differ from the profile of congeners in food and the environment. In view of the inadequacies in the toxicological databases it was not possible to establish a tolerable daily intake, and a Margin of Exposure (MoE) approach was adopted. The most sensitive effect of pentaBDE was considered to be neurodevelopmental, with a lowest observed adverse effect level (LOAEL) of 600 µg/kg bw obtained from a study in which BDE-99 was administered by a single oral dose to mice on postnatal days 3 or 10. However, the focus of the 2004 Statement was on exposure to PBDEs from fish, and the available data did not allow an assessment of exposure to infants of a comparable developmental stage (up to one month), and therefore a relevant MoE could not be calculated for neurodevelopmental effects.

8. Liver toxicity was considered the most relevant and sensitive effect for older children and adults. The COT identified a no observed adverse effect level (NOAEL) for liver effects induced by a pentaBDE formulation in the rat (450 µg/kg bw/day) as a reference point (point of departure). In 2006, the COT considered exposure to PBDEs in fish and shellfish collected over 2003-2004 (Fernandes, 2005a) and from the rest of the diet using the 2003 Total Diet Study (Fernandes, 2005b), again taking a MoE approach with the same reference point for liver effects.

### EFSA

Following oral exposure, BDE-209 is absorbed to a limited extent (<25% of dose), whereas absorption of other congeners for which data are available is higher (50-80%), with distribution primarily into lipophilic tissues. Debromination and hydroxylation are the major metabolic pathways. Elimination half-lives for individual congeners in the rat ranged from about 2 to 20 days, whereas much larger values had been reported in humans for lower

---

<sup>1</sup> <http://cot.food.gov.uk/pdfs/bfrstatement.pdf>

<sup>2</sup> <http://multimedia.food.gov.uk/multimedia/pdfs/cotstatementfishsurveys.pdf>

brominated congeners (e.g. 926 days for BDE-47, 4,530 days for BDE-153). The elimination half-life of BDE-209 was reported as 2.5-8.6 days for rats, and about 15 days for humans. (EFSA, 2011)

9. EFSA (2011) reviewed a number of epidemiological studies relating PBDE exposure to effects on thyroid hormones, neurodevelopmental effects, cancer, diabetes and metabolic syndrome, and fertility and offspring. Limitations in the study designs, inconsistencies between the outcomes of different studies, and co-exposure to other halogenated contaminants prevented firm conclusions.

10. EFSA concluded that relevant toxicological data were available for only four individual congeners (BDE-47, BDE-99, BDE-153 and BDE-209) of the eight identified as being predominant in PBDE technical mixtures (Table 2). From studies of neurodevelopmental behavioural changes, mainly in mice, following a single oral administration, EFSA calculated benchmark dose lower confidence limits for a 10% increase in the relevant activity (BMDL<sub>10s</sub>). The critical neurodevelopmental effect of BDE-47 was locomotor activity, whilst those for BDE-99, -153 and -209 were total physical activity.

11. For BDE-47, -99 and -153, the much slower rate of elimination in humans compared to rodents led EFSA to take body burden into account by estimating human intake associated with the body burden at the BMDL<sub>10</sub> (assuming 75% uptake of the single oral dose). The body burdens were then converted into human intakes estimated to result, following attainment of steady state, in the body burden at the BMDL<sub>10</sub>. In the case of BDE-209, the elimination half-life did not differ markedly between humans and rodents, and therefore the BMDL<sub>10</sub> value was applied without adjustment for body burden. These estimated human intakes at the BMDL<sub>10s</sub> for BDE-47, -99 and -153, and the external dose for BDE-209, were used as the reference points (see Table 3) in an MoE approach.

Table 3: BMDL and references points derived by EFSA (2011)

Congener	Critical endpoint	BMDL <sub>10</sub> (µg/kg bw)	Body burden at BMDL <sub>10</sub> (µg/kg bw)	Reference point (µg/kg bw/day)	Reference
BDE-47	Locomotion (mouse)	309	232	172 <sup>b</sup>	Eriksson et al. (2001)
BDE-99	Total activity (mouse)	12	9	4.2 <sup>b</sup>	Viberg, et al. (2004)
BDE-153	Total activity (mouse)	83	62	9.6 <sup>b</sup>	Viberg et al. (2003)
BDE-209	Total activity (rat)	1700	n/r <sup>a</sup>	1700	Viberg et al. (2007)

<sup>a</sup> n/r: not reported because the elimination half-life in rodents and humans is similar, and therefore the external dose was used as the reference point

<sup>b</sup> Daily human intake estimated to result in the body burden at the BMDL<sub>10</sub>.

12. EFSA expressed reservations about the protocol of a single administration of PBDE to neonatal animals and the relevance of the findings. Limitations included the single dose, not taking into account the litter effect, and that most studies were conducted in a single laboratory with no independent verification of the results. EFSA took a conservative approach for individual congeners in using these data, despite the limitations noted, to calculate BMDL<sub>10</sub> values and the body burden at the BMDL<sub>10</sub> for the PBDE congeners for which data were available.

13. The potential for additive effects of PBDEs was considered by EFSA (2011) but based on observations of divergent toxicological responses and limitations in the available information, it was not possible to conclude there was a common mode of action between congeners. Therefore a cumulative risk assessment was not performed.

### **New toxicological and epidemiological data**

14. A literature review found one new toxicological study of neurodevelopmental effects of BDE-47 published since those included in the EFSA evaluation. Groups of nine male and nine female 10-day old rats were given a single administration by gavage of 1, 5 or 10 mg/kg bw BDE-47 alone or in combination with 5 mg/kg bw polychlorinated biphenyl (PCB)-153 (He *et al.*, 2011). Serum/plasma concentrations of BDE-47 and thyroid hormones, organ to body weight ratios and performance in tests for learning and memory were performed when the rats were 2 months old. The relative uterus weights were significantly decreased at all doses, relative ovary weights were increased at 5 and 10 mg/kg bw and relative thyroid weight was decreased at the top dose. Plasma thyroxine (T4) concentration was significantly increased at 5, but not 1 or 10 mg/kg bw BDE-47. Tri-iodothyronine (T3) and thyroid stimulating hormone (TSH) did not differ significantly from control. It is notable that BDE-47 was detected in the serum of control animals at the age of 2 months, and was significantly elevated at 5 and 10 mg/kg bw. Performance in learning and memory tests was reported to be impaired at all doses, with possible enhancement by PCB153 at the top dose. The lowest dose in this study is higher than the BMDL calculated by EFSA.

15. In vitro studies have indicated that some hydroxylated PBDEs could be more neurotoxic than their parent congeners (Dingemans *et al.*, 2011). Two main modes of action have been proposed for the neurodevelopmental effects of PBDEs: an indirect effect mediated by modulation of thyroid hormone homeostasis; and direct interaction of PBDEs with nerve cells (Dingemans *et al.*, 2011; Gilbert *et al.*, 2012; Costa *et al.*, 2013).

16. A recent developmental neurotoxicity study, conducted according to international guidelines, did not provide evidence of adverse effects of BDE-209 on neurodevelopment following administration by oral gavage to dams from gestation day 6 to weaning at doses of 0, 1, 10, 100 and 1000 mg/kg bw/day. No treatment-related neurobehavioural changes were observed in detailed clinical observations, startle response, learning and memory tests, or



in motor activity assessments up to 6 months of age. Furthermore, there were no treatment-related neuropathological or morphometric alterations, and the authors concluded that the NOAEL was 1000 mg/kg bw/day, the highest dose tested (Biesemeier *et al.*, 2011). It should be noted that this NOAEL relates to maternal dose, whereas the BMDL<sub>10</sub> of 1700 µg/kg bw/day relates to direct exposure to the neonatal animal, and is therefore more relevant to the risk assessment of the infant diet.

17. Roth and Wilks (2014) conducted a systematic review of the epidemiological literature since January 2006 relating neurodevelopmental and neurobehavioural outcomes to exposure to polybrominated and polyfluorinated chemicals. The review identified 10 articles for PBDEs meeting the inclusion criteria. The studies addressed endpoints including reduced head circumference, motor function and cognitive development, attention and hyperactivity disorders, internalising and externalising behaviour, socio emotional skills, and social competence. The authors noted the difficulty in appraising the body of evidence for a given neurodevelopmental or neurobehavioural outcome due to inconsistencies across studies. However, they concluded that the epidemiological evidence currently did not support a strong causal association between PBDEs and adverse neurodevelopmental and neurobehavioural outcomes in infants and children.

18. Literature searching did not identify additional epidemiological studies relating to post-natal exposure (i.e. via breastfeeding) to PBDEs that was not included in either the systematic review of Roth and Wilks (2014) or the EFSA opinion.

19. Overall, the COT concluded that the new data did not contradict the reference points identified by EFSA for BDE-47, BDE-99, BDE-153 and BDE-209, and did not provide a basis for identifying reference points for other congeners.

## **Sources of exposure to PBDEs**

### ***Environmental occurrence of PBDEs***

20. Because of their low vapour pressure, PBDEs preferentially partition to dust in the indoor environment (Law *et al.*, 2014). Concentrations of selected PBDEs have been measured in dust sampled in homes, offices and cars in the UK with widely varying results depending on the specific PBDEs measured, and the location of sampling, but generally showed higher levels of BDE-209 and the nonaBDEs. Concentrations of most target compounds were in the order cars > offices ≥ homes. However, it is likely that the levels of specific PBDE congeners have altered over time due to progressive phasing out of usage of the technical mixtures and/or degradation of the components. Table 4 presents results for dust vacuumed from carpet or bare floors from homes, which are of more relevance than offices or cars for prolonged exposure of infants.

Table 4: Concentrations of some PBDEs in dust sampled in UK homes during 2006 (Harrad *et al.*, 2008)

Congener	PBDE concentrations in dust (µg/kg)	
	Mean	Maximum
BDE-28	0.70	2.10
BDE-47	15.0	58.0
BDE-99	36.0	180
BDE-100	5.60	17.0
BDE-153	14.0	110.0
BDE-154	4.40	16.0
BDE-183	71.0	550
Sum of BDE-28 to -183	146.7	933
BDE-209	260,000	2,200,000

21. The median sum concentration of BDEs 28, 47, 49, 66, 99, 100, 153, and 154, in air sampled in the 31 UK homes (reported in 2006), was 24 pg/m<sup>3</sup> (range 4-245 pg/m<sup>3</sup>), which was higher than in outdoor air (median 0.49, range 8.7-30 pg/m<sup>3</sup>). Concentrations of BDE-209 were not reported (Harrad *et al.*, 2010).

### ***Dietary occurrence of PBDEs***

#### ***Breast milk***

22. Fürst (2006) noted that PBDE levels in milk samples collected in the early 2000s were approximately 60% higher than those sampled 10 years before. A review by Costa *et al.* (2008) reported that levels of PBDEs in breast milk had been increasing in the past 20-30 years, along with serum levels in the general population though a slight decline had started to emerge in more recent years. The more recent review by EFSA did not find a consistent trend (EFSA, 2011).

23. Data for breast milk sampled in the UK during 2010-12 are summarised in Table 5. The congener profiles differ in the two studies with BDE-154 detected at the highest levels in the Birmingham study, and BDE-47 in the North-East England study. In contrast with the findings in dust and food (tables 4 and 7), BDE-209 was not detected at markedly higher levels than the other congeners, which is consistent with it being less bioaccumulative.

Table 5. PBDE congeners in breast milk (n=54) sampled in the UK in 2010 (Abdallah and Harrad, 2014) and 2011-2012 (Bramwell *et al.*, 2014)



This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Location sampling date (number of samples) Reference	Congener	Concentration in breast milk (ng/kg whole weight) <sup>a</sup>		
		Median	Minimum	Maximum
Birmingham  Jan-Feb 2010  (n=35)  Abdallah and Harrad, 2014	BDE-47	98	5.95	513
	BDE-49	<1.75	<1.75	15.8
	BDE-85	<1.75	<1.75	29.1
	BDE-99	24.2	<2.1	120
	BDE-100	13.3	<1.75	65.1
	BDE-153	31.9	<2.1	156
	BDE-154	7.35	<2.1	389
	SumTri-hexa <sup>b</sup>	175	<7	914
	BDE-209	8.75	<2.1	32.2
North-East England  April 2011-Feb 2012  (n=6)  Bramwell et al., 2014	DBE28	3.15	0.7	10.9
	BDE-47	67.2	11.2	458
	BDE-49	1.05	<0.7	<3.85
	BDE-66	1.05	<1.05	4.55
	BDE-85	1.4	<0.35	12.3
	BDE-99	30.8	4.2	131
	BDE-100	22.4	2.45	76.7
	BDE-138	0.7	<0.35	1.4
	BDE-153	35.4	24.5	58.8
	BDE-154	2.45	0.35	6.3
	BDE-183	1.75	0.7	8.05
	Sum exc 209 <sup>c</sup>	173	44.8	736
	BDE-209	18.2	<7	36.4

<sup>a</sup> Data converted to whole milk basis from fat weight basis assuming breast milk contains 3.5% fat.

<sup>b</sup> Sum of BDEs 28, 47, 85, 99, 100, 153, 154.

<sup>c</sup> Sum of all measured congeners except BDE-209

### *Infant formulae and complementary foods*

24. Table 6 presents mean occurrence data for some PBDE congeners in foods classified as “food for infants and small children” as reported by EFSA (2011). These relate to 42 samples, of which 29 were “ready-to-eat meal for infants and young children”, 8 were “infant formulae” and “follow on formulae”, 2 were “cereal based food for infants and young children” and one was unspecified. Data are not available for infant formula and commercially-produced infant foods purchased in the UK.

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table 6: Mean concentrations of some PBDEs in “food for infants and small children” reported in EFSA (2011)

	Mean occurrence (ng/kg whole weight)							
	BDE-28	BDE-47	BDE-99	BDE-100	BDE-153	BDE-154	BDE-183	BDE-209
Lower bound	1	207	76	21	2	5	2	115
Upper bound	2	208	78	23	6	7	5	127

## Food

25. The most recent measurements of PBDEs in food sampled in the UK were for the composite food groups of the 2012 Total Diet Study (TDS) (Fernandes *et al.*, 2012). The congeners measured in the 2012 TDS were BDE-17, -28, -47, -49, -66, -71, -77, -85, -99, -100, -119, -126, -138, -153, -154, -183 and -209. Table 7 shows the concentrations of the key PBDE congeners.

Table 7. Levels of selected or total PBDE congeners in food expressed on a whole weight basis

Food group	PBDE concentrations in food (ng/kg food) <sup>a</sup>				
	BDE-47	BDE-99	BDE-153	Total excluding BDE-209 <sup>b</sup>	BDE-209
Bread	5.38	5.71	1.66	23.0-25.0	<200 <sup>c</sup>
Canned vegetables	0.65	0.47	<0.14	1.34-2.07	20.1
Carcase meat	17.9	22.5	7.06	61.4-62.1	<130
Cereals	6.31	7.63	2.07	20.9-22.6	<190
Dairy products	23.1	25.4	5.83	64.8-66.9	21.0
Eggs	12.8	16.2	4.97	45.0-45.7	89.8
Fats+oils	36.9	34.7	8.12	97.4-103	<391
Fish	134	22.7	7.08	304	170
Fresh fruit	1.22	0.91	0.24	3.19-3.66	142
Fruit products	1.25	0.99	0.41	2.86-4.42	30.2
Green vegetables	1.54	1.48	0.16	4.07-4.21	50.2
Meat products	17.7	19.2	4.01	52.0-52.8	<140
Milk	1.79	1.95	0.49	5.30-54.8	120
Nuts	5.86	4.60	1.26	13.4-20.1	100
Offal	7.34	8.83	2.98	25.4-27.4	<120
Other vegetables	5.13	7.75	1.37	21.1-21.2	50.2
Potatoes	4.67	5.19	0.67	12.9-13.4	49.8
Poultry	5.34	5.86	1.39	18.0-19.0	220
Sugar and preserves <sup>d</sup>	121	62.1	7.08	263	1948

<sup>a</sup> Concentrations in food were calculated from values for concentrations in fat and for the fat content of food, which were both reported in Fernandes *et al.*, 2012.

<sup>b</sup> Total BDE-17, -28, -47, -49, -66, -71, -77, -85, -99, -100, -119, -126, -138, -153, -154, -183, -209. Where a range is given it represents lower bound to upper bound (treating values <LOD as 0 and LOD, respectively)

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

<sup>c</sup> Below the limit of detection (LOD), which follows the arrow, and varies with food group and congener.

<sup>d</sup> Includes sugar, sugar confectionery, jam, syrup, honey, jelly and chocolate.

### *Drinking water*

26. Concentrations of PBDEs in water were not reported in EFSA (2011). In a series of international studies (Crookes *et al.*, 2009), the only EU findings for PBDEs in water were for Sweden and all were below the limits of detection (LOD), which ranged from 0.6 to 2.9 ng/L for different congeners. If PBDEs are present in water, levels are expected to be low due to the lipophilicity of these compounds.

## **Exposure to PBDEs**

27. The exposure assessments for air, soils and dust and the diet presented here are based on external exposure. Bodyweight data are from the UK Dietary and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013), with average bodyweights of 7.8, 8.7 and 9.6 kg for infants aged >4.0-6.0, >6.0-9.0 and >9.0-12.0 months old, respectively. Since DNSIYC did not include infants younger than 4 months, in this statement a value of 5.9 kg for infants aged 0-3 months from an earlier survey (DH, 1994), is assumed for infants aged 0-4 months.

## ***Environmental exposure to PBDEs***

### *Dust and soil*

28. Table 8 shows potential exposures of infants to PBDEs through ingestion of soil/dust, calculated assuming ingestion of 100 mg dust/day (WHO, 2007) based on the occurrence data in Table 4. The assessment focuses on infants aged 9-12 months because they are likely to have more contact with floors and other surfaces than younger infants. Since the dust was sampled in 2006, and there have been changes in uses of PBDE technical mixtures since then, it is possible that these estimates are not representative of current exposures.

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table 8: Potential exposures of infants aged 9-12 months to PBDEs in dust in UK homes

Congeners	PBDE exposures from consumed dust (ng/kg bw/day)	
	At mean conc	At maximum conc
BDE-28	0.01	0.02
<b>BDE-47<sup>a</sup></b>	<b>0.16</b>	<b>0.60</b>
<b>BDE-99</b>	<b>0.38</b>	<b>1.88</b>
BDE-100	0.06	0.18
BDE-153	0.15	1.15
BDE-154	0.05	0.17
BDE-183	0.74	5.73
Sum of BDE-28 to -183	1.53	9.72
<b>BDE-209</b>	<b>271,000</b>	<b>2,292,000</b>

<sup>a</sup> Congeners for which reference points have been calculated are highlighted in bold

29. Potential exposures of UK infants to PBDEs in air, calculated assuming a ventilation rate of 3 m<sup>3</sup>/day (US EPA, 1989), and the median reported occurrence of 0.49 pg/m<sup>3</sup> for the sum of BDEs 28, 47, 49, 66, 99, 100, 153, and 154 in home air (paragraph 23), are 12, 9, 8 and 8 ng/kg bw/day at age 0-4, 4-6, 6-9 and 9-12 months respectively.

### ***Dietary exposure to PBDEs***

#### ***Breast milk***

30. Table 9 shows estimated exposure of exclusively breastfed infants based on the maximum values identified from the data of Abdallah and Harrad (2014) and Bramwell *et al.* (2014) for average (800 mL) and high-level (1200 mL) daily consumption of breast milk.

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table 9 PBDE exposure ( $\mu\text{g/kg bw/day}$ ) from exclusive breastfeeding by infants estimated for average and high level consumption

PBDE concentration ( $\mu\text{g/L}$ whole weight)	Exposures to PBDE from exclusive breastfeeding for age 0-4 and 4-6 months (ng/kg bw/day)			
	0-4.0 (800 mL)	0-4.0 (1200 mL)	>4.0-6.0 (800 mL)	>4.0-6.0 (1200 mL)
BDE-28 (10.9) <sup>b</sup>	1.47	2.21	1.11	1.67
<b>BDE-47<sup>a</sup> (513) <sup>c</sup></b>	<b>69.5</b>	<b>104</b>	<b>52.6</b>	<b>78.9</b>
BDE-49 (15.8) <sup>c</sup>	2.14	3.20	1.62	2.42
BDE-66 (4.55) <sup>b</sup>	0.62	0.93	0.47	0.70
BDE-85 (29.1) <sup>c</sup>	3.94	5.91	2.98	4.47
<b>BDE-99 (131) <sup>b</sup></b>	<b>17.8</b>	<b>26.6</b>	<b>13.4</b>	<b>20.1</b>
BDE-100 (76.7) <sup>b</sup>	10.4	15.6	7.86	11.8
BDE-138 (1.4) <sup>b</sup>	0.19	0.28	0.14	0.22
<b>BDE-153 (160) <sup>c</sup></b>	<b>21.7</b>	<b>32.5</b>	<b>16.4</b>	<b>24.6</b>
BDE-154 (389) <sup>c</sup>	52.7	79.0	39.9	59.8
BDE-183 (8.05) <sup>b</sup>	1.09	1.64	0.83	1.24
Sum exc 209d (736) <sup>b</sup>	99.8	150	75.5	113
<b>BDE-209 (36.4) <sup>b</sup></b>	<b>4.94</b>	<b>7.40</b>	<b>3.73</b>	<b>5.60</b>

<sup>a</sup> Congeners for which reference points have been calculated are highlighted in bold

<sup>b</sup> Bramwell *et al.* (2014)

<sup>c</sup> Abdallah and Harrad (2014)

<sup>d</sup> Sum of all measured congeners except BDE-209

## Food

31. UK Data on PBDE in infant formula and commercially-produced infant foods are not available. Table 10 summarises upper bound total infant dietary exposure to PBDEs estimated using the 19 composite food groups of the 2012 TDS (see table 7) together with consumption data from the DNYSIC (DH, 2013). The detailed data are included in Annex 1. The food groups contributing most to total exposure vary with the age group and appear to be related to frequency of consumption.

Table 10: Estimated exposure of infants to PBDEs from food

PBDE	Dietary exposure to PBDEs (ng/kg bw/day)					
	4-6 months (n=102)		6-9 months (n=602)		9-12 months (n=684)	
	Mean	P97.5	Mean	P97.5	Mean	P97.5
BDE-47	1.00	3.39	1.04	3.29	1.00	2.48
BDE-99	1.10	3.73	1.10	3.51	1.01	2.66
BDE-153	0.251	0.856	0.25	0.833	0.23	0.612
Sum exc 209 <sup>a</sup>	2.94	9.83	3.14	9.57	3.21	7.25
BDE-209	1.55	4.16	2.94	7.71	4.48	10.9

<sup>a</sup> Sum of all measured congeners except BDE-209

## Risk characterisations for PBDEs

32. Table 11 shows the MoEs calculated for exclusively breastfed infants, by dividing the reference point for the different congeners (table 3) by the estimated exposure. The reference point used for the sum of all measured congeners except BDE-209, is 4.2 µg/kg bw/day, i.e. the lowest of the available reference points (for BDE-99). This is a pragmatic approach taken to allow for the congeners for which reference points are not available due to the lack of relevant toxicity data, and is likely to be a worst case since some of the congeners are less toxic. BDE-209 is excluded from this approach since it is less toxic and has a much higher reference point than the lower congeners.

Table 11. MoEs for exclusively breastfed infants

PBDE	MoEs for PBDEs for exclusive breastfeeding for age 0-4 and 4-6 months			
	0-4.0 (800 mL)	0-4.0 (1200 mL)	>4.0-6.0 (800 mL)	>4.0-6.0 (1200 mL)
BDE-47	2,475	1,654	3,270	2,180
BDE-99	236	158	313	209
BDE-153	442	295	585	390
Sum exc 209 <sup>a</sup>	42	28	56	37
BDE-209	344,130	229,730	455,764	303,571

<sup>a</sup> Reference point for BDE-99 divided by upper bound sum of all measured congeners except BDE-209

33. Tables 12 and 13 show the MoEs calculated for infant exposure to PBDEs via the diet and dust, respectively

Table 12. MoEs for dietary exposure of infants

PBDE	4-6 months		6-9 months		9-12 months	
	Mean	P97.5	Mean	P97.5	Mean	P97.5
BDE-47	172000	50737	165385	52280	172000	69355
BDE-99	3818	1126	3818	1197	4158	1579
BDE-153	38247	11215	38400	11525	41739	15686
Sum exc 209 <sup>a</sup>	1429	427	1338	439	1308	579
BDE-209	1096774	408654	578231	220493	379464	155963

<sup>a</sup> Reference point for BDE-99 divided by upper bound sum of all measured congeners except BDE-209

Table 13: MoEs for PBDEs in consumed dust from homes

Congeners	Margins of Exposure	
	At mean conc.	At maximum conc.
BDE-47	1,075,000	286,667
BDE-99	11,053	2,234
BDE-153	64,000	8,348
Sum of BDE-28 to 183 <sup>a</sup>	2,745	432
BDE-209	6.27	0.74

<sup>a</sup> Reference point for BDE-99 divided by upper bound sum of measured BDEs in the range 28-183

34. EFSA (2011) provides the following comment on the MoE values for PBDEs. “Usually for non-genotoxic compounds a MoE of 100 is considered sufficient to conclude that there is no health concern (WHO, 2009). This MoE covers uncertainties and variability with regard to both kinetic and dynamic differences between experimental animals and humans (factor  $4 \times 2.5 = 10$ ), and within the human population (factor  $3.2 \times 3.2 = 10$ ). Given the fact that the MoE approach is based on a body burden comparison between animals and humans, the potential kinetic differences have been accounted for. Equally, by focussing on the body burden associated with a BMDL10 for neurobehavioural effects in mice induced during a relevant period for brain development (i.e. representing the most sensitive fraction of the population), and applying this body burden for the entire life span in humans, individual difference in susceptibility has been covered by the approach taken. Therefore, the calculated MoE should be sufficient to cover inter-species differences in sensitivity for the effects observed (i.e. toxicodynamic differences between experimental animals and humans). Usually, in a traditional hazard characterization a default assessment factor of 2.5 (WHO, 2009) is applied to account for inter-species difference in toxicodynamics. In addition, by using the longest half-lives as reported for the various individual PBDE congeners, the estimation of the chronic human intake associated with the body burden calculated at the respective BMDLs is on the conservative side. This implies that in the case of PBDEs in principle any MoE larger than 2.5 indicate that there is unlikely to be a health concern. The larger the MoE is, the smaller is the potential health concern.”

35. Occurrence data for PBDEs are limited to a relatively small proportion of the 209 possible congeners. Whilst different research groups have measured and reported different subsets of congeners, they generally focus on those that are most likely to be present based on their prevalence in technical mixtures of PBDEs that have been used as BFRs, and all include the key congeners (BDE-47, BDE-99, BDE-153 and BDE-209), for which reference points are available. There is a lack of toxicity data on many of the congeners. EFSA concluded there was not enough information on modes of action to support a cumulative risk assessment approach. However, on a pragmatic worst case basis the sum of measured congeners have been compared to the lowest reference point in this assessment. On the basis of



the above considerations, exposure to PBDEs from breastfeeding and from complementary food does not represent a health concern.

36. There is a possible concern with respect to exposure from dust at the highest level measured in UK homes. However, there is uncertainty with respect to whether this high level is representative of current occurrence levels in the UK and whether infants would be repeatedly exposed to dust containing high levels of PBDEs. There are no clear time trends for exposure to PBDEs, which is likely to be due to changing uses and differences in the persistence of congeners in the environment.

## Conclusions

[To be drafted following COT discussion]

**Secretariat**  
**July 2014**

## References

- Abdallah MA, Harrad S (2014). Polybrominated diphenyl ethers in UK human milk: implications for infant exposure and relationship to external exposure. *Environ Int.* 2014 Feb;63:130-6.
- Biesemeier JA, Beck MJ, Silberberg H, Myers NR, Ariano JM, Radovsky A, Freshwater L, Sved DW, Jacobi S, Stump DG, Hardy ML, Stedeford T (2011). An oral developmental neurotoxicity study of decabromodiphenyl ether (DecaBDE) in rats. *Birth Defects Res B Dev Reprod Toxicol.* Feb;92(1):17-35.
- Bramwell L, Fernandes A, Rose M, Harrad S, Pless-Mulloli T (2014). PBDEs and PBBs in human serum and breast milk from cohabiting UK couples. *Chemosphere.* Apr 15. pii: S0045-6535(14)00409-3. doi: 10.1016/j.chemosphere.2014.03.060. [Epub ahead of print]
- Costa LG, Giordano G, Tagliaferri S, Caglieri A, Mutti A (2008). Polybrominated diphenyl ether (PBDE) flame retardants: environmental contamination, human body burden and potential adverse health effects. *Acta Biomed.* 2008. 79(3):172-83.
- Costa LG, de Laat R, Tagliaferri S, Pellacani C (2013). A mechanistic view of polybrominated diphenyl ether (PBDE) developmental neurotoxicity. *Toxicol Lett.* Nov 20. pii: S0378-4274(13)01417-3. doi: 10.1016/j.toxlet.2013.11.011.
- Crookes M, Brooke D and Burns J (2009). Brominated flame retardants - risks to UK drinking water sources. Client report number 242844, Building Research Establishment Ltd, Garston UK, Available at:

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

[http://dwi.defra.gov.uk/research/completed-research/reports/DWI70\\_2\\_219.pdf](http://dwi.defra.gov.uk/research/completed-research/reports/DWI70_2_219.pdf)

Dingemans MM, van den Berg M, Westerink RH (2011). Neurotoxicity of brominated flame retardants: (in)direct effects of parent and hydroxylated polybrominated diphenyl ethers on the (developing) nervous system. *Environ Health Perspect.* Jul;119(7):900-7.

DH (Department of Health) (2013). Diet and Nutrition Survey of Infants and Young Children (DNSIYC), 2011. Available at: <http://transparency.dh.gov.uk/2013/03/13/dnsiyc-2011/>

EFSA (2011). Scientific Opinion on Polybrominated Diphenyl Ethers (PBDEs) in Food. *EFSA Journal*; **2156**:1-274.

Eriksson P, Jakobsson E, and Fredriksson A (2001). Brominated Flame Retardants: A Novel Class of Developmental Neurotoxins in Our Environment? *Environ Health Perspect*;109:903–908.

Fernandes A, D'Silva K, Driffield M, White S and Rose M (2005a). FD 05/07. Brominated Flame Retardants and Brominated Dioxins in wild and farmed fish and shellfish. A Central Science Laboratory report for the Food Standards Agency.

Fernandes A, D'Silva K, Driffield M, White S and Rose M (2005b). FD 04/37. Brominated Flame Retardants and Brominated Dioxins in 2003 Total Diet Samples. A Central Science Laboratory report for the Food Standards Agency.

Fernandes A, Rose M, Smith F, and Holland M (2012). FD 12/04. Organic Environmental Contaminants in the 2012 Total Diet Study Samples Report to the Food Standards Agency. Available at: [http://www.foodbase.org.uk/admintools/reportdocuments/848-1-1561\\_FS241031\\_TDS\\_2012\\_final.pdf](http://www.foodbase.org.uk/admintools/reportdocuments/848-1-1561_FS241031_TDS_2012_final.pdf)

Fürst P. Dioxins, polychlorinated biphenyls and other organohalogen compounds in human milk. Levels, correlations, trends and exposure through breastfeeding. *Mol Nutr Food Res.* 2006. 50(10):922-33.

Gilbert ME, Rovet J, Chen Z, Koibuchi N (2012). Developmental thyroid hormone disruption: prevalence, environmental contaminants and neurodevelopmental consequences. *Neurotoxicology.* Aug;33(4):842-52.

Harrad S, de Wit CA, Abdallah MA, Bergh C, Bjorklund JA, Covaci A, Darnerud PO, de Boer J, Diamond M, Huber S, Leonards P, Mandalakis M, Ostman C, Haug LS, Thomsen C and Webster TF (2010). Indoor contamination with hexabromocyclododecanes, polybrominated diphenyl ethers, and perfluoroalkyl compounds: an important exposure pathway for people? *Environmental Science & Technology*;44:3221-3231.

Harrad S, Ibarra C, Abdallah MA, Boon R, Neels H and Covaci A. (2008). Concentrations of brominated flame retardants in dust from United Kingdom cars, homes, and offices: causes of variability and implications for human exposure. *Environ Int.*;34:1170-1175.

He P, Wang A, Niu Q, Guo L, Xia T, Chen X (2011). Toxic effect of PBDE-47 on thyroid development, learning, and memory, and the interaction between PBDE-47 and PCB153 that enhances toxicity in rats. *Toxicol Ind Health.* Apr;27(3):279-88.

Law RJ, Covaci A, Harrad S, Herzke D, Abdallah MA, Fernie K, Toms LM, Takigami H (2014). Levels and trends of PBDEs and HBCDs in the global environment: status at the end of 2012. *Environ Int.* Apr;65:147-58.

Roth N, Wilks MF (2014). Neurodevelopmental and neurobehavioural effects of polybrominated and perfluorinated chemicals: A systematic review of the epidemiological literature using a quality assessment scheme. *Toxicol Lett.* Feb 25. pii: S0378-4274(14)00093-9.

US EPA (U. S. Environmental Protection Agency). (1989). Review of the National ambient air quality standards for lead: exposure analysis methodology and validation. OAQPS staff report. *EPA-450/2-89-011*

Viberg H, Fredriksson A and Eriksson P (2003). Neonatal exposure to polybrominated diphenyl ether (PBDE 153) disrupts spontaneous behaviour, impairs learning and memory, and decreases hippocampal cholinergic receptors in adult mice. *Toxicology and Applied Pharmacology*;192:95–106.

Viberg H, Fredriksson A and Eriksson P (2004). Neonatal exposure to the brominated flame-retardant, 2,2',4,4',5-pentabromodiphenyl ether, decreases cholinergic nicotinic receptors in hippocampus and affects spontaneous behaviour in the adult mouse. *Environmental Toxicology and Pharmacology*;17:61–65.

Viberg H, Fredriksson A and Eriksson P (2007). Changes in spontaneous behaviour and altered response to nicotine in the adult rat, after neonatal exposure to the brominated flame retardant, decabrominated diphenyl ether (PBDE 209). *NeuroToxicology*;28:136–142.

WHO (World Health Organization). (2007). Health risks of heavy metals from long-range transboundary air pollution. Joint WHO/Convention Task Force on the Health Aspects of Air Pollution, Copenhagen

WHO (World Health Organization). (2009) Principles and Methods for the Risk Assessment of Chemicals in Food, *Environmental Health Criteria* 240 WHO, Geneva, Switzerland.

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

## Abbreviations

COT	Committee on Toxicity of Chemicals in Food Consumer Products and the Environment
BDE	Brominated diphenyl ether
BMDL	Benchmark dose lower confidence limit
BFR	Brominated flame retardant
bw	Body weight
DH	Department of Health
DNSIYC	Dietary and Nutrition Survey of Infants and Young Children
EFSA	European Food Safety Authority
FERA	Food and Environment Research Agency
LOAEL	Lowest observed adverse effect level
LOD	Limit of detection
MoE	Margin of Exposure
NOAEL	No observed adverse effect level
PBDE	Polybrominated diphenyl ether
PCB	Polychlorinated biphenyl
SACN	Scientific Advisory Committee on Nutrition
T3	Tri-iodothyronine
T4	Thyroxine
TSH	Thyroid stimulating hormone

Additional abbreviations can be found within Tables 1 and 2.

## Annex 1

### Dietary exposure to PBDEs for different foods and infant age groups

Table A1: Estimate mean dietary exposure for age 4-6 months

Food group	Number of Consumers	4.00 to 5.99 months - PBDE Mean Exposure (ng/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	11	0.0041	0.0044	0.0013	0.0191	0.1531
Canned vegetables	4	0.0013	0.0009	0.0003	0.0039	0.0387
Carcase meat	10	0.0149	0.0187	0.0059	0.0516	0.1079
Cereals	59	0.0072	0.0087	0.0023	0.0257	0.2157
Dairy products	76	1.2842	1.412	3.241	3.7191	1.1167
Eggs	2	0.0079	0.0099	0.0031	0.0281	0.0526
Fats+oils	14	0.0045	0.0042	0.0009	0.0126	0.0478
Fish	6	1.5489	0.0262	0.0082	0.3514	0.1965
Fresh fruit	36	0.0046	0.0034	0.0009	0.0138	0.5346
Fruit products	29	0.0028	0.0022	0.0009	0.0097	0.0666
Green vegetables	33	0.0034	0.0032	0.0004	0.0092	0.1098
Meat products	1	0.0132	0.0143	0.0029	0.0393	0.1042
Milk	17	0.0055	0.006	0.0015	0.1689	0.3698
Nuts	0	0	0	0	0	0
Offal	0	0	0	0	0	0
Other vegetables	57	0.0128	0.0193	0.0034	0.0528	0.125
Potatoes	36	0.0108	0.012	0.0016	0.031	0.1154
Poultry	11	0.0084	0.0093	0.0022	0.03006	0.348
Sugar and preserves	10	0.0272	0.0139	0.0016	0.059	0.4374
<b>Total</b>	<b>102</b>	<b>1.0049</b>	<b>1.0981</b>	<b>0.2514</b>	<b>2.9418</b>	<b>1.5512</b>

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table A2: Estimate mean dietary exposure for age 6-9 months

Food group	Number of Consumers	6.00 to 8.99 months - PBDE Mean Exposure (ng/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	242	0.0066	0.0070	0.0020	0.0305	0.2441
Canned vegetables	131	0.0011	0.0008	0.0002	0.0035	0.0335
Carcase meat	217	0.0267	0.0335	0.0105	0.0926	0.1938
Cereals	496	0.0194	0.0235	0.0064	0.0695	0.5847
Dairy products	535	1.0104	1.1110	0.2550	2.9261	0.9185
Eggs	88	0.0163	0.0207	0.0063	0.0584	0.1147
Fats+oils	282	0.0069	0.0065	0.0015	0.0193	0.0733
Fish	175	0.1607	0.0272	0.0085	0.3646	0.2039
Fresh fruit	385	0.0050	0.0037	0.0010	0.0150	0.5823
Fruit products	235	0.0023	0.0018	0.0007	0.0080	0.0549
Green vegetables	338	0.0029	0.0028	0.0003	0.0079	0.0938
Meat products	93	0.0267	0.0289	0.0060	0.0795	0.2108
Milk	270	0.0100	0.0109	0.0027	0.3061	0.6702
Nuts	19	0.0013	0.0010	0.0003	0.0043	0.0215
Offal	6	0.0030	0.0036	0.0012	0.0112	0.0491
Other vegetables	453	0.0178	0.0269	0.0048	0.0736	0.1742
Potatoes	389	0.0130	0.0144	0.0019	0.0372	0.1382
Poultry	252	0.0059	0.0065	0.0015	0.0211	0.2440
Sugar and preserves	172	0.0449	0.0231	0.0026	0.0976	0.7231
Total	602	1.0419	1.0986	0.2523	3.1433	2.9442

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table A3: Estimate mean dietary exposure for age 9-12 months

Food group	Number of Consumers	9.00 to 11.99 months - PBDE Mean Exposure (ng/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	502	0.0101	0.0107	0.0031	0.0468	0.3742
Canned vegetables	271	0.0015	0.0011	0.0003	0.0048	0.0463
Carcase meat	372	0.0280	0.0352	0.0111	0.0973	0.2036
Cereals	656	0.0269	0.0326	0.0088	0.0965	0.8112
Dairy products	661	0.7943	0.8734	0.2005	2.3004	0.7221
Eggs	207	0.0184	0.0233	0.0071	0.0656	0.1290
Fats+oils	456	0.0106	0.0100	0.0023	0.0297	0.1127
Fish	305	0.1998	0.0339	0.0106	0.4534	0.2535
Fresh fruit	574	0.0062	0.0046	0.0012	0.0187	0.7253
Fruit products	322	0.0026	0.0021	0.0009	0.0092	0.0630
Green vegetables	436	0.0028	0.0027	0.0003	0.0076	0.0908
Meat products	262	0.0261	0.0283	0.0059	0.0779	0.2065
Milk	426	0.0188	0.0205	0.0051	0.5756	1.2604
Nuts	29	0.0020	0.0016	0.0004	0.0070	0.0349
Offal	9	0.0072	0.0087	0.0029	0.0270	0.1182
Other vegetables	595	0.0175	0.0264	0.0047	0.0721	0.1708
Potatoes	546	0.0161	0.0179	0.0023	0.0462	0.1715
Poultry	400	0.0075	0.0082	0.0019	0.0265	0.3069
Sugar and preserves	297	0.0552	0.0283	0.0032	0.1200	0.8890
Total	684	1.0001	1.0136	0.2340	3.2088	4.4796



This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table A4: Estimate high level (97.5<sup>th</sup> percentile) dietary exposure for age 4-6 months

Food group	Number of Consumers	4.00 to 5.99 months - PBDE 97.5 Percentile Exposure (ng/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	11	0.0087	0.0093	0.0027	0.0407	0.3252
Canned vegetables	4	0.0015	0.0011	0.0003	0.0048	0.0465
Carcase meat	10	0.0412	0.0517	0.0162	0.1428	0.2989
Cereals	59	0.0266	0.0322	0.0087	0.0953	0.8009
Dairy products	76	3.4152	3.7552	0.8619	9.8907	3.1047
Eggs	2	0.0174	0.0205	0.0068	0.0622	0.1222
Fats+oils	14	0.0122	1.2000	0.0028	0.0358	0.1358
Fish	6	0.2890	0.0490	0.0153	0.6558	0.3667
Fresh fruit	36	0.0166	0.0124	0.0033	0.0498	1.9338
Fruit products	29	0.0113	0.0090	0.0037	0.0400	0.2735
Green vegetables	33	0.0103	0.0099	0.0011	0.0281	0.3355
Meat products	1	0.0132	0.0143	0.0030	0.0393	0.1042
Milk	17	0.0248	0.0245	0.0062	0.6881	1.5069
Nuts	0	0	0	0	0	0
Offal	0	0	0	0	0	0
Other vegetables	57	0.0399	0.0604	0.0107	0.1652	0.3912
Potatoes	36	0.0261	0.0291	0.0038	0.0750	0.2788
Poultry	11	0.0283	0.0310	0.0074	0.1007	1.1657
Sugar and preserves	10	0.0591	0.0303	0.0035	0.1285	0.9520
Total	102	3.39232	3.73067	0.85618	9.82632	4.16336

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table A5: Estimate high level (97.5<sup>th</sup> percentile) dietary exposure for age 6-9 months

Food group	Number of Consumers	6.00 to 8.99 months - PBDE 97.5 Percentile Exposure (ug/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	242	0.0235	0.0249	0.0072	0.1090	0.8720
Canned vegetables	131	0.1409	0.0723	0.0082	0.3063	2.2686
Carcase meat	217	0.0116	0.0111	0.0012	0.0316	0.3768
Cereals	496	0.0485	0.0539	0.0070	0.1392	0.5173
Dairy products	535	0.0618	0.0933	0.0165	0.2553	0.6045
Eggs	88	0.0045	0.0033	0.0010	0.0144	0.1395
Fats+oils	282	0.0174	0.0130	0.0034	0.0522	2.0237
Fish	175	0.0095	0.0076	0.0031	0.0337	0.2306
Fresh fruit	385	0.0320	0.0349	0.0088	0.9794	2.1448
Fruit products	235	3.2813	3.6080	0.8281	9.5030	2.9830
Green vegetables	338	0.0781	0.0945	0.0256	0.2798	2.3521
Meat products	93	0.0040	0.0032	0.0009	0.0138	0.0689
Milk	270	0.1125	0.1414	0.0444	0.3902	0.8168
Nuts	19	0.0038	0.0045	0.0015	0.0141	0.0618
Offal	6	0.0928	0.1006	0.0210	0.2767	0.7338
Other vegetables	453	0.0243	0.0266	0.0063	0.0863	0.9998
Potatoes	389	0.6028	0.1021	0.0318	1.3676	0.7647
Poultry	252	0.0281	0.0264	0.0062	0.0784	0.2977
Sugar and preserves	172	0.0687	0.0869	0.0267	0.2452	0.4817
Total	602	3.29291	3.50835	0.83306 1	9.56762	7.71142

This is a draft statement for discussion.  
It does not reflect the final views of the Committee and should not be cited.

Table A6: Estimate high level (97.5<sup>th</sup> percentile) dietary exposure for age 9-12 months

Food group	Number of Consumers	9.00 to 11.99 months - PBDE 97.5 Percentile Exposure (ng/kg bw/d)				
		BDE-47	BDE-99	BDE-153	Sum exc BDE-209	BDE-209
Bread	502	0.0338	0.0359	0.0104	0.1571	1.2565
Canned vegetables	271	0.0056	0.0040	0.0012	0.4338	0.1729
Carcase meat	372	0.1263	0.1588	0.0498	0.0178	0.9173
Cereals	656	0.0899	0.1087	0.0295	0.4382	2.7064
Dairy products	661	2.3175	2.5482	0.5849	0.3219	2.1068
Eggs	207	0.0707	0.0894	0.0274	6.7116	0.4958
Fats+oils	456	0.0393	0.0370	0.0086	0.2523	0.4164
Fish	305	0.7344	0.1244	0.0388	0.1097	0.9318
Fresh fruit	574	0.0208	0.0155	0.0041	1.6662	2.4257
Fruit products	322	0.0125	0.0099	0.0041	0.0625	0.3009
Green vegetables	436	0.0127	0.0122	0.0013	0.0440	0.4148
Meat products	262	0.1029	0.1116	0.0233	0.0348	0.8139
Milk	426	0.1065	0.1161	0.0292	0.3070	7.1419
Nuts	29	0.0068	0.0054	0.0015	3.2615	0.1165
Offal	9	0.0160	0.0193	0.0065	0.0234	0.2619
Other vegetables	595	0.0512	0.0774	0.0137	0.0598	0.5011
Potatoes	546	0.0532	0.0591	0.0076	0.2116	0.5674
Poultry	400	0.0257	0.0282	0.0067	0.1527	1.0602
Sugar and preserves	297	0.1996	0.1024	0.0117	0.0916	3.2131
Total	684	2.4809	2.6602	0.6120	7.2518	10.9439