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M.J. Zeilmaker | B.G.H. Bokkers | C.W. Noorlander | J.D. te Biesebeek | A.A. Jekel | R.C. Schothorst

Polybrominated diphenyl ethers: occurrence in Dutch duplicate diets and comparison with exposure from European house dust

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M.J. Zeilmaker
B.G.H. Bokkers
C.W. Noorlander
J.D. te Biesebeek
A.A. Jekel
R.C. Schothorst

Contact:
M.J. Zeilmaker
Centre for Substances and Integrated Risk Assessment
Marco.Zeilmaker@rivm.nl

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Abstract

Polybrominated diphenyl ethers: occurrence in Dutch duplicate diets and comparison with exposure from European house dust

Flame retardants, like polybrominated diphenyl ethers (PBDEs), are applied to consumer products and have entered the human food chain via the environment. At the moment the exposure to PBDE-99 from food is around its maximum allowed human intake level. This indicates that adverse effects on public health may be expected when the level of this compound in food would increase. In this respect monitoring results have revealed that the level of PBDE-99 in food has somewhat increased in the last 3 decades. For this reason the monitoring of PBDEs in food remains necessary.

The presence of PBDEs has been investigated in food items which are consumed by individuals over 24 hours ('24-hour duplicate diets'). In the Netherlands, 24-hour duplicate diets have been collected in 1978, 1984, 1994 and 2004.

Three PBDEs have been found in the duplicate diets: PBDE-47, PBDE-99 and PBDE-209. In the mentioned period the food levels of PBDE-47 have remained constant. However, food levels of PBDE-99 just showed a consistent, increasing pattern from 1978 to 1994, which seems to level off after 1994.

Taking additional exposure to PBDEs from house dust and bioaccumulation into account a maximum allowed human intake level for PBDE-99 has been estimated by the RIVM. The exposure to PBDE-99 from food is around this maximum intake level. Maximum allowed intake levels for other PBDEs still need to be determined. We therefore recommend performing additional studies on exposure assessment and the estimation of maximum allowed intake levels of other PBDEs.

Key words: brominated flame retardants, food, dietary intake, house dust, risk assessment

Rapport in het kort

Blootstelling aan polygebromeerde difenylethers uit Nederlandse duplicaat voedingen en uit Europese huisstof

Vlamvertragers als polygebromeerde difenylethers (PBDE's) hebben zich vanuit consumenten producten in het milieu verspreid. Van daaruit zijn zij in de voedselketen terechtgekomen. Op dit moment ligt de blootstelling aan PBDE-99 rond de maximaal toelaatbare inname voor deze verbinding. Er kan dus een gezondheidsrisico ontstaan wanneer het gehalte van PBDE-99 in voeding toe zou nemen. Monitoring onderzoek heeft aangegeven dat het gehalte van PBDE-99 in de laatste 30 jaar enigszins is toegenomen. Onderzoek naar PBDE's in voeding blijft daarom nodig.

In dit onderzoek is al het voedsel dat iemand in 24 uur consumeert ('24-uurs duplicaat dieet') onderzocht op de aanwezigheid van PBDE's. In Nederland zijn gegevens over deze voedingsmonsters verzameld in 1978, 1984, 1994 en 2004.

In de monsters zijn drie PBDE's aangetoond: PBDE-47, PBDE-99 en PBDE-209. In de onderzochte jaren is de hoeveelheid PBDE-47 in de diëten onveranderd gebleven. PBDE-99 laat een consistente toename zien van 1978 tot 1994, waarna het gehalte zich lijkt te stabiliseren.

Rekening houdend met blootstelling van PBDEs uit huisstof en bioaccumulerend vermogen is door het RIVM een maximaal dagelijkse toelaatbare humane inname voor PBDE-99 berekend. De blootstelling aan PBDE-99 via voeding blijkt rond deze maximaal toelaatbare inname te liggen. Wat het risico is voor andere PBDE's moet nog onderzocht worden. Aanbevolen wordt daarom om het blootstellingonderzoek te vervolgen en ook maximaal toelaatbare innamen voor andere PBDE's te berekenen.

Trefwoorden: PBDE, gebromeerde vlamvertragers, blootstelling, voeding, huisstof, risicoschatting

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Summary

Brominated flame retardants, like polybrominated diphenyl ethers (PBDEs), are environmental contaminants which have entered the human food chain. This report considers the dietary intake of the polybrominated diphenyl ethers (PBDEs) between 1978 and 2004 in the Netherlands. In addition, human health risk and the time trends of PBDEs in food are investigated.

Dietary intake of PBDEs was estimated using 24-hour duplicate diets, collected in 1978, 1984, 1994 and 2004. Three PBDEs could be analyzed in the duplicate diets: PBDE-47, PBDE-99 and PBDE-209. Food levels of PBDE-47 did not show a clear time pattern. However, food levels of PBDE-99 just showed a consistent, increasing pattern from 1978 to 1994, which seems to level off after 1994. Since PBDE-209 was only detected in 2004, no conclusion can be drawn on the time pattern.

Taking additional exposure to PBDEs from house dust and bioaccumulation into account a maximum allowed human intake level for PBDE-99 has been estimated by the RIVM. The exposure to PBDE-99 from food, measured in 24-hour duplicate diets, is just above this maximum intake level. When PBDE-99 is measured with the so-called total diet method, the exposure was found to be just below the maximum intake level (Bakker et al., 2008). For the other PBDEs, a corresponding risk assessment can only be carried out when suitable results of toxicity studies will become available.

The main routes of exposure to PBDEs are food and house dust. Food showed to be the dominant route of exposure for adults (determined for PBDE-47, 99, 100 and 183). In case of two-year-old children, the exposure to PBDE-99 and -100 via dust is of the same order or even greater than the exposure from food. For PBDE-47 and -183, the exposure via house dust was lower than dietary exposure. Hence, the amount accumulated in the body and/or breast milk (mainly) reflects the time-trend of long-term dietary exposure. The observed time trend in, mainly Swedish, breast milk is not comparable with the time trend of 24-hour Dutch duplicate diets, which could be due to a lack of measurements of PBDEs in food (time-span every 10 year). The time trend of PBDEs in breast milk shows that monitoring should be rather fine-meshed, i.e. to be once every two years, in order to achieve a full-scale overview of PBDEs in the body over time.

In conclusion, while the use of PBDEs in Europe has been restricted, it is assumed that there will be a time-delay before this will result in lower PBDE concentrations in food. A regular monitoring program of PBDEs in Dutch food is therefore recommended. The potential risk due to exposure to PBDE-99 indicates that caution should be exercised with other PBDEs and/or brominated flame retardants. Therefore, more toxicity studies should become available which allow the determination of maximum allowed intake levels for other PBDEs.

1 Introduction

Brominated flame retardants (BFRs) are a group of brominated organic substances that have an inhibitory effect on the ignition of combustible organic materials. BFRs are applied to textiles, wiring, furniture, industrial paints and incorporated into plastics and foams, and they are commonly used in electronic products to reduce the flammability of the product. About one-third of the total world production of BFRs consists of polybrominated diphenylethers (PBDEs) (De Wit, 2002; Domingo, 2004). Use of pentabromodiphenylether (penta-BDE) technical products was voluntarily phased out by industry within the European Union over the last 10 years. The use of penta-BDE and octa-BDE technical products in all applications for the European Union market has officially been banned since August 2004 (De Winter-Sorkina et al., 2006).

PBDEs are additives mixed into polymers and are not chemically bound to the plastic or textile. Therefore, they may be released relatively easily from consumer products. Humans may be exposed to PBDEs via food, ingestion of house dust and inhalation of indoor air (Wilford et al., 2005). Just as for PCBs and dioxins, food products of animal origin with high fat content (fatty fish, meat and dairy products) are expected to be major contributors to dietary exposure. The contamination of human food products by PBDEs is not well known. However in recent years a number of studies have been carried out to measure the PBDE concentrations in food. A large number of measurements in fish is reported, but mainly as an indicator of environmental pollution and to a much less extent in fish for consumption. Recently, a number of new studies assessing the dietary intake of PBDEs is reported. These studies demonstrate that the Dutch population is exposed to a considerable amount of PBDEs (De Mul et al., 2005).

Exposure assessment of persistent, lipophilic contaminants is usually estimated *indirectly* by combining the results of monitoring of food categories (butter, milk, edible oils, etc.) with habitual food consumption patterns as revealed by food consumption surveys (“total diet method”)(De Mul et al., 2005). Alternatively, exposure to chemical contaminants may be *directly* assessed by means of the so-called 24-hour duplicate diet method: all food items consumed by an individual over 24 hours are collected and mixed into one composite 24-hour sample. In the Netherlands, duplicate diets have been collected in 1978, 1984, 1994 and 2004. This series is well suited to:

- estimate the exposure to PBDEs from actually consumed food anno 2004 and to compare this with corresponding exposure as determined with the “total diet method” anno 2003/2004 (Bakker et al., 2008; De Mul et al., 2005; De Winter-Sorkina et al., 2006).

- a time trend analysis of PBDE-exposure via the 24-hour duplicate diet method over the period 1978 – 2004 and to compare this trend in related matrices (such as breast milk) over time.

This report describes the occurrence of PBDEs in 24-hour duplicate diets which have been collected in 1978, 1984, 1994 and 2004 and its corresponding human exposure. Both occurrence and exposure were analyzed on their time-trend characteristics. In the case of PBDE-99, this exposure is compared with the maximum allowed intake level for this congener as calculated by the RIVM.

Unfortunately, no comparable data on the time trend of PBDEs in food are available, thereby preventing a direct comparison of the data presented in this report with data from other sources. However, here, data in breast milk may be a substitute for food data. The reason for this is that PBDEs, being persistent contaminants, tend to accumulate in the body. The accumulated amount in the body ("body burden"), rather than the daily exposure, determines the toxic risk. The amount in the body, or entities arising from it such as breast milk, thus reflects the long-term history of exposure.

In the case of PBDEs two routes of exposure are of practical importance: food and house dust (Wilford et al., 2005). When food would be the dominant route, the time trend of PBDEs in food would reflect the time trend of breast milk. In order to evaluate whether breast milk data of PBDEs reflect those in food, this report evaluates food and house-dust as routes of PBDE exposure in the Netherlands. Finally, the time trend of PBDEs as revealed in Dutch 24-hour duplicate diets is compared with the time trend of these compounds in, mainly Swedish, breast milk.

2 PBDEs in 24-hour duplicate diets

2.1 Materials and Methods

2.1.1 Sample collection

Duplicate diet samples in 1978 were collected from 101 RIVM employees aged between 18 and 65 years. In 1984, 1994 and 2004, the collection of food focused on representative sampling from the Dutch population. In these years, 123 participants (18 and 74 years) collected a duplicate diet of a complete day (24 hours). The homogenized wet material was freeze-dried and the dried product obtained re-homogenized and stored under refrigeration until analysis. For analysis of PBDEs, food samples from 10 (1978, 1984, 1994) or 35 participants (2004) were randomly selected. From the concentrations measured in the duplicate diets the 24-hour intake of PBDE-47 and PBDE-99 was calculated by multiplying the concentrations with the amount of food consumed, followed by division by body-weight.

2.1.2 Analytical method

The analytical method used is described in detail in Schothorst et al. (in preparation). Briefly, duplicate diet samples, corresponding with 200 mg fat, together with the internal standard, is mixed with 25 ml acetone. The mixture is filtered and the acetone extract is collected, evaporated and re-dissolved in 8 ml methanol. The test portion is saponified with 1 ml saturated KOH in water. Afterwards, 25 ml iso-octane is added and the solution is centrifuged. The extract is cleaned, transferred in an autosampler vial and evaporated. The residue is re-dissolved in 0.1 ml iso-octane. The final extract was analysed for PBDEs by GC-MS with negative chemical ionization.
As (certified) reference materials or analytical quality control samples for PBDEs in duplicate diet samples are lacking, in house validation experiments established the performance characteristics of the method. The following parameters were investigated: the limit of detection, the limit of quantification, the recovery and the within laboratory reproducibility. Also an estimate of the uncertainty of measurement was made. All in-house validation experiments were carried out with different duplicate diet samples. The limit of detection is defined as 3 times and the limit of quantification as 9 times the noise in the time windows for the PBDEs. Recovery experiments were done by adding standards of the particular PBDE to a duplicate diet sample. The level at which standards were added to the test portion was 50 ng/kg. Experiments were carried out on five different days. The reproducibility was determined by analysing duplicate diet samples on different days. The estimate of the uncertainty of measurement is based on the within laboratory reproducibility. A coverage factor of 2 is used.

2.1.3 PBDEs analysed

The following PBDEs were analysed:

- 2,2',4-triBDE (PBDE17)
- 2,4,4'-triBDE (PBDE28)
- 2,2',4,4'-tetraBDE (PBDE47)
- 2,3',4,4'-tetraBDE (PBDE66)
- 2,2',3,4,4'-pentaBDE (PBDE85)
- 2,2',4,4',5-pentaBDE (PBDE99)
- 2,2',4,4',6-pentaBDE (PBDEl00)
- 2,2',3,4,4',5'-hexaBDE (PBDE138)
- 2,2',4,4',5,5'-hexaBDE (PBDE153)
- 2,2',4,4',5,6'-hexaBDE (PBDE154)
- 2,2',3,4,4',5',6-heptaBDE (PBDE183)
- 2,2',3,3',4,4',5,5',6,6'-decaBDE (PBDE209)

2.1.4 Time trend modeling

In order to present a time trend of PBDE concentration in 24-hour duplicate diets, time trend modeling is used. Time trend modeling and model selection is described in detail in Slob (2002). In short, a family of five nested time trend models is used to describe a data set. Mutually the models differ in complexity in that they contain a different number of unknown parameters which have to be estimated from the data. The reason for having different model complexity is that more complex data sets need more complex models. The extension of a model is analyzed by means of the likelihood ratio test.

2.2 PBDE exposure from 24-hr duplicate diets anno 2004

The analysis showed that PBDE-47 and PBDE-99 could be quantified in almost all of the 65 analyzed duplicate diets. PBDE-209 could be quantified in the samples of 2004 only, due to a high variable background levels. Only in rare cases the other PBDEs could be quantified. For this reason intake calculations were restricted to PBDE-47, PBDE-99 and PBDE-209.

In total, 35 duplicate diet samples collected in 2004 were analyzed. Though two slightly different analytical methods (without and with lipid extraction as first step of the clean-up) were used, the results of the separate analyses did not indicate a systematic difference (for details, see Appendix 1). Figures 1, 2 and 3 show the corresponding 24-hour intake distributions of PBDE-47, PBDE-99 and PBDE-209, respectively.

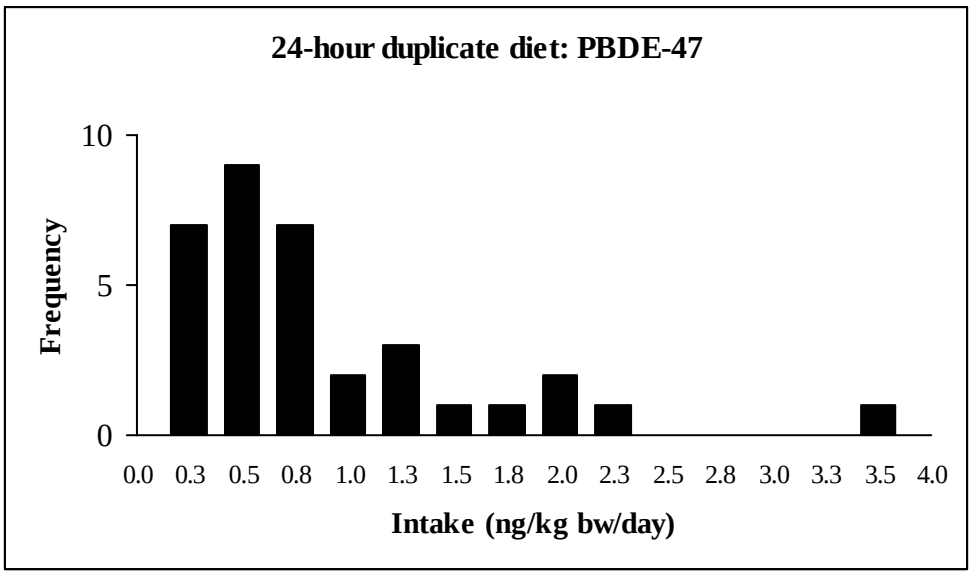

Figure 1. The intake distribution of PBDE-47 as determined in 24-hour duplicate diets anno 2004 (mean: 0.77; SD: 0.72; median: 0.54 ng/kg bw/day; N = 35)

The results of PBDE-47 agreed with the intake distribution based on the "total diet method" anno 2003/2004, in which the median life-long exposure was 0.4 ng/kg bw/day and the 97.5th percentile 1.1 ng/kg bw/day (De Winter-Sorkina et al., 2006, see also Appendix 2).

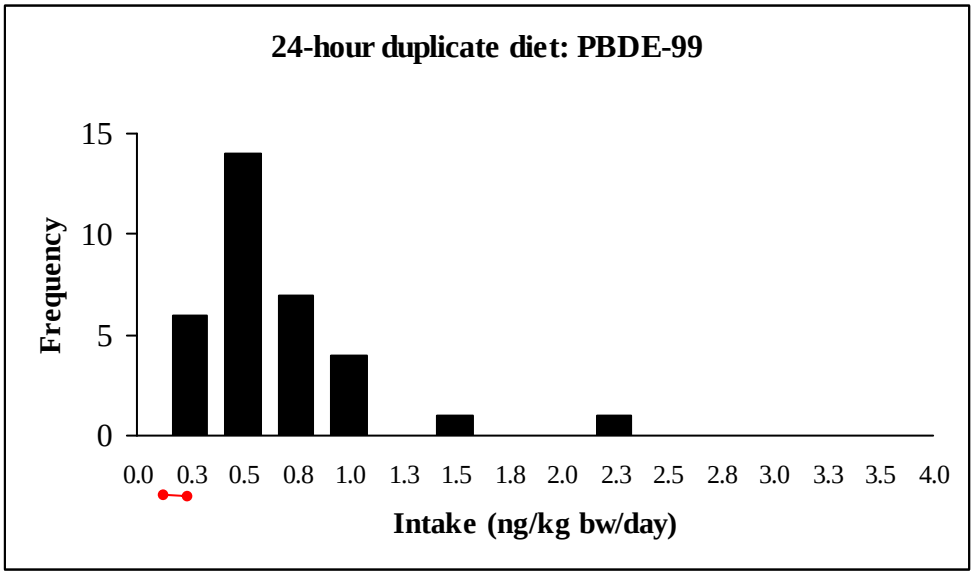

Figure 2. The intake distribution of PBDE-99 as determined in 24-hour duplicate diets anno 2004 (mean: 0.50; SD: 0.39; median: 0.41 ng/kg bw/day; N=35). ⊷ represents the maximum allowable intake level of 0.23-0.30 ng PBDE-99/kg bw/day.

As presented by de De Winter-Sorkina et al. (2006) and Bakker et al. (2008) a risk assessment can only be performed for PBDE-99. For this PBDE, RIVM calculated (a range) for the maximum allowed chronic human intake level of 0.23-0.30 ng PBDE-99/kg bw/day (the range mainly reflects uncertainties in the half-life of PBDE-99 in humans).

When compared with the exposure as determined in 2004 duplicate diets (see Figure 2) it appears that the majority of the samples exceeds the set intake level, with the maximum exposure exceeding the set intake level by a factor of 7 to 9.

The results of PBDE-99 are about 5-times higher than the intake distribution based on the "total diet method" anno 2003/2004, in which the median life-long exposure was 0.08 – 0.11 ng/kg bw/day and the 97.5th percentile 0.17 – 0.21 ng/kg bw/day (De Winter-Sorkina et al., 2006, see also Appendix 2).

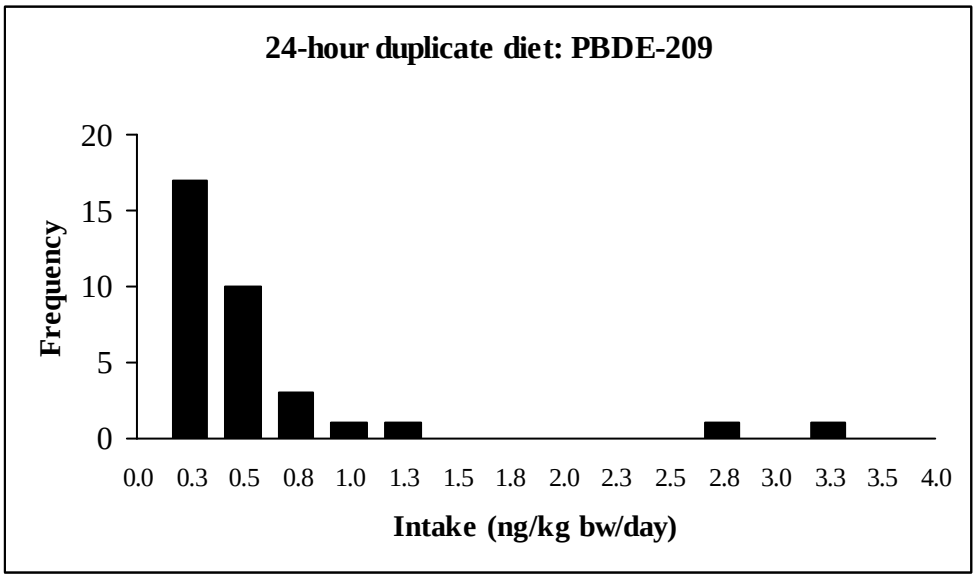

Figure 3. The intake distribution of PBDE-209 as determined in 24-hour duplicate diets anno 2004 (mean: 0.48; SD: 0.68; median: 0.26 ng/kg bw/day; N = 35)

In the 24-hour duplicate diets of 2004, three PBDEs are detected: PBDE-47, 99 and 209. The predominant PBDEs in the environment and human tissue are PBDE congeners 47, 99, 100, 153, 154, 183 and 209 (Darnerud et al., 2001; Hites, 2004). Many studies performed in Europe have reported the presence of PBDE congeners 47, 99, 100, 153 and 154 in food (Bakker et al., 2008; Darnerud et al., 2001; Harrad et al., 2004; Kiviranta et al., 2004; Lind et al., 2002). In a Canadian study, PBDE-28 was also included in the intake via food, although the contribution of PBDE-28 to the sum of the congeners was very small (Ryan and Patry, 2001). A British diet study reported dietary intakes of 17 individual PBDEs. PBDE-209 was the most abundant PBDE congener, followed by PBDE-47. These and PBDEs 49, 66, 99, 100, 153 and 183 were detected in most of the

food groups (Food Standards Agency, 2006). Schecter et al. (2006) reported concentrations of 13 PBDE congeners in food products purchased in the USA. The reported concentrations of the PBDE congeners in the USA are generally similar to those in the Netherlands, which is in agreement with the results of Lorber, who reported that levels in food in the USA are comparable to those in Europe. The difference in body burden between Europe and the USA may be explained by differences in house dust ingestion, as concentrations in indoor dust are higher in the USA than in Europe (De Boer et al., 2007; Ibarra et al., 2006; Pless-Mulloli et al., 2006; Knoth et al., 2003; Santillo et al., 2003; Stapleton et al., 2005; Harrad et al., 2008; Wilford et al., 2005).

2.3 PBDE exposure from 24-hr duplicate diets 1978 - 2004

Duplicate diets of adults were collected in 1978, 1984, 1994 and 2004 and analyzed on their concentrations/exposure of PBDE-47 and PBDE-99 (see Tables 1 and 2 for exposure).

Table 1. Summary of the exposure to PBDE-47 (ng/kg-bw) as determined in 24-hour duplicate diets anno 1978, 1984, 1994 and 2004.

Year	Median	Mean	Standard deviation	Number of samples/ Number of samples above detection limit
1978	0.62	0.57	0.29	10/9
1984	0.03	0.08	0.10	10/2
1994	0.21	0.14	0.07	10/2
2004	0.54	0.77	0.72	35/31

Table 2. Summary of the exposure to PBDE-99 (ng/kg-bw) as determined in 24-hour duplicate diets anno 1978, 1984, 1994 and 2004.

Y ear	Median	Mean	Standard deviation	Number of samples/ Number of samples above detection limit
1978	0.03	0.12	0.22	10/2
1984	0.34	0.30	0.26	10/6
1994	0.49	0.61	0.52	10/9
2004	0.41	0.50	0.39	35/33

Concentrations and intakes were analyzed on their time-trend characteristics in the following way.

As persistent chemicals, like PBDEs, have entered the food chain from spreading in the environment their time trend in food is believed to follow that in the environment. Starting from a background level the latter consists of steep increasing levels in environmental matrices such as soil, water and air (Phase 1: reflecting increased environmental emissions

leading to an increase in environmental levels) followed by a slowing down of the rate of increase until a maximum level is reached (Phase 2: reflecting the decrease in environmental emissions due to restrictive use or phasing out of the contaminant). Thereafter environmental levels may steadily decrease until the background level is reached again (Phase 3: reflecting the removal of the contaminant from the environment due to chemical and/or biological turn-over).

A full environmental time trend cycle of persistent chemicals may span decades rather than years and its full-scale monitoring needs a long sample collection period (see figure 9 for an example of the full-scale monitoring of PBDEs in breast milk, which spans over three decades). Clearly, such full-scale monitoring is merely exception than rule. In practice, monitoring is often limited to either one of the phases mentioned above. In this the monitoring of Phases 1 or 3 requires less demanding data, i.e. only increasing or decreasing data, when compared with the monitoring of Phase 2, which needs data on the reversal of an increasing into a decreasing trend. Furthermore, as the monitoring of dioxin like contaminants in food have shown, (a minimum) of 4 time-points suffices to characterise an increasing/decreasing time trend (De Mul et al., 2008). The detection of a time trend is facilitated when (even) background levels and trend-data are well above the detection limits of the applied analytical-chemical methods.

In conclusion, not counting exceptions, the full-scale monitoring of PBDEs in food is virtually impossible. However, part of such modelling, i.e. the detection of an increasing/decreasing trend, is possible. In this case the background level of the contaminant should lie significantly above its analytical detection limit. Actual trend data should lie well above the analytical detection limit. Furthermore at least four time points should be available.

Given these criteria most of the 1984 – 2004 data of PBDE-99 are well above the analytical detection limit (1984 6/10; 1994 9/10; 2004 33/35). In contrast only 2 out the 10 1978 measurements apply to this criterion. As shown in Figure 4A these observations are compatible with a steady increase of the (mean) concentrations of PBDE-99 in food from 1978 to 1994, after which it appears to level off. This is emphasized by the trend-line through these data[1]. Continuous monitoring beyond 2004 will be useful to confirm this levelling off. Similar conclusions can be drawn regarding the 24-hour intake data of PBDE-99 (Figure 4B).
Clearly the relative high number of measurements below the analytical detection limit in the 1978 data is a major source of uncertainty in the analysis. This uncertainty can only be reduced by analyzing more of these 1978 samples.

[1] Various models were assessed (Slob, 2002) to obtain the solid line, which represents the time trend of the PBDEs in food over the years. In this report the most appropriate model is used to describe each time trend.

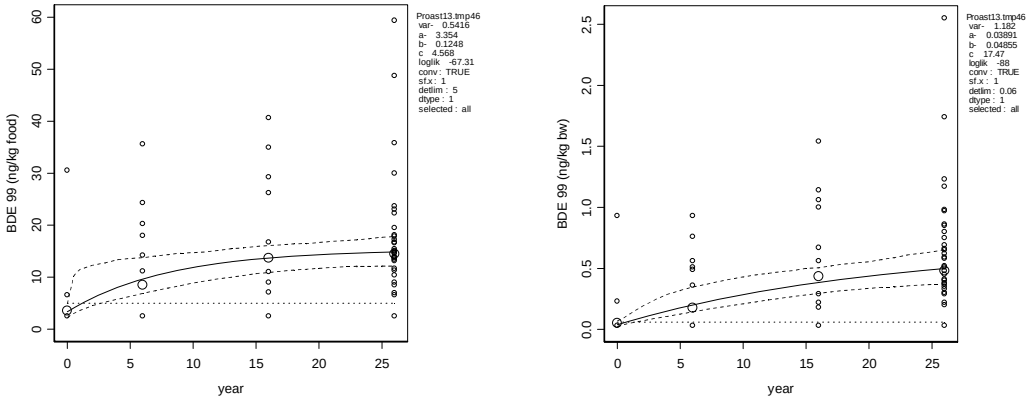

Figure 4. Left panel: Concentration of PBDE-99 (solid line) in food plotted against the year of sampling. Right panel: 24-hour intake of PBDE-99 (solid line) plotted against the year of sampling. The years 1978, 1984, 1994, and 2004 are transformed into 0, 6, 16, and 26 years respectively. The horizontal dotted line represents the detection limit[2]. The lower (5th percentile) and upper (95th percentile) confidence bounds of the trend line are represented by the curved, dotted lines.

In the case of PBDE-47, only a small part of the 1984 and 1994 measurements lie above the analytical detection limit: 1984 2/10, 1994 2/10, to be compared with 9/10 and 31/35 in the 1978 and 2004 measurements. As can be deducted from Table 1, these data are incompatible with either an increasing or decreasing time trend. The data merely suggest the absence of any time trend between 1978 and 2004. Again, this uncertainty can only be reduced by analyzing more of the 1984 and 1994 samples.

[2] The detection limit for the intake was calculated with the equation for the 24-hour intake where concentration in food equals the detection limit in food (5 ng/kg food), amount of food consumed equals the lowest amount consumed (1.186 kg), and body weight equals the highest body weight (117 kg). This results in detection limits of 0.05 ng/ kg bw for PBDE-99

3 PBDEs in house dust

3.1 Calculation of the combined exposure to PBDEs from food and household dust

Total daily uptake of PBDEs, i.e. the amount of PBDE that passes the intestine and thereby enters the body was calculated using the following equation:

$$\text{Daily uptake} = I_f * F_{abs,\,f} + I_d * F_{abs,\,d}$$

in which:

I_f = daily intake of PBDE in food (obtained from de Winter-Sorkina et al., 2006)
$F_{abs,\,f}$ = absorbed fraction PBDE in food (obtained from Geyer et al., 2004)
I_d = daily intake of PBDE in dust (see below)
$F_{abs,\,d}$ = absorbed fraction PBDE in dust (see below)

The daily intake of PBDE in dust was calculated with the formula:

$$I_d = C_d * A_d$$

in which:

C_d = concentration PBDE in dust (ng/g dust)
A_d = average daily intake of dust (adults: 50 mg/day; child: 100 mg/day, obtained from Oomen et al., 2008)

In converting the absolute PBDE intake I_d from dust to intake per kg body weight 70 kg was used for adults and 14 kg for 2-year old children.

Preliminary results of Hakk et al. (2007) showed that *in vivo* administered PBDEs in corn oil or house dust resulted in similar bioconcentration in adipose tissue, suggesting similar bioavailability from both matrices. Geyer et al. (2004) mention a fraction absorbed between 0.9 and 1.0 for PBDEs from food. This strongly suggests that the bioavailability of PBDEs from food and dust is high and almost identical. Hence, it seems reasonable to assume that the contribution of food and dust to the daily *uptake* of PBDEs is determined by the *intake* of PBDEs from these matrices.

3.2 Materials and Methods

3.2.1 Data collection of house dust

As the concentration of PBDEs in European house dust is much lower than in North-America (De Boer et al., 2007; Ibarra et al., 2006; Pless-Mulloli et al., 2006; Knoth et al., 2003; Santillo et al., 2003; Stapleton et al., 2005; Harrad et al., 2008; Wilford et al., 2005) and dust concentrations from various European countries show considerable overlap (data not shown) pooled PBDE values as reported in collected individual European household dust samples (Table 3) were used as the starting point for the calculation of the PBDE exposure from house dust. This collection also contains the (preliminary) results of the analysis of house dust from the Netherlands.

Table 3. Overview of the papers used for the calculation of the European exposure to PBDEs

Location	Number of samples	Reference
The Netherlands	12	De Boer et al., 2007
England	11	Ibarra et al., 2006
England	7	Pless-Mulloli et al., 2006
Germany	40	Knoth et al., 2003
England	10	Santillo et al., 2003

3.2.2 Statistical analysis of house dust data

Statistical analysis of the data was conducted using Excel (Microsoft Office XP) to calculate summarizing statistics (mean, SD, geometrical mean, P_{05}, median, P_{90}, P_{95}, $P_{97.5}$ and P_{99}) for PBDE-47, -99, -100, -183 and -209 with total sample sizes of n = 76, 72, 78, 69 and 68, respectively. Outliers above 1000 ng/g dust were removed for PBDE-47, -99 and -100. No log transformation was conducted on the data. Non-detect values for PBDE-47, -100 and -183, respectively n = 1, 3 and 5, were set at 0 and included in all the calculations except for the geometrical mean.

Both the exposures from dust and food were not calculated as single values, but merely as statistical distributions. Hence, their mutual comparison was made on statistical index numbers, i.e. percentiles, median and average values. Finally a comparison was made between the Dutch, European and North-American exposure via house dust (Harrad et al., 2008).

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3.2.3 Daily intake of PBDE congeners via dust

Table 4 presents the statistical index numbers of PBDE congeners in European household dust. Clearly, PBDE-209 is by far the dominant congener.

Table 4. Statistical index numbers of PBDE congeners in European dust (ng/g dry weight)

PBDE	47	99	100	183	209
5th percentile	4.2	3.5	0.76	0	80
50th percentile (median)	16	25	5	9.7	500
90th percentile	61	79	22	41	9160
95th percentile	86	147	65	60	18225
97.5th percentile	104	275	167	79	19631
99th percentile	264	332	249	208	31415
Geometrical mean	18	22.0	7.2	7.4	735
Mean	33	43	19	20	3554
Standard deviation	67	65	48	57	7953
Total number of samples	76	72	78	69	68

Tables 5 and 6 present the calculated daily intake of PBDE congeners from dust for adults and two-year-old children, respectively.

Table 5. Statistical index numbers of the daily intake of PBDE congeners via dust in adults (ng/kg bw/day)

PBDE	47	99	100	183	209
5th percentile	0.00	0.00	0.00	0.00	0.06
50th percentile (median)	0.01	0.02	0.00	0.01	0.36
90th percentile	0.04	0.06	0.02	0.03	6.54
95th percentile	0.06	0.10	0.05	0.04	13.02
97.5th percentile	0.07	0.20	0.12	0.06	14.02
99th percentile	0.19	0.24	0.18	0.15	22.44

Table 6. Statistical index numbers of the daily intake of PBDE congeners via dust in two-year-old children (ng/kg bw/day)

PBDE	47	99	100	183	209
5th percentile	0.03	0.02	0.01	0.00	0.57
50th percentile (median)	0.11	0.18	0.04	0.07	3.57
90th percentile	0.44	0.57	0.15	0.29	65.43
95th percentile	0.61	1.05	0.47	0.43	130.18
97.5th percentile	0.74	1.97	1.19	0.56	140.22
99th percentile	1.89	2.37	1.78	1.48	224.40

3.2.4 Intake from food

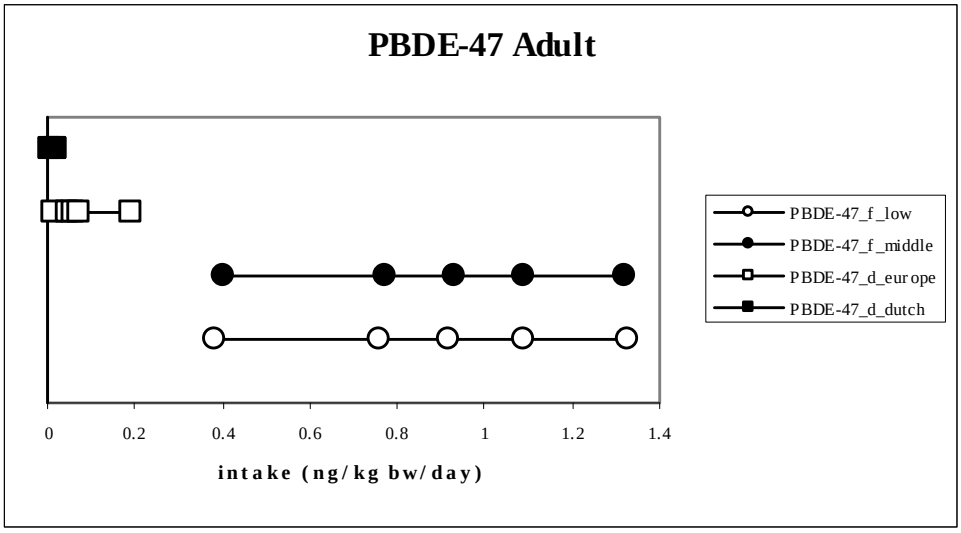
The calculated dust exposures were compared to the daily dietary intake of PBDE as assessed by means of the "total diet method". The latter was preferred over the 24-hour duplicate diets because of the availability of a thorough statistical analysis which results in a representative estimation of the exposure (distribution) of the whole Dutch population to PBDEs from food.
The calculation of the life-long dietary daily intake with the "total diet method" is complex and consists of a combination of measured concentrations of PBDEs in various food items and food consumption data. As PBDE levels in food are in general rather low, quite some food measurements may well be below the so-called analytical level of detection (LOD). To incorporate the uncertainty adhering to such levels in intake calculations their values are usually set at zero or at half the LOD. In this way a low estimate or a middle estimate is obtained (for details, see De Winter-Sorkina, 2006 and Appendix 2). Exposure calculations are presented for adults and two-year-old children.

3.3 PBDE exposure: food *versus* dust

As mentioned the exposures from dust and food were not calculated as single values, but as statistical distributions. Their mutual comparison was made on statistical index numbers, i.e. percentiles, median and average values. This comparison, which was made for adults and 2-year olds separately, is shown graphically in Figures 6 – 13.

3.3.1 Adults

As shown in Figure 5 the exposure of PBDE-47 and -183 from dust is substantially lower than that from food. Therefore, the contribution of dust to the total exposure of PBDE-47 and -183 is small compared to that from food.



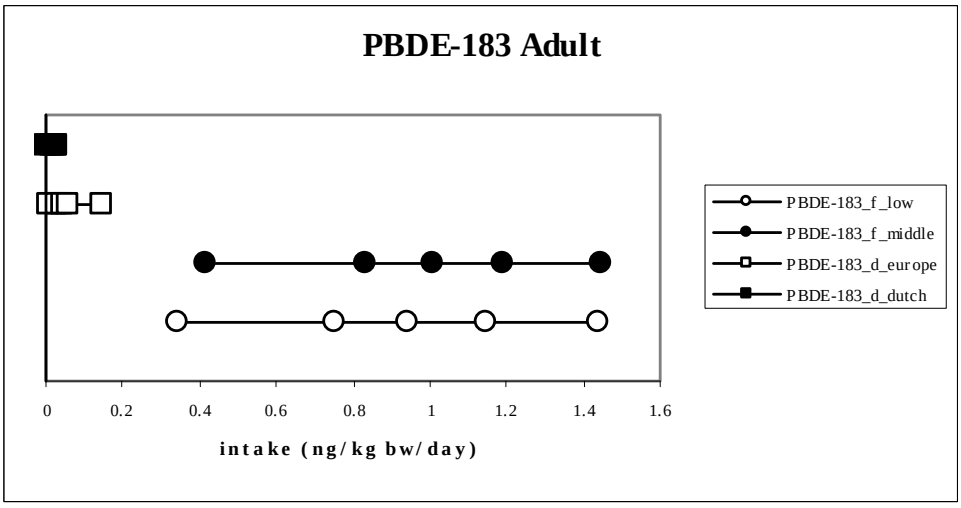


Figure 5. Comparison of the exposure of PBDE-47 (upper panel) and -183 (lower panel) from house dust and food in adults. Statistical index values shown (from left to right): 50th percentile (median), 90th, 95th, 97,5th and 99th percentiles. Dietary intake: low estimate (open circle), middle estimate (closed circle). Daily dust intake: based on pooled European data (open square); based on Dutch data (closed squares).

In contrast to PBDE-47 and -183, Figure 6 shows that the exposure to PBDE-99 and -100 from dust shows significant overlap with the exposure from food (note that, when only Dutch data were used, such overlap was not found). Therefore, a substantial contribution of dust to the PBDE exposure cannot be excluded.

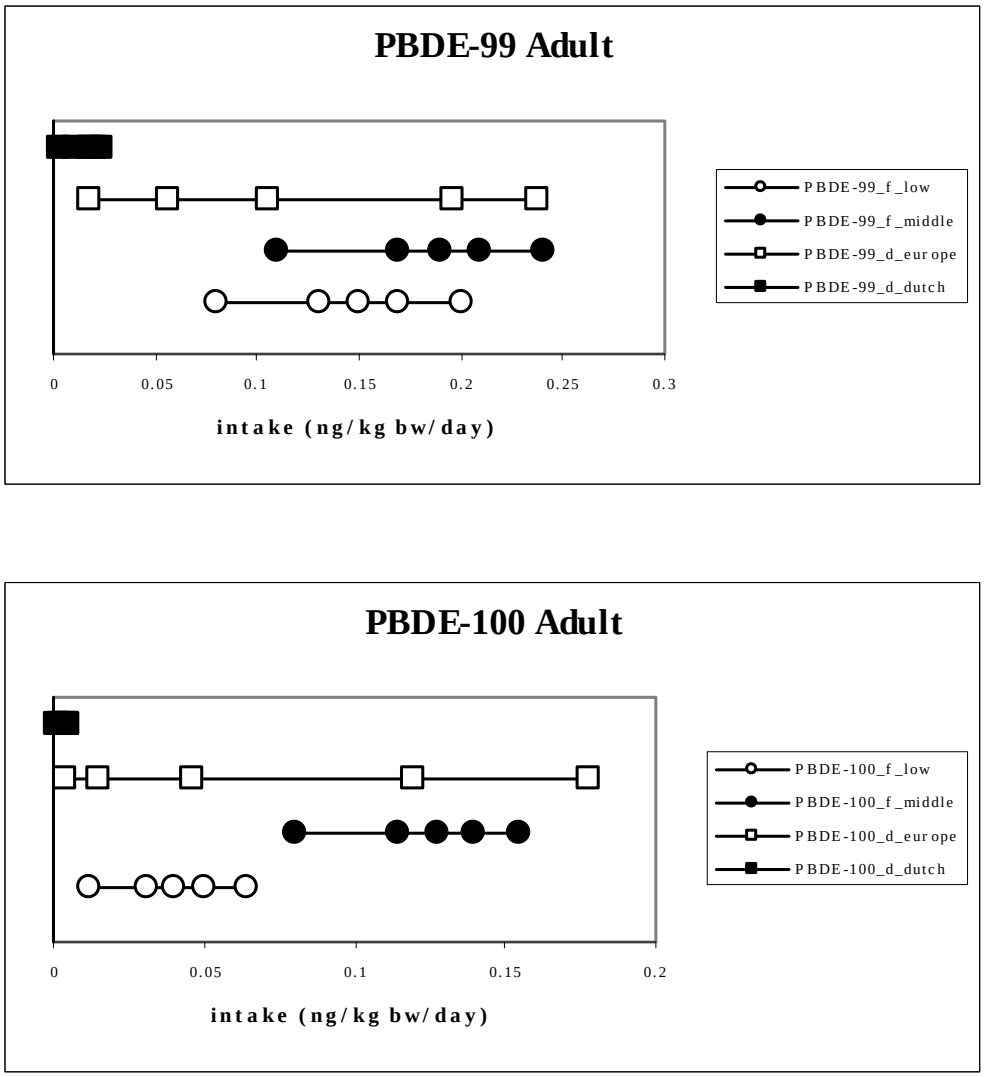
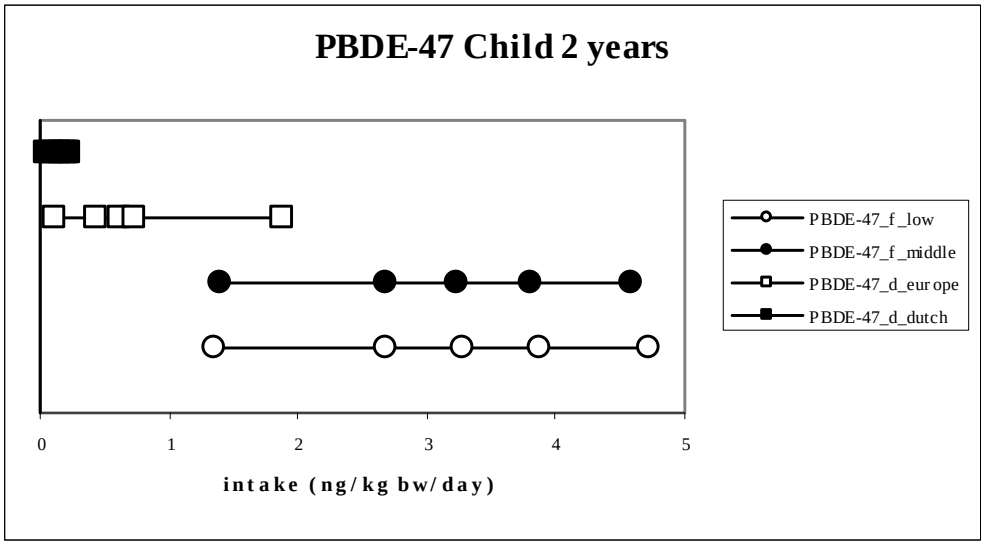


Figure 6. Comparison of the exposure of PBDE-99 (upper panel) and -100 (lower panel) from house dust and food in adults. Statistical index values shown (from left to right): 50th percentile (median), 90th, 95th, 97,5th and 99th percentiles. Dietary intake: low estimate (open circle), middle estimate (closed circle). Daily dust intake: based on pooled European data (open square); based on Dutch data (closed squares).

3.3.2 Children

As in adults the exposure of PBDE-47 and -183 from dust in children is substantially lower than that from food (see Figure 7).



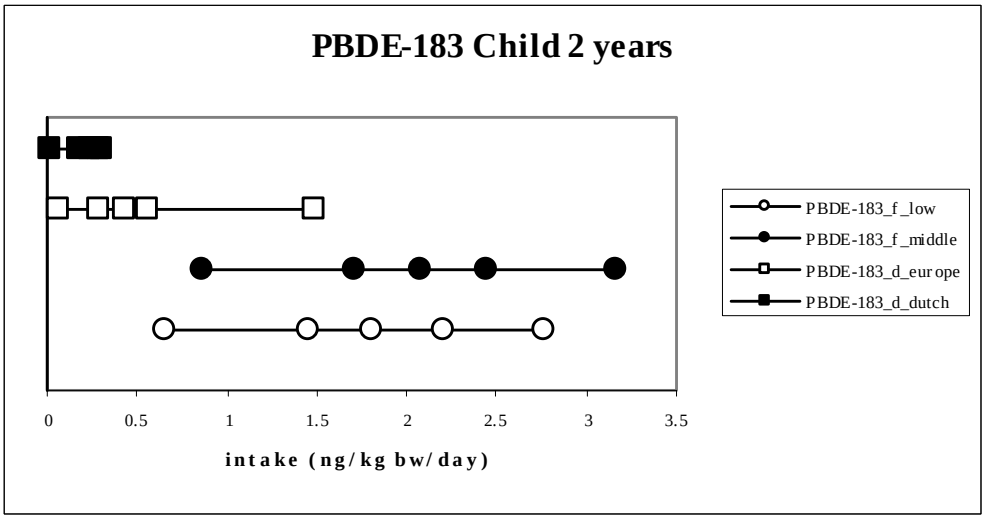
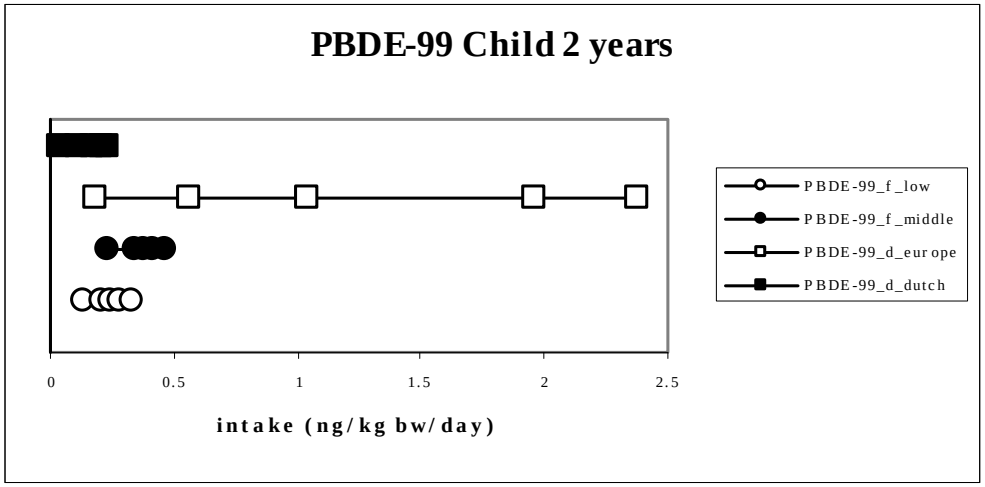


Figure 7. Comparison of the exposure of PBDE-47 (upper panel) and -183 (lower panel) from house dust and food in 2-year olds. Statistical index values shown (from left to right): 50th percentile (median), 90th, 95th, 97,5th and 99th percentiles. Dietary intake: low estimate (open circle), middle estimate (closed circle). Daily dust intake: based on pooled European data (open square); based on Dutch data (closed squares).

Figure 8 shows that the exposure to PBDE-99 and -100 from dust is in the same order or even greater than the exposure from food. Clearly, the exposure of children to PBDE-99 and -100 from dust may account for at least half of their total PBDE exposure.



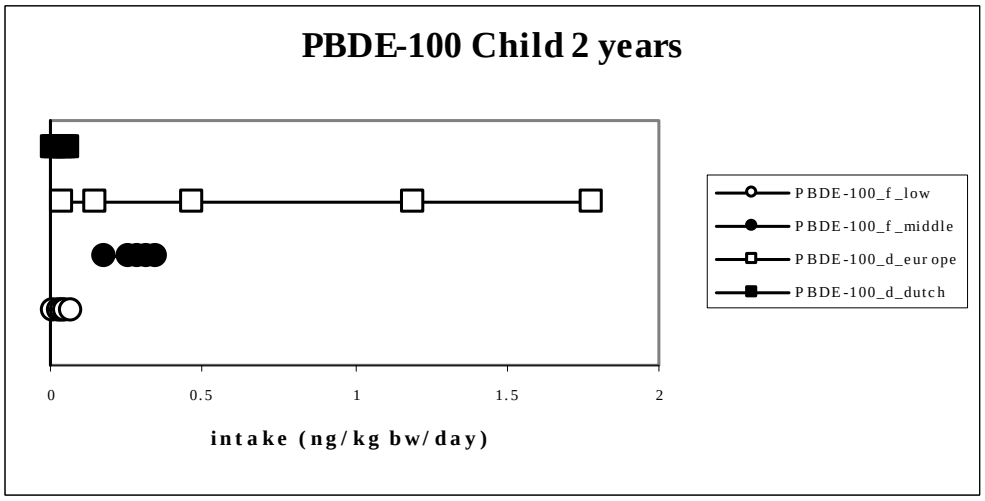


Figure 8. Comparison of the exposure of PBDE-99 (upper panel) and -100 (lower panel) from house dust and food in 2-year olds. Statistical index values shown (from left to right): 50th percentile (median), 90th, 95th, 97,5th and 99th percentiles. Dietary intake: low estimate (open circle), middle estimate (closed circle). Daily dust intake: based on pooled European data (open square); based on Dutch data (closed squares).

4 Time trend of PBDE-47 in breast milk

Literature data on the time-trend of PBDEs in food are not available. In the present study the time-trend of PBDEs in breast milk of, mainly Swedish, women was taken as a substitute for the time-trend of PBDE exposure from food. The reason for taking breast milk for this purpose lies in food being the predominant long-term factor in determining the uptake of PBDEs in the body (see Chapter 3) and, consequently, their excretion in breast milk. The time-trend in breast milk is therefore expected to closely follow the time-trend in food.

4.1 Materials and Methods

European time-trend data of PBDEs have been reported for European serum/blood (Thomsen et al., 2002, 2003; Schröter-Kermani et al., 2000) and European breast milk (Thomsen et al., 2003; Fängström et al., 2005, 2008; Lind et al., 2003; Meironyté et al., 1999, 2003; De Winter-Sorkina et al., 2006). In addition European breast milk data on a single time point are available (Ingelido et al., 2007; Pirard et al., 2003; Kalantzi et al., 2004; Weber et al., 2004; Fürst, 2001; Vieth et al., 2004)(see Appendix 3 for the characteristics of the individual studies).
Though the breast milk data are dominated by data from Sweden, all studies indicate PBDE-47 to be the dominant PBDE component in breast milk. PBDE-47 was therefore chosen as a reference for PBDEs in breast milk.

4.2 Results of the time trend data in breast milk

Figure 9 presents the long term time trend of PBDE-47 in breast milk as compiled from thirteen independent European studies. Though dominated by data from Sweden, data from other European countries fit in well. The overall picture shows, after an increase in the period 1972 until late 90ties, a decrease in the period late 90ties to 2004. The Dutch values, however, tend to follow a different pattern. The three measurements suggest an increase from 1990 until 2005.

The data furthermore show that the monitoring of the time trend needs rather frequent sampling frequency of breast milk samples, i.e. a frequency of every other year. Clearly the time trend shown would not have been detected with a monitoring frequency of once per decade.

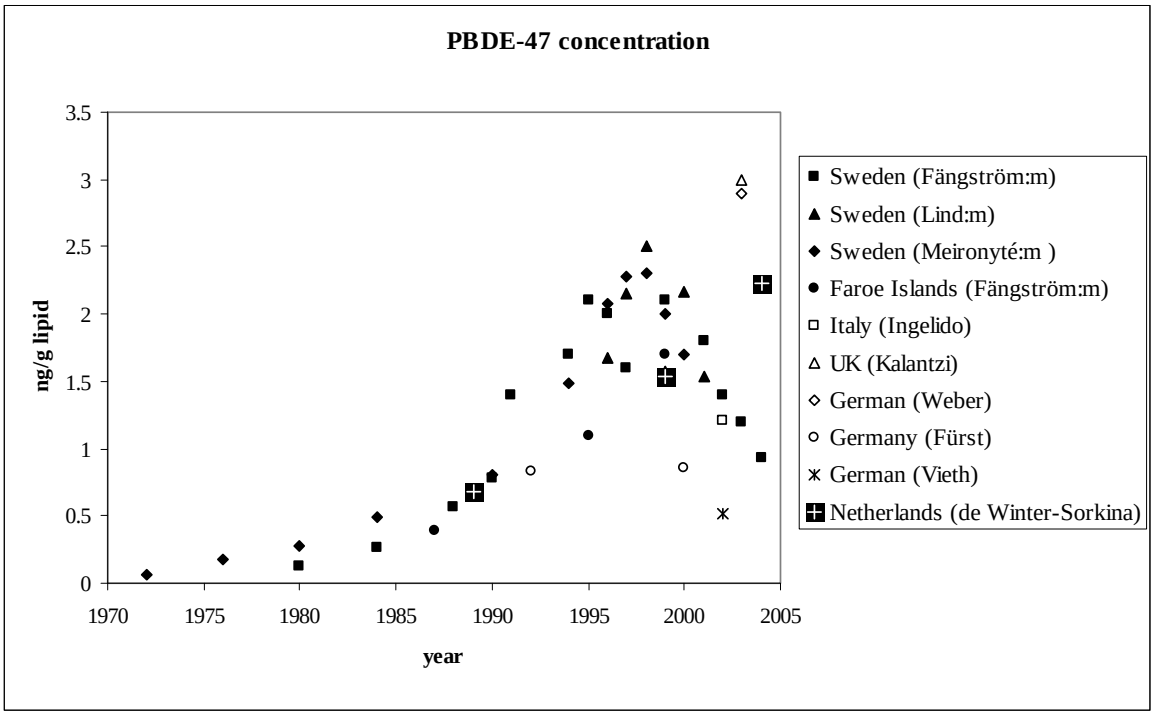


Figure 9. The concentration of PBDE-47 (ng/g lipid) in European breast milk (mainly Swedish data) versus time (years) and the Netherlands (solid box with cross), complemented by the individual time points of German (open diamond, open circle and astrix), Italian (open square) and UK (open triangle) studies.

4.3 Time trend analysis: comparison with 24-hour duplicate diets

Levels of PBDE-47 in European breast milk clearly increased in the period between 1990 and 2000, after which a decline occurred. European data show a peak level of PBDE-47 in 10 years (1990-2000). No clear time-trend of PBDEs was observed in 24-hour duplicate diets. This can be due to the fact that samples are collected once every 10 year and an increase can be missed in this way. In conclusion, the absence of a time-trend of PBDEs as revealed in 24-hour duplicate diets is not comparable with the clear time-trend of PBDE-47 in European breast milk.

5 Conclusions and recommendations

- PBDE-47 and PBDE-99 proved to be the dominant PBDE-congeners in 24-hour duplicate diets. Additionally, PBDE-209 was found.

- In 1974 – 2004 food levels of PBDE-47 did not show a clear time pattern. However, food levels of PBDE-99 showed a consistent, increasing pattern from 1978 to 1994, which seems to level off after 1994.

- In general, the exposure as determined in 24-hour duplicate diets anno 2004 was in the same order for PBDE-47 and 5-fold higher for PBDE-99 than the corresponding exposure as determined by the "total diet method" anno 2003/2004.

- Whether assessed by the "total diet method" or in 24-hour duplicate diets, the human exposure to PBDE-99 lies around the maximum allowed intake level. The potential risk due to exposure to PBDE-99 indicates that caution should be exercised with other PBDEs. Therefore, additional toxicity studies which are suited for the derivation of maximum allowed intake levels should become available for other PBDEs as well.

- Food and house dust are the main routes of exposure to PBDEs. Food showed to be the dominant route of exposure for adults (determined for PBDE-47, -99, -100 and -183). In case of (two-year-old) children the exposure of PBDE-99 and -100 via dust is in the same order or even higher than the exposure from food. For PBDE-47 and -183, the exposure via house dust was lower than dietary exposure.

- The observed time-trend in 24-hour duplicate diets is not comparable with that in European breast milk, the former showing no trend, whereas PBDE levels in breast milk show a peak level around 1998.

- The time-trend of PBDEs in breast milk shows that monitoring should be fine-meshed, i.e. to be once every one or two years, in order to be detected. As levels in breast milk are expected to follow those in food closely, this conclusion holds for the monitoring of levels in food too.

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rivm

Appendix 1. RIVM measurements in 24-hour duplicate diets

Measurements 2004

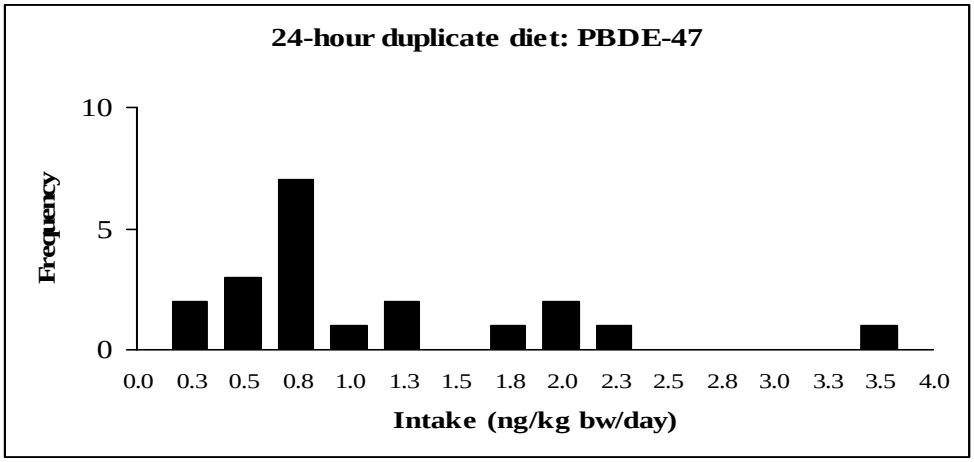
As the 2004 series consists of two independent series of measurements which were performed with slightly different analytical chemical methods both series are presented separately together with their accompanying frequency distribution.

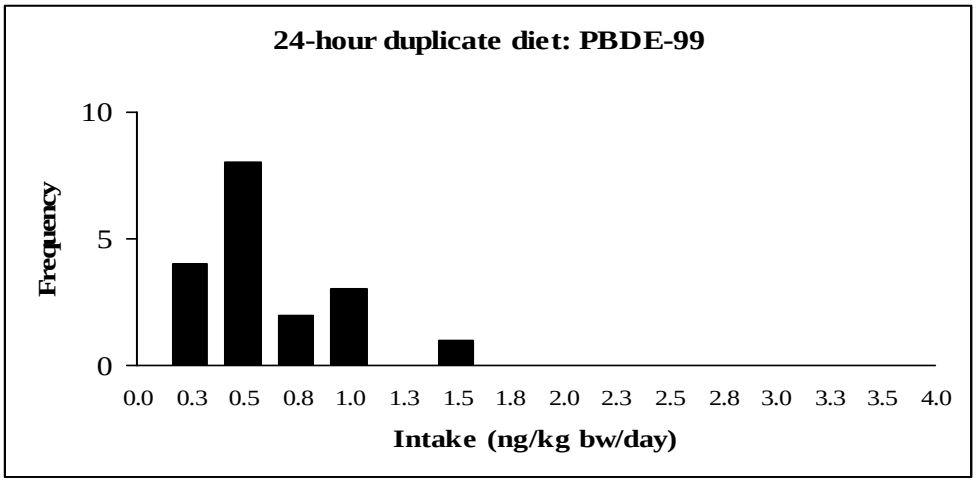
Series 1. The exposure to PBDE-47, PBDE-99 and PBDE-209 as determined with 24-hour duplicate diets (20 samples, ARO SOP 495 (clean-up without lipid extraction)).

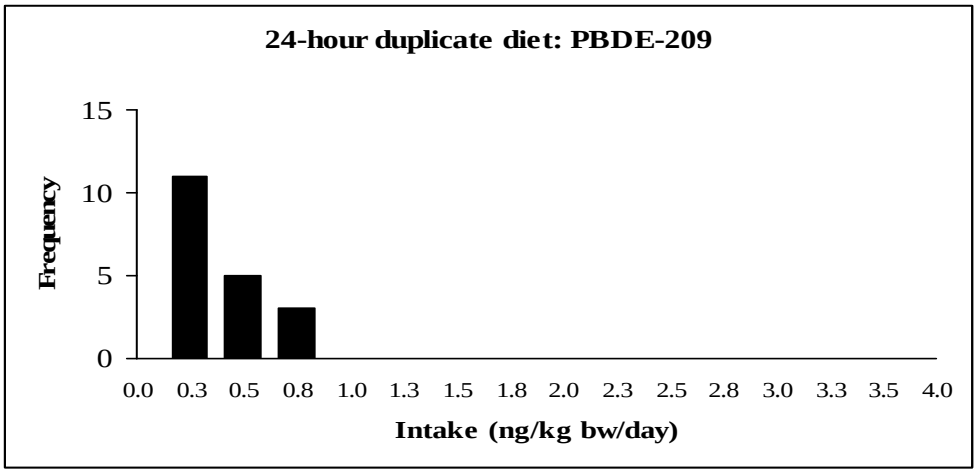
	Intake (ng/day)					Intake (ng/kg bw/day)			Concentration (ng/kg food, wet weight)			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
2004M0709	50	33	8	< LOD	92	0.54	0.36	0.09	21.6	14.3	3.6	< LOD
2004M0710	39	< LOD	18	< LOD	69	0.57	-	0.26	10.6	< LOD	4.8	< LOD
2004M0711	20	24	28	< LOD	80	0.25	0.30	0.35	9.6	11.6	13.3	< LOD
2004M0715	59	36	26	< LOD	80	0.74	0.46	0.32	14.9	9.2	6.5	< LOD
2004M0758	151	66	63	53	85	1.78	0.77	0.75	43.2	18.7	18.1	15.1
2004M0759	66	38	35	< LOD	94	0.70	0.41	0.38	19.8	11.5	10.7	< LOD
2004M0771	41	22	22	< LOD	94	0.43	0.24	0.23	13	7.1	7	< LOD
2004M0772	28	21	< LOD	< LOD	95	0.30	0.23	-	14.4	11	< LOD	< LOD
2004M0773	152	70	18	< LOD	72	2.11	0.97	0.25	25.7	11.8	3	< LOD
2004M2419	58	43	53	< LOD	108	0.54	0.40	0.49	14.7	11	13.4	< LOD
2004M2424	66	< LOD	46	< LOD	84	0.78	-	0.55	14.1	< LOD	9.9	< LOD
2004M2426	63	34	12	15	53	1.18	0.64	0.23	20.3	10.9	3.9	4.9
2004M2429	122	49	42	12	63	1.93	0.78	0.67	46.5	18.7	16.1	4.6
2004M2434	191	76	8	53	55	3.46	1.38	0.15	96.8	38.5	4.1	26.9
2004M2440	38	28	9	58	60	0.63	0.47	0.14	16	11.9	3.7	24.7

2004M2443	103	59	20	18	100	1.03	0.59	0.20	26.8	15.4	5.1	4.7
Intake (ng/day)						**Intake (ng/kg bw/day)**			**Concentration (ng/kg food, wet weight)**			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
2004M2443	103	59	20	18	100	1.03	0.59	0.20	26.8	15.4	5.1	4.7
2004M2458	33	14	7	14	82	0.40	0.18	0.09	11.8	5.2	2.6	5.0
2004M2474	22	14	15	14	92	0.24	0.16	0.17	8.4	5.5	5.9	5.3
2004M2480	51	29	10	< LOD	92	0.55	0.32	0.11	18	10.4	3.5	< LOD
2004M2486	154	48	24	51	98	1.57	0.49	0.25	42.4	13.2	6.7	14.0
					Mean	*0.99*	*0.46*	*0.28*				
					SD	*0.82*	*0.33*	*0.20*				
					Median	*0.66*	*0.40*	*0.24*				

Accompanying histograms of the 24-hour duplicate diets of PBDE-47, PBDE-99 and PBDE-209 (20 samples):



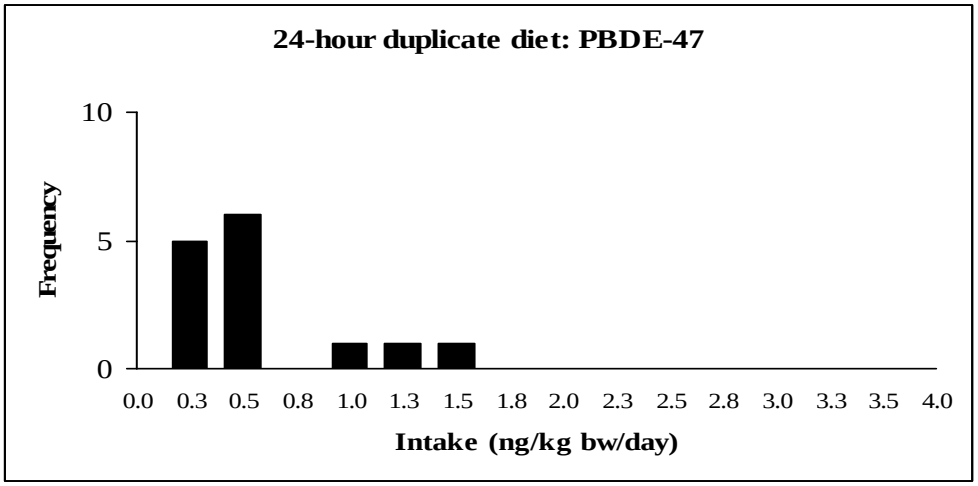


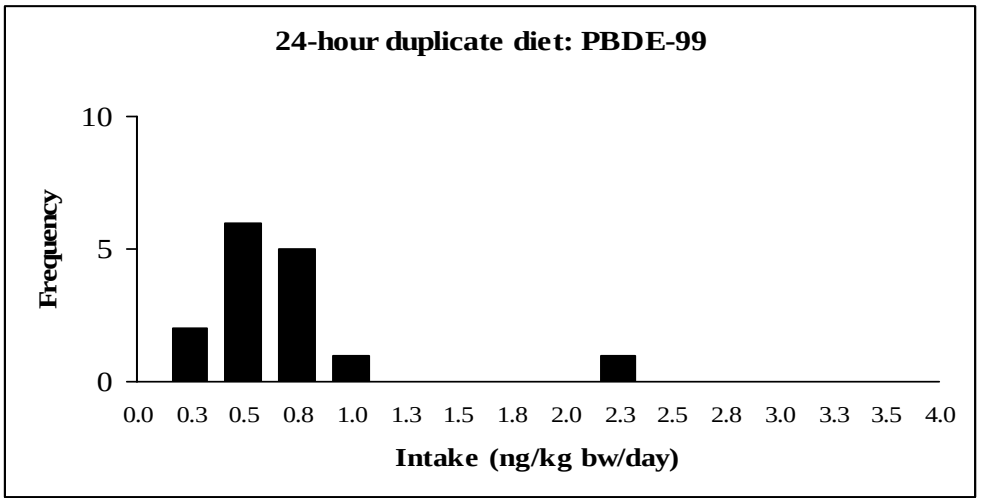


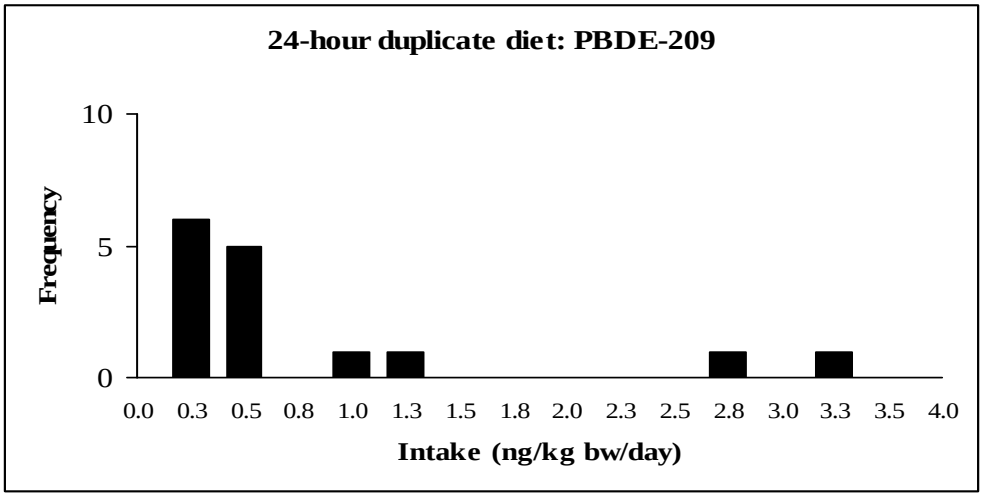
Series 2. The exposure to PBDE-47, PBDE-99 and PBDE-209 as determined with 24-hour duplicate diets (15 samples, ARO SOP 495 (revised: clean-up including lipid extraction)).

	Intake (ng/day)					Intake (ng/kg bw/day)			Concentration (ng/kg food, wet weight)			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
2004M0703	75	49	108	10	90	0.83	0.54	1.20	36.2	23.7	52.1	4.8
2004M0734	62	123	12	14	61	1.02	2.02	0.20	23.7	46.9	4.6	5.3
2004M0736	34	37	15	< LOD	71	0.48	0.52	0.21	12.1	13.1	5.3	< LOD
2004M0742	5	38	29	< LOD	78	0.06	0.49	0.37	1.4	10.6	8.1	< LOD
2004M0744	15	33	26	< LOD	65	0.23	0.51	0.40	5.5	12.2	9.6	< LOD
2004M0751	99	49	24	13	72	1.38	0.68	0.33	35.6	17.6	8.6	4.7
2004M0761	29	85	72	10	92	0.32	0.92	0.78	9.6	28.3	24.0	3.3
2004M0770	19	23	24	< LOD	88	0.22	0.26	0.27	6.8	8.2	8.6	< LOD
2004M2422	1	19	< LOD	< LOD	68	0.01	0.28	2.74	0.4	6.7	65.5	< LOD
2004M2425	< LOD	24	11	< LOD	97	-	0.25	0.11	< LOD	8.2	3.7	< LOD
2004M2437	37	28	236	< LOD	73	0.48	0.38	3.23	11.9	8.9	75.6	< LOD
2004M2444	31	43	26	11	64	0.48	0.67	0.41	9.8	13.6	8.2	3.5
2004M2463	43	30	12	< LOD	92	0.47	0.33	0.13	15.5	10.8	4.3	< LOD
2004M2472	6	24	16	< LOD	81	0.07	0.30	0.20	3.5	14.1	9.4	< LOD
2004M2487	32	34	19	< LOD	117	0.27	0.19	0.16	16.9	18.2	10.2	< LOD
					Mean	*0.45*	*0.56*	*0.72*				
					SD	*0.39*	*0.46*	*0.33*				
					Median	*0.39*	*0.49*	*0.97*				

Accompanying histograms of the 24-hour duplicate diets of PBDE-47, PBDE-99 and PBDE-209 (15 samples):







Measurements 1994

The exposure to PBDE-47 and PBDE-99 as determined with 24-hour duplicate diets (10 samples, ARO SOP 495 (revised: clean-up including lipid extraction)).

	Intake (ng/day)					Intake (ng/kg bw/day)			Concentration (ng/kg food, wet weight)			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
94M4190	67	24	n.m.	64	103	0.65	0.23	n.m.	20.0	7.1	n.m.	19.1
94M0821	< LOD	16	n.m.	< LOD	110	-	0.15	n.m.	< LOD	5.6	n.m.	< LOD
94M0807	28	70	n.m.	< LOD	77	0.36	0.91	n.m.	12.9	32.1	n.m.	< LOD
94M0824	< LOD	58	n.m.	< LOD	73	-	0.79	n.m.	< LOD	23.1	n.m.	< LOD
94M4193	2	94	n.m.	10.4	51	0.04	1.84	n.m.	1.0	41.5	n.m.	4.6
94M0829	6	12	n.m.	< LOD	68	0.09	0.18	n.m.	2.8	5.6	n.m.	< LOD
94M0794	0.62	65	n.m.	43	77	0.01	0.84	n.m.	0.2	20.7	n.m.	13.8
94M0830	< LOD	14	n.m.	< LOD	84	-	0.17	n.m.	< LOD	4.9	n.m.	< LOD
94M0826	9	37	n.m.	< LOD	83	0.11	0.44	n.m.	2.1	8.7	n.m.	< LOD
94M4192	16	45	n.m.	< LOD	85	0.19	0.53	n.m.	4.6	13.2	n.m.	< LOD
					Mean	*0.14*	*0.61*	*n.m.*				
					SD	*0.07*	*0.52*	*n.m.*				
					Median	*0.21*	*0.49*	*n.m.*				

Measurements 1984

The exposure to PBDE-47 and PBDE-99 as determined with 24-hour duplicate diets (10 samples, ARO SOP 495 (revised: clean-up including lipid extraction)).

	Intake (ng/day)					Intake (ng/kg bw/day)			Concentration (ng/kg food, wet weight)			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
84217	< LOD	< LOD	n.m.	40	72	-	-	n.m.	< LOD	< LOD	n.m.	20.0
84220	< LOD	< LOD	n.m.	< LOD	58	-	-	n.m.	< LOD	< LOD	n.m.	< LOD
84214	< LOD	23	n.m.	< LOD	59	-	0.39	n.m.	< LOD	14.2	n.m.	< LOD
84211	10	43	n.m.	20	72	0.14	0.60	n.m.	6.8	28.1	n.m.	13.3
84333	12	51	n.m.	< LOD	70	0.17	0.73	n.m.	4.6	19.2	n.m.	< LOD
84209	< LOD	< LOD	n.m.	76	65	-	-	n.m.	< LOD	< LOD	n.m.	64.5
84335	20	32	n.m.	12	72	0.28	0.44	n.m.	10.2	16.0	n.m.	5.8
84218	4	9	n.m.	< LOD	80	0.05	0.11	n.m.	1.6	3.5	n.m.	< LOD
84337	9	22	n.m.	< LOD	78	0.12	0.28	n.m.	4.4	11.2	n.m.	< LOD
83344	< LOD	26	n.m.	< LOD	64	-	0.41	n.m.	< LOD	8.8	n.m.	< LOD
					Mean	*0.08*	*0.30*	*n.m.*				
					SD	*0.10*	*0.26*	*n.m.*				
					Median	*0.03*	*0.34*	*n.m.*				

Measurements 1978

The exposure to PBDE-47 and PBDE-99 as determined with 24-hour duplicate diets (10 samples, ARO SOP 495 (revised: clean-up including lipid extraction)).

	Intake (ng/day)					Intake (ng/kg bw/day)			Concentration (ng/kg food, wet weight)			
Sample code	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**	**Body Weight**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**PBDE-47**	**PBDE-99**	**PBDE-209**	**Other PBDEs**
71	15	< LOD	n.m.	24	80	0.19	-	n.m.	9.8	< LOD	n.m.	16.0
100	51	< LOD	n.m.	< LOD	64	0.80	-	n.m.	20.6	< LOD	n.m.	< LOD
69	49	< LOD	n.m.	< LOD	62	0.79	-	n.m.	21.4	< LOD	n.m.	< LOD
22	24	< LOD	n.m.	< LOD	110	0.22	-	n.m.	10.3	< LOD	n.m.	< LOD
8	81	4	n.m.	116	78	1.04	0.05	n.m.	30.4	1.4	n.m.	43.3
5	< LOD	41	n.m.	45	56	-	0.73	n.m.	< LOD	24.1	n.m.	26.6
73	19	1	n.m.	40	57	0.33	0.02	n.m.	12.6	0.6	n.m.	26.0
32	34	3	n.m.	< LOD	68	0.50	0.04	n.m.	16.3	1.3	n.m.	< LOD
12	48	13	n.m.	< LOD	78	0.62	0.17	n.m.	14.0	3.7	n.m.	< LOD
88	50	11	n.m.	40	74	0.68	0.15	n.m.	19.5	4.2	n.m.	15.8
					Mean	*0.57*	*0.12*	*n.m.*				
					SD	*0.29*	*0.22*	*n.m.*				
					Median	*0.62*	*0.03*	*n.m.*				

Appendix 2. Dietary intake of PBDEs

This table shows the statistical index numbers of the dietary intake of PBDE congeners in the Dutch population as determined by the "total diet method" (ng/kg bw/day, De Winter-Sorkina et al., 2006):

PBDE-47	Median	P90	P95	P97.5	P99
two-year-olds (low)	1.35	2.69	3.27	3.88	4.73
Life-long (low)	0.38	0.76	0.92	1.09	1.33
two-year-olds (middle)	1.4	2.69	3.24	3.8	4.58
Life-long (middle)	0.4	0.77	0.93	1.09	1.32
PBDE-99	Median	P90	P95	P97.5	P99
two-year-olds (low)	0.13	0.21	0.25	0.28	0.33
Life-long (low)	0.08	0.13	0.15	0.17	0.2
two-year-olds (middle)	0.23	0.34	0.38	0.42	0.47
Life-long (middle)	0.11	0.17	0.19	0.21	0.24
PBDE-100	Median	P90	P95	P97.5	P99
two-year-olds (low)	0.012	0.031	0.04	0.05	0.065
Life-long (low)	0.012	0.031	0.04	0.05	0.064
two-year-olds (middle)	0.181	0.261	0.29	0.317	0.352
Life-long (middle)	0.08	0.115	0.128	0.14	0.155
PBDE-183	Median	P90	P95	P97.5	P99
two-year-olds (low)	0.66	1.45	1.81	2.2	2.76
Life-long (low)	0.34	0.75	0.94	1.15	1.44
two-year-olds (middle)	0.87	1.71	2.07	2.44	3.17
Life-long (middle)	0.42	0.83	1.01	1.19	1.45

Appendix 3. Details of breast milk and blood studies

Auther	Country	PBDE standard	Analysis	Type	Number of PBDEs	PBDE-47	Time trend	Trend line	P/M *	Remark
Thomsen et al., 2002	Norway	Wellinton	GC/MS	serum	6	yes	yes	1977-1999	-	Pooled samples
Thomsen et al., 2003	Norway	Wellinton	GC/MS	serum/milk	11	no	yes	1986-1993-2001	P	Pooled samples
Schröter-Kermani et al., 2000	Germany	Promochem	GC/MS	blood	11	yes	yes	1985-1999	-	Mean values
Fängström et al., 2008	Sweden	in-house	GC/MS	milk	4	yes	yes	1980-2004	P 55-80%	Pooled samples
Fängström et al., 2005	Faroe Islands	in-house	GC/MS	milk	4	yes	yes	1987-1995-1999	unknown	Pooled samples
Lind et al., 2003	Sweden	Stockholm University	GC/MS	milk	5	yes	yes	1996-2001	P	Mean and median values
Meironyté et al., 1999	Sweden	in-house, CIL and Riedel de Haën	GC/MS	milk	8	yes	yes	1972-1997	P 55-75%	Mean values of pooled samples
Meironyté-Guvenius et al., 2003	Sweden	unknown	unknown	milk	5	yes	yes	1998-2000	unknown	Mean values
Ingelido et al., 2007	Italy	unknown	GC/MS	milk	10	yes	no	2000	unknown	Pooled samples
Pirard et al., 2003	Belgum	unknown	GC/MS	milk	6	yes	no	2000-2001	P/M	Mean values
Kalantzi et al., 2004	UK	Promochem	GC/MS	milk	15	yes	no	2002	unknown	Geometric mean values
De Winter-Sorkina et al., 2006	Netherlands	unknown	GC/MS	milk	11	yes	yes	1988-1998-2003	P	Mean values
Weber et al., 2004	Germany	Promochem	GC/MS	milk	7	yes	no	2002	7 P + 1 M	Mean values
Fürst et al., 2001	Germany	unknown	GC/MS	milk	9	yes	no	1992-2000	unknown	Pooled samples in 1992, mean values in 2000
Vieth et al., 2004	Germany	Wellington	GC/MS	milk	9	yes	no	2002	P	Mean values

* P: primiparae origin, M: multiparae origin

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National Institute
for Public Health
and the Environment

P.O. Box 1
3720 BA Bilthoven
The Netherlands
www.rivm.com