

Polybrominated diphenyl ethers (PBDEs) as food contaminants

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Abstract: Polybrominated diphenyl ethers (PBDEs) show similarities on their behavior in the environment with classical persistent organic pollutants, especially polychlorinated biphenyls. Therefore, their increasing levels reported for humans are based on their presence at elevated levels in various environmental matrices like food, dust or airborne particles. Since sources for human contamination with PBDEs are multiple, this material is based on reviewing the presence of these chemicals in food samples. The contribution of several food categories to human exposure to PBDEs was discussed with emphasis on dietary intake.

Keywords: polybrominated diphenyl ethers, PBDEs, BFRs, food, contaminants.

Introduction

Brominated flame retardants (BFRs) are currently the largest market group of flame retardants (FRs) because of their low cost and high efficiency. Currently, there are more than 75 different BFRs recognized

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commercially.¹ The five major BFRs (Figure 1) are tetrabromobisphenol-A (TBBP-A), hexabromocyclododecanes (HBCDs), and three technical mixtures of polybrominated diphenyl ethers (PBDEs): penta-BDE, octa-BDE, and deca-BDE.

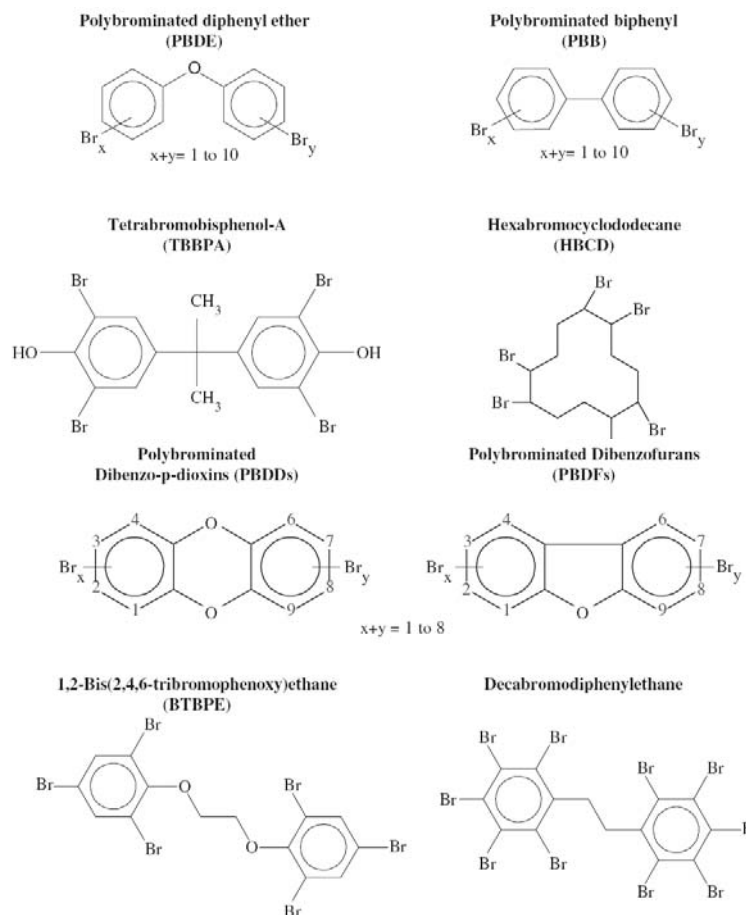


Figure 1. Chemical structures of the most important brominated flame retardants.

The commercial products are waxy solids, have boiling points between 310°C and 425°C and have low vapour pressures ranging from 3.9 to 13.3 Pa at $20\text{--}25^\circ\text{C}$.^{2,3} Their solubility in water is low, especially that of the higher BDEs. The *n*-octanol/water partition coefficient ($\log K_{ow}$) ranges between 4.3 and 9.9.³ PBDEs are thermally labile and break down readily

with heat, which enables them to act as effective FRs. Both aliphatic and aromatic carbon–bromine covalent bonds are relatively weak due to the large atomic size of bromine. As a result, polybrominated organics, including PBDEs, tend to break down often. Brominated flame retardants under thermal degradation conditions release hydrogen bromide. These reaction products react with highly reactive H• and HO• radicals present in the flame resulting in the formation of inactive molecules and of Br• radicals. The halogen radical has much lower energy than H• and HO•, and therefore much lower potential to propagate the radical oxidation reaction and therefore the flame.⁴

Higher BDEs, such as BDE 209 in particular, can also degrade upon exposure to UV light.^{5,6} This can be particularly problematic during analysis if no proper measures are taken to prevent UV degradation.

PBDEs are additive FRs which are used in a wide array of household products in concentrations up to 30% by weight, typically between 2 and 6%. They are structurally related to polychlorinated biphenyls (PCBs) and are produced commercially as mixtures. However, PBDE mixtures contain fewer congeners than the commercial PCB mixtures. The three PBDE mixtures (penta-BDE, octa-BDE, and deca-BDE) have different applications, such as polystyrene foams, high-impact polystyrene and epoxy resins, which are further used in a wide range of consumer products, including computers, electronics and electrical equipment, televisions, textiles, foam-padded furniture, insulating foams, and other building materials. BFR production has increased dramatically over the past 20 years, with the largest relative increase in market demand at this time being found in Asia. More than 200,000 metric tons of BFRs are produced each year.⁷

Despite of their benefits for reducing fire-related injury and property damage, growing concern for BFRs has risen because of their occurrence and persistence in the environment, biota and humans, in a similar way to other persistent organic pollutants.^{8,9}

Due to the lipophilic nature of these man-made chemicals, PBDEs are mainly found in lipid-rich food of animal origin, such as meat, fish and dairy products, which are a part of our daily diet. Because PBDEs are lipophilic synthetic halogenated chemicals that resemble PCBs to a great extent, it is expected that also exposure to PBDEs will occur mainly through the diet. The importance of the different routes of human exposure to PBDEs (diet and inhalation/ingestion) is not completely understood at present and opinions about the contribution of the dietary intake vary among publications.

Analytical methods for determination of PBDEs in food

The methods used for the determination of PBDEs in different matrices are very sensitive and thus able to detect extremely low amounts of PBDEs in any type of matrix and particularly in food products. Since dietary intake studies consider most of the time that $\frac{1}{2}$ of LOQ as contaminants concentration to be further used for intake calculations when concentrations are below LOQ, it is extremely important that existing methods have to be able to detect as low as possible PBDEs in such matrix. The methods described in the literature have been recently reviewed and therefore, only the most important details related to necessary steps to be followed in order to apply a correct analytical methodology for an accurate determination of PBDEs in food products will be addressed.¹⁰⁻¹³

Sample preparation and instrumental analysis

In the last years there was an increased number of publications which reported various analytical methodologies for PBDE analysis in various matrices, including food.¹⁴⁻²⁰ Moreover, there are also published several reviews focussed in analytical methodologies for PBDEs determination which pointed out the improvements of such methodologies.¹⁰⁻¹³ As a general rule, the protocols developed for the analysis of BFRs in general, and PBDEs in particular, were based on GC-MS methodologies previously validated for trace POPs, such as OCPs or PCBs.²¹ Some basic steps of the BFR determination are sample pre-treatment, extraction, clean-up and instrumental analysis. Since recent studies^{10-13,20,22} published in detail each step of the analytical procedures applied on determination of PBDEs from environmental matrices, which are applicable also for food samples, in this paper there will be no more discussion related to this section than the general information presented above.

However, being present in all environmental compartments, laboratory contamination during each analysis step can easily occur.

Quality assurance/ quality control

Quality Assurance (QA) is a set of procedures, which include the quality control (QC) activities that are undertaken to affirm the quality of analytical results. As a general rule, between 10 and 20 % of the analysis time is spent to ensure good quality of the performed analyses.²³ To assure sufficient quality, a number of measures should be taken during the pre-analysis quality control (or validation) and in-process quality control. These measures can be divided into three major areas: calibrants, analytical procedure control, system performance/long-term stability.

Because all essential steps of analytical procedure are matrix-specific, the analyte recovery, the use of procedural blanks and the determination of limits of detection and quantification should be performed for each compound and matrix to be investigated.

The analytical characteristics of the method should be also considered as an in-ternal quality control by determining the following parameters: repeatability (same operating conditions over a short time), intermediate precision (within-laboratory variation), reproducibility (precision between laboratories) and accuracy (estimated through the use of certified reference materials). The external quality control is usually assessed through the participation in interlaboratory tests which facilitates the evaluation and assessment of the overall method performance.

Occurrence of PBDEs in food products

An important pathway for human exposure to PBDEs is dietary intake via food consumption. It was clearly shown in the literature the bioaccumulative effect of these chemicals, and because of humans position in trophic level it is expected that higher concentrations of PBDEs may be found in human samples (human tissues, serum and milk). Indeed, a large number of human samples have been analyzed for PBDEs and, it could be concluded that the PBDE concentrations have increased in people by a factor of ~100 during the last 30 years, especially in USA. For Europe, the increase was not clearly evident. Moreover, a level-off or even decrease has been observed in Europe in the last 10 years.²⁴ Figure 2 present the regression analysis of these data as a function of year. Despite the disparate sample types, the different continents of origin, and the various congeners measured, a good regression was obtained when all of these data were

plotted together (Figure 2).

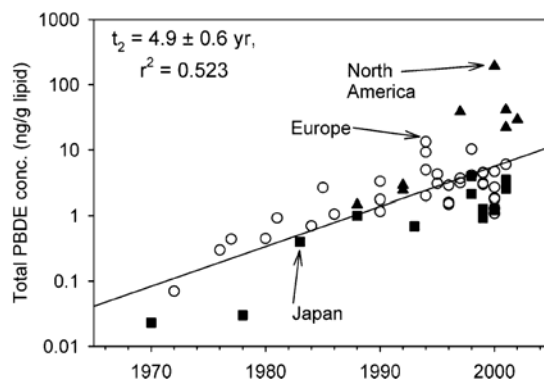


Figure 2. Total PBDE concentrations in human blood, milk and tissue (in ng/g lipid) shown as a function of the year in which the samples were taken. The three symbol types indicate the location from which the samples were collected.²⁴

Despite restrictive measurements on their use, PBDEs were detected in various food products for human consumption, such as fish²⁵⁻²⁸, meat products^{14,15,29} and eggs^{14,19,30}. Combined with the reported increased levels in human tissues (Figure 2)^{24,31,32}, this has led to an additional interest in market basket surveys.^{14,29,33} Besides food intake, other important exposure routes, such as dust ingestion³⁴⁻³⁷ and inhalation of airborne particles³⁸, have recently been acknowledged for BFRs.

Therefore, even if trends for PBDE concentrations in humans are shown to be increasing over the last years in USA, but not necessarily in Europe, there is a need to elucidate the pathways for human contamination with BFRs, especially for children (0-10 years). A recent publication focussed on assessment of sources and pathways of human exposure to PBDEs in US shown that breastfed infants are exposed to higher levels of PBDEs than are adults.³⁹ The cumulative daily exposure dose of PBDEs for breastfed infants was, on average 3–5 times greater than the dose estimated for the other age groups. Diet accounted for, on average, 91% of the total

PBDE exposure in breastfed infants whereas for the other age groups, including adults, diet accounted for 21–39% of the total PBDE intake. House dust ingestion was the predominant pathway for PBDE exposure, accounting for 56–77% of the total PBDE intake in toddlers, children, teenagers, and adults. The contributions of various sources to PBDE intake in the various age groups are summarized in Figure 3.³⁹

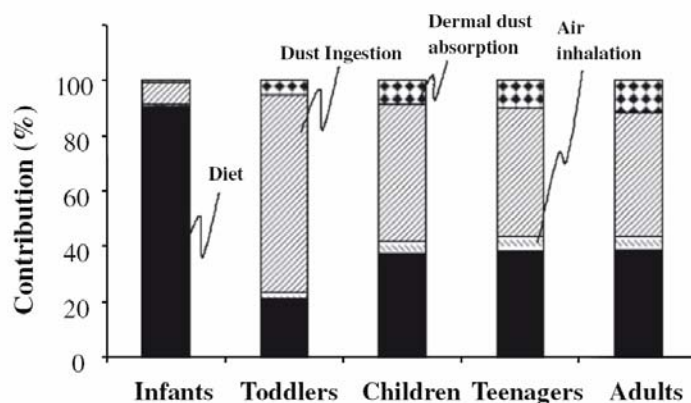


Figure 3. Per cent contributions from multiple sources to cumulative exposure to PBDEs, according to five age groups in the US population.³⁹

The hypothesis of higher human exposure to PBDEs through dust for the adult US population (as presented in Figure 3)³⁹ is supported also by the reported levels of these contaminants in indoor dust samples collected from various locations including USA, but also other locations, like Canada, New Zealand or Europe, as presented in Table 1.^{22,37} Therefore, it might be seen that sum PBDE levels in samples collected from USA are higher when compared to similar samples collected from Canada, New Zealand or Europe.^{22,37}

Table 1. Summary of concentrations (ng/g) of sum PBDE congeners in indoor dust samples from different countries (adapted from^{22,37}).

Location	N	Σ PBDEs ^a
Toronto, Canada	- ^b	970
Wellington, New Zealand	- ^c	-
Birmingham, UK	- ^d	3000
Amarillo and Austin, TX, US	- ^e	4000
Ottawa, Canada	68	1800
Various regions, UK	10	-
Newcastle, UK	10	10000
Various locations, US	17	4250
Various locations, Romania	18	490

^a sum of PBDEs 28, 47, 49, 66, 99, 100, 153, 154, 183, 196, 197, 203, and 209

^b ten samples analysed for tri-hexa BDEs; seven samples analysed for tri-deca BDEs.

^c twenty samples analysed for tri-hexa BDEs; hepta-deca BDEs not analysed.

^d twenty eight samples analysed for tri-hexa BDEs; sixteen samples analysed for tri-deca BDEs.

^e twenty samples analysed for tri-hexa BDEs; seventeen samples analysed for tri-deca BDEs.

Due to the lipophilic nature of these chemicals, PBDEs are mostly found in lipid-rich food of animal origin, such as meat, fish and dairy products, which are a part of our daily diet. It has been shown that food, and more in particular food of animal origin, is responsible for more than 90% of the average human intake of polychlorinated biphenyls (PCBs)⁴⁰ and therefore for BFRs, is expected to be similar. However, considering recent studies focussed on PBDE levels in food items, it seems that the contribution of some food categories, such as plant-based comestibles is not negligible for total human PBDEs intake as it was assumed/shown for classical POPs. Human exposure via a vegan diet was indeed shown to be lower than that via an omnivorous diet, but still appreciable.⁴¹ Whereas available measurements support the idea that exposures via ingestion of

fruit and vegetables are low, this is not the case for oils and fats of vegetable origin, and it would be prudent to include such foodstuffs in future studies.⁴² D'Silva also published similar findings when reporting results of the analysis of archived total diet survey samples from the United Kingdom, 70% of dietary exposure to PBDEs in the UK in 2000 came from non-meat sources: vegetables, fruit, bread, and dairy products.⁴³

Some important findings obtained from both Europe and Asia show the important role played by the consumption of fish and seafood, with its ingestion typically comprising around 50% of overall dietary exposure.²⁰ Related to this, there have been several studies that indicate the potential for exposure via ingestion of fish oil dietary supplements.²⁰ Other important food groups, with respect to dietary exposure to PBDEs, include dairy products (including eggs) and meats. Table 2 lists the dietary intake based on food shopping basket surveys performed in Europe and America. Except one case, data were calculated using the "median-bound" scenario by assuming that values below the limit of detection were equal to one half of the LOD. This comparison clearly shows that the dietary intake in Europe is in the same range as that for Canada.⁴⁸ An expanded US survey reported that the total contamination level in European food samples was comparable to that in the US.^{33,49} In most European countries fish products are the main contributors to total PBDE intake, but in the US meat and meat products tend to account for most of the exposure to these chemicals.¹⁷

A special attention should be addressed to BDE 209, which shows a different profile when speaking about food samples analyzed from different continents. It seems that BDE-209 has been reported as ranging from nondetectable to very low levels (100 pg/g ww) in fish, meat and dairy products from the United States.³³ Likewise, while a market basket study of

Spanish foodstuffs found BDE-209 to be the dominant congener in eggs and oils, it was present only in low concentrations in other food samples.⁵⁰

Table 2. Calculated daily PBDE dietary intake from various studies in ng/kg body weight (values < LOD=0.5×LOD, as not otherwise stated) [adapted from ¹⁷].

Dietary intake	Study location	Year	Sum of PBDE congeners	References
1.40	Spain	2000	47, 99, 100, 153, 154, 183	42
3.55 ^a	The Netherlands	2001–2002	28, 47, 99, 100, 153, 154	44
1.06	Norway	2002–2006	47, 99, 100, 153, 154	45
0.79	The Netherlands	2003–2004	47, 99, 100, 153, 154	46
1.2 ^{b,c}	UK	2003–2004	47, 99, 100, 153, 154	47
1.3 (m) ^d 0.9 (f) ^d	USA (Texas)	2003–2004	17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 209	33
0.58 ^a	Belgium	2005	BDE 28, 47, 99, 100, 153, 154, 183	14

(m): male; (f): female. ^aFor an adult of 60 kg bw; ^bAverage consumption behaviour; ^c<LOD=0; ^dAdults 20–39 years.

In contrast, BDE-209 was the dominant congener in all food groups, except canned vegetables and fish in the 2003 UK total diet study.⁵¹ Data from a recent Belgium study showed a similar profile with the UK reported study, and although BDE-209 was detected in only ~25% of the samples, sometimes just above LOQ, it was the major contributing congener (70-90% of ΣPBDE) in food samples collected from Antwerp, Belgium.⁵² However, BDE 209 was found at levels below method LOQ in all food samples included in a Romanian study, showing that regarding human exposure to

that contaminant has a different pathway for this region. Most probably other sources are more important when addressing exposure of Romanian population to BDE 209.⁵³

An important aspect for reporting PBDEs in food samples should also consider that most of the studies give the levels of these contaminants based on unprocessed foods, which were not boiled, cooked, baked or fried prior to analysis. Therefore, the dietary exposure to BFRs might slightly overestimate real dietary exposure.¹⁸ In one study it was shown that broiling, with fat dripped from the foods (ground beef, ground lamb, catfish, trout, and salmon) reduces the amount of PBDEs in these foods. This suggests that calculations of food intake need to take into consideration levels in the cooked food rather than in the uncooked food.¹⁸

Concluding remarks and future perspectives

The bioaccumulative character for PBDEs among trophic levels was evidenced with emphasis on increasing levels for these contaminants in humans. Sources for human exposure to PBDEs are multiple and they should be carefully investigated in order to obtain accurate data for minimizing their adverse health effects acknowledged for these contaminants in biologic organisms.

Because of their relative recent ban on EU and also other markets like USA, Canada or Asia, it should be expected that PBDEs will be introduced on priority monitoring lists on different matrices (especially food) and therefore, accurate analytical methodologies for their measurement are necessary in order to estimate/control human exposure to such chemical formulations. All aspects related to each step which is applied in order to accurately determine PBDEs in any environmental matrix should be

carefully addressed following specific steps on quality assurance/ quality control procedures.

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