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Review

Human exposure to polybrominated diphenyl ethers through the diet

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Abstract

Polybrominated diphenyl ethers (PBDEs), a class of brominated flame retardants, are used in a variety of consumer products being produced in notable quantities. PBDEs have been detected in environmental samples. In recent years, a marked increase in the levels of PBDEs in human biological tissues and fluids, especially breast milk, has been observed in some countries. As for other persistent organic pollutants (POPs), dietary intake is very probably the main route of exposure to PBDEs for the general population. This paper reviews the state of the science regarding human exposure to PBDEs through the diet. Because of the scarce information about it, it is concluded that studies focused on determining PBDE exposure for the population of a number of countries are clearly required. The correlation of PBDE body burdens and dietary intake of PBDEs are also necessary.

Keywords: Reviews; Human exposure; Dietary intake; Food analysis; Polybrominated diphenyl ethers

Contents

1. Introduction

The polybrominated diphenyl ethers (PBDEs) are a class of chemicals widely used as flame retardants. The PBDEs belong to a family of polyhalogenated compounds with long 2–10-year half-lives, which includes also polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/PCDFs), polychlorinated biphenyls (PCBs), polychlorinated naphthalenes (PCNs), polychlorinated diphenyl ethers (PCDEs) and polybrominated biphenyls (PBBs) [1]. More specifically, PBDEs and their metabolites are structurally similar to PCBs and DDT. Therefore, their chemical properties, persistence and distribution in the environment follow similar patterns [2–4]. The stability and lipophilicity of PBDEs causes them to biomagnify up the food chain, increasing in concentration at each successively higher trophic level [5,6].

Three major commercial mixtures of PBDEs are produced: deca-BDEs (mostly deca-BDE with some nona- and octa-BDE congeners), octa-BDEs (mostly hepta- and octa-BDE congeners), and penta-BDEs (mostly penta- and tetra-BDE congeners). Fully brominated deca-BDE is the major product accounting for 75% of the PBDE production [1–3,7]. Generally, the penta-BDEs seem to cause toxic effects at the comparably lowest dose, whereas much higher doses are needed for adverse effects of deca-BDEs [8]. Although the toxicology of PBDEs is still under investigation, it has been already established that PBDEs can be persistent, bioaccumulative and toxic [8–11]. In general terms, PBDEs can cause liver and neurodevelopmental toxicity,

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and affect thyroid hormone levels [12–14]. The critical effects of penta-BDEs are those on neurobehavioral development (from 0.6 mg kg^{-1} body weight) and, at somewhat higher dose affect thyroid hormone levels in rats and mice. Adverse effects of octa-BDEs have been also found on fetal toxicity/teratogenicity in rats and rabbits (from 2 mg kg^{-1} body weight), while critical effects of deca-BDEs have been noted on thyroid, liver, and kidney morphology in adult animals from 80 mg kg^{-1} body weight [8].

On the other hand, it is still unclear whether current concentrations of PBDEs in human tissues would be expected to adversely impact human health [3]. However, there is evidence that, in recent years, PBDE concentrations are rising in human tissues in which PBDEs have been identified: blood, adipose tissue, liver and milk [15–26]. Generally, in biological samples the congeners BDE-47 and BDE-99 occurred at the highest levels [15–26]. These are the most important congeners within the penta-products, which each make up approximately 37% of the total composition [27].

It seems that for the general population, one of the main routes of exposure to PBDEs, particularly the lower brominated congeners, is through the diet, as it also occurs with PCDD/PCDFs and PCBs [28,29]. This is especially relevant when the compounds are persistent enough to be biomagnified in the food web similarly to other persistent chemicals. Consequently, consumption of foodstuffs such as fatty fish from contaminated sources is a major way of exposure to PBDEs [30–32]. In turn, inhalation of air polluted by PBDEs in the work environment can be also an important route of occupational exposure to these compounds [28,33].

With respect to human PBDE exposure through the diet, data on the levels of these environmental pollutants in food are only relatively abundant in fish. However, much less is known on PBDE concentrations in other major food groups, or about possible differences in food levels between countries or regions. This paper presents an overview on the available scientific information concerning PBDE concentrations in foodstuffs, as well as data on human exposure to these pollutants through the diet.

2. PBDEs in freshwater fish and marine species

2.1. Freshwater fish

An analysis of fish tissue samples from selected locations in Washington State (USA) showed that PBDE concentrations ranged from 1.4 μg kg^{-1} (wet weight) in rainbow trout from a remote spring-fed stream to 1250 μg kg^{-1} in mountain whitefish from the urbanized Spakane River [34]. The highest concentrations were found in areas draining urbanized watersheds compared to undeveloped watersheds. In all analyzed species, tetra- and penta-BDEs were the major congeners.

Because of the relative scarcity of reports on the North American environment, the high USA demand for products containing tetra- to hexabrominated diphenyl ethers, and possible exposure to human via fish consumption, Hale et al. [35] determined the concentrations of these PBDE congeners in edible fish tissues (332 fish samples belonging to 33 different species) from two large Virginia watersheds. Concentrations of total tetra- to hexabrominated congeners in filets ranged from <5 to 47,900 μg kg^{-1} (lipid basis). BDE-47, one of the major constituents of penta-BDEs, was detected in 89% of samples and constituted 40–70% of the total PBDEs observed. The highest total PBDE concentration, 47,900 μg kg^{-1} (1140 μg kg^{-1} wet weight), was detected in a carp from the Hyco River.

In another study, Manchester-Neesvig et al. [36] reported several PBDE congeners in 21 coho and chinook salmon samples taken in 1996 from Lake Michigan tributaries. Six PBDE congeners were detected in all 21 samples, and the rank order of concentration of these congeners was similar to that found in commercial mixtures of PBDEs. The average concentration across all samples of the sum of PBDE congeners was 80.1 ng g^{-1} wet weight, or 2440 ng g^{-1} lipid. This level was much less than the average sum PCB concentration (1450 ng g^{-1} wet weight), which was also quantified in this study.

Rice et al. [37] identified PBDEs in fish collected from the Detroit River, MI, USA (carp and large mouthbass) and the Des Plaines River, IL, USA (carp). These sites were selected because they had high levels of industrial and municipal effluents contributing to their flow. The average total concentrations (wet weight basis) in carp and bass from the Detroit River were very similar (5.25 and 5.39 ng g^{-1} in the bass and carp, respectively). However, expressed on a lipid basis there was a greater average level of PBDE in the bass (163 ng g^{-1}) than the carp (40.7 ng g^{-1}). Average total PBDE concentration in the carp from the Des Plaines River (12.48 ng g^{-1} wet weight) was significantly higher than that in carp from the Detroit River.

Dodder et al. [38] determined the concentrations and spatial distributions of PBDEs in fish (white crappie, carp, smelt and bluegill) collected near a suspected source and from more remote locations. Fish from four lakes, two small lakes in the northeastern USA and two of the Great Lakes were analyzed. Three of these lakes were considered to have background levels of PBDEs. At these background locations, the sum PBDE concentrations ranged from 6.9 to 18.8 ng g^{-1} wet weight, or 150 to 300 ng g^{-1} lipid. At the lake near the suspected source, the sum PBDE concentration was 65 ng g^{-1} wet weight (2400 ng g^{-1} lipid).

Recently, Zennegg et al. [39] measured PBDE concentrations in pooled whitefish (*Coregonus* sp.) samples from eight Swiss Lakes and in rainbow trout samples from four Swiss fish farms. Sum of PBDE congeners in filet from whitefish was between 36 and 165 ng g^{-1} lipid, or 1.6 and 7.4 ng g^{-1} wet weight. PBDE contents in filet from farmed rainbow trout were significantly lower than in wild whitefish (12–24 ng g^{-1} lipid, or 0.74–1.3 ng g^{-1} wet weight). The PBDE congener patterns were different for both species.

2.2. Marine species

Akutsu et al. [40] analyzed the concentrations of seven PBDEs (BDE-28, 47, 66, 99, 100, 153 and 154) in seven species of edible marine fish (conger eel, flounder, gray mullet, horse mackerel, red sea bream, sea bass and yellowtail) collected from the Inland Sea of Seto (Japan). In all samples, BDE-47 was the most abundant congener. The sum concentration of total PBDEs varied between 110 (red sea bream) and 3300 (gray mullet) ng kg^{-1} wet weight, or 2400 (conger eel) and 60,000 (gray mullet) ng kg^{-1} lipid.

In a screening of blue mussels (*Mytilus edulis*) collected in 15 locations from Denmark during the autumn of 2000, BDE-47 constituted 64% of the sum of the four PBDE congeners analyzed (BDE-47, 99, 100 and 153). The sum of these four lower brominated congeners was in the range 80–811 ng kg^{-1} wet weight. The highest contamination with PBDEs was found close to populated areas, probably due to washout and emission from flame retarded plastics [41].

Christensen et al. [42] determined the levels of PBDEs in shorthorn sculpin collected at three locations in southern Greenland in July–August 2000. Uvak, spotted wolffish, starry ray and blue mussels were also collected in one (no population) of these locations. Sum of PBDEs in fish varied between 1800 and 12,000 ng kg^{-1} wet weight, depending on the location and species, while for mussels it was 110 ng kg^{-1} wet weight. The highest concentrations corresponded to uvak, a top-predator on fish indicating that PBDEs were biomagnifying. The researchers emphasized that PBDE levels were 15–24 times lower than the levels of PCBs measured also in the same individuals of fish, except for shorthorn sculpin collected in one location, where the level of PCBs was 40 times higher than that of PBDEs. This finding was attributed to a local emission of PCBs, which was higher than the local PBDE emission.

The concentrations of 16 individual PBDE congeners were investigated in animals representing different trophic levels of the North Sea food web [32]. Six congeners (BDE-28, 47, 99, 100, 153 and 154) were present as major compounds in most analyzed species of invertebrates (sea star, hermit crab, whelk and shrimp), fish (herring, cod and whiting), and marine mammals (harbor porpoise and harbor sea). The general order of decreasing concentrations was BDE-47 > BDE-99, BDE-100 > BDE-153, BDE-154 > BDE-28. However, the sum of PBDEs was not reported. The major biomagnification step in the food chain occurred from fish to marine mammals. In these animals, the lipid-normalized PBDE levels were generally more than an order of magnitude higher than in the invertebrates and fish [32].

Samples of edible fish from the San Francisco Bay (2000) and from California coastal waters (2001) were analyzed for PBDEs. Each sample was a composite of several individual fish of the same species (white croaker, California halibut, diamond turbot, surf perch, shiner perch and stripped bass). With very few exceptions, BDE-47 was the dominant congener, followed by BDE-100. PBDE levels were 429 (257–752) and 394 (19–1144) ng g^{-1} lipid weight in fish composite samples from the San Francisco Bay and other California coastal waters, respectively [43].

In October–November 2001, benthic invertebrates such as shrimp, crab and starfish, benthic fish such as goby, dab, plaice and sole, and gadoid fish such as bib and whiting were sampled in the Belgian North Sea (BNS), a presumably non-polluted area, and the Scheldt estuary (SE), an area subjected to a variety of suspected PBDE sources. Samples of these species were analyzed to determine the concentrations and spatial variations of a number of PBDEs. In BNS, the sum of six PBDE congeners (BDE-28, 47, 99, 100, 153 and 154) ranged from 0.02 to 1.5 ng g^{-1} wet weight in benthic invertebrates and goby, from 0.06 to 0.094 ng g^{-1} wet weight in fish muscle, and from 0.84 to 128 ng g^{-1} wet weight in fish liver, respectively. For the SE samples, the sum of the six congeners ranged from 0.20 to 29.9 ng g^{-1} wet weight in benthic invertebrates and goby, from 0.08 to 6.9 ng g^{-1} wet weight in fish muscle, and from 15.0 to 984 ng g^{-1} wet weight in fish liver. For each species, the levels of PBDEs were significantly higher for all SE locations when compared to the BNS locations, while the lipid percentages of each species were similar for the two areas. The concentrations found in this study clearly suggested a source of PBDEs in or near the Scheldt estuary [44].

The above results in freshwater and marine species clearly show that PBDE concentrations in these species vary considerably according to, basically, the levels of PBDEs in the waters where they were collected.

3. PBDEs in foodstuffs

With the exception of the studies concerning PBDE levels in a number of freshwater and marine species, little data are available on PBDE concentrations in other major food groups. Preliminary data from mixed meat products from Sweden (market basket samples) revealed a mean level of 360 ng kg^{-1} fat [45], while the mean PBDE level in Swedish eggs found in the same study was 420 ng kg^{-1} fat.

Huwe et al. [46] investigated the presence of PBDEs in chickens. Fat samples of these animals were collected in 1997. The PBDEs were isolated by a modified version of EPA Method 1613. All chromatography was performed using disposable columns and an automated system of Fluid Management Systems. Analyses were performed by HRGC/HRMS using on-column auto-injection. Relative response factors (rrfs) together with the recoveries determined for the $^{13}C_{12}$-surrogates were used to quantitate the PBDEs in all samples. Solvent or matrix blanks and a performance check sample were run interspersed with the samples. Matrix and laboratory blanks contained low but detectable levels of several PBDEs. The total concentrations of PBDEs on a whole weight basis ranged from 1.76 ng g^{-1} in North Dakota (USA) chickens to 39.43 ng g^{-1} in a chicken from Arkansas (USA). On a lipid weight basis, these levels were

lower than those generally found in fish and fish-eating mammals. However, they were higher than levels found in the local North Dakota chicken composite (matrix blank). The PBDE pattern found in this study was also different from other samples reported. Thus, penta-BDEs rather than tetra-BDEs were the most predominant congeners.

Recently, an investigation was carried in Japan in order to establish the relationship between dietary intake of fish, meat, and vegetables and tubers, and the concentrations of PBDEs in human milk [29]. The purification method used to extract PBDEs from fish, meat and vegetable samples was similar to the multi-layer column used for dioxin purification developed by Miyata et al. [47]. An isotope internal standard method typically used to quantify dioxin concentrations in environmental samples was adopted for the analysis of PBDEs, which were performed by HRGC/HRMS in ES-SM mode. Identification and determination were carried out by comparing the retention times and mass spectra of the different PBDE isomers with those of authentic standards. The concentrations of PBDEs were corrected with the recoveries of their respective ^{13}C-internal standards. PBDE levels in edible tissues of four species of fish (yellowtail, salmon, mackerel and yellow tuna), and one species of shellfish (clam) purchased from two food markets ranged between 17.7 and 1720 ng kg^{-1} wet weight. In vegetables and tubers, PBDE concentrations were between 38.4 (carrot) and 134 (spinach) ng kg^{-1} wet weight, while the levels of PBDEs in meat varied from 6.25 (chicken) to 63.6 (pork) ng kg^{-1} wet weight.

In a recent study performed in Catalonia (Spain), the concentrations of PBDEs were measured in a number of foodstuffs (vegetables, tubers, pulses, cereals, fruits, fish and shellfish, meat and meat products, eggs, milk and dairy products, and fats and oils) [48]. Composite samples were lyophilized previously to analyses of PBDEs, which were performed in accordance to the US EPA 1625 method (semivolatile organic compounds by isotope dilution GC/MS). The cleanup procedure and fractionation of the sample aliquot was carried out as a multiple cleanup, using adsorption chromatography, a multilayer silica column (from top to bottom: sodium sulfate, silica, silica-sulfuric acid, silica, silica-potassium hydroxide, silica), alumina columns, and gel permeation columns (BioBeads SX3). The final step involved the reduction of the PBDE-containing fractions to the volume necessary for the analysis. Recovery rates were calculated against external reference standards. Standards were used to calculate the amounts of PBDE of their own congener group (e.g., tetra- for tetras, penta- for pentas, etc.), with the exception of Octa-BDE that was calculated using $^{13}C_{12}$-Hepta-BDE #183. The cleaned extract was analyzed by high-resolution gas chromatography/high-resolution mass spectrometry. Mean recovery rates ranged from 79%, for the sum of penta-BDEs (range 55–106%) and hexa-BDEs (range 54–115%), to 85% (54–114%) for the sum of hepta-BDEs. The highest PBDE levels (sum in ng kg^{-1} wet weight) were found in fats and oils (587.7), fish and shellfish (339.2), meat and meat products (109.2) and eggs (64.5). In contrast, the lowest levels corresponded to fruits (5.8), vegetables (7.9) and tubers (7.4). In all these groups, a predominance of the homologues tetra- and penta-BDEs, followed by hexa-BDEs, could be observed in the sum of total PBDEs [48].

Table 1
Dietary intake of PBDEs by the general population of Catalonia, Spain[a]

Food group	Daily consumption[b] (g)	PBDE intake[c] (ng per day)
Vegetables	226 (15.7)	1.8 (1.2)
Tubers	74 (5.1)	0.6
Fruits	239 (16.6)	1.4
Pulses	24 (1.7)	0.3 (0.05)
Cereals	206 (14.3)	7.4
Fish and shellfish	92 (6.4)	30.7 (29.9)
Meat and meat products	185 (12.8)	20.2 (18.9)
Eggs	34 (2.4)	2.2 (2.0)
Milk	217 (15.0)	3.7 (2.9)
Dairy products	106 (7.3)	5.1 (3.6)
Fats and oils	41 (2.8)	24.1 (23.3)
Total intake	1444 (100)	97.3 (81.9)

[a] Daily intake was estimated for a male adult of 70 kg body weight. Data from Bocio et al. [48].
[b] In parentheses, percentages of total consumption per day.
[c] PBDE intake was estimated assuming that when a congener was below the limit of detection (LOD), the concentration was equal to one-half of the respective LOD. In parentheses, PBDE intake calculated assuming that ND = 0.

4. Human exposure to PBDEs through the diet

In an extensive search of the scientific literature, only six studies concerning human exposure to PBDEs through the diet were found. Among these reports, the most wide study corresponds to that of Bocio et al. [48], in which 54 food samples belonging to 11 food groups were analyzed in a previous step to estimate the dietary intake of PBDEs in Catalonia (Spain). For a standard male adult of 70 kg body weight, total dietary intake of PBDEs was 97.3 ng per day (assuming not detected (ND) = 1/2 limit of detection), or 81.9 ng per day (assuming ND = 0). These values are equivalent to 1.4 and 1.2 ng kg^{-1} body weight per day, respectively. The highest contribution to this intake corresponded to fish and shellfish, with approximately one-third of the total intake, followed by fats and oils, and meat and meat products, with a contribution of about one-fourth each group. The lowest percentages corresponded to vegetables, tubers, fruits and cereals (Table 1). Although in Catalonia the consumption of these food groups is very notable (Mediterranean diet), the low PBDE content in these foodstuffs would explain their small contributions to the total intake of PBDEs through the diet.

In an estimation of PBDE exposure from food in Sweden, the dietary intake was 51 ng per day (calculations were done assuming that ND = 1/2 limit of detection) [49]. This

Table 2
A summary of recent data of human exposure to PBDEs through the diet

Country	Characteristics of the study	PBDE intake (ng per day)	Remarks	Reference
Sweden	Market basket samples: fish, meat, dairy products, eggs, fats/oils, pastry	51 Sum of congeners 47, 99, 100, 153, 154	Calculations for intake were done assuming that ND = 1/2(LOD)	Darnerud et al. [49]
Sweden	Foods of animal origin, Diet National Swedish Inventory	Females (18–74 years), mean: 40.8 Sum of congeners 47, 99, 100, 153, 154	Calculations for intake were done assuming that ND = 0	Lind et al. [50]
Canada	Food basket study, most food samples of animal origin	44	–	Ryan and Patry [51]
UK	Duplicate diet samples	Median: 90.5 Sum of congeners 47, 99, 100, 153, 154	Calculations for intake were done assuming that ND = 0	Wijsekera et al. [33]
Catalonia (Spain)	Total diet study, 54 samples belonging to 11 food groups	81.88 (lower), 112.65 (upper), sum of tetra-to-octa-BDEs	ND = 0, ND = LOD	Bocio et al. [48]
Switzerland	Analysis of pooled whitefish samples from eight Swiss lakes and farmed rainbow trout	150 (whitefish), 26 (trout) Sum of congeners 28, 47, 99, 100, 153, 154 and 183	Calculations for intake were done assuming a daily fish consumption of 20 g. Intake was only estimated for fish consumption	Zennegg et al. [39]

intake was higher that that found in a subsequent study, in which only food of animal origin was included [50]. Total dietary intake for Swedish females (18–74 years old) was 40.8 ng per day. However, in that case, when the concentration of a PBDE congener was below the detection limit, that concentration was assumed to be zero (ND = 0). It is important to remark that in both Swedish studies only congeners BDE-47, BDE-99, BDE-100, BDE-153 and BDE-154 were determined. A similar dietary intake, 44 ng per day, was also found for Canadian adults in a food basket study [51]. About 75% of the daily intake of PBDEs through the diet corresponded to meat, while dairy products and fish contributed with approximately 7 and 4%, respectively. In turn, in a recent survey carried out in the United Kingdom, the median dietary intake of five PBDE congeners (BDE-47, 99, 100, 153 and 154) was 90.5 ng per day (calculations were done assuming ND = 0) [33].

On the other hand, in a recent study based on an average daily consumption of 20 g whitefish from Swiss lakes, with a PBDE content of 7.4 ng g^{-1} wet weight (highest PBDE concentration detected in the study), a maximum PBDE intake of 148 ng per day was estimated for the population consuming this fish [39]. In turn, for consumption of the same amount of Swiss farmed trout, intake of PBDEs was estimated 26 ng per day. Anyhow, it should be noted in both cases that PBDE intake corresponded to fish consumption only. However, in relation to it Ohta et al. [29] found a strong positive relationship between PBDE concentrations in human milk and the dietary intake of fish and shellfish.

A summary of data on human exposure to PBDEs through the diet is given in Table 2. The comparison of these daily intakes with the suggested low observed adverse effect level (LOAEL) value of 1 mg kg^{-1} per day for PBDEs [49], results in a safety factor of some orders of magnitude in relation to exposure to these pollutants through the diet. However, in a recent investigation on the overall daily exposure to PBDEs, Wijesekera et al. [33] found that diet and inhalation contributed with 73 and 27%, respectively. It would indicate that in addition to special dietary habits, the safety factor could be also remarkably influenced by other routes of PBDE exposure.

5. Summary and research directions

A number of studies in human tissues and fluids have shown that, in general terms, PBDE levels have remarkable increased in recent years [16,18,25,52]. However, some PBDEs have been banned in Europe, and levels in countries in which their use has already been discontinued are dropping [53]. For example, Lind et al. [26] recently reported a peak in PBDE concentrations found in breast milk of Swedish women around 1998 and thereafter decreasing levels. Anyhow, PBDE concentrations are still comparatively lower than those of other environmental pollutants of similar chemical characteristics such as PCBs [16].

However, rising body burdens of endocrine-disrupting chemicals (including PBDEs) may pose a potential public health threat. The diet is very probably the main way of human exposure to PBDEs. Taking into account that information concerning the occurrence of PBDEs in foodstuffs and the dietary intake of these pollutants is scarce, studies to determine PBDE exposure through the diet by the general population of a number of countries are clearly necessary. At the same time, the systematic monitoring of body burdens of PBDEs and the correlation with the dietary intake of these contaminants would be also an issue of interest.

Moreover, to determine individual toxic equivalency factors (TEF) and the contribution to the most predominant PBDE congeners to the TEQs (toxic equivalents), would be especially relevant. It should be noted that as it occurs with other structurally similar classes of compounds, at least some PBDE congeners are endocrine disrupters.

With respect to PBDE analyses, two world-wide interlaboratory studies were recently organized. Biota and sediment samples together with standard solutions containing unknown concentrations of PBDEs were provided to an important number of laboratories in different countries [54,55]. The results showed a good agreement between the laboratories for BDE 47 and 100, while for the BDEs 99, 153 and 154 further information is required. Particularly for BDE 99, a better resolution is required to separate this BDE from interferences or other BDEs. In turn, the analysis of BDE 209 was not under control in most laboratories, while most other BDEs were present at concentration between the current detection limits of the laboratories. Recently, analytical methods for the determination of brominated flame retardants, with a special emphasis on PBDEs were widely reviewed by Covaci et al. [56].

References

[1] K. Hooper, T.A. McDonald, Environ. Health Perspect. 108 (2000) 387.
[2] F. Rahman, K.H. Langford, M.D. Scrimshaw, J.N. Lester, Sci. Total Environ. 275 (2001) 1.
[3] T.A. McDonald, Chemosphere 46 (2000) 745.
[4] M. Alaee, P. Arias, A. Sjödin, A. Bergman, Environ. Int. 29 (2003) 683.
[5] G. Lindström, H. Wingfors, M. Dam, B. van Bavel, Arch. Environ. Contam. Toxicol. 36 (1999) 355.
[6] S. Rayne, M.G. Ikonomou, B. Antcliffe, Environ. Sci. Technol. 37 (2003) 2847.
[7] C.A. de Wit, Chemosphere 46 (2002) 583.
[8] P.O. Darnerud, Environ. Int. 29 (2003) 841.
[9] A.M.C.M. Pijnenburg, J.W. Everts, J. de Boer, J.P. Boon, Rev. Environ. Contam. Toxicol. 141 (1995) 1.
[10] P.O. Darnerud, A. Thuvander, Toxicol. Environ. Chem. 70 (1999) 229.
[11] J. Legler, A. Brouwer, Environ. Int. 29 (2003) 879.
[12] P. Eriksson, E. Jakobsson, A. Fredriksson, Environ. Health Perspect. 109 (2001) 903.
[13] T. Zhou, M.M. Taylor, M.J. de Vito, K.M. Crofton, Toxicol. Sci. 66 (2002) 105.
[14] I. Branchi, F. Capone, E. Alleva, L.G. Costa, Neurotoxicology 24 (2003) 449.
[15] D. Meironyté Guvenius, A. Bergman, K. Norén, Arch. Environ. Contam. Toxicol. 40 (2001) 564.
[16] D. Meironyté Guvenius, A. Aronsson, G. Ekman-Ordeberg, A. Bergman, K. Norén, Environ. Health Perspect. 111 (2003) 1235.
[17] M. Meneses, H. Wingfors, M. Schuhmacher, J.L. Domingo, G. Lindström, B. van Bavel, Chemosphere 39 (1999) 2271.
[18] K. Akutsu, M. Kitagawa, H. Nakazawa, T. Makino, K. Iwazaki, H. Oda, S. Hori, Chemosphere 53 (2003) 645.
[19] K. Hooper, J. She, Environ. Health Perspect. 111 (2003) 109.
[20] M. Petreas, J. She, F.R. Brown, J. Winkler, G. Windham, E. Rogers, G. Zhao, R. Bhatia, M.J. Charles, Environ. Health Perspect. 111 (2003) 1175.
[21] A. Schecter, M. Pavuk, O. Päpke, J.J. Ryan, L.S. Birnbaum, Environ. Health Perspect. 111 (2003) 1723.
[22] D. Meironyté, K. Norén, A. Bergman, J. Toxicol. Environ. Health 58 (1999) 329.
[23] A. Covaci, J. de Boer, J.J. Ryan, P. Voorspoels, P. Schepens, Environ. Res. 88 (2002) 210.
[24] M. Alaee, Environ. Monit. Assess. 88 (2003) 327.
[25] J.W. Choi, S. Fujimaki, K. Kitamura, S. Hashimoto, H. Ito, N. Suzuki, S.I. Sakai, M. Morita, Environ. Sci. Technol. 37 (2003) 817.
[26] Y. Lind, P.O. Darnerud, S. Atuma, M. Aune, W. Becker, R. Bjerselius, S. Cnattingius, A. Glynn, Environ. Res. 93 (2003) 186.
[27] R.E. Alcock, A.J. Sweetman, K. Prevedouros, K.C. Jones, Environ. Int. 29 (2003) 691.
[28] I. Watanabe, S.I. Sakai, Environ. Int. 29 (2003) 665.
[29] S. Ohta, D. Ishikuza, H. Nishimura, T. Nakao, A. Aozasa, Y. Shimidzu, F. Ochiai, T. Kida, M. Nishi, H. Miyata, Chemosphere 46 (2002) 689.
[30] A. Sjödin, L. Hagmar, E. Klasson-Wehler, J. Björk, A. Bergman, Environ. Health Perspect. 108 (2000) 1035.
[31] A. Sjödin, D.G. Patterson Jr., A. Bergman, Environ. Int. 29 (2003) 829.
[32] I.P. Boon, W.E. Lewis, M.R.T.A. Choy, C.R. Allchin, R.J. Law, J. de Boer, C.C.T. Hallers-Tjabbes, B.N. Zegers, Environ. Sci. Technol. 36 (2002) 4025.
[33] R. Wijsekera, C. Halliwell, S. Hunter, S.A. Harrad, Organohalogen Compd. 55 (2002) 239.
[34] A. Johnson, N. Olson, Arch. Environ. Contam. Toxicol. 41 (2001) 339.
[35] R.C. Hale, M.J. La Guardia, E.P. Harvey, T.M. Manoir, W.H. Duff, M.O. Gaylor, Environ. Sci. Technol. 35 (2001) 4585.
[36] J.B. Manchester-Neesvig, K. Valters, W.C. Sonzogni, Environ. Sci. Technol. 35 (2001) 1072.
[37] C.P. Rice, S.M. Chernyak, L. Benoche, R. Quintal, J. Hickey, Chemosphere 49 (2002) 731.
[38] N.G. Dodder, B. Strandberg, R.A. Hites, Environ. Sci. Technol. 36 (2002) 146.
[39] M. Zennegg, M. Kohler, A.C. Gerecke, P. Schmid, Chemosphere 51 (2003) 545.
[40] K. Akutsu, H. Obana, M. Okihashi, M. Kitagawa, H. Nakazawa, Y. Matsuki, T. Makino, H. Oda, S. Hori, Chemosphere 44 (2001) 1325.
[41] J.H. Christensen, J. Platz, J. Environ. Monit. 3 (2001) 543.
[42] J.H. Christensen, M. Glasius, M. Pécseli, J. Platz, G. Pritzl, Chemosphere 47 (2002) 631.
[43] F.R. Brown, J. Winkler, P. Visita, J. Dhaliwal, M. Petreas, Organohalogen Compd. 61 (2003) 117.
[44] S. Voorspoels, A. Covaci, P. Schepens, Environ. Sci. Technol. 37 (2003) 4348.
[45] P.O. Darnerud, S. Atuma, M. Aune, W. Becker, A. Wicklund-Glynn, Toxicol. Lett. 116 (Suppl. 1) (2000) 28.
[46] J.K. Huwe, M. Lorentzsen, K. Thuresson, A. Bergman, Chemosphere 46 (2002) 635.
[47] H. Miyata, T. Nakao, O. Aozasa, S. Ohta, Organohalogen Compd. 32 (1997) 130.
[48] A. Bocio, J.M. Llobet, J.L. Domingo, J. Corbella, A. Teixidó, C. Casas, J. Agric. Food Chem. 51 (2003) 3191.
[49] P.O. Darnerud, G.S. Eriksen, T. Jóhannesson, P.B. Larsen, M. Viluksela, Environ. Health Perspect. 109 (Suppl. 1) (2001) 49.
[50] Y. Lind, M. Aune, S. Atuma, W. Becker, R. Bjerselius, A. Glynn, P.O. Darnerud, Organohalogen Compd. 58 (2002) 181.
[51] J.J. Ryan, B. Patry, Organohalogen Compd. 51 (2001) 226.
[52] R.A. Hites, Environ. Sci. Technol. 38 (2004) 945.
[53] K. Betts, Environ. Sci. Technol. 37 (2003) 164A.
[54] J. de Boer, W.P. Cofino, Chemosphere 46 (2002) 625.
[55] J. de Boer, D. E Wells, K. Noren, Organohalogen Compd. 58 (2002) 197.
[56] A. Covaci, S. Voorspoels, J. de Boer, Environ. Int. 29 (2003) 735.