# DIETARY EXPOSURE AND SERUM PBDE CONCENTRATIONS CORRELATE WELL AMONG CONSUMERS OF FISH FROM A PBDE CONTAMINATED LAKE

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**Abstract**

Very high concentrations of polybrominated diphenyl ethers (PBDEs) have been reported in fish from Lake Mjøsa in Norway. This study on 66 hobby fishermen and women shows that the individual calculated dietary exposure based on PBDE concentrations measured in food and food frequencies correlate quite well with the measured serum concentrations for several of the lower brominated PBDEs. The median dietary intake of Sum 7 PBDEs was 30 ng/kg body weight/day, the highest dietary intake of PBDEs reported. The contribution from fish caught from the contaminated lake comprised 98.7% of the total dietary exposure. The HBCD serum concentration was also correlated to the calculated PBDE intake. The BDE-209 serum level on the other hand, was neither correlated with the calculated BDE-209 dietary exposure nor to any of the exposure estimates for the other PBDEs. This clearly suggests that sources other than the diet are important for human BDE-209 exposure.

## Introduction

Brominated organic compounds such as polybrominated diphenyl ethers (PBDEs) are used as flame retardants to protect a wide variety of products from catching fire. Several PBDEs have been shown to be persistent organic pollutants (POPs) and are found widespread in the environment, in wildlife and in humans[1,2]. PBDEs are both lipophilic and able to biomagnify, thus intake of fatty fish is supposed to be a major source of human exposure. However, only a few studies have found positive associations between fish consumption and body burdens of PBDEs[3,4].

In Lake Mjøsa, the largest lake in Norway located in the south-eastern part of the country, especially high concentrations of PBDEs have been reported in trout[5,6]. High PBDE levels were also found in perch, pike and burbot[6] from this lake. All these fish species are part of the diet for many people living near the lake. The aim of this study was to calculate the dietary exposure to PBDEs in a group of high consumers of fish from Lake Mjøsa and investigate how well the intake estimates correlate with the subjects' blood levels of PBDEs.

## Materials and Methods

*Study subjects*

This investigation was conducted on 66 subjects from a study organised by the Norwegian Institute of Public Health, to investigate the body burdens of POPs in high consumers of inland fish caught in Lake Mjøsa. The participants were recruited among local hobby fishermen and women. Participants provided serum samples and filled in detailed questionnaires regarding personal background data and dietary habits concerning their regular diet and intake of fish from the lake. The project was approved by the Regional Committees for Medical Research Ethics.

*Calculation of PBDE intake*

An extensive database comprising PBDE concentrations in Norwegian foods has recently been established[7]. A separate database, with concentrations in different fish species from Lake Mjøsa, was also created. Species or foods for which no PBDE measurements were available were assigned a lipid adjusted PBDE content based on a comparable food item. Food frequencies from both questionnaires were converted into consumption (g/day) by multiplying with gender specific portion sizes. The individual dietary intake of PBDEs was calculated by multiplying the consumption with the PBDE level in the respective food. Compounds not detected were assigned the value zero (lower bound (LB) approach). The dietary intake of HBCD could not be calculated due to limited information on HBCD concentrations in fish from Lake Mjøsa. The questionnaires were filled in satisfactory by

65 of the participants, who all fulfilled the inclusion criteria of reporting a daily food consumption corresponding to between 1000 and 4000 kcal/day.

*Chemical analysis*
The PBDE standards (BDE-18, 28, 37, 47, 51, 85, 99, 100, 103, 119, 138, 153, 154, 181, 183 and 209) and hexabromocyclododecane (HBCD) were obtained from Wellington Laboratories (Guelph, Ontario, Canada), CIL (Andover, MA) or AccuStandard (New Haven, CT). All solvents used were of pesticide grade from sds (Peypin, France). The concentrations of PBDEs and HBCD were determined according to a previously described method[8] utilizing automated solid phase extraction and quantification on GC-ECMS. The lipids were determined enzymatically at Haukeland University Hospital (Bergen, Norway) and the total lipid content of the samples calculated according to the method described by Grimvall et al.[9].

**Results and Discussion**
*Calculated dietary exposure*
The participants in this study consisted of a total of 41 men and 25 women of age 9 to 87 years (mean 58 and 54 years). Their mean and median calculated dietary intake of Sum 7 PBDEs from different food groups are presented in Table 1. As can be seen, the contribution from fish caught in the contaminated lake exceeds the other food groups by several orders of magnitude, comprising 98.7% of the total exposure. The mean calculated dietary intake of Sum 7 PBDEs was 47 ng/kg body weight/day and the median 30 ng/kg body weight/day for the whole study group. In comparison, the corresponding mean intake of Sum 7 PBDEs was found to be 1.1 ng/kg body weight/day in a reference group of 44 Norwegians eating only food with background levels of contamination[7]. The two studies utilized the same food frequency questionnaires and database containing PBDE levels in food for calculation of the exposure. In contrast, the calculated mean dietary intake of BDE-209 was in the same order of magnitude in the two groups of Norwegians, i.e. 1.1 ng/kg body weight/day in the present study and 1.4 ng/kg body weight/day in the other study, respectively. An almost equal BDE-209 exposure in the two groups was expected, since the fish in Lake Mjøsa was not particularly contaminated with BDE-209. For the anglers living around Lake Mjøsa, the second most important food sources of Sum 7 PBDEs were commercial oily and semi oily fish (0.54%), followed by fish liver and roe (0.18%) and diary products (0.17%). This relative contribution is in accordance with the findings in the above mentioned study[7]. The average daily dietary intake of PBDEs has recently been estimated in several other countries in the range 23 to 107 ng/day[10-13]. The corresponding mean value for the local hobby fishermen and women is 3981 ng/day (median 2549 ng/day), which clearly shows their strongly elevated exposure due to consumption of fish from the contaminated Lake Mjøsa.

Table 1. The calculated dietary intake of Sum 7 PBDEs (ng/kg body weight/day) from different food groups.

| | Mean (all) | Median (all) | Mean (men) | Median (men) | Mean (women) | Median (women) | Min | Max |
|---|---|---|---|---|---|---|---|---|
| **Dairy products** | 0.081 | 0.066 | 0.083 | 0.065 | 0.079 | 0.067 | 0 | 0.30 |
| **Hen eggs** | 0.025 | 0.018 | 0.024 | 0.018 | 0.027 | 0.020 | 0 | 0.10 |
| **Meat** | 0.068 | 0.063 | 0.068 | 0.067 | 0.067 | 0.054 | 0 | 0.16 |
| **Fish, lean** | 0.038 | 0.031 | 0.038 | 0.032 | 0.037 | 0.029 | 0 | 0.18 |
| **Fish, oily and semi oily** | 0.26 | 0.22 | 0.31 | 0.24 | 0.18 | 0.18 | 0 | 1.9 |
| **Fish, liver and roe** | 0.086 | 0.001 | 0.089 | 0.0007 | 0.082 | 0.0033 | 0 | 0.86 |
| **Shellfish** | 0.0017 | 0.0005 | 0.0019 | 0.0005 | 0.0013 | 0.0005 | 0 | 0.027 |
| **Vegetable oils and fats** | 0.014 | 0.013 | 0.016 | 0.014 | 0.012 | 0.0086 | 0 | 0.043 |
| **Seagull eggs** | 0.031 | 0 | 0.035 | 0 | 0.026 | 0 | 0 | 0.74 |
| **Bread, cereals, nuts, greens** | 0.0090 | 0.0066 | 0.0044 | 0.0029 | 0.016 | 0.013 | 0 | 0.039 |
| **Sweets, dry foods, beverages** | 0.0048 | 0.0029 | 0.0042 | 0.0027 | 0.0057 | 0.0042 | 0 | 0.024 |
| **Fish from lake Mjøsa** | 47 | 29 | 58 | 36 | 31 | 20 | 0 | 260 |
| **Total diet** | 48 | 30 | 58 | 37 | 32 | 20 | 0.3 | 260 |

*Correlations between calculated dietary exposure and serum levels*

The calculated exposure to Sum 7 PBDEs of fish from Lake Mjøsa and the measured Sum 7 PBDE concentrations in serum are clearly correlated as shown in Figure 1. The correlations between serum levels and the total calculated dietary exposure for all the individual brominated flame retardants (BFRs) are presented in Table 2. For men, the calculated dietary exposure on congener basis correlated well with the measured serum level for the major congeners in the pentaBDE mixture, BDE-47, 99, 100, 153 and 154, both on individual congener basis and between the different congeners. The relationships were more pronounced than what was found in the previously mentioned study on persons eating only food with background levels of contamination[7]. This may be explained by the relatively high exposure from the trout. The dietary exposure to HBCD was not calculated due to limited data on HBCD levels in fish from Lake Mjøsa. Nevertheless, HBCD serum levels correlated well with intakes of all PBDEs except for BDE-209 (Table 2), indicating that HBCD exposure occurs through the same pathways as the lower brominated PBDEs. The BDE-209 serum level on the other hand, was neither correlated with the calculated BDE-209 dietary exposure nor to any of the exposure estimates for the other PBDEs. This clearly suggests that sources other than the diet are important for human BDE-209 exposure. Several recent publications have pointed towards the indoor environment as a likely exposure source that should be further investigated[13-14].

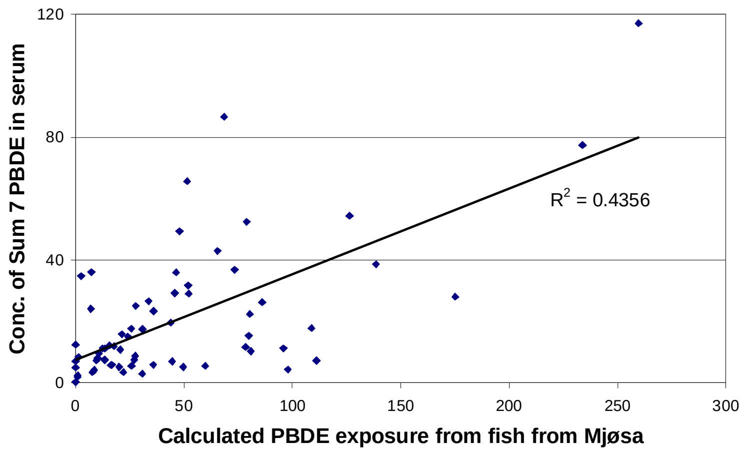


Figure 1. The relationship between calculated exposures to Sum 7 PBDEs (ng/kg body weight/day) from fish from Lake Mjøsa and the Sum 7 PBDEs concentrations in serum (ng/g lipids).

For women, only serum levels of BDE-47 and 99 were significantly correlated with the calculated dietary exposure (Table 2). We suspect that the striking and substantial differences between males and females may be caused by excretion of PBDEs during lactation periods, however, we found no correlations between the numbers of breast fed children and serum levels in this study. As shown in Table 1, the calculated dietary intake of Sum 7 PBDEs were about twice as high for men compared to women, which might explain why weaker correlations are found between calculated dietary exposure and serum levels for women.

The significant correlations in Table 2 have been confirmed using adjusted linear regression, where also age and gender was taken into account. When including age and gender, a significant relation between the observed serum level and the calculated dietary exposure was found also for BDE-28. In addition, age was positively and significantly related to the serum level for BDE-47, 99, 100 and 153. The serum levels of several of the lower brominated PBDEs in this study group are significantly higher than in a reference group of 44 Norwegians eating only food with background levels of contamination[15].

Table 2. Spearman rank correlation coefficients between BFR concentrations in serum and the calculated dietary intake of the corresponding congener for males (M) and females (F).

| Serum concentration | | Calculated dietary intake: BDE-28 | BDE-47 | BDE-100 | BDE-99 | BDE-154 | BDE-153 | BDE-209 | Sum 7 PBDEs |
|---|---|---|---|---|---|---|---|---|---|
| BDE-28 | M | 0.29 | 0.29 | 0.27 | **0.32**[b] | 0.28 | 0.29 | 0.01 | 0.30 |
| | F | 0.24 | -0.16 | -0.17 | -0.21 | -0.18 | -0.17 | -0.36 | -0.18 |
| BDE-47 | M | **0.57**[a] | **0.57**[a] | **0.58**[a] | **0.59**[a] | **0.57**[a] | **0.58**[a] | -0.07 | **0.58**[a] |
| | F | **0.45**[b] | **0.50**[b] | **0.48**[b] | **0.47**[b] | **0.48**[b] | **0.47**[b] | -0.36 | **0.49**[a] |
| BDE-100 | M | **0.55**[a] | **0.56**[a] | **0.56**[a] | **0.57**[a] | **0.55**[a] | **0.55**[a] | -0.04 | **0.56**[a] |
| | F | 0.31 | 0.32 | 0.30 | 0.29 | 0.30 | 0.28 | -0.19 | 0.31 |
| BDE-99 | M | **0.52**[a] | **0.52**[a] | **0.53**[a] | **0.54**[a] | **0.52**[a] | **0.52**[a] | -0.12 | **0.53**[a] |
| | F | **0.44**[b] | **0.48**[b] | **0.47**[b] | **0.45**[b] | **0.47**[b] | **0.45**[b] | -0.20 | **0.47**[b] |
| BDE-154 | M | **0.56**[a] | **0.57**[a] | **0.57**[a] | **0.57**[a] | **0.56**[a] | **0.57**[a] | -0.04 | **0.57**[a] |
| | F | 0.17 | 0.13 | 0.12 | 0.09 | 0.12 | 0.08 | -0.15 | 0.13 |
| BDE-153 | M | **0.46**[a] | **0.46**[a] | **0.45**[a] | **0.47**[a] | **0.45**[a] | **0.45**[a] | 0.10 | **0.46**[a] |
| | F | -0.002 | 0.004 | -0.02 | -0.04 | -0.02 | -0.04 | 0.04 | -0.01 |
| BDE-209 | M | -0.28 | -0.28 | -0.28 | -0.23 | -0.28 | -0.27 | 0.10 | -0.27 |
| | F | -0.03 | -0.01 | -0.005 | 0.02 | -0.01 | 0.02 | 0.05 | 0.002 |
| Sum 7 PBDEs[c] | M | **0.56**[a] | **0.57**[a] | **0.57**[a] | **0.58**[a] | **0.56**[a] | **0.57**[a] | -0.01 | **0.57**[a] |
| | F | 0.27 | 0.28 | 0.26 | 0.25 | 0.26 | 0.24 | -0.33 | 0.27 |
| HBCD | M | **0.48**[a] | **0.47**[a] | **0.48**[a] | **0.49**[a] | **0.46**[a] | **0.48**[a] | -0.10 | **0.47**[a] |
| | F | 0.23 | 0.25 | 0.23 | 0.22 | 0.23 | 0.23 | -0.15 | 0.24 |

[a]Correlation is significant at the 0.01 level (2-tailed).
[b]Correlation is significant at the 0.05 level (2-tailed).
[c]Sum 7 PBDEs comprises BDE-28, 47, 99, 100, 153, 154 and 183.

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