PBDE Contamination of U.S. Food and Human Milk; and PBDE, PCDD/F, PCB, and Levels in U.S. Human Blood (1973 and 2003)

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Introduction

Historical exposure trends to persistent organic pollutants (POPs) indicate that the primary route of exposure in humans is traditionally dietary intake, contributing to a steady-state body burden of these lipophilic chemicals, which are also transported to offspring during nursing. Concentrations found in humans often mimic what is found in the consumer product or environment and typically display a lag in the time trend, representing time for transport of the chemicals from the source of release to the source of exposure. Recently, the increase of polybrominated diphenyl ethers (PBDEs) in humans has raised particular concern due to their association with endocrine disruption, reproductive toxicity, and developmental neurotoxicity in rodent studies¹. PBDEs have been used commercially for several decades in the US; however the primary route of exposure is presently unclear, but is assumed to be through dietary intake. In this study, blood levels of several POPs, including PBDEs, PCBs, and PCDD/Fs, are compared and a preliminary analysis of PBDEs in US food is presented. These data can be compared to previously published human blood and breast milk PBDE levels; this comparison provides additional information which correlates human exposure and resultant PBDE body burdens in the US.

Materials and Methods

Sample Collection: Milk (N=52, 2003) and blood samples (1973 and 2003) were obtained from medical facilities in Texas (serum N=100, one pooled analysis per time period) or Mississippi (N=29, individual analyses)². Food samples of animal origin were purchased from three different supermarket chains in Dallas, Texas and presumed to be representative of the foods eaten by the people from which the milk and blood samples were collected.
Analysis: Chemical evaluation of individual PCB, PBDE, and PCDD/F congeners were determined. Briefly, thirteen individual PBDE congeners (BDEs 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, and 209) were analyzed in human milk, food and blood samples by an isotope dilution method using GC-MS as previously described\(^2\). Dioxin analytic methods have been described elsewhere\(^3\). PCDD, PCDF and PCB TEQ values were calculated using current WHO TEFs\(^4\). For food NDs were calculated as equaling zero. For human milk and blood, ½ DL was used for NDs.

Results

U.S. blood serum (1973 and 2003) of several POPs collected over time are compared in Figure 1. Results indicate that PCB (TEQ) levels have declined in the past three decades as demonstrated by pooled serum concentrations of 64.7 ppt lipid in 1973 and 9.3 in 2003. PCDD/F (TEQ) levels have also declined rapidly; in 1973, the pooled concentration was 85 ppt lipid and was 15.8 in 2003. In contrast, PBDEs have increased dramatically: these chemicals were not detected in samples from 1973 (and therefore calculated as 0.77 ppb, which is equal to half the limit of detection). In 2003, pooled serum concentrations were 61.7 ppb lipid. These blood levels can be compared to previously published breast milk data\(^2\). Concentrations in milk vary from 6.2 to 419 with a median of 34 ppb, whereas concentrations in whole blood (N=29, data not shown) vary from 5.5 to 351 ppb with a median of 31 ppb lipid in this sample.

Currently, it is assumed that dietary exposure is the primary route through which humans are exposed to PBDEs. In this market basket survey, food samples of animal origin were analyzed for the presence and concentration of the same PBDEs that were analyzed in blood and milk. Results and congener profiles are shown in Figures 2-4 on a wet weight (ww) basis. Collectively, fish have the highest levels of PBDEs (median = 1,725 ppt) while also displaying a large variation in total PBDE concentrations (8.5 to 3,078 ppt). Salmon and catfish have the highest levels where as tilapia have the lowest, this is true even if concentrations are expressed on a lipid basis. Meat products had a median PBDE concentration of 283 ppt. The variation in these samples was also large: bacon had the lowest levels (ND) while pork sausage (1373 ppt) was the highest on a wet weight basis. Figure 5 shows PBDE levels in dairy products (median value = 31.5 ppt). When the samples are collectively analyzed, congener profiles are dominated by BDE 47, followed by BDE 99. It is interesting to note that in several samples, BDE 209 was the dominant congener or was a significant contributor to the sum PBDE concentration. Further analysis can be used to estimate dietary intake from a U.S. based market survey.

Table 1 shows PBDE estimated intake of nursing infants in Germany and the USA. These calculations indicated that nursing infants are exposed to 355 ng/kg/day total PBDEs in the US; where as German infants are only exposed to 11 ng/kg/day\(^5\).
Discussion

In agreement with reported trends for POPs, the results of this study indicate that concentrations of PBDEs in milk and blood samples from the US are much higher in 2003 than compared to thirty years ago whereas concentrations of dioxins, dibenzofurans and PCBs are declining. Levels in Sweden have already peaked which is most likely due to the voluntary ban on PBDEs several years prior to the observed peak.6

To date, there have not been any studies reporting levels of PBDEs in food consumed by the U.S. general population. Here we report that PBDEs are found in almost all foods of animal origin; and some have very high levels of these chemicals. These results indicate that dietary intake is mostly likely a primary route of exposure. Furthermore, concentrations of PBDEs in food are much higher in the US than other parts of the world.7

Dietary intake for nursing infants was also calculated. Because of the predicted toxicity, it is important to understand the levels of exposure to developing offspring. It is clear that humans are exposed to PBDEs through a variety of sources, one of which is dietary intake, and that nursing mothers’ milk contains relatively high levels of these lipophilic chemicals. The higher levels consumed by U.S. nursing infants than by German infants are striking. These results should not come as a surprise considering that the available literature all suggest that exposure levels in the US are several orders of magnitude higher than Europeans.2,6,7 PBDE contamination of food probably contributes significantly to the steady state body burdens currently detected in humans, which ultimately leads to placental transport, as well as lactational exposure, to developing infants.

Acknowledgements

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References


**Figure 1.** PCDD/PCDF TEQ (ppt), PCB TEQ (ppt), and PBDE (ppb) concentrations in serum samples from 1973 and 2003.
Figure 2. PBDE congener levels (median value = 1725 ppt) and profiles in USA fish products (ppt w.w.).

Figure 3. PBDE congener levels (median value = 283 ppt) and profiles in USA meat products (ppt w.w.).
Figure 4. PBDE congener levels (median value = 31.5 ppt) and profiles in USA dairy products (ppt w.w.).

Table 1. Estimated PBDE intake in nursing infants in Germany and USA*

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<tr>
<th></th>
<th>Germany</th>
<th>USA</th>
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<tr>
<td>Consumed milk/day (ml)</td>
<td>800</td>
<td>800</td>
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<tr>
<td>Lipid content of milk (%)</td>
<td>3</td>
<td>3</td>
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<tr>
<td>Body weight of infant (kg)</td>
<td>5</td>
<td>5</td>
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<tr>
<td>Concentration (ng/g milk fat)</td>
<td>2.3</td>
<td>74</td>
</tr>
<tr>
<td>Total PBDE intake ng/day</td>
<td>55.2</td>
<td>1774</td>
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<tr>
<td>PBDE intake/day in ng/kg BW</td>
<td>11</td>
<td>355</td>
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*USA milk from 52 samples of human milk.