Polybrominated diphenyl ethers (PBDEs) are used as flame retardant additives in polymers with a wide variety of applications, for example, electronic equipment, construction materials, and textiles [World Health Organization (WHO) 1997]. In recent years it has become evident that certain PBDEs are generally found in humans, as are other well-known environmental pollutants such as polychlorinated biphenyls (PCBs) (WHO 1992) and pentachlorophenol (PCP) (WHO 1987). Although decabromodiphenyl ether is the dominating commercial PBDE product, the environmental occurrence is dominated by lower brominated PBDE congeners, substituted with fewer than seven bromine atoms (reviewed by Bergman et al. 2002; Darnerud et al. 2001; de Boer et al. 2000; de Wit 2002). Several studies have reported PBDE levels in human blood (Schröter-Kermani et al. 2000; Sjödin et al. 1999, 2001; Thomsen et al. 2001), liver (Meironyté Guvenius et al. 2001), and milk (Fürst 2001; Meironyté et al. 1999, 2001), liver (Meironyté Guvenius et al. 2001), and milk (Fürst 2001; Meironyté et al. 1999, 2001), breast milk, blood plasma, and breast milk. In the present study, we investigated exposures to PBDEs, PCBs, OH-PCBs, and PCP by comparing the levels of these compounds in human maternal blood plasma, cord blood plasma, and breast milk. Our overarching aim was to determine fetal and infant exposures for these compounds.

### Materials and Methods

**Samples.** Samples of maternal blood plasma, cord blood plasma, and breast milk were collected from 72 pregnant women during the third trimester of pregnancy. The samples were collected between July 2000 and February 2001. The mothers were recruited from the Department of Obstetrics and Gynecology at Karolinska Hospital in Stockholm, Sweden.

**Experimental design.** Samples were collected at the following time points: 1. **Weekly** during pregnancy to assess levels in maternal blood plasma and cord blood plasma, 2. **Monthly** until 6 months postpartum to assess levels in maternal blood plasma, cord blood plasma, and breast milk. The samples were stored at −20°C until analysis.

**Analytical methods.** PBDEs, PCBs, OH-PCBs, and PCP were determined using gas chromatography/mass spectrometry (GC/MS) analysis. The PBDEs were analyzed using electron capture negative ionization mode (ECNI) with a Thermoquest Finnigan (San Jose, CA) Trace GC/Trace MS system. The PBDEs were identified by comparing their mass spectra with those of standards. The PCBs were analyzed using electron impact ionization mode (EI) with a Thermoquest Finnigan Trace GC/Thermoquest Trace MS system. The PCBs were identified by comparing their mass spectra with those of standards.

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collected during 2000–2001 from 15 mothers living in Stockholm, 13 of whom were native Swedish. The study population was randomly chosen from those who voluntarily agreed to participate in the investigation. All mothers delivered by cesarean surgery, according to their own wish. The mothers were healthy and delivered healthy babies. The participants were asked to answer a questionnaire about age, number of children, earlier place of residence, fish consumption, and other factors. The average maternal age was 32 years (range, 28–38); 53% of them gave birth to their first child, 33% their second, and 14% their third. One mother reported eating eight meals of fatty fish per month; the others reported zero to two. Their consumption of fatty fish from the Baltic was low; one mother reported two meals per month.

Blood samples were collected at the Karolinska Hospital into heparinized Venovject glass tubes (Terumo Europe N.V., Leuven, Belgium). Blood samples (30 mL) from the mothers were collected when they arrived at the hospital for delivery, and cord blood (15–20 mL) was collected at delivery. The fraction containing PCBs and PBDEs was Concentrated under reduced pressure to 2 mL and transferred to a glass tube. The flask was rinsed with methanol (2 x 0.5 mL), followed by hexane (3 x 1 mL). The mixture was shaken cautiously with water (3 mL) and centrifuged. The hexane phase was transferred to another tube, and the aqueous phase was shaken twice more with hexane (3 and 2 mL). The combined hexane phases were concentrated with a gentle stream of nitrogen to approximately 100 µL. Five drops of methanol were added, and the phenolic compounds were derivatized with diazomethane in diethyl ether (0.5 mL). The mixture was left to react overnight. The solvent was evaporated with.

Table 1. PBDE concentrations (median and range, ng/g lipids) in maternal blood plasma, cord blood plasma, and breast milk from 15 individuals.

| PBDE congeners | Maternal blood plasma | Cord blood plasma | Breast milk |
|----------------|-----------------------|-------------------|------------|
|                | Median | Range | Median | Range | Median | Range |
| BDE-17         | <0.01  | <0.01–0.03 | <0.01  | <0.01–0.1 | <0.01  | <0.01 |
| BDE-28         | 0.07   | 0.01–0.2  | 0.07   | 0.01–0.31 | 0.06   | 0.02–0.18 |
| BDE-47         | 0.83   | 0.3–5.1  | 0.98   | 0.3–3.28 | 1.15   | 0.26–4.01 |
| BDE-66         | 0.02   | 0.01–0.14 | 0.01   | 0.01–0.11 | 0.02   | 0.01–0.07 |
| BDE-100        | 0.17   | 0.01–0.52 | 0.07   | 0.01–0.27 | 0.14   | 0.01–0.09 |
| BDE-99         | 0.19   | <0.01–1.43 | 0.07   | <0.01–0.89 | 0.21   | 0.07–2.20 |
| BDE-85         | <0.01  | <0.01–0.07 | <0.01  | <0.01–0.09 | 0.04   | <0.01–0.17 |
| BDE-154        | 0.04   | 0.01–0.16 | <0.01  | 0.01–0.17 | 0.02   | <0.01–0.14 |
| BDE-153        | 0.56   | 0.27–1.03 | 0.17   | 0.01–3.32 | 0.32   | 0.03–1.16 |
| Sum            | 0.06   | 0.01–0.44 | 0.01   | <0.01–0.01 | 0.01   | <0.01–0.14 |

Values in parentheses are on fresh-weight basis (pg/g fresh weight).

Methods. Organic solvents and adsorbents used in the analysis were prepared as previously described (Meironyté et al. 1999; Meironyté Guvenius et al. 2002).

PBDE standards BDE-85, BDE-99, BDE-153, and BDE-77 were purchased from CIL (Andover, MA, USA). BDE-17, BDE-28, BDE-47, BDE-66, BDE-100, and BDE-154 were synthesized as described elsewhere (Marsh et al. 1999; Orn et al. 1996). PCB congeners were purchased from Ehrenstorff (Augsburg, Germany). The PBDE and PCB congeners are numbered as suggested by Ballschmiter et al. (1992) for single PCB congeners. The OH-PCB congeners were purchased from Larodan Fine Chemicals AB (Gothenberg, Sweden) or synthesized as described elsewhere (Bergman et al. 1995). OH-PCB congeners in the present work are numbered according to the recommendations of Letcher et al. (2000).

Methoxy-PCBs used for identification and quantification of methylated OH-PCBs were synthesized (Bergman et al. 1995) or derivatized from OH-PCBs as described below. PCP was purchased from Riedel-de Haën AG (Seelze-Hannover, Germany).

Instruments. We used gas chromatography (GC) on a Chrompack CP 2009 instrument (Middelburg, The Netherlands) equipped with an on-column injector and electron capture detector to detect and measure PCBs. We used a mass spectrometer (model VG 70-250; Fisons Instruments, VG Analytical, Manchester, UK), equipped with a Hewlett-Packard gas chromatograph (model HP 5890A; Geneva, Switzerland), for determination of PBDEs, methylated OH-PCBs, and PCP. Further details are given elsewhere (Meironyté et al. 1999; Meironyté Guvenius et al. 2002).

Analysis of blood plasma. The previously described method for analysis of organochlorine compounds in blood plasma (Weistrand et al. 1995) was modified to incorporate determination of PBDEs, OH-PCBs, and PCP. The method was also adapted for a smaller sample volume. Blood plasma (10 mL) was weighed into a 100-ml flask with polytetrafluoroethylene-lined screw cap. In case of smaller sample volumes, water was added to the total volume of 10 mL. A blank sample (10 mL water) was run with each set of samples. Internal standards for each group of analytes (50 µL of 10 pg 13C6-BDE-177/µL hexane, 50 µL of 10 pg 4-OH-CB-162/µL hexane, 100 µL of 90 pg CB-198/µL hexane) were added to all samples. After addition of formic acid (10 µL), the samples were left for 15 min. Then, 2-propanol (4 mL), water (4 mL), and Lipidex 5000 (3 g) were added, and the mixture was shaken in a water bath (35°C) for 3 hr.

After extraction, the mixture was transferred to a glass column, and the gel was eluted with solvents of decreasing polarity. Polar compounds were eluted with 30% methanol in water (10 mL) and 50% methanol in water (10 mL). Organohalogen compounds and some lipids were eluted with acetonitrile (50 mL), and the rest of the lipids, with a mixture of methanol/chloroform/hexane (1/1/1, vol/vol/vol, 20 mL). The fraction containing OH-PCBs was further purified on aluminum oxide column. PBDEs and PCBs were eluted in the subsequent fraction with acidified methanol (1/100 vol/vol sulfuric acid/methanol, 20 mL) and methanol (10 mL). The fraction containing PCBs and PBDEs was concentrated and applied to a silica gel column (0.6 g). PCBs were eluted in the first fraction with dry hexane (4 mL); PBDEs were eluted in the second fraction with 25% dichloromethane in hexane (5 mL). The column containing PBDEs was dried onto a Pasteur pipette column packed with silica gel (0.1 g) and sulfuric acid–prepared silica gel (0.3 g, 2:1 silica gel:90% sulfuric acid). The second column was eluted with an additional 25% dichloromethane in hexane (1.5 mL). The volume of the fraction was reduced by evaporation with a gentle stream of nitrogen to approximately 50 µL. Injection standard (100 µL of 1.1 pg CB-209/µL hexane) was then added, and the sample was analyzed by high-resolution GC/mass spectrometry (GC/MS), using selected ion monitoring (Meironyté et al. 1999).

The fraction containing OH-PCBs was concentrated under reduced pressure to 2 mL and transferred to a glass tube. The flask was rinsed with methanol (2 x 0.5 mL), followed by hexane (3 x 1 mL). The mixture was shaken cautiously with water (3 mL) and centrifuged. The hexane phase was transferred to another tube, and the aqueous phase was shaken twice more with hexane (3 and 2 mL). The combined hexane phases were concentrated with a gentle stream of nitrogen to approximately 100 µL. Five drops of methanol were added, and the phenolic compounds were derivatized with diazomethane in diethyl ether (0.5 mL). The mixture was left to react overnight. The solvent was evaporated with.
nitrogen gas, and the residue was desorbed in hexane. The methylated phenolic compounds were purified on a Pasteur pipette column packed with silica gel (0.1 g) and sulfuric acid–prepared silica gel (0.5 g, 2:1 silica gel:90% sulfuric acid). The analytes were eluted with 70% dichloromethane in hexane (6 mL). The fraction was concentrated with nitrogen to approximately 50 µL; then, injection standard (100 µL of 1.1 pg CB-209/µL hexane) was added before analysis by high-resolution GC/MS (Meironyté Guvenius et al. 2002).

**Analysis of human milk.** Breast milk was analyzed as described elsewhere (Meironyté et al. 1999; Norén and Sjövall 1987), with minor modifications. PBDEs were separated from PCBs using silica gel and purified on a sulfuric acid–prepared silica gel column as described above. Some changes were introduced in order to include analysis of phenolic compounds. OH-PCBs and PCP were eluted from the aluminum oxide column with acidified methanol (1/100 vol/vol sulfuric acid/methanol, 30 mL) and methanol (10 mL) and derivatized as described for the blood plasma samples. The residue obtained after derivatization was dissolved in hexane (2 mL) and shaken with sulfuric acid (90%, 1 mL), and the phases were separated by centrifugation. The sulfuric acid fraction was shaken with hexane (1 mL). The combined hexane phases were concentrated and purified on silica gel and sulfuric acid–prepared silica gel as described above.

### Results

The modified analytical methods were evaluated by recovery studies. Samples were fortified with the PBDE congeners listed in Table 1 (50 pg/g plasma), with PCP (400 pg/g plasma and milk), and with the OH-PCB congeners 4-OH-CB107, 4-OH-CB121, 4-OH-CB130, 4-OH-CB146, 4-OH-CB162, 4-OH-CB172, 4-OH-CB187, and 4-OH-CB193 (50 pg/g plasma and milk) before extraction. The mean recoveries of PBDEs and phenolic compounds were 67–88% (SD, 4–11; n = 4) and 69–97% (SD, 6–24; n = 5), respectively. The average recoveries of the internal standards added before extraction to all samples of maternal blood plasma, cord blood plasma, and breast milk were, respectively, 75, 77, and 84% of [13C]-BDE-77; 77, 80, and 87% of 4-OH-CB162; and 70, 75, and 76% of CB-198.

Samples of maternal blood plasma, cord blood plasma, and breast milk from 15 mothers were analyzed for PBDEs, PCBs, OH-PCBs, and PCP. The median concentrations and ranges of 10 PBDE congeners are given in Table 1, and those of 15 PCB congeners are shown in Table 2. The PCB congeners CB-123 and CB-189 were not detected in any of the samples (detection limit, 0.5 ng/g lipids). The median sums of PBDEs were 2.07, 1.69, and 2.14 ng/g lipids in maternal blood plasma, cord blood plasma, and breast milk, respectively. The PCB levels were two orders of magnitude higher than those of PBDEs, with median values of 176, 104, and 190 ng/g lipids in maternal blood plasma, cord blood plasma, and breast milk, respectively. The median lipid content was 0.7% (range, 0.5–1.4%) in maternal blood plasma, 0.2% (0.2–0.3%) in cord blood plasma, and 1.9% (0.8–4.9%) in breast milk. Because phenolic compounds are retained in blood mainly due to their affinity to plasma proteins and not due to their lipophilic properties (Letcher et al. 2000), the concentrations of OH-PCBs and PCP are given on a fresh-weight basis (Table 3). The median sums of 12 OH-PCB congeners in maternal and cord blood plasma were 124 and 88 pg/g plasma, respectively. 4-OH-CB121 and 3’-OH-CB188 were not found in the samples (detection limit, 0.1 pg/g sample). The OH-PCB levels in breast milk were very low; the median sum was 3 pg/g milk. Calculated on a fresh-weight basis, the ratios of PBDEs to OH-PCBs were 13, 3, and 1,400 in maternal blood plasma, cord blood plasma, and breast milk, respectively. PCP was the predominant phenolic compound in all sample matrices (Table 3). The median levels in maternal blood plasma, cord blood plasma, and breast milk were 2.83, 1.96, and 0.02 ng/g fresh weight, respectively. Figure 1 shows the 10th through 90th percentiles of PBDEs, PCBs, OH-PCBs, and PCP in the blood and breast milk samples. The individual concentrations are shown in Figures 2 and 3. No influence of maternal age, number of nursed children, or time of milk collection was ascertained.

### Discussion

**PBDEs and PCBs.** Although there were large differences in the concentrations of PBDEs and PCBs, the distribution of these classes of compounds was similar between the sample matrices. The levels (nanograms per gram...
lipids) were comparable in maternal blood and breast milk, whereas the levels in the cord blood were generally lower (Figure 1). The sums of PBDEs and PCBs in cord blood plasma constituted, on average, 72 and 70% of the sums in maternal blood plasma, respectively, calculated on lipid weight. The differences were more obvious (21 and 19%) when the comparison was made on a fresh-weight basis. Consequently, the lower lipid content of the fetal blood “protects” the fetus, at least to some extent, from these contaminants from the mother. Further, we found no correlation between PBDE and PCB levels. The highest PCB levels were found in the samples from the mother who consumed fatty fish most frequently (eight meals per month). This is in accordance with previously reported findings that fish is an important source of human exposure (Asplund et al. 1994; WHO 1992). The lowest PCB levels were in samples from two immigrant mothers (Figure 2). No such relations were found for PBDEs. We assume that exposures to PCBs and PBDEs differ, even though the major proportion of PBDEs may also be ingested via the diet, as are PCBs. Further, PCBs have been regulated and not produced for decades, whereas PBDEs are still produced and present in many goods around us. It therefore cannot be ruled out that inhalation may play a more important role for PBDEs than for PCBs. The individuals participating in the present study were from the general population with no known specific exposure to PBDEs. Therefore, the concentrations in breast milk and blood plasma may be considered as background levels and are in the range of recently reported levels in human milk from Sweden (Meironyté et al. 1999), Finland (Strandman et al. 2000), and Germany (Fürst 2000; Sala et al. 2001). The present study confirms the transfer of PCBs to the fetus.

**Hydroxylated PCB metabolites and PCP.** The distribution of OH-PCBs and PCP in the samples differed from those of PBDEs and PCBs. The highest levels of phenolic compounds were found in maternal blood plasma, and the lowest in breast milk (Figure 1). This is not surprising because the distribution in the blood is entirely different, the former being bound to proteins and the latter localized to the blood lipids.

PCP was the dominating phenolic compound in all samples, with notably high concentrations in the blood plasma samples (Table 3). The levels of PCP in cord blood plasma correlated well with those in maternal blood plasma \((r = 0.73, p < 0.01)\) and constituted 67% of the levels in maternal blood plasma. The PCP levels in maternal and cord blood plasma were, on average, 30 and 36 times higher than the sum of OH-PCBs. The OH-PCB levels in cord blood plasma correlated well to those in maternal blood plasma \((r = 0.60, p < 0.05)\) and constituted 62% of the levels in maternal blood plasma. The concentrations of OH-PCB congeners in cord blood plasma were lower than the previously reported levels in cord blood plasma from coastal populations in Quebec, whereas the PCP levels were similar (Sandau et al. 2002). It is evident from the present study that exposure to PCP and OH-PCBs is only slightly higher in the mother than in the fetus. This behavior of OH-PCBs stands in contrast to that of parent PCBs. The results imply that the potential health impact of halogenated phenolic compounds may have hitherto been underestimated compared with the impact of neutral persistent chemicals.

The predominant OH-PCB congeners in blood samples were 4-OH-CB187 and 4-OH-CB146, followed by 4-OH-CB107 and 3′-OH-CB138 (Table 3). The similar congener pattern was previously reported in other studies from Sweden (Bergman et al. 1994; Sjödin et al. 2000), Faroe Islands (Fängström et al. 2001), and coastal populations in Quebec (Korrick et al. 2000; Bjerregaard and Hansen 2000; Sjödin et al. 2000; Sala et al. 2001). The present study confirms the transfer of PCBs to the fetus.

**Figure 1.** The 10th–90th percentiles (boxes), median (solid line), and range (whiskers) of the sums of (A) PBDEs, (B) PCBs, (C) OH-PCBs, and (D) PCP in maternal blood plasma, cord blood plasma, and breast milk. OH-PCB and PCP levels in breast milk have been multiplied by 10 and 100, respectively. In (D), the cut-off level of PCP (0.57 ng/g milk).
et al. 2002), and Canada (Sandau et al. 2000, 2002). A somewhat different pattern of OH-PCB congeners was reported in blood from Latvian men (Sjödin et al. 2000), where 4-OH-CB107 occurred at the highest levels. Hydroxylated metabolites of PCBs are formed from PCBs by cytochrome P450–mediated direct hydroxylation or via formation of an arene oxide (reviewed by Letcher et al. 2000). Several OH-PCBs may be formed from certain PCB congeners; for example, 4-OH-CB146 can be formed from CB-138 and CB-153, 4-OH-CB107 from CB-118 and CB-105, and 4-OH-CB187 from CB-187 (Sjödin et al. 1998b). Possibly, 4-OH-CB187 may also be formed from CB-183. However, it is not yet possible to predict the relative contribution of different PCB congeners to each one of the specific OH-PCB metabolites. All the identified OH-PCB congeners have an OH group in para- or meta-position, with two chlorine atoms on the neighboring carbon atoms. These OH-PCBs have structural similarities to T₄ and have high competitive binding potency to transthyretin (Lans et al. 1993). Even though thyroid-binding globulin is the major T₄ transporting protein in humans, transthyretin also plays a role, particularly during fetal development (Brouwer et al. 1998). The interaction between transthyretin and OH-PCBs and other halogenated phenolic compounds suggests a plausible mode of endocrine-mediated actions of these compounds that could make them important for in-depth studies.

The levels of OH-PCBs and PCBs correlated well in cord blood plasma samples ($r = 0.78, p < 0.01$). OH-PCBs constituted 5–26% and 14–53% of the sum of PCBs in maternal and cord blood plasma samples, respectively, calculated on a fresh-weight basis. The high percentage of OH-PCBs in cord blood suggests that OH-PCBs may pass the placenta to a higher extent than do PCBs or possibly that they may be formed to some extent on the fetal side. An efficient transfer of the halogenated phenolic compounds is supported by the similar high concentrations of PCP in the maternal and cord blood plasma. In this case, the major contribution is from PCP, even though a slight contribution of PCP may originate from metabolized hexachlorobenzene (Renner 1988).

The levels of OH-PCBs and PCP in breast milk were approximately 35 and 100 times lower than in maternal and cord blood plasma (Figure 1), confirming poor transfer of halogenated phenolics via lipids. The dominating OH-PCB congeners were 4-OH-CB107 and 4-OH-CB193 (Table 3). The congener 4-OH-CB187 has been previously reported in Canadian breast milk samples (Newsome and Davies 1996).

The PCP levels in breast milk were an order of magnitude lower than previously reported in Swedish breast milk collected in 1980 (Norén 1988). The levels of PCP in blood plasma reported in this study were similarly lower than PCP concentrations determined in blood from Stockholm women sampled around 1980 (Jensen S. Personal communication). However, one individual diverged by having a PCP concentration in breast milk almost 30 times higher (0.57 ng/g.
fresh weight) than any of the other subjects (Figure 3).

The low levels of halogenated phenolic compounds in breast milk compared with their levels in blood are most likely explained by their low accumulation in lipids (e.g., in human adipose tissue, OH-PCBs as well as to other persistent organochlorines) that the fetus is probably continuously exposed to from their levels in maternal blood, there are differences in congener distribution of, for example, PBDEs and OH-PCBs that must be considered. This has yet to be done through congener-specific analysis. Our results show that the fetus is probably continuously exposed to PBDEs, PCBs, OH-PCBs, and PCP during development. However, more work needs to be done to describe the exposure situation for the fetus during the entire developmental period. Exposure to PBDEs and PCBs as well as to other persistent organohalogen pollutants continues by breastfeeding and possibly at a higher level than during fetal development, whereas the exposure to halogenated phenols is strongly reduced via this route.

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