Persistent organochlorine (OC) contaminants were first detected in breast milk in the early 1950s (Laug et al. 1951). Although many OCs were restricted in North America and Europe during the 1970s, specific requirements such as the control of malaria with 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane (p,p'-DDE) or hexachlorocyclohexanes (HCHs) remain in some parts of the world. Although polychlorinated biphenyls (PCBs), predominantly industrial chemicals, were also banned in many countries in the 1970s, these are still present in the environment, and uncertainties exist regarding their current distribution (Breivik et al. 2004). Human tissue levels of PCBs and p,p'-DDE peaked in the 1970s but, probably because of restrictions on use, are now decreasing (Norén and Meironyté 2000). In contrast, polybrominated diphenyl ether (PBDE) levels, first detected in breast milk in 1972, have been increasing throughout the 1980s and 1990s (Meironyté et al. 1999; Norén and Meironyté 2000; Solomon and Weiss 2002).

Brominated diphenyl ethers (BDEs) are commercially available as pentabromodiphenyl ether (pentaBDE), octabromodiphenyl ether (octaBDE), and decabromodiphenyl ether (decaBDE), all of which have different PBDE profiles. DecaBDE is used in high-impact polystyrene for electronic enclosures and as a flame retardant in upholstery textiles (Hardy 2002a). OctaBDE is used in business equipment housings made of acrylonitrile–butadiene–styrene (ABS) resins (Hardy 2002b). PentaBDE is used as a flame retardant for flexible polyurethane foam (Hardy 2002b). As additive flame retardants, PBDEs are used in consumer items such as polyurethane foams, television sets, computers, radios, textiles, paints, and plastics. These brominated compounds act in the gas phase of the fire by reacting with free radicals generated during combustion, thus terminating the reaction (Rhame et al. 2001). PBDEs are dissolved in materials, not covalently bonded. This has led to suggestions that they leak out or volatilize into the environment (McDonald 2002). PBDEs investigated in this study are predominantly indicative of the pentaBDE commercial product.

PBDEs, PCBs, and OCs are hydrophobic, persistent chemicals that may bioaccumulate through food chains. The main route of exposure to PCBs or OCs in the general human population is via the diet (Duarte-Davidson and Jones 1994). In the case of PBDEs, the relative importance of different exposure routes remains obscure. It has been suggested that exposure may occur mainly through the consumption of PBDE-contaminated fish (Meironyté et al. 1999). However, PBDEs do appear to contaminate indoor human environments (Jakobsson et al. 2002), giving rise to the possibility of transfer via inhalation of vapor-phase chemicals and chemicals attached to indoor air dust.

The human toxicity of PCBs and OCs is well documented [World Health Organization (WHO) 1993]. The toxic effects of PBDEs in humans remain to be properly ascertained (Hardy 2002b). Despite apparent similarities in chemical structure between PBDEs and PCBs, there are important three-dimensional differences due to the ether linkage found in the former (Hardy 2002a). Although currently found to be lower than PCB levels in human tissues, PBDE levels have been increasing (Hites 2004; Norén and Meironyté 2000). PBDEs, particularly congeners present in the pentaBDE product, may possess toxic properties, including endocrine-disrupting activity (Hallgren and Dannerud 2002) and interference with brain development (Branchi et al. 2003), and there is also a suggested association with incidence of non-Hodgkin lymphoma (Dannerud et al. 2001). Some evidence exists
of PBDE carcinogenicity in animal bioassays, but the results of these studies remain at best unequivocal (Hardy 2002b; McDonald 2002).

A current profile of PBDE congeners in U.K. breast milk (as measured in extracts of milk fat) in relation to other contaminants remains to be established (Kalantzi et al. 2003). In the present study, our primary objective was to report in detail current U.K. PBDE levels (associated with the pentaBDE product) and, in the same samples, to relate these to current and temporal PCB and OC levels of the United Kingdom. The samples from which milk-fat extracts were obtained originated from London in the southeast and Lancaster in the northwest. We also investigated whether nuclear magnetic resonance (NMR)–based metabolomics (Nicholson et al. 1999), combining high-resolution NMR spectroscopy with pattern recognition, might be a potential platform to study the chemical constituents of milk-fat extracts shown to contain different contaminant profiles. This methodology allows the identification, quantification, and cataloguing of time-related changes in integrated biologic systems and thus may be employed to screen for profiles in tissue extracts (Lindon et al. 2003; Nicholson et al. 1999; Viant et al. 2003). Finally, we consider whether the quantification of such contaminants in biologic matrices remains sufficient, or whether it is more appropriate to analyze their biologic effects, either individually or as complex mixtures.

Materials and Methods

Sample collection. Individual breast milk samples from U.K.-resident women, donated anonymously and without criteria (i.e., no prior selection or bias), were obtained with appropriate ethical approval. A total of 54 samples were collected between late 2001 and January 2003 from the maternity units of hospitals based in the Lancaster (northwest England; \( n = 27 \), via a neonatal unit) and London (southeast England; \( n = 27 \), via a milk bank) regions. Donor ages ranged from 24 to 34 years, and milk samples (=100 mL from a single expression; in a small number of cases, samples expressed on different days were donated) were collected by manual expression into sterile collection bottles. Samples were immediately frozen and stored at \(-20^\circ\text{C} \) before analysis.

Donors originating from Lancaster completed simple lifestyle questionnaires; such information was not available for London samples. Lancaster samples were donated within the first month of parturition, several being donated within the first 2–3 days. From the information obtained on Lancaster samples, it was noted that all but two donors were nonsmokers, and cumulative past lactation ranged from 0 to 21 months. All consumed meat, a healthy amount of fresh fruit, and low amounts of alcohol (five donors, 1–2 units/day; one unit is defined as 7.9 g ethanol). Lifestyle data thus collected were not found to correlate with the levels of contaminants measured (data not shown). In the absence of more details, it is difficult to comment on similarities or differences between the two cohorts originating from the London or Lancaster regions. However, experience would suggest that no marked differences in range of socioeconomic class, outlets through which food might be sourced, age, or parity would exist. Although the London cohort would be envisaged to be a more heterogeneous population with an input from foreign donors, this was not expected to have an overwhelming influence.

Extraction of milk for PBDE and PCB/OC analyses. All solvents were of HPLC or glass-distilled grade. Silica gel (0.063–0.200 mm; Merck, Poole, U.K.) and \( \text{Na}_2\text{SO}_4 \) were heated at 450°C overnight and stored in sealed containers. Standards were purchased from Promochem (Welwyn Garden City, U.K.) and QMx (Thaxted, U.K.).

After thawing, milk samples that originated from a single expression were centrifuged at 3,000 rpm for 15 min. After separation of the milk-fat layer from the aqueous phase, a mixture of milk fat (0.5 g), \( \text{Na}_2\text{SO}_4 \) (5 g), and hexane (50 mL) was boiled for 10 min and allowed to cool before lipid determinations. Evaporated to 5 mL, these mixtures were applied to 25-mm inner-diameter columns containing 15 g acidified silica gel (2:1 silica gel:acid by weight) and eluted with hexane. Eluted samples were evaporated to 1 mL under a gentle stream of nitrogen and applied to gel permeation chromatography columns packed with Biobeads S-X3 (Biorad Laboratories, Hercules, CA, USA) and eluted with hexane: dichloromethane (1:1 by volume). \( ^{13}\text{C}_2\text{H}_4 \)-labeled PCB and dioxin recovery standards (added at the beginning of the procedure) and internal standards (added at the end of the procedure) in dodecane were incorporated when subsequent gas chromatography–mass spectrometry (GC-MS) analysis was carried out on whole milk-fat extracts but were excluded when extracts were generated for subsequent \(^1\text{H}-\text{NMR} \) spectroscopy.

Gas chromatography for PBDE analysis was performed on a Finnigan Trace GC2000 series gas chromatograph equipped with a 30-m DB-5MS 0.25-mm inner-diameter capillary column (J&W Scientific, Stockport, U.K.) fitted with a retention gap (2 m long, 0.53 mm inner diameter). Sample aliquots (2 mL) were injected by a Thermoquest AS2000 autoinjector (Finnigan, Hemel Hempstead, U.K.), with the injection port at 270°C, in splitless mode, with 100 kPa pressure surge. The carrier gas was helium at a flow rate of 1 mL/min. The oven temperature program was as follows: 100°C for 2 min, 20°C/min to 140°C, 4°C/min to 200°C, 200°C for 13 min, 4°C/min to 300°C, and 300°C for 10 min. The quadrupole MD800 mass spectrometer (Fisons) was set in selected ion recording mode, in electron impact positive ionization (EI+) mode, with a source temperature of 250°C, interface temperature of 300°C, and electron energy of 70 eV. A screen for the following PCB congeners and OC pesticides was carried out: PCB congeners 18, 22, 28, 31, 41/64, 44, 49, 52, 54, 60/56, 70, 74, 87, 90/101, 95, 99, 104, 105, 110, 114, 118, 123, 132, 138, 141, 149, 151, 153, 155, 156, 157, 158, 167, 170, 174, 180, 183, 187, 188, 189, 194, 199, 203; \( \alpha \)-HCH, \( \beta \)-HCH, \( \gamma \)-HCH, \( \delta \)-HCH, hexachlorobenzene (HCB), \( \alpha \)-chlordane, \( \gamma \)-chlordane, \( \delta \)-chlordane, \( \alpha \)-dichlorodiphenyldichloroethene (DDE), \( \beta \)-dichlorodiphenyldichloroethene (DDD), \( \alpha \)-dichlorodiphenyldichloroethane (DE), \( \beta \)-DDD, \( \alpha \)-DDD, and \( \gamma \)-DDD. The following were regularly detected in the milk samples: PCB congeners 41/64, 44, 49, 50, 56, 70, 74, 87, 90/101, 95, 99, 105, 110, 114, 118, 138, 149, 151, 153, 156, 157, 160, 180, 183, 187, 189, 194, 203; \( \alpha \)-HCH, \( \beta \)-HCH, \( \gamma \)-HCH, HCB, \( \alpha \)–DDD, \( \beta \)–DDD, \( \gamma \)–DDD, and \( \delta \)–DDD. A seven-point calibration was used for quantifying all PBDEs, PCBs, and OCs. Detection limits were defined as three times the standard deviation of the levels found in the analytical blanks or the instrument detection limit in the absence of detectable levels in the blanks.

Quality assurance/quality control. Milk samples were analyzed in batches of six. Along with each batch a blank sample was included, and with every third batch an in-house
reference material (a butter sample) was also extracted. This reference material had been analyzed on 12 separate occasions before these analyses and was considered a useful determinant of the reproducibility and rigorous nature of the analytical procedure employed. A control chart was employed to identify the mean and warning limits (defined as plus two times the standard deviation of the average concentration found in the 12 reference material samples previously analyzed) of the reference materials analyzed throughout the study. The comparison quantity was defined as the sum of the levels of PBDE-47, PBDE-99, PBDE-100, PBDE-153, and PBDE-154 in the reference butter sample. Seven reference material samples were analyzed during the course of this study, and the value of the sum of the five PBDEs never exceeded the warning limits. Recoveries ranged from 50 to 119%, with an average of 90%. Extracted milk fat ranged from 29 to 85% of centrifuged milk fat fresh weight (mean, 55%). Centrifuged milk fat fresh weight ranged from 0.7 to 5.7% (mean, 2%) of whole milk fresh weight. 1H-NMR spectroscopy: Extracts in hexane were transported to Imperial College (London) for analysis. Extracts of milk samples containing the lowest contaminant levels (n = 7, all from Lancaster) were compared with extracts of milk samples containing the highest levels (n = 7, all from London). These two sets of samples were chosen on the basis of the sum of all contaminants. To these, an equal volume of CDCl3 (99.9% deuterium; Sigma, Poole, U.K.) was added, and samples were purged with nitrogen gas. To ensure the removal of hexane, this purge process was repeated twice. Then samples were reconstituted in 500 µL of CDCl3 and transferred into 5-mm NMR tubes. 1H-NMR spectra were recorded on a Bruker DRX600 NMR spectrometer, at 300 kelvin, with a 5-mm inverse probe using a standard one-dimensional pulse sequence [RD-90°-1 rs-90°-τ90°-acquisition]. The recycle delay (RD) was 3 sec; mixing time, tms 150 msec; t1, 3 µsec. The 90° pulse length was adjusted to approximately 10 µsec. Transients (128) were collected into 32,768 data points for each spectrum with the spectral width of 12 kHz. Data reduction and principal components analysis. All free induction decays were multiplied by an exponential function equivalent to a 1 Hz line-broadening factor before Fourier transformation. NMR spectra were then phase baseline corrected before being divided into 245 buckets, over the chemical shift range 0.2–10 ppm, using AMIX (Bruker Analytik, Rheinstetten, Germany). Each bucket was 0.04 ppm wide, and the area in each region was integrated. The regions 68.6–87.5 ppm and 1.5–2.5 ppm containing residual solvents (chloroform and water, respectively) were discarded. The value of each bucket was normalized relative to the total sum of the spectral integral before principal components analysis (PCA), which was carried out with the software Simca-P 8.0 (Umetrics, Umeå, Sweden).

Results
PBDE congener levels in milk-fat extracts obtained from U.K. breast milk samples (n = 54) obtained between late 2001 and January 2003 are shown in Table 1. ΣPBDE levels ranged from 0.1 to 69 ng/g lipid, with a geometric mean of 6.6 ng/g lipid (median, 6.3 ng/g lipid). PBDE-47 (geometric mean, 3 ng/g lipid; median, 2.7 ng/g lipid; 45% of ΣPBDE content) was always the most abundant congener present in breast milk. As a percentage of ΣPBDE content, other congeners were found in the following order: PBDE-153 (21%) > PBDE-99 (14%) > PBDE-100 (9.0%) > PBDE-154 (7%), respectively. Making up much smaller proportions of ΣPBDE levels were PBDE congeners 28, 32, 35, 17, and 71.

Published levels of ΣPBDE congeners in breast milk are shown in Table 2. Selected congeners have been included in the ΣPBDE values presented in Table 2 to facilitate comparisons with the results of different studies. Compared with European and Japanese milk samples, markedly higher (~10-fold) levels appear to occur in U.S. milk samples. Current ΣPBDE levels in U.K. milk samples, although not as high as those found in U.S. milk samples, are still much higher than in milk samples from other European countries and Japan. As a consequence of stringent fire regulations since 1988, the United Kingdom has seen the use of larger quantities of the pentaBDE mixture than many of her European counterparts. The United Kingdom is currently the fourth largest PBDE producer in the world, with an output of ~25,000 metric tons annually (Alaee et al. 2003).

PCB levels in milk-fat extracts obtained from U.K. breast milk are shown in Table 3. ΣPCB levels ranged from 26 to 530 ng/g lipid, with a geometric mean of 150 ng/g lipid (median, 180 ng/g lipid). The most commonly occurring PCB congeners were found to be present in the following order:

| Table 1. PBDE congener levels in milk-fat extracts obtained from U.K. breast milk (n = 54), 2001–2003. |
|-----------------------------------------------|
| PBDE congener | Geometric mean (ng/g lipid) | Median (ng/g lipid) | Minimum (ng/g lipid) | Maximum (ng/g lipid) | Percent samples positive |
| 17 | 0.1 | ND | ND | 1 | 32 |
| 28 | 0.3 | 0.2 | ND | 2.1 | 89 |
| 32 | 0.1 | ND | ND | 0.3 | 38 |
| 35 | 0.2 | ND | ND | 0.6 | 28 |
| 37 | 0.4 | ND | ND | 0.5 | 9 |
| 47 | 3 | 2.7 | 0.1 | 37 | 100 |
| 49 | 0.2 | ND | ND | 0.9 | 11 |
| 71 | 0.2 | ND | ND | 0.5 | 11 |
| 75 | 0.5 | ND | ND | 0.6 | 4 |
| 85 | 0.5 | ND | ND | 1.4 | 9 |
| 99 | 0.9 | 0.8 | ND | 13 | 92 |
| 100 | 0.6 | 0.5 | ND | 7 | 94 |
| 119 | 0.2 | ND | ND | 0.4 | 11 |
| 153 | 1.4 | 1.3 | ND | 4.9 | 89 |
| 154 | 0.5 | 0.4 | ND | 2.5 | 81 |
| ΣPBDE | 6.6 | 6.3 | 0.3 | 69 | — |

Abbreviations: —, not applicable; ND, not detected. For raw data, see Supplemental Material online (http://ehp.niehs.nih.gov/members/2004/6991/supplemental.pdf).

| Table 2. A comparison of ΣPBDE levels in breast milk samples from different countries. |
|-----------------------------------------------|
| Country | No. of samples analyzed | Year of sampling | ΣPBDE [ng/g, mean (range)] | PBDE congeners included in ΣPBDE | Reference |
| Canada | 10 | 1992 | 5.7 (0.7–28a | 28, 47, 99, 100, 153, 154 | Ryan and Patry 2000 |
| Finland | 11 | 1994–1998 | 2.3 (0.9–5.9 | 28, 47, 99, 153 | Strandman et al. 2000 |
| Japan | 12 | NA | 0.7–2.8 | 28, 47, 99, 100, 153, 154 | Ohta et al. 2000 |
| Sweden | 40 | 1997 | 4 | 28, 47, 66, 100, 99, 85, 153, 154 | Norén and Meironyté 2000 |
| United Kingdom | 54 | 2001–2003 | 8.9 (0.1–63)b | 47, 99, 100, 153, 154 | This study |
| United States | 47 | 2001 | 73.9 (6.2–418.8 | 17, 28, 47, 66, 77, 85, 99, 100, 138, 153, 154, 183, 209 | Schecter et al. 2003 |

NA, data not available.

aSum includes PBDE congeners listed only to facilitate interstudy comparisons.

bArithmetic mean and concentration range in ng/g lipid.
PCB-153 > PCB-138 > PCB-180 > PCB-170 > PCB-118 > PCB-187 > PCB-99 > PCB-156. PCB-153 constituted some 26%, PCB-138 some 20%, and PCB-180 some 13% of PCB levels. A comparison of the results from this study with previously reported U.K. levels (Table 4) suggests that ΣPCB levels are currently decreasing, probably as a consequence of bans imposed on the use of these contaminants.

| Period of sampling | Mean ΣPCB (ng/g lipid) | Range ΣPCB (ng/g lipid) | No. of samples | Tissue source | Reference |
|--------------------|------------------------|-------------------------|----------------|--------------|-----------|
| 1976–1977          | 600                    | 100–1,500               | 81             | Adipose fat  | U.K. MAFF 1983 |
| 1979–1980          | 500                    | 100–2,100               | 102            | Milk fat     | U.K. MAFF 1983 |
| 1990–1991          | 522                    | 140–1,697               | 32             | Milk fat     | Duarte-Davidson et al. 1994 |
| 2001–2003          | 200                    | 26–530                  | 54             | Milk fat     | This study |
| p,p'-DDD          | 0.3                    | ND                      | 0.9            | 4 |
| p,p'-DDT          | 0.7                    | 0.6                     | 55             | 80 |
| p,p'-DDE          | 0.2                    | 0.1                     | 5.8            | 69 |
| p,p'-DDD          | 0.3                    | 0.3                     | 11             | 82 |
| p,p'-DDT          | 6.2                    | 6.2                     | 760            | 100 |
| ΣDDX              | 150                    | 26–190                  | 520            | — |

| Period of sampling | Mean p,p'-DDT (ng/g lipid) | Range p,p'-DDT (ng/g lipid) | No. of samples | Tissue source | Reference |
|--------------------|---------------------------|-----------------------------|----------------|--------------|-----------|
| 1976–1977          | 50                       | 10–290                      | 85             | Adipose fat  | Abbott et al. 1972 |
| 1978–1977          | 220                      | 20–3,200                    | 81             | Adipose fat  | Abbott et al. 1981 |
| 1979–1980          | 140                      | 10–1,000                    | 102            | Milk fat     | Collins et al. 1981 |
| 1982–1983          | 110                      | 30–320                      | 187            | Adipose fat  | Abbott et al. 1985 |
| 1997–1998          | 43                       | 12–333                     | 188            | Milk fat     | Harris et al. 1999 |
| 2001–2003          | 20                       | ND–180                      | 54             | Milk fat     | This study |
| β-HCH              | 16                       | 18–22                      | 760            | 100 |

Abbreviations: NA, data not available; ND, not detected.

Total HCB levels ranged from below the detection limit to 180 ng/g lipid (geometric mean, 17 ng/g lipid; median, 18 ng/g lipid), whereas ΣHCH (defined as the sum of α-, β-, and γ-HCH) levels ranged from 1.2 to 1,500 ng/g lipid (geometric mean, 16 ng/g lipid; median, 18 ng/g lipid; Table 3). ΣDDX (p,p'-DDT and its metabolites) levels ranged from 24 to 2,300 ng/g lipid, giving rise to a geometric mean of 160 ng/g lipid (median, 160 ng/g lipid). The most commonly occurring isomers were p,p'-DDE (geometric mean, 150 ng/g lipid; median, 150 ng/g lipid; range, 20–1,600 ng/g lipid) and p,p'-DDT (geometric mean, 6.2 ng/g lipid; median, 6.2 ng/g lipid; range, 1.1–760 ng/g lipid). Of two milk samples that exhibited exceptionally high ΣDDX levels, one also exhibited exceptionally high ΣHCH levels and the other exhibited exceptionally high ΣHCB levels (data not shown). Table 4 again demonstrates that levels in U.K. breast milk of p,p'-DDE, HCB, and β-HCH have been falling for some time.

Geometric means, medians, and ranges of various contaminant concentrations in milk-fat extracts obtained from breast milk samples originating from the London region (n = 27) and the Lancaster region (n = 27) are compared in Table 5 and Figure 1A, B. The data were log-transformed, and a t-test was used to investigate differences in contaminant levels between the samples from London and those from the Lancaster region. Significantly higher (p < 0.0001) levels of PBDE-47 were found in London milk samples compared with those originating from Lancaster, but no differences were found in ΣPBDE and the levels of four other PBDE congeners (Table 5, Figure 1B). With regard to PCBs and OCs, significantly higher (p < 0.05) levels of PCB-138, PCB-153, PCB-180, ΣPCB, p,p'-DDE, ΣDDX, HCB, β-HCH, and ΣHCH were found in milk samples from London (Table 5, Figure 1A). Breast milk samples originating from London tended to exhibit higher geometric mean levels for all the contaminants analyzed in comparison with Lancaster levels (Figure 1A, B).

1H-NMR spectra showed that the major chemical constituents of extracts analyzed in this study were lipids (data not shown). Taking into account the remaining, unidentified NMR signals, seven of the milk-fat extracts shown to contain the lowest contaminant levels (all from the Lancaster region) were compared with seven extracts shown to contain the highest levels (all from the London region). NMR with pattern recognition methods gave rise to a multivariate PCA score plot (Figure 2) in which each point represents an individual extract. The observation that clustering occurred based on contaminant levels highlights the differences in the chemical composition of these two groups of milk-fat extracts. Excellent separation for the two
groups of extracts was clearly evident along the first principal component, thus implying that the chemical constituents of both groups are significantly different.

**Discussion**

The occurrence of PBDE-47 in breast milk (Meironyté et al. 1999), human blood plasma (Klasson-Wehler et al. 1997), and human adipose tissue (She et al. 2000) has been reported. In Sweden, a PBDE congener pattern consisting of PBDE-47 > PBDE-99 > PBDE-153 > PBDE-100 > PBDE-154 in terms of relative content has been noted, whereas others have reported the presence of equal levels of PBDE-99 and PBDE-153 (Darnérud et al. 1998; Meironyté et al. 1999). Other studies from Japan, Sweden, the United States, and now the United Kingdom (present study) have found PBDE-153 to be present at higher levels than PBDE-99 (Meironyté et al. 2003; Ohta et al. 2000; She et al. 2002).

Estimates of market demand for major brominated flame retardants suggest that some 58% of world market volume occurs in Asia, followed by the Americas (26%) and then Europe (14%) [Bromine Science and Environmental Forum (BSEF) 2001]. Of three commercial PBDE mixtures—pentaBDE, octaBDE and decaBDE—the latter is the most commonly found in Europe in recent years with approximately 7,600 metric tons of decaBDE in current use (BSEF 2001; Hardy 2002b). PBDE congeners 47, 99, 100, and 153 are all components of pentaBDE. It has been tentatively suggested that since 1970 a cumulative sum of 3,000 metric tons of pentaBDE may have been manufactured in the United Kingdom (Alcock et al. 2003).

That lower brominated PBDEs are still present in U.K. milk samples (Table 1) suggests historical use of the pentaBDE product still plays a role in determining current congener patterns. It has been hypothesized that in sand, soil, or sediment decaBDE photodegrades and may to a minor degree give rise to lower brominated congeners (Sellström et al. 1998; Söderström et al. 2004). Diet may be an important route of exposure to PBDEs, particularly through the consumption of fish (Lind et al. 2002). Whether diet is the dominant route of uptake remains to be ascertained.

As PBDEs are used in indoor environments, the potential for transfer via inhalation of indoor dusts is a possibility. Foam from furniture and cars in the United Kingdom is likely to have been treated with the technical pentaBDE mixture profile in the past (Wilford et al. 2003), and the possibility of PBDEs volatilizing from foam has been previously demonstrated (Wilford et al. 2003). The pattern of PBDE congeners in U.K. breast milk suggests higher PBDE-47, PBDE-100, PBDE-153, and PBDE-154 levels but lower PBDE-99 levels (Table 1). Indoor dust samples from two U.K. parliamentary buildings (Santillo et al. 2001) and from houses in the United Kingdom (Santillo et al. 2003) exhibited similar patterns. PBDE patterns in fish from the U.K. North Sea (Thomas et al. 2003) also gave rise to a closely matching profile. U.S. data suggest a similar profile for foam (Hale et al. 2002) but a different indoor dust pattern (Rudel et al. 2003).

London (population > 10 million) contrasts markedly with Lancaster, a semi-rural town (population ~100,000). Pearson correlations of the logged \( \Sigma PCB, \Sigma PBDE, \Sigma \text{HCH}, \Sigma \text{DDX} \).
Despite the presence of contaminants in U.K. breast milk, it must be emphasized that breast-feeding is protective to the neonate (Dundaroz et al. 2002; Oddy 2001) and also appears to confer a protective effect against breast cancer in the mother (Grover and Martin 2002). The value of this resource is that it potentially allows us to noninvasively analyze ongoing exposures. This work shows for the first time the presence of PBDEs in U.K. breast milk obtained from two different locations and also points to significant regional differences in contaminants in this biologic fluid. Future work is required to ascertain the underlying reasons for these regional differences and also the biologic effects of such exposures.

Correction

Values in the tables and in the corresponding text in the original manuscript published online were incorrect and have been corrected here.

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