

# Polychlorinated Biphenyls (PCBs) and Dichlorodiphenyl Dichloroethene (DDE) in Human Milk: Effects of Maternal Factors and Previous Lactation

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**Abstract:** The authors measured polychlorinated biphenyls (PCBs) and dichlorodiphenyl dichloroethene (DDE) in maternal serum, cord blood, placenta, and serial samples of breast milk from 868 women. Almost all samples of breast milk showed detectable levels of both chemicals. Overall, values for DDE in this study are within the range of those found previously, whereas those for PCBs are somewhat higher. Possible causes of variation in levels were investigated. For DDE, older women, Black women, cigarette smokers, and women who consumed sport fish during pregnancy had higher levels; only age and race showed large effects. For PCBs,

older women, women who regularly drink alcohol, and primiparae had higher levels. In addition, both chemicals showed modest variation across occupational groupings. Casual exposure to a PCB spill did not result in chemical levels different from background. In general, women have higher levels in their first lactation and in the earlier samples of a given lactation, and levels decline both with time spent breast-feeding and with number of children nursed. These striking declines are presumably a measure of exposure to the child. (*Am J Public Health* 1986; 76:172-177.)

## Introduction

Persistent halogenated hydrocarbons have been used extensively in this country and are now widespread in the environment. This class of chemicals has produced major economic and public health benefits. Dichlorodiphenyl trichloroethane (DDT) was a widely used and effective pesticide from its introduction in the 1930s to its ban in 1972.<sup>1</sup> The polychlorinated biphenyls (PCBs), whose main uses were as nonflammable dielectrics in heavy electrical transformers, were also used as vehicles for pesticide application, as pigment suspension agents in carbonless copy paper, and as the dielectric in an assortment of small electronic parts. Until manufacture ceased in 1974, about eight million pounds of PCBs were made in the United States.<sup>2</sup> Much of this was discarded with no thought to environmental impact. DDT was sprayed or applied directly into the environment in large quantities. These kinds of chemicals are not readily degraded in the environment, nor are they completely metabolized or excreted by organisms. Instead, they come into a steady state, bioconcentrate in the food chain, and produce low-level but ubiquitous contamination of human beings. There also have been incidents involving higher-dose exposures to PCBs,<sup>3</sup> DDT,<sup>4</sup> and other members of the class of halogenated hydrocarbons through accidents or improper disposal. Because of the amounts now in the environment, continued exposure to these chemicals for the next decades is certain in spite of the fact that many are no longer produced or being put into use.

Exposure to and consequent storage of these chemicals by human beings has been documented repeatedly. The chemicals are stored in fat, and thus are not excreted under physiological circumstances by human beings, except during

lactation. Breast milk is about 3 per cent fat; this fat appears to be in approximate equilibrium with the body fat stores. Surveys of human milk from unselected and presumably non-occupationally exposed women in the United States and worldwide have shown widespread contamination by DDT and its metabolites, PCBs, and other chemicals like them.<sup>5</sup>

The largest US survey was done in 1975<sup>6</sup>; women were volunteers at a representative sample of US hospitals. Of 1,436 women tested for DDT, essentially all had p,p'-DDT and p,p'-DDE (dichlorodiphenyl dichloroethene, the metabolite most stable in tissue).<sup>7</sup> PCBs were present in at least "trace" amounts in 99 per cent of 1,038 samples, and about one-fourth were above the Food and Drug Administration's "action level" (2.5 parts per million) at which a commercial food would have been removed from the market.<sup>8,9</sup>

Since these breast milk analyses had been undertaken for purposes of monitoring exposure to and storage of persistent environmental chemicals, the studies did not include monitoring of morbidity in the children. Both PCBs and DDT are toxic, but data on their toxicity for human beings is sparse and limited to high-dose and mixed exposures.

There have now been two outbreaks of mass poisonings due to PCBs and the thermal degradation products that contaminate them as they are used; one, called Yusho (oil disease), was in Japan in 1968,<sup>10</sup> and the other, called Yucheng, was in Taiwan in 1979.<sup>11</sup> The chemicals leaked into cooking oil during processing, and went undetected until the oil was analyzed chemically after the disease outbreaks. The principal sign of toxicity was chloracne, which is a highly cystic, relatively non-inflammatory acne-like rash. Children born to exposed mothers tended to be of low birthweight, to have hyperbilirubinemia, and to have pigmentation of the gingiva, nails, nose, and axillary and groin folds.<sup>12</sup> Few data are available on levels in breast milk; analytic sensitivity was not great in 1968, and patients in Taiwan were told not to breast-feed. One Japanese woman who was tested did not show remarkably high levels.<sup>13</sup> Harada reported on several cases of Yusho produced by breast milk exposure alone,<sup>14</sup> and at least one case of Yucheng is believed to have been produced that way (Hsu S-T: Center for Control of Communicable Diseases, Taipei, personal communication).

In the laboratory, PCBs are perhaps the best studied inducers of mixed function oxidase enzymes. At high doses,

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they produce a fatal "wasting syndrome", the mechanism of which is poorly understood. They produce hepatic tumors in mice and are probably promoters of carcinogenesis. The toxicity of various chemicals which are contaminants of PCBs (polychlorinated terphenyls, quatraphenyls, and dibenzofurans) is broadly similar but less well studied. There are extreme differences in sensitivity to all of these compounds from species to species; in addition, the individual congeners of each of the parent compounds differ in their toxicity, and there is a wide range of toxicity across the parent compounds, with the furans generally being the most potent.<sup>15</sup>

When female rhesus monkeys are fed 2.5 ppm of commercial-grade PCBs in their total diets, they have difficulty carrying infants to term. In addition, their offspring are small-for-dates, develop chloracne on breast-feeding, suffer increased early mortality,<sup>16,17</sup> and have a variety of neurobehavioral sequelae.<sup>18-20</sup> The relative susceptibility of human beings compared to the rhesus monkey is not known.

Of the systems thus far tested, the ones most sensitive to PCBs are the induction of microsomal enzymes of the P450 class<sup>21</sup> or the covalent binding to macromolecules in microsomes.<sup>22</sup> Both effects occur in a dose-related fashion beginning at about 1 to 4  $\mu\text{g}/\text{kg}/\text{day}$ ; we have shown elsewhere that the typical dose to a fully breast-fed newborn in our data would be about 6  $\mu\text{g}/\text{kg}/\text{day}$ .<sup>23</sup>

At quite high doses, DDT is an excitatory neurotoxin in man. It is not clear that a fatal ingestion has taken place, since at the doses involved the toxicity of the diluent, typically kerosene, becomes important. Moderate exposures over months to years have been studied in dosed prisoners<sup>24</sup> and exposed workers,<sup>25</sup> and few toxic effects have been seen. Kreiss, *et al*, studied consumers of fish heavily contaminated with DDT. Their data include the highest serum DDE values recorded in human beings. They observed some associations with blood lipid levels but no dose-related clinical illness. There were no data on exposed newborns.<sup>4</sup>

In the laboratory at high doses, DDT can produce central nervous system excitability and seizures. It is also an inducer of mixed function oxidase enzymes, and is a carcinogen (probably a promoter) in mice.<sup>26</sup> DDT has a complicated effect on estrogen metabolism. Because of induction, some estrogen may be metabolized faster. However, o,p-DDT and o,p-DDE are weak but persistent estrogens.<sup>27</sup>

We report here the results of the chemical analysis of samples from 868 women using very sensitive and well-controlled chemical analysis techniques developed especially for this study. We also followed the children longitudinally; their experience will be reported separately.

## Materials and Methods

### Study Design

The study was conducted in North Carolina and coordinated by the National Institute of Environmental Health Sciences (NIEHS). The East Carolina University School of Medicine at Pitt County Hospital in Greenville, the Durham Women's Clinic (a large private practice), and the Wake Area Health Education Center at Wake County Medical Center in Raleigh each enrolled about 300 subjects. The study was approved by institutional review boards at the three institutions and by the clinical review panel of the Clinical Center at the National Institutes of Health.

Women were recruited into the study from hospital familiarization tours, from Lamaze classes, and from both private and public prenatal clinics. No attempt was made to

assemble a random sample, and women interested in participation were disqualified only if they planned to move from the area within six months. At or near term, a questionnaire was administered to the mother. Breast milk, colostrum or formula (whatever the child was being fed) was collected, as well as blood from the mother, cord blood, and placenta. Initial follow-up visits took place at six weeks, three months, and six months postpartum. At each visit, formula or breast milk was collected. At the six-week visit, another blood sample was taken from the mother. Children were seen again at six-month intervals until age 2, and then yearly. Samples of breast milk were collected from women who continued to lactate. In some cases, an extra milk sample was taken at nine months although no regular visit was done at that time.

### Chemical Methods

Specimens for chemical analysis were collected on a protocol developed by the Chemistry Branch of NIEHS. Samples were shipped on dry ice to NIEHS, where they were split, archived if possible, and sent off for analysis. Raltech Scientific Services Inc., (now known as Hazelton Raltech Inc.) in Madison, Wisconsin performed lipid estimation and analyzed for PCBs and p,p-DDE by gas liquid chromatography. Details of the chemical methods and the quality control procedures are presented elsewhere.<sup>28</sup> All biologic samples were analyzed. A 10 per cent sample of formula specimens was also analyzed.

Since the chemicals of interest are stored in fat, the amount of chemical found will depend on the fat content of the sample. The fat content of placenta is low and does not vary substantially. Thus, chemical levels are simply reported as grams of chemical per gram of placenta. Serum has a variable but relatively small amount of complex fat, and there is not general agreement on whether or how to adjust for it; we report simply the concentration found in the whole sample without adjustment. The fat content of milk varies considerably from woman to woman and also over the course of a feeding, with hind milk typically about 4 per cent fat and fore milk 1 per cent. We thus analyzed all milk samples separately for fat content, and report the values as "fat adjusted", i.e., as grams chemical per gram milk fat.

### Statistical Methods

The chemical data were analyzed using analysis of variance techniques applied to the logarithms of the original data, since the distributions were substantially skewed to the right. Descriptive statistics such as means were also obtained on the log scale and then retransformed.

In some cases, the levels of chemicals were below the quantitation limit of the method. In order to account for these unquantifiable observations, techniques for censored data were employed. The percentiles presented in Tables 1 and 2 are Kaplan-Meier estimates.<sup>29</sup>

In order to examine whether or not the levels of chemicals depend on various characteristics of the mother, it is necessary to use comparable levels for all women. However, not all women had each type of sample. Thus, using a single sample, such as milk at birth, would eliminate many women. Also, we wished to use as much information as possible on each woman in order to get as much accuracy as we could. Therefore, values from all samples from a given woman were combined and expressed as an estimated amount of chemical in milk at birth. The high correlations we found (Tables 3-4) made such a procedure attractive. This combining involved three steps. First, any levels below the quantitation limit were changed to an estimated amount. (We

**TABLE 1—PCB Levels in Various Types of Samples**

Sample	Number	Median	95th Percentile	Maximum	Per Cent Less Than Quantitation Limit
Milk at birth	733	1.77	3.91	16.00	13
Milk at 6 weeks	617	1.53	3.44	14.80	6
Milk at 3 months	498	1.46	3.35	15.00	9
Milk at 6 months	362	1.38	2.90	17.10	12
Milk at 9 months	62	1.18	2.70	3.20	6
Milk at 1 year	101	1.17	2.34	2.54	11
Milk at 18 months	32	1.02	2.55	3.28	16
Cord serum	744	<4.27	7.49	410.00	88
Maternal serum at birth	872	9.06	19.70	88.80	13
Maternal serum at six weeks	802	6.98	14.60	44.60	26
Placenta	790	<12.00	<12.00	33.00	97
Estimated milk at birth	915	1.74	3.64	15.83	—

Units for milk are ppm (fat basis).  
Units for serum and placenta are ppb.

**TABLE 2—DDE Levels in Various Types of Samples**

Sample	Number	Median	95th Percentile	Maximum	Per Cent Less Than Quantitation Limit
Milk at birth	733	2.43	6.72	25.4	<1
Milk at 6 weeks	617	2.19	5.84	25.7	<1
Milk at 3 months	498	2.07	5.51	23.4	1
Milk at 6 months	362	1.85	4.69	22.5	1
Milk at 9 months	62	1.39	4.91	11.7	0
Milk at 1 year	101	1.51	3.37	12.7	0
Milk at 18 months	32	1.29	4.44	11.9	0
Cord serum	744	3.95	11.80	76.0	8
Maternal serum at birth	872	12.60	34.60	180.0	<1
Maternal serum at 6 weeks	802	9.69	28.20	102.0	1
Placenta	790	6.77	20.15	73.9	11
Estimated milk at birth	919	2.51	7.59	23.8	—

Units for milk are ppm (fat basis).  
Units for serum and placenta are ppb.

assumed that the chemical levels had a log normal distribution, estimated its parameters, and then used as an estimate the expected value conditional on being less than the quantitation limit). Second, levels were multiplied by a scale factor to make them all comparable to milk at birth. This scale factor is the median ratio of birth milk samples to the sample in question. It adjusts for the declining milk values over time and also for the fact that blood levels are substantially lower than milk levels. Third, all available levels were averaged. Because most of the PCB levels for cord blood and placenta were below the quantitation limits, the estimation in the first step could not be done reliably, and these two samples were not used for the PCB calculation. Four women whose only samples were cord blood and/or placenta thus have DDE levels but no PCB levels. This method allows comparisons involving as many women as possible using as much data as

possible. The correlation between the estimated amount in milk and the actual amount (where the actual amount was available and above the quantitation limit) was .86 for PCBs and .94 for DDE.

**Results**

**Characteristics of Participants**

There were 931 children (from 880 women) whose mothers agreed to participate in the study. A number of these women dropped out almost immediately, and, in particular, 12 gave no samples. Furthermore, there were three perinatal deaths. Of the original group, 856 children (807 women) participated past the original neonatal contact. The children originally enrolled include six sets of twins and 45 pairs of non-twin siblings, born between April 1978 and October 1982.

The mothers are not a representative sample. Most of the women (92 per cent) are White, ranging in age from 16 to 41, with a median age of 27. The women are well educated: 53 per cent have a college education or the equivalent. Only 16 per cent of the mothers identify themselves as housewives; 41 per cent are professionals, and 27 per cent are white collar workers; 18 per cent of the women smoke; 40 per cent drink alcohol at least once a week; 21 per cent report eating sport fish (i.e., any fish which were caught rather than purchased) at least once during pregnancy.

Forty-three per cent of the women were primiparous. Most (88 per cent) of the women breast-fed their study child to at least some extent. Of those who started on the breast,

**TABLE 3—Correlations among PCB Measurements**

Sample	Milk at 6 wk	Milk at 3 mo	Milk at 6 mo	Maternal Serum at Birth	Maternal Serum at 6 wk
Milk at birth	.74	.68	.59	.64	.57
Milk at 6 weeks		.77	.70	.66	.65
Milk at 3 months			.71	.60	.57
Milk at 6 months				.56	.56
Maternal serum at birth					.56

TABLE 4—Correlations among DDE Measurements

Sample	Milk at 6 wk	Milk at 3 mo	Milk at 6 mo	Cord Serum	Maternal Serum at Birth	Maternal Serum at 6 wk	Placenta
Milk at birth	.89	.87	.86	.82	.82	.83	.79
Milk at 6 weeks		.88	.88	.78	.83	.85	.79
Milk at 3 months			.88	.78	.81	.82	.75
Milk at 6 months				.75	.81	.82	.71
Cord serum					.79	.81	.77
Maternal serum at birth						.88	.79
Maternal serum at 6 weeks							.80

10 per cent continued for less than a month, while 21 per cent continued more than a year. The median time on the breast was 29 weeks. Twenty-nine per cent of the women had breast-fed a child previously.

#### Chemical Levels

Tables 1 and 2 show the levels of chemicals that were found. The number of samples of each type varies substantially, since not all mothers breast-fed, and not all samples could be collected and analyzed. Note that mothers with two children in the study are included in these Tables twice. We also analyzed 104 formula samples; all but one had no detectable PCBs or DDE.

PCB levels in milk at birth averaged around 1.8 ppm (fat basis). Levels of DDE were generally 40 per cent higher than levels of PCBs, with a median of 2.4 ppm. Levels of both chemicals declined substantially over the course of lactation. In milk, both dropped about 20 per cent over six months and 40 per cent over 18 months. This implies that excretion in milk is a major factor in lessening the mother's body burden; however, it also implies substantial exposure to the child.

Levels of both chemicals were higher in milk than in serum, and higher in maternal serum than in placenta. To some extent, this represents a difference in fat content; our analyses show that milk usually has 1–6 per cent fat, while placenta has 1–1.5 per cent fat. Blood typically has about 0.5 per cent fat. For DDE, levels in cord blood are only about one-third of the levels in maternal blood at birth. For PCBs, levels in cord blood were almost always below the quantitation limit, but the samples that were quantifiable show the same relationship to maternal blood. This may simply be because cord blood has only one-third the fat,<sup>30</sup> or it may indicate the existence of a placental barrier.

The correlations among the various samples taken from the same woman were quite high (Tables 3–4). Correlations between milk samples taken at different times were about 0.7–0.8 for PCBs and 0.9 for DDE. The correlation between milk and blood taken at the same time was about 0.7 for PCBs and 0.8 for DDE. This indicates a substantial degree of coherence in the body burden of these chemicals; a woman's value for one sample is a very good predictor of her value on another.

#### Relation of Chemical Levels to Characteristics of Mothers

Using the summary measure of estimated amount in milk at birth, we found substantial racial differences in DDE levels, but not PCB levels. Table 5 shows the magnitude of the difference; almost half the Blacks had over 6 ppm (fat basis), while only 5 per cent of the Whites reached this level. PCBs show no racial difference. In spite of the fact that pregnant women come from a restricted age range, levels of both chemicals can be seen to increase with increasing

TABLE 5—Distribution of Estimated Chemical Levels in Milk at Birth by Race

ppm (fat basis)	DDE			PCB		
	White	Black	Asian	White	Black	Asian
0–1.49	20%	2%	0%	34%	38%	33%
1.5–2.99	46	12	0	57	54	66
3–4.49	22	27	33	8	8	0
4.5–5.99	7	14	33	1	0	0
6+	5	45	33	0.4	0	0
No. Women	852	51	3	849	50	3

TABLE 6—Mean Chemical Levels in Milk at Birth among White Women by Age

Mother's Age (years)	PCB	DDE
16–19	1.41 (1.16, 1.72)	1.90 (1.45, 2.48)
20–24	1.58 (1.49, 1.68)	1.94 (1.79, 2.10)
25–29	1.88 (1.80, 1.95)	2.41 (2.28, 2.55)
30–34	1.83 (1.75, 1.92)	2.78 (2.58, 3.00)
35–41	1.71 (1.55, 1.90)	2.31 (1.80, 2.97)

Entries shown are means and 95% confidence intervals, obtained by calculating means and standard deviations on the log scale, and transforming the results back to the original scale. Units are ppm (fat basis). Figures are based on the estimated amount in milk at birth.

TABLE 7—Mean Chemical Levels in Milk at Birth among White Women by Occupational Grouping

Occupation	PCB	DDE
Laborer/farmer	1.41 (1.24, 1.61)	2.33 (1.91, 2.83)
Professional	1.89 (1.81, 1.97)	2.45 (2.32, 2.60)
White collar	1.71 (1.62, 1.80)	2.60 (2.42, 2.80)
Paraprofessional	1.81 (1.65, 1.98)	2.06 (1.82, 2.34)
Housewife	1.69 (1.58, 1.81)	2.09 (1.87, 2.33)
Student	1.99 (1.70, 2.33)	1.94 (1.58, 2.39)

Entries shown are means and 95% confidence intervals, obtained by calculating means and standard deviations on the log scale, and transforming the results back to the original scale. Units are ppm (fat basis). Figures are based on the estimated amount in milk at birth.

maternal age; the higher ages have about 30 per cent higher levels (Table 6).

Levels of both chemicals are lower in women who have breast-fed previously; PCB levels are about 12 per cent lower, while DDE levels are about 17 per cent lower. Analysis of variance shows that in addition to the effects of race, age, and prior breast-feeding, DDE levels show effects of smoking, with smokers having 15 per cent higher levels, occupation (Table 7), and of consumption of sport fish during pregnancy, with fish-eaters having levels 14 per cent higher.

PCB levels, in addition to the effects of age and prior breast-feeding, showed effects of occupation (Table 7), prior pregnancies (with those with no prior pregnancies having levels 11 per cent higher), and alcohol (with women consuming at least one drink per week having 13 per cent higher levels). Neither chemical was affected significantly by education or mother's weight.

Data are available on 45 pairs of non-twin siblings who were both in the study. In general, levels for the second recorded lactation are lower than for the first. Based on the summary exposure measure, all but seven of the pairs show lower PCBs for the second child, while all but three show lower DDE for the second child. The median per cent change is a 26 per cent drop for PCBs (23 per cent for DDE). This difference between the two siblings is presumably related, like the decline over the course of lactation, to the excretion of chemicals by the mother. (Data not shown in Tables are available on request to authors.)

### Discussion

Since the women who participated in the study were not drawn randomly from a defined population, assuming they are typical or representative is not strictly justified. There are not, however, many known risk factors for unusual exposures to PCBs or DDT, and none are so strong and so well-known that women could choose to participate in the study because they suspected that their levels were high. It thus seems reasonable to assume that the values found are fairly typical.

We asked the women whether they had ever had any unusual exposures. Thirteen women reported contact with a PCB spill along the North Carolina roadside; their chromatographic patterns but not their PCB levels differed from those of other women in the study.<sup>31</sup> Four women reported some other exposure to PCBs; only one of these had particularly high values. Three women reported exposure to DDT; only one had high values.

The values we find for PCBs are somewhat higher than those reported by others. Schwartz, *et al.*, reported a median of 0.74 ppm fat basis (1260 standard) in 138 neonatal samples, compared with 1.8 ppm reported here.<sup>32</sup> About 75 per cent of the women in their study reported regular consumption of (presumably contaminated) Lake Michigan fish. The correlations between fish consumption and PCB levels in their study, while in some cases statistically significant, were rather modest; this fact, combined with the low levels seen, may mean that fish consumption was not an important source of PCBs for these women. Smith, *et al.*, in a similar study of consumers of contaminated fish in Wisconsin, found a mean of 1.13 ppm.<sup>33</sup> Wickizer, *et al.*, reported on a group of 1057 women with a median value of 1.35 ppm fat basis.<sup>34</sup> The US Environmental Protection Agency's (EPA) national human milk study found 30 per cent of women with values greater than 50 ppb (whole milk basis)<sup>8</sup>; we have similar figures at birth.

The DDE values we find are typical of what other studies have found. The EPA national milk study found a mean of 3.5 ppm fat basis for p,p'-DDE.<sup>7</sup> Jensen cites other US studies with DDE means ranging from 1.2 to 4.8 ppm, plus a study done in a "pesticide area" of Mississippi with a mean of 14.7.<sup>5</sup> As noted above, our group of women is not "representative", but there does not seem to be any special exposure that accounts for the higher PCB levels found in this area. The most likely difference is in the sensitivity of the analytical method we used. The strength of this method is in

the extraction and clean-up procedures designed to remove PCBs quantitatively from the sample matrix.

One of the more striking of our findings is the demonstration of a substantial decline in the levels of both chemicals over the course of lactation. Jensen reviews three studies of serial samples for DDT which show declines similar to the one seen here. In addition, he cites many studies which find that levels are highest for the first child.<sup>5</sup> For PCBs, Kodama and Ota showed a decline in blood levels over the course of lactation, but they saw an inconsistent pattern for milk.<sup>35</sup> Mes, *et al.*, saw no trends over the course of lactation, but they studied only 16 women.<sup>36</sup>

Our finding of a decline in levels over the course of lactation, coupled with the findings that previous breast-feeding is associated with lower chemical levels and that serial pregnancies in the study also show declines, is consistent with the quantitatively important excretion of chemicals by the mother, and consequent exposure of the child, over the course of lactation. Wickizer, *et al.*, have calculated that a child breast-feeding for eight months would have a body burden of about 8 mg.<sup>34</sup> We did not draw blood from the children in this study, since our analytical methods required 20cc and we did not feel justified ethically in attempting to draw such samples serially from asymptomatic children. In other studies, children have been documented to absorb and store PCBs from breast-milk exposure.<sup>35,37,38</sup> Eight mg should not be an obviously acutely toxic dose. Taiwanese Yucheng patients who became clinically ill were estimated to have consumed about a gram of PCBs, and were also exposed to other chemicals.<sup>11</sup> The course of the children in this study is the subject of a separate report.

We and others have found lower levels of these compounds in cord blood than in maternal blood at term.<sup>32,35,38</sup> However, there is a difference in the amount of fat in term maternal and cord blood, with maternal blood about three-fold higher. Polishuk, *et al.*, analyzed lipid extracts from maternal and cord serum and found higher values for PCBs and other organochlorine compounds in the cord side<sup>39</sup>; it is not known, however, whether the amount of these compounds in total extractable lipid from the complex lipid of serum is an estimate of meaningful biological exposure. On a quantitative basis, it appears that transfer through breast milk has much greater potential for a long-term exposure, but that there is some exposure all during gestation, and that if a placental barrier other than a simple solubility differential exists, it is not completely effective.

Besides previous lactation, the most important predictors of chemical levels were race and age. There are studies of serum residues in which Blacks are higher than Whites for PCBs<sup>40</sup> and for DDT,<sup>41</sup> and a study of DDT in adipose tissue where Blacks are higher than Whites.<sup>42</sup> The reason for these differences is unknown. The rise of chemical levels with age was anticipated, since body burdens of these chemicals are slowly accumulated over a woman's lifetime, and excretion or metabolism is rare.

The relationship of DDE values to smoking has been seen in several previous studies.<sup>5</sup> The association of regular alcohol consumption with PCB levels was not expected; if anything, one might hypothesize some degree of increased metabolism due to alcohol consumption, with a consequent decrease in levels. Fein, *et al.*, also found a positive association with alcohol consumption.<sup>43</sup>

Consumption of sport fish from contaminated waters has been shown to influence PCB levels<sup>44</sup>; it has also been shown

to be an important source of DDT.<sup>45</sup> In our data, we see an association with DDE but not with PCBs.

Overall, our data are consistent with the notion that general population exposure to PCBs and DDE is the result of widespread low-level contamination. The most likely source is low-level food contamination, but dermal absorption or lung absorption may play a role. The levels seen here and in other similar surveys are below those that we expect to be associated with overt toxicity in the mother. Our findings of clear declines with both current and prior breastfeeding indicate exposure of the child. For the infant, the PCB values at least are in a range that overlap exposures at which the most sensitive *in vitro* systems respond. Similar low-dose experiments have not been carried out for DDE.

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