The Transfer of Polychlorinated Biphenyls (PCBs) and Polybrominated Biphenyls (PBBs) Across the Human Placenta and into Maternal Milk

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Abstract: Cord serum and maternal milk levels of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) were examined in relation to maternal serum levels. Maternal serum levels were significantly higher than cord serum levels for both types of compounds. Placental passage was indicated by significant maternal to cord serum correlations for both PCBs (r = .42) and PBBs (r = .81). Correlations between maternal serum and milk levels were similar. Higher PBB correlations were probably due to greater reliability in the measurement of PBB levels in serum and milk. (*Am J Public Health* 1984; 74:378–379.)

Introduction

Polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) are chemically related families of synthetic hydrocarbons. Both are lipophilic and poorly metabolized and, therefore, have a long half-life in human tissue. PCBs, used until recently in a wide range of industrial products, are a worldwide and persistent pollutant. They are found at relatively high concentrations in the fatty tissue of predatory sports fish feeding in polluted waters and in the tissue of humans who consume such fish.[1–3] PBBs, used primarily as a fire-retardant, entered the food chain in Michigan in 1973 when accidentally mixed with livestock feed. They are currently found at low concentrations in the tissue of most Michigan residents.[4]

Evidence concerning the placental passage of PCBs is contradictory. While only a small proportion of the PCBs ingested by the dam have been found in the fetal tissue of laboratory rodents,[5,6] higher concentrations have been found in the tissue of the offspring of exposed rhesus monkeys.[7] Three studies of placental passage in humans, based on small samples drawn from the general Japanese population, have yielded inconsistent results.[8–10] Transfer to milk was documented in one of these studies.[10] Data regarding the transfer of PBBs have not been reported.

Materials and Methods

The sample consisted of 313 women and their newborn infants delivered at four hospitals in western Michigan. The women resided in counties which were sites of the 1973 PBB incident and which also offered easy access to PCB-contaminated Lake Michigan sports fish. Details regarding subject recruitment and sample characteristics are reported elsewhere.[11]

Cord and maternal serum samples were collected following delivery. Sixty-one per cent of the mothers breast fed and, of these, 70 per cent provided milk samples between 1 and 16 weeks postpartum. Serum and milk samples were analyzed for the presence of PCBs and PBBs at the Michigan Department of Public Health by means of packed column gas chromatography.[11] Quantification of PCB peaks was provided by adapting the Webb-McCall method[12] to a computer data system, in which Aroclors 1016 and 1260 were used as calibration standards. Serum lipids were determined colorimetrically[13]; milk lipids, gravimetrically. Since virtually all of the serum samples matched to the PCB Aroclor 1016 standard were below the laboratory's detection limit (5.0 ng/mL), only values matched to the Aroclor 1260 standard were used in the data analysis. Any serum samples containing lipid levels below 200 mg/dL were omitted, since measurement techniques were judged insufficiently sensitive at lipid values of that magnitude.

The PCB and PBB measures were log transformed to compensate for skewness in the original distributions. Means and confidence limits were determined on the basis of the transformed distributions.

Results

Mean PCB and PBB levels in cord and maternal serum and milk are shown in Table 1. Maternal serum levels are significantly higher than cord serum levels for both PCBs ($t(147) = -13.90$, $p < .001$) and PBBs ($t(152) = -18.99$, $p < .001$), in part reflecting the greater concentration of lipids found in maternal serum. When calculated on a fat basis, PCB levels in maternal and cord serum are not significantly different ($t(147) = -1.17$, $p > .20$) but PBB concentrations in maternal serum are still about three times greater than in cord serum ($t(152) = -7.41$, $p < .001$). PCB levels are higher than PBB levels in all fluid samples.

Tables 2 and 3 present Pearson product moment correlations relating concentrations in cord serum, maternal serum, and milk for PCBs and PBBs, respectively. The data indicate significant transfer to fetus and milk for both types of compounds.

Discussion

The maternal to cord serum correlation for PCBs is similar in magnitude to that reported in one Japanese study[9] but higher than those reported in two others.[8,10] The lower correlations in the latter two studies may be due to restriction of range associated with the lower PCB-body burdens of the mothers participating in those studies. The maternal serum to milk correlation found here is similar to that reported by Masuda, *et al.*[10]

Given the chemical similarity of these substances, the substantially higher PBB correlations were not expected. These higher correlations are not due to the passage of greater quantities of PBBs across the placenta and into the milk, since the ratios of cord to maternal serum concentrations and of milk to maternal serum concentrations are actually lower for PBBs than for PCBs. The differences may be attributable to differential reliability of measurement.

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TABLE 1—Mean PCB, PBB, and Lipid Levels in Cord Serum and Maternal Serum and Milk

	N	Mean	95% confidence limits
PCBs (1260 standard)			
Cord serum (ng/mL)	198	2.0	0.1–7.2
Maternal serum (ng/mL)	196	4.7	1.1–14.3
Maternal milk (ng/mL)	138	19.3	5.4–63.1
Maternal milk—fat basis (n/g)	138	732.6	293.4–1827.0
PBBs			
Cord serum (ng/mL)	230	0.3	0.0–1.7
Maternal serum (ng/mL)	205	1.7	0.0–9.7
Maternal milk (ng/mL)	138	3.6	0.0–23.0
Maternal milk—fat basis (n/g)	138	105.1	4.38–2089.8
Lipid levels			
Cord serum (mg/dL)	230	324.0	128.8–519.2
Maternal serum (mg/dL)	206	618.1	322.9–913.3
Maternal milk (%)	138	0.029	0.005–0.053

PBBs are easier to measure accurately because few other compounds appear in their region on the gas chromatograms, whereas several common chlorinated pesticides appear in the same region as PCBs. In addition, only one mixture of PBB isomers entered the Michigan food chain, whereas a variety of PCB mixtures are found in the environment. Assuming that certain isomers in each mixture are transferred more readily than others, those PBB isomers that cross readily are a relatively constant proportion of any maternal PBB serum sample, so that total PBB level should estimate their transfer relatively accurately. By contrast, the proportion of PCB isomers that are transferred readily may vary from one individual to another depending on the particular PCB mixtures to which she has been exposed.

The lower concentration of these substances in cord serum as compared with maternal serum is consistent with the notion that the placenta may function as a partial barrier.[10] Laboratory animal studies indicate that substantially greater amounts of PCBs are transferred postnatally via nursing than as a result of placental passage.[6,7] The present data suggest the same may be true for humans, given the substantially higher fat content of milk and the higher concentrations of these substances in milk as compared with maternal and cord serum. Nevertheless, even at low concentrations, intrauterine exposure may be significant for several reasons. In contrast to postnatal ingestion, prenatal exposure is continuous. Although small in comparison to maternal body burden, the quantities found in cord serum may be large relative to the size of the developing fetus. Finally, intrauterine exposure occurs during a period when the organism has been found to be particularly vulnerable to a variety of teratogenic agents[14,15] and lacks protective barriers that are found postnatally (e.g., the blood-brain barrier) as well as fat deposits that might cushion the impact.

TABLE 2—Intercorrelations of PCB Levels in Cord Serum, Maternal Serum, and Maternal Milk

	Maternal serum	Maternal milk (fat basis)
Cord serum	.42*	.16
	(148)	(95)
Maternal serum		.35*
		(91)

Values are Pearson product moment correlations. Sample size is given in parentheses.
*$p < .001$

TABLE 3—Intercorrelations of PBB Levels in Cord Serum, Maternal Serum, and Maternal Milk

	Maternal serum	Maternal milk (fat basis)
Cord serum	.81*	.39*
	(153)	(107)
Maternal serum		.71*
		(92)

Values are Pearson product moment correlations. Sample size is given in parentheses.
*$p < .001$

REFERENCES

1. Humphrey HEB: Population studies of PCBs in Michigan residents. *In:* D'Itri FM, Kamrin M (eds): PCBs: Human and Environmental Hazards. Boston: Butterworth, 1983.
2. Kreiss K, Zack MM, Kimbrough RD, Needham LL, Smrek AL, Jones BT: Association of blood pressure and polychlorinated biphenyl levels. JAMA 1981; 245:2505–2509.
3. Schwartz PM, Jacobson SW, Fein GG, Jacobson JL, Price HA: Lake Michigan fish consumption as a source of polychlorinated biphenyls in human cord serum, maternal serum, and milk. Am J Public Health 1983; 73:293–296.
4. Schwartz EM, Rae WA: Effect of polybrominated biphenyls (PBBs) on developmental disabilities in young children. Am J Public Health 1983; 73:277–281.
5. Takagi Y, Otake T, Kataoka M, *et al:* Studies on the transfer and distribution of [^{14}C]polychlorinated biphenyls from maternal to fetal and suckling rats. Toxic Appl Pharmac 1976; 38:549–558.
6. Masuda Y, Kagawa R, Tokudome S, Kuratsune M: Transfer of polychlorinated biphenyls to the foetuses and offspring of mice. Food Cosmet Toxicol 1978; 16:33–37.
7. Allen JR, Barsotti DA: The effects of transplacental and mammary movement of PCBs on infant rhesus monkeys. Toxicology 1976; 6:331–340.
8. Akiyama K, Ohi G, Fujitani K, Yagyu H: Polychlorinated biphenyl residues in maternal and cord blood in Tokyo metropolitan area. Bull Environ Contam Toxicol 1975; 14:588–592.
9. Kodama H, Ota H: Studies on the transfer of PCB to infants from their mothers. Jpn J Hyg 1977; 32:567–573.
10. Masuda Y, Kagawa R, Kuroki H, *et al:* Transfer of polychlorinated biphenyls from mothers to foetuses and infants. Bull Environ Contam Toxicol 1978; 16:543–546.
11. Jacobson SW, Jacobson JL, Schwartz PM, Fein GG: Intrauterine exposure of human newborns to PCBs: measures of exposure. *In:* D'Itri FM, Kamrin M (eds): PCBs: Human and Environmental Hazards. Boston: Butterworth, 1983.
12. Sawyer LD: Quantitation of polychlorinated biphenyl residues by electron capture gas-liquid chromatography: reference material characterization and preliminary study. J Assoc Off Anal Chem 1978; 61:272–281.
13. Fringe CS, Fendley TW, Dunn RT, Queen CA: Improved determination of total serum lipids by the sulfo-phosphoro-vanillin reaction. Clin Chem 1972; 18:673–674.
14. Wilson JG: Environment and Birth Defects. New York: Academic Press, 1973.
15. Fein GG, Schwartz PM, Jacobson SW, Jacobson JL: Environmental toxins and behavioral development: a new role for psychological research. Am Psychologist, in press.

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