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Concentrations of PCDDs, PCDFs and coplanar PCBs in blood fat of a breast-fed and a formula-fed infant

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1. Introduction

In infants an accumulation (compared to the mother) of lipophilic compounds like PCDDs, PCDFs or PCBs is expected⁵⁰ after several months of breast-feeding which caused concern about possible adverse health effects. Tissue levels in infants who died from Sudden Infant Death Syndrome (SIDS) did not show concentrations higher than the range measured in adults^{2,3)}. However, in these infants nothing is known about concentrations in mother's milk or blood fat.

In order to investigate toxicokinetics of PCDDs, PCDFs and PCBs, intake and fecal excretion of these compounds were measured at the age of one and five months in a breast-fed and a formula-fed infant. Results reported last year (DIOXIN '93) showed a clearly (up to 40 times) higher intake in the breast-fed infant and an almost complete absorption of the most toxic congeners¹⁾. Here we report on concentrations of PCDDs, PCDFs and coplanar PCBs in blood fat of the same infants at the age of 11 months compared to the values of their mothers.

2. Experimental conditions

Basic information about the children was presented earlier¹⁾. Investigations of the blood were performed at the age of 11 months when the breast-fed and the formula-fed infant had a body weight of 10.4 kg and 11.2 kg, respectively. The breast-fed infant had been fully suckled for 4 months and in part for another 4 months. Whole blood was obtained by venipuncture (20 ml in infants, 80 ml in mothers) before breakfast and collected in heparinised vials which were immediately frozen at -18°C until analysed for PCDDs, PCDFs and coplanar PCBs. The analytical methods applied have been described elsewhere^{6,7,8,9,10)} and will not be reported here. 2378-T4CDD toxicity equivalents (TEs) for PCDDs/PCDFs were calculated using I-TEFs⁴⁾. Concentrations below the limit of detection (LD) were taken in consideration as one half of the value defining the limit of detection.

3. Results and discussion

Concentrations of PCDDs, PCDFs and coplanar PCBs in blood fat of the infants and their mothers are listed in the table. Additionally, concentrations in mother's milk are shown (mean values of the analysis of two samples at the age of one month which did not differ much from the values at the age of 5 months). These samples were analysed by Dr. Hille and Dr. Ende, Lebensmitteluntersuchungsamt Oldenburg, Germany.

Ηυτοχ

Compound	breast-fed infant			formula-fed infant				
(Conc. in pg/g fat)	Mother's milk	Mother's blood	infants blood		Mother's blood		infants blood	
2378-T4CDF 2378-T4CDD	1.0 1.9	<1.7 1.9	<2.7 3.7		<1.7 2.0		<3.0 <1.0	
12378-P5CDF 23478-P5CDF 12378-P5CDD	0.3 19.9 7.7	<1.0 8.6 4.1	<1.2 23.1 11.1		<1.0 11.7 6.4		<1.2 1.5 <1.0	
123478-H6CDF 123678-H6CDF 234678-H6CDF 123478-H6CDD 123678-H6CDD	3.6 3.8 0.7 2.4 24.2	4.0 3.7 <1.9 4.1 18.9	9.8 8.1 <3.4 7.8 43.0		5.3 4.6 <2.9 6.3 29.2		<2.2 <1.0 <2.3 <1.1 2.5	
123789-H6CDD 1234678-H7CDF 1234678-H7CDD	1.9 6.0 14.6	2.1 13.3 21.8	7.1 13.1 24.3		3.2 10.1 27.8		<1.2 <5.8 8.8	
OCDF OCDD I-TE (<ld=0.5*ld)< td=""><td>5.1 63.6 <i>19.7</i></td><td><8.0 189.3 <i>12.3</i></td><td><5.0 148.7 29.2</td><td></td><td><7.1 372.0 16.9</td><td></td><td><5.0 79.3 2.4</td><td></td></ld=0.5*ld)<>	5.1 63.6 <i>19.7</i>	<8.0 189.3 <i>12.3</i>	<5.0 148.7 29.2		<7.1 372.0 16.9		<5.0 79.3 2.4	
PCB 77 PCB 126 PCB 169	n.a. n.a. n.a.	13 105 111	(m) 23 287 270	(m)	19 193 191	(m)	26 24 7	(m)

* analysed by LUA Oldenburg

n.a. = not analysed

(m) = maximum value, due to possible contribution of a contaminent

In the breast-fed infant a distinct accumulation of PCDDs, PCDFs and coplanar PCBs in blood fat was observed compared to mother's values. A factor of 2 to nearly 3 was found for the most toxic congeners (due to higher concentrations lower values were obtained when compared to mother's milk). Using a) the known intake of PCDDs/PCDFs during the first months of life and b) the baby's body weight and assumed body composition at the age of 11 months for a (theoretical) calculation, an accumulation factor of 1.8 had to be expected. The values measured seem to be in good agreement with this assumption.

In the formula-fed infant which had a much lower intake of PCDDs/PCDFs (2.1 and 1.6 pg I-TE/kg/day versus 82.2 and 38.3 pg I-TE/kg/day in the breast-fed infant, at one and five months, respectively) only four PCDD/PCDF congeners were detectable in blood fat. Values were clearly lower than those of the mother. Since concentrations of both mothers were in the same range, prenatal exposure of the infants can be assumed to be comparable.

From the preliminary results presented here we conclude that breast-feeding for several months in deed leads to an accumulation of PCDDs, PCDFs and PCBs, as expected from theoretical considerations. In contrast, prenatal exposure seems to play a minor role for the body burden of a breast-fed child.

4. References

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