Plasma PCB levels and dietary intake of PCBs and PCDD/Fs in Dutch preschool children

Svati Patandin[1], Nynke Weisglas-Kuperus[1], Pieter C. Dagnelie[2], Corine Koopman-Esseboom[1] and Pieter J.J. Sauer[1]

[1] Department of Paediatrics, Division of Neonatology, Erasmus University and Academic Hospital/Sophia Children's Hospital Dr. Molewaterplein 60, 3015 GJ Rotterdam, The Netherlands

[2] Institute of Internal Medicine II, Erasmus University of Rotterdam, P.O.Box 1738, 3000 DR, Rotterdam, The Netherlands.

Abstract

Polychlorinated biphenyls (PCBs) in plasma of Dutch preschool children are reported. The relationship of these levels and their exposure to PCBs through placental and lactational transfer as well as their dietary intake at 3.5 years is described. Plasma PCB levels in children breast-fed (n=91) in infancy were 4 times higher than plasma PCB levels measured in children fed formula (n=82) during infancy. Preschool children have plasma PCB levels mainly dictated by lactational and placental transfer of maternal PCBs. Dietary intake of PCBs at preschool age was significantly associated with plasma PCB levels in children from the formula-fed group. The main contributors of PCBs and polychlorinated dibenzo-dioxins/furans (PCDD/Fs) in Dutch preschool children are dairy products (46%), processed foods (27%) and meat (16%). Strategies should be directed towards reducing PCB accumulation through the food chain. Mothers today would have to lower their longterm dietary intake of PCBs to reduce PCB exposure to their children.

1. Introduction

Polychlorinated biphenyls (PCBs), chlorinated dibenzo-para-dioxins, -di-benzo-furans (PCDD/Fs) are widespread and persistent environmental pollutants[1)]. Human exposure to PCBs and PCDD/Fs is mainly through the food chain. They are stored in adipose tissue, and not readily excreted, except through lactation[2,3)]. The Netherlands belong to countries with the highest environmental levels of PCBs and PCDD/Fs measured in human milk as well as in animal products[4)]. Tissues in human newborns contain PCBs and PCDD/Fs through placental transport[3,5,6)], however much larger quantities of PCBs and PCDD/Fs are transferred to the infant during breast-feeding[3,6-8)]. We report plasma PCB levels measured in Dutch preschool children, either breast-fed or formula-fed in infancy. Their current dietary intake of PCBs and PCDD/Fs is estimated and related with plasma PCB levels at 3.5 years of age.

2. Methods

From 1990-1992, 207 mother-infant pairs were recruited in Rotterdam and surroundings, a highly industrialized area in the Netherlands. Infants, 1st or 2nd, born at term, West-European origin, without perinatal complications, were included for follow-up from birth until preschool age. To study the effects of perinatal exposure to PCBs and PCDD/Fs, two groups of women were included, a group of women who intended to breast-feed their child for at least 6 weeks (breast-fed group = BF) and a group of women who intended to give formula (formula-fed group = FF) directly after birth. The study design, chemical analysis and findings until 18 months of age have been described elsewhere[7,9,10]. Participating children were invited at 3.5 years of age to the Childrens Hospital for blood collection and anthropometric assessment. Parents were asked to fill out a food questionnaire designed to estimate intake of PCBs and PCDD/Fs. Plasma PCB levels at 3.5 years of age were analysed at the Nutrition and Food Research Institute, Zeist, The Netherlands. Four PCB congeners, IUPAC nrs 118, 138, 153 and 180 were measured by gas chromatography with electron capture detection[10]. The sum of PCBs (∑PCB) was defined as the sum of PCB 118, 138, 153 and 180. From 193 subjects, 173 plasma samples were analysed for PCBs. Prenatal PCB exposure was estimated by the ∑PCB in maternal plasma collected during the last trimester of pregnancy and in cord plasma[10]. Postnatal PCB exposure was estimated by the ∑PCB of these 4 congeners in breast milk. Formula given to children had negligible concentrations of PCBs and PCDD/Fs.
Dietary intake of PCBs and PCDD/Fs at preschool age, was assessed by a validated semi-quantitative food frequency questionnaire (FFQ). The daily intake of PCBs and PCDD/Fs was estimated by calculating the toxic equivalents (TEQ) for planar PCBs (IUPAC nr 77, 126 and 169)[11] and 2,3,7,8-tetra-chloro-dibenzo-p-dioxin (TCDD) in each food item[11,12] using reference data for food products provided by the Dutch National Institute of Public Health and Environmental Protection (RIVM)[13,14]. Energy and fat intake were calculated using the Dutch Food Database 1993[15]. The total TEQ intake was defined as the sum of planar PCB TEQ and TCDD TEQ in picogrammes per day (pg/d). There were 183 FFQs available for analysis. Information regarding education and occupation of parents were obtained at the 3.5 year assessment. Weight (kg), height (m) and skinfolds-thicknesses (mm) at 4 sites, bicipital, tricipital, subscapular and supra-iliacal skinfolds were measured. Total body fat percentage was calculated from the 4 skinfolds[16].
Mean and 95%-CI the 4 PCB congeners in plasma at 3.5 years of age are shown. The student t-test, χ-square test and Mann-Whitney test were used to compare differences between the BF and FF group. Multiple linear regression analysis was performed for the BF and the FF group, separately. Results were considered statistically significant at a p-value $\leq$ 0.05.

3. Results

The follow-up response at 3.5 years of age was 93%, (n=193). There were 100 children in the BF group and 93 in the FF group. PCB levels were measured in plasma of 173 children, 91 from the BF group and 82 from the FF group. In 19 children no permission for blood collection was given and 1 sample was lost during PCB analysis. Polychlorinated biphenyls were detectable in all plasma samples. In figure 1 means and errorbars are presented of the 4 PCB congeners at 3.5 years of age in the BF and FF group. Children in

the BF group had 4 times higher plasma PCB levels compared to the FF group (t-test, $p<0.0001$).
Plasma PCB levels of individual congeners and $\sum$PCB at 3.5 years of age in the BF and FF group, were significantly correlated with the levels in their maternal and cord plasma (data not shown, $p<0.05$). In the BF group, correlations between plasma PCB levels at preschool age and PCB levels in breast milk, was also significant ($p<0.01$). No significant correlations were found for PCDD/Fs TEQ in breast milk and plasma PCB levels at 3.5 years.
Regression analysis for the $\sum$PCB measured in plasma of children at 3.5 years of age, showed that in the BF group, duration of breast-feeding ($p<0.0001$) and ΣPCB level in breast milk ($p=0.02$) were important predicting variables for plasma PCB levels at 3.5 years. In the FF group, plasma ΣPCB levels at 3.5 years of age, were significantly influenced by their maternal ΣPCB levels ($p=0.009$) and the dietary intake of PCBs and PCDD/Fs at preschool age ($p=0.04$). Body weight of the child was negatively associated with the PCB levels at 3.5 years in the BF and FF group ($p= 0.02$ and $p=0.0002$, respectively). Similar results were found when body fat percentage was entered in the regression analysis instead of body weight.
Dietary intake of planar PCB TEQ and 2,3,7,8 TCDD TEQ was not significantly different for the BF and the FF group. Mean daily intake of TCDD TEQ in the BF and the FF group was 46 and 47 pg/d respectively, and for planar PCB TEQ was 60 and 57 pg/d respectively. Seven children from this study (4%) exceeded the tolerable daily intake (TDI) level of 10 pg TEQ/kg body weight per day.
Multiple regression analysis showed that after adjustment for gender and feeding type (BF vs FF) during infancy, the consumption of PCB TEQ and PCDD/F TEQ was significantly higher in children of mothers with lower levels of education (all p-values <0.001).
Multiple regression analysis showed that energy, fat and carbohydrate intake was significantly higher in children with lower educated mothers (all p-values < 0.001). Energy and carbohydrate intake was significantly lower in girls ($p=0.004$ and $p=0.001$, respectively), however this was not significant for fat intake.
In figure 2, the contribution of dietary intake in preschool children for planar PCB-TEQ and PCDD/F-TEQ for several groups of food items is given. Dairy products, processed foods and meat products are the major contributors for PCBs and PCDD/Fs dietary intake for preschoolers (73% and 84%, respectively). Processed foods and sea fish consumption contributed to a higher dietary PCB intake compared to dietary PCDD/F intake (22.6% vs 15.5% and 10.7% vs 5%, respectively).

4. Discussion and conclusions

Significant amounts of PCBs are present in plasma of Dutch preschool children exposed to background levels of PCBs and PCDD/Fs. Children breast-fed in infancy have plasma PCB levels that are times higher compared to their peers who have been formula-fed during infancy. Our results confirm that plasma PCB levels in children breast-fed during infancy are mainly dictated by lactational transfer. However our data also show that placental transfer of maternal PCBs has a significant influence on plasma PCB levels in preschool children. To lower plasma PCB levels in childhood, total PCB body burden of mothers would have to be reduced. Future mothers, have to reduce their long-term dietary intake of PCBs.

For preschool children the main contributors of PCBs and PCDD/Fs through dietary intake are dairy products, processed foods and meat. The effect of dietary PCB intake after weaning in these children was less important for their plasma PCB levels measured at 3.5 years of age. The influence of long-term dietary intake however, is important for the total body burden of PCBs and PCDD/Fs. We found that 4% of our study group exceeded the TDI level of 10 pg TEQ/ kg body weight. According to a Dutch survey performed by the RIVM [13,14], 1% of all children below 6 years of age exceed the TDI level. We believe that strategies should be directed towards reducing PCB accumulation through the food chain. We also observed that PCB levels are negatively associated with body weight and total body fat percentage, which does not imply that total body burden of PCBs in fat children would be lower. This association reflects PCB levels in the fat compartment and may be explained by a dilutional effect. Therefore, it would be better if PCBs were expressed per gram lipids in plasma or in adipose tissue.

Acknowledgments
This research was funded by the European Commission for Environmental and Health Programmes. We wish to thank all families participating in this study. We are very grateful to the students Juul van der Veen and Eline Op de Coul from the Department of Human Nutrition, Agricultural University Wageningen for the development of the food frequency questionnaire.

5. References

1) de Voogt P and U.A.T. Brinkman. Production, properties and usage of polychlorinated biphenyls (1989): Kimbrough RD, Jensen A, eds. Halogenated biphenyls, terphenyls, naphtalenes, dibenzodioxins and related products. 2 ed. Amsterdam: Elsevier, 3-46.
2) Yakushiji T., I. Watanabe, K. Kuwabara, R. Tanaka, T. Kashimoto, N. Kunita, I. Hara (1984): Postnatal transfer of PCBs from exposed mothers to their babies: influence of breast-feeding. Arch Environ Health 39, 368-75.
3) Yakushiji T. (1989): Contamination, clearance, and transfer of PCB from human milk. Rev Environ Contam Toxicol 101, 139-64.
4) World Health Organization (1989): Levels of PCBs, PCDDs, and PCDFs in breast milk; results of WHO-coordinated interlaboratory quality control studies and analytical field studies. Regional office for Europe, FADL, Copenhagen.
5) Masuda Y., R. Kagawa, H. Kuroki, M. Kuratsune, T. Yoshimora, I. Taki, M. Kusuda, F. Yamashita, M. Hayashi (1978): Transfer of polychlorinated biphenyls from mothers to foetuses and infants. Food Cosmet Toxicol 16, 543-6.
6) Jacobson J.L., G.G. Fein, S.W. Jacobson, P.M. Schwartz, JK. Dowler (1984): The transfer of polychlorinated biphenyls (PCBs) and polybrominated biphenyls (PBBs) across the human placenta and into maternal milk. Am J Public Health 74, 378-9.
7) Koopman-Esseboom C., N. Weisglas-Kuperus, M.A.J. de Ridder, C.G. van der Paauw, L.G.M.Th. Tuinstra, P.J.J. Sauer. Effects of PCB/Dioxin exposure and feeding type on the infant's mental and psychomotor development. Pediatrics, in press.
8) Niessen K.H., J. Ramolla, M. Binder, G. Brugmann, U. Hofmann (1984): Chlorinated hydrocarbons in adipose tissue of infants and toddlers: inventory and

tudies on their association with intake of mothers' milk. Eur J Pediatr 142, 238-44.
9) Huisman M., C. Koopman-Esseboom, V. Fidler, M. Hadders-Algra, C.G. van der Paauw, L.G. Tuinstra, N. Weisglas-Kuperus, P.J. Sauer, B.C. Touwen, E.R. Boersma (1995): Perinatal exposure to polychlorinated biphenyls and dioxins and its effect on neonatal neurological development. Early Hum Dev 41, 111-27.
10) Koopman-Esseboom C., M. Huisman , N. Weisglas-Kuperus, C.G. Van der Paauw, L.G.M.Th. Tuinstra, R. Boersma , P.J.J. Sauer (1994): PCB and Dioxin levels in plasma and human milk of 418 Dutch women and their infants. Predictive value of PCB congener levels in maternal plasma for fetal and infant's exposure to PCBs and Dioxins. Chemosphere 28, 1721-32.
11) Ahlborg U.G., G.C. Becking, L.S. Birnbaum, A. Brouwer, H.J.G.M. Derks, M. Feeley, G. Golor, A. Hanberg, J.C. Larsen, A.K.D. Liem, S.H. Safe, C. Schlatter, F. Wærn, M. Younes, E. Yrjänheikki (1994): Toxic Equivalency Factors for Dioxin-like PCBs. Report on a WHO-ECEH and IPCS consultation, December 1993. Chemosphere 28, 1049-67.
12) Safe S.H. (1994): Polychlorinated Biphenyls (PCBs): Environmental Impact, Biochemical and Toxic Responses, and Implications for Risk Assessment. Crit Rev Toxicol 24, 87-149.
13) Liem A.K.D., R.M.C. Theelen, W. Slob, J.H. van Wijnen (1991): Dioxinen en planaire PCB's in voeding. Gehalten in voedingsproducten en inname door de Nederlandse bevolking. Bilthoven: Rijksinstituut voor volksgezondheid en milieuhygiene (RIVM).
14) Theelen R.M.C., A.K.D. Liem, W. Slob, J.H. van Wijnen (1993): Intake of 2,3,7,8, chlorine substituted dioxins, furans, and planar PCBs from food in The Netherlands: median and distribution. Chemosphere 27, 1625-1635.
15) NEVO-tabel. Nederlandse Voedingsstoffenbestand (1993): Stichting NEVO, Voorlichtings bureau voor voeding Den Haag.
16) Weststrate J.A. and P. Deurenberg (1989): Body composition in children: proposal for a method for calculating body fat percentage from total body density or skinfold-thickness measurements [published errata appear in Am J Clin Nutr 1991 Aug;54, 428 and 1991 Sep;54, 590]. Am J Clin Nutr 50, 1104-15.

Fig.1 Plasma PCB levels
in Dutch children at 3.5 years of age

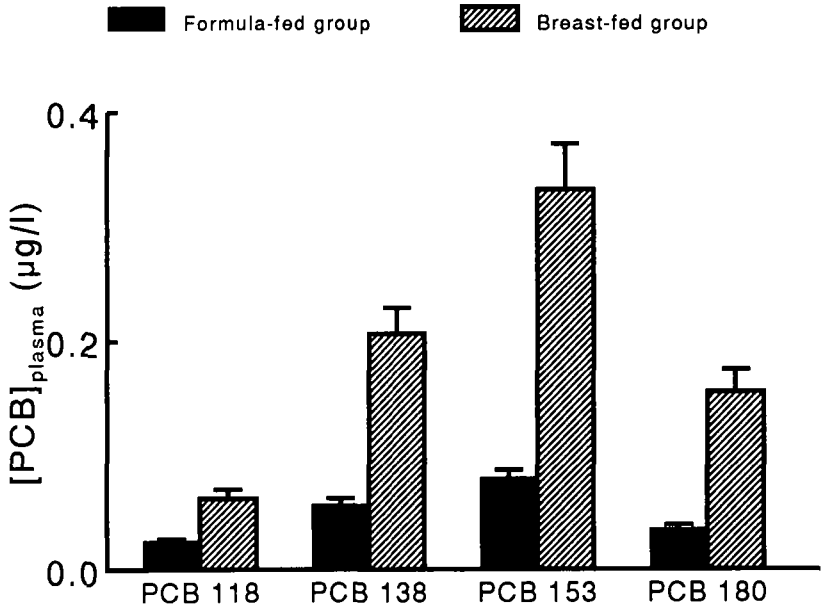
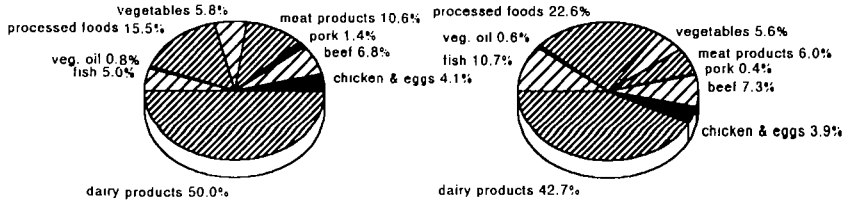


Fig. 2 Intake in preschool children
Contribution of categories food products



PCDD/F-TEQ intake

PCB-TEQ intake