

Birth weight and growth in Dutch newborns exposed to background levels of PCBs and Dioxins

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Abstract

Two hundred and seven healthy newborns were followed from birth until pre-school age. Anthropometric measurements were collected at birth, 10 days, 3-, 7-, 18- and 42 months of age. Growth was defined as change in standard deviation score (SDS) for weight and length between two successive measurements. Prenatal PCB exposure was estimated from cord plasma PCB levels. After adjustment for covariates, cord plasma PCB levels had a significant negative effect on birth weight as well as growth from birth to 3 months of age. Growth beyond 3 months of age was not related to prenatal PCB exposure. We conclude that in utero exposure to PCBs has a negative effect on birth weight as well as growth until 3 months of age.

Introduction

Polychlorinated biphenyls (PCBs), and Polychlorinated dibenzo-p-dioxins (PCDDs) and -dibenzo-furans (PCDFs), summarized as dioxins, are widespread environmental contaminants. Intrauterine growth retardation was reported in the Yusho and YuCheng incidents in which pregnant women consumed cooking oil accidentally contaminated with PCBs and related compounds¹. Reduced birth weight and a shorter gestation was observed in infants whose mothers consumed contaminated fish from Lake Michigan, USA². We investigated the influence of pre- and postnatal exposure to background PCB and dioxin levels on birth size and postnatal growth. Results from the Rotterdam part of the "Dutch/PCB dioxin study" are presented in this paper.

Methods

From 1990 to 1992, infants born at term (37-42 weeks of gestation), without congenital anomalies or diseases were recruited for this study. Pregnancy and delivery had to

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be without complications or serious illnesses. All mother-infant pairs included were of the Caucasian race. To study the effects of prenatal and postnatal exposure to PCBs and dioxins, women were included who intended to give formula-feeding (formula-fed group) next to women who intended to breast-feed their child for at least 6 weeks (breast-fed group). In the formula, concentrations of both PCBs and dioxins were negligible. Participants lived in Rotterdam or its immediate surroundings, a highly industrialized and densely populated area in the western part of the Netherlands.

Maternal and umbilical cord plasma samples were obtained in the last month of pregnancy and shortly after delivery. Four non-planar PCB congeners, IUPAC n's 118, 138, 153 and 180 were analysed in maternal and cord plasma. The PCB sum (\sum PCB) was calculated by adding up the 4 congeners in each plasma sample. A 24-hour representative breast milk sample was collected 2 weeks after delivery from each breast-feeding mother, and analysed for 17 dioxin and 26 PCB congeners. Prenatal PCB exposure was estimated from PCB levels measured in maternal and cord plasma. Postnatal PCB and dioxin exposure was estimated by PCB and dioxin TEQ (toxic equivalents) levels measured in breast milk multiplied by the number of weeks of breast-feeding³.

Birth weight and weight, length and head circumference were collected at 10 days, 3, 7, 18 and 42 months of age. Weight, length or height, and head circumference were converted into standard deviation scores (SDS) using weight, height and head circumference standards of healthy Dutch children as reference data. The SD Scores were calculated with the following formula; $SDS = (X - \text{Mean})/SD$, $X = \text{Weight or Height or Head circumference}$, $\text{Mean} = \text{mean for age and sex of reference data}$ and $SD = \text{standard deviation for age and sex of reference data}$ ⁴. Growth rate was defined as change in SD score of weight ($\Delta wSDS$), length ($\Delta hSDS$) and head circumference ($\Delta hcSDS$) between 2 successive measurements, e.g. birth/10 days and 3 months.

Potential confounding variables for birth size and growth were selected from data on socio-economic background, maternal (health) history, pregnancy and delivery, gestational age, parity, gender and fetal exposure to alcohol and cigarette smoking. Parent's height served to calculate the target height (cm) (TH) as predicting variable for birth size and growth. Linear regression analysis was carried out to study the influence of prenatal PCB exposure on birth weight. The influence of pre- as well as postnatal PCB and dioxin exposure on growth per interval, e.g. from 0-3 months was also studied by linear regression analysis. Results were significant if $p < 0.05$.

Results and Discussion

In this study 207 mother-infant pairs were enrolled, of whom 105 were in the breast-fed group (BF) and 102 in the formula-fed group (FF). Linear regression analysis showed that after adjustment for gestational age, parity, target height, smoking and alcohol use during pregnancy, the \sum PCB in cord plasma negatively influenced birth weight (β (SE) = -119 (54), $p=0.03$, $n=179$). Similar results were found when maternal \sum PCB levels was entered in the regression analysis as a measure for prenatal exposure.

The effect of pre- as well as postnatal PCB and dioxin exposure on growth between 0-3, 3-7, 7-18 and 18-42 months was studied by multiple linear regression analysis. Cord plasma

PCB levels and PCB- and dioxin-TEQ multiplied by breast feeding weeks, were entered as pre- respectively, postnatal exposure variables. Cord plasma PCB levels were negatively associated with the change in SD score of body weight (Δw SDS) between 0-3 months ($p=0.03$), however the influence on change in SD score of length (Δh SDS, $p=0.06$) and head circumference (Δhc SDS, $p=0.17$) were not significant. Postnatal PCB and dioxin exposure were not related to growth after birth, nor was prenatal PCB exposure related to growth beyond 3 months of age (see table).

Our findings are consistent with the results found in animal⁵ as well as human studies^{1,2}, showing that prenatal exposure to PCBs is associated with reduced birth weight. We also found that weight gain (growth) during the first 3 months after birth, is negatively influenced by prenatal PCB exposure. Although lactational exposure to PCBs and dioxins is much higher, the effects described are associated with prenatal PCB exposure. We must keep in mind that the effects are small and within the normal range, however they are described in a population exposed to background PCB and dioxin levels.

Table: Linear regression analysis for growth from 0-3 months of age.

	Weight growth [*]		Length growth ^{**}		Head growth ^{***}	
	β (se) [†]	p-value	β (se)	p-value	β (se)	p-value
Constant	3.5 (2.33)		-0.69 (1.84)		5.6 (1.9)	
Ln(\sum PCB) _{cord} [#]	-0.27 (0.13)	0.03	-0.18 (0.10)	0.06	-0.14 (0.10)	0.17
Total TEQ _@ [#]	2.07 (1.53)	0.89	1.34 (1.24)	0.28	-6.55 (1.25)	0.60
ng/g fat x weeks						
weight-SDS at birth	-0.38 (0.07)	<0.001				
height-SDS at 10 days			-0.32 (0.06)	<0.001		
head-SDS at 10 days					-0.23 (0.05)	<0.001

*: (Δw SDS) change in standard deviation score of body weight **:(Δh SDS) change in standard deviation score of body length ***:(Δhc SDS) change in standard deviation score of head circumference. #: Sum of Polychlorinated Biphenyls, IUPAC nrs 118, 138, 153 and 180 in cord blood @: Postnatal exposure; sum of toxic equivalents (TEQs) of 8 dioxin like PCB and 17 dioxin congeners measured in breast milk, multiplied by the number of weeks of breast-feeding until 3 months. †: β (se)=Regression coefficient (standard error)= change in Δ SD score per unit change in variable. All regression analyses are adjusted for alcohol and cigarette smoking during pregnancy and gestational age.

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