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Perinatal exposure to PCBs and dioxins and the neurological status at 3½ years of age

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Abstract

We investigated the effect of perinatal exposure to PCBs and dioxins on the neurological condition at 3½ years of age. PCB levels were determined in cord and maternal plasma, and used as a measure of prenatal exposure. To measure postnatal exposure, breast milk was analyzed for PCBs and dioxins. In addition, PCBs were determined in child's 3½-year plasma. The neurological condition was assessed in 394 children by means of the Touwen/Hempel method. Neither prenatal exposure to PCBs nor lactational exposure to PCBs and dioxins nor the child's body burden at 3½ years was found to be related to the neurological condition at 3½ years of age after adjustment for covariates.

Introduction

As part of the 'Dutch PCB/Dioxin Study', we have studied the effects of 'background' levels of perinatal exposure to PCBs and dioxins on the neurological condition in the second week after birth[1)], and at 1½ years of age[2)]. Therefore, we followed a group of healthy term infants from birth. In the second week after birth, it was found that a high prenatal exposure together with a high lactational exposure resulted in an adverse effect on the neurological condition and a higher incidence of hypotonia. At 1½ year, higher levels of prenatal PCB exposure were found to be related to a less optimal neurological condition, and no effect of lactational exposure to PCBs and dioxins was found.

In the present abstract, we report on the effect of prenatal exposure to PCBs, exposure to PCBs and dioxins through breast milk, and the PCB level measured in the child's 3½-year plasma on the neurological condition at 3½ years of age.

Methods

Healthy pregnant women were asked to participate in the study. The study was performed in two study centres, Groningen and Rotterdam, The Netherlands. It was planned to include 50% breast-feeding and 50% formula-feeding mother/infant pairs in each study centre. Only healthy, first or second born term children who did not suffer from complications during fetal life and birth were included in the final study group. Moreover, in addition to the formula-feeding mother/infant pairs, we included only those who could sustain breast-feeding for at least six weeks. For each mother, the duration of breast-feeding was noted. We used a 72-item questionnaire to record information on the obstetrical, socioeconomic, pre-, intra-, and immediate postpartum conditions. A compound score of this, the obstetrical optimality score, was made by counting the number of items that fulfilled pre-set optimality criteria.

We collected both maternal and cord blood. Child's blood was sampled at 3½ years. In plasma we determined the non-planar PCB congeners 118, 138, 153, and 180. The sum of the levels of these four congeners (PCBsum) was calculated for cord, maternal, and child's plasma. PCB levels in cord and maternal plasma are considered to be a reflection of prenatal exposure. To asses lactational exposure, human milk samples were collected, and analyzed for three planar and 23 non-planar PCB congeners and 17 ubiquitous 2,3,7,8-substituted dioxins. We calculated a total PCB/dioxin TEQ, a dioxin TEQ, a planar PCB TEQ (nrs. 77, 126, and 169), a mono-ortho PCB TEQ (nrs. 105, 118, and 156), and a di-ortho PCB TEQ (nrs. 170, and 180).

At 3½ years, the neurological condition was assessed according to Touwen/Hempel[3]. This age-adequate technique is directed at the observation of the motor functions prehension, sitting, crawling, standing and walking. It is conducted in a free-field situation. The neurological examination led to a clinical diagnosis: normal, mildly abnormal, or abnormal. In addition, we evaluated the neurological findings in terms of optimality by means of a list of 56 predefined criteria for optimality. A neurological optimality score (NOS) was calculated for each child. The quality of movements was scored as a 15-item fluency cluster score. The examiners were unaware of the levels of pre- and postnatal exposure to PCBs and dioxins and the type of feeding during early life.

To investigate the effect of pre- and postnatal exposure to PCBs and dioxins on the neurological condition at 3½ years, we performed a linear regression analysis. Results were considered to be significant if $p \leq 0.05$.

Results and Discussion

The final study group consisted of 418 mother infant pairs, of which 209 were in the breast-feeding group and 209 in the formula-feeding group. At 3½ years, the neurological condition was assessed in 394 (94%). Twelve (3%) children were considered to be mildly abnormal. One child was diagnosed as abnormal. The remaining 3½-year-olds (n=381; 97%) were considered to be neurologically normal. The children classified as mildly abnormal or abnormal did not differ from the group of neurological normal children in terms of PCB levels in plasma, and PCB and dioxin levels in breast milk. The median NOS at 3½ years was 52 (range: 30-56), whereas the mean fluency cluster score was 13 ± 2. Table 1 shows the PCB levels in plasma, and table 2 presents the PCB and dioxin levels in breast milk.

Table 1: PCB levels (μg/l) in plasma.

PCBsum*	Maternal plasma (n=394)	Cord plasma (n=352)	3½-year plasma (n=298)
Median	2.0	0.4	0.4
5th percentile	1.0	0.2	0.1
95th percentile	3.8	0.9	1.9

* PCBsum = sum of the levels of the PCB congeners 118, 138, 153, and 180.

Table 2: PCB and dioxin levels (ng TEQ/kg fat) in breast milk.

PCB/dioxin levels	Dioxins (n=170)	Planar PCBs (n=186)	Mono-ortho PCBs (n=186)	Di-ortho PCBs (n=186)
Median	28.8	14.5	14.2	4.2
5th percentile	14.9	6.8	6.9	2.1
95th percentile	51.5	31.9	24.8	7.8

Neither the PCBsum in cord plasma nor the PCBsum in maternal plasma nor the PCBsum in child's 3½-year plasma was found to be significantly related to the 3½-year NOS. Adjustments were made for the study centre and the obstetrical optimality score. No significant effect of the levels of dioxins, planar-, mono-ortho, di-ortho PCBs, and the total PCB/dioxin TEQ on the NOS was found. The final model consisted of the study centre and the obstetrical optimality score. Neither the PCBsum in cord plasma nor the PCBsum in maternal plasma nor the PCBsum in child's 3½-year plasma nor the levels of dioxins, planar-, mono-ortho, and di-ortho PCBs nor the total PCB/dioxin TEQ was significantly related to the fluency cluster score.

Our results are in accordance with the findings by Rogan and Gladen who, in the United States, studied the effect of pre- and postnatal exposure to PCBs on cognitive development; deficits seen through two years of age were no longer apparent at three, four or five years of age[4]. On the other hand, also in the US, Jacobson and co-workers[5] found that higher levels of prenatal exposure predicted poorer cognitive functioning at four years. It should be kept in mind that cognitive tests measure the child's abilities in a quantitative fashion, whereas the neurological examination is a qualitative measure of brain function.

In conclusion, adverse effects of prenatal exposure on the neurological condition could not be detected at 3½ years. Moreover, postnatal exposure to PCBs and dioxins was not found to be related to the neurological condition at 3½ years.

Acknowledgments

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