codex alimentarius commission

FOOD AND AGRICULTURE ORGANIZATION OF THE UNITED NATIONS

WORLD HEALTH ORGANIZATION

JOINT OFFICE: Viale delle Terme di Caracalla 00100 ROME Tel: 39 06 57051 www.codexalimentarius.net Email: codex@fao.org Facsimile: 39 06 5705 4593

Agenda Item 16 H **CX/FAC 03/33-Add. 1**
February 2003

JOINT FAO/WHO FOOD STANDARDS PROGRAMME

CODEX COMMITTEE ON FOOD ADDITIVES AND CONTAMINANTS
Thirty-fifth Session
Arusha, United Republic of Tanzania, 17 - 21 March 2003

PROPOSED DRAFT CODE OF PRACTICE FOR SOURCE DIRECTED MEASURES TO REDUCE DIOXIN AND DIOXIN LIKE PCB CONTAMINATION OF FOODS

COMMENTS

The following comments have been received from IBFAN:

IBFAN (International Baby Food Action Network)**:**

We suggest to add the following text to the proposed draft code:
Countries shall sign and ratify the Stockholm Convention on Persistent Organic Pollutants.
We are convinced that a code of source directed measures is a good way to reduce the environmental load and the load that humans are exposed to as being at the top of the foodchain. We are specially concerned by the prenatal and postnatal exposure of human infants , who are especially vulnerable to the adverse developmental and long term disease effects of dioxins and PCBs. Studies have proven that source directed measures, where they are in force, have brought a reduction in the amount found present in breastmilk. The prenatal transfer to the unborn is of greater concern and has shown to cause measurable neurological and developmental effects in newborns and babies. The transfer through breastmilk has not shown similar effects see study below:

Adv Exp Med Biol 2000;478:271
Environmental exposure to polychlorinated biphenyls (PCBs) and dioxins. Consequences for longterm neurological and cognitive development of the child lactation.
Boersma ER, Lanting CI.
Department of Pediatrics/Obstetrics and Gynecology, University Hospital Groningen, The Netherlands.

Polychlorinated biphenyls (PCBs) and dioxins are environmental pollutants. Prenatally, as well as postnatally through breast feeding, large amounts are transferred from mother to the child. Formula is free of these substances. Considering their potential developmental neurotoxicity, we investigated long term effects of perinatal exposure to PCBs and dioxins on neurological and cognitive development. Given the evidence that PCBs exert oestrogenic effects, and oestrogens are known to suppress lactation, we investigated the effect of maternal PCB body load on lactation performances as well.
METHODS: A group of 418 infants were followed from birth up to 6 years of age. Half of them were fully breast fed (BF) for at least 6 weeks. Prenatal PCB exposure was measured from cord and maternal blood.

Postnatal exposure was reflected by PCB and dioxin levels in breast and formula milk and plasma PCB levels at 42 months of age. Both neurological and cognitive development were taken as outcome variable at 18, 42 months and at 6 years of age. At 18 and 42 months of age neurological condition was evaluated according to Hempel and at 6 years of age according to Touwen. Condition was evaluated in terms of optimality. Separately, the fluency of movements was scored. Cognitive abilities were measured at 18 months by the Bayley Scales of Infant Development, at 42 months of age by the Kaufman Assessment Battery for Children (K-ABC) and at 6 years of age by the McCarthy Scales. Daily breast milk volume and milk fat content in relation to PCB body load was evaluated in 102 mothers. Multivariate regression models were applied to analyse associations of measured exposure variables with independent variables adjusted for confounders.

RESULTS: At 18 months of age cognitive development was not affected by either pre- or postnatal exposure to the measured PCBs and dioxins. However, neurological examination showed an adverse effect of prenatal exposure to the measured pollutants on neurological optimality score. At 42 months of age we found negative associations between prenatal PCB exposure on cognitive development. However no effect was demonstrated on postnatal exposure to the measured pollutants. Neurological development was not affected by either pre- or postnatal exposure to PCBs and dioxins. At 6 years of age the preliminary results revealed evidence that cognitive development is affected by prenatal exposure to these pollutants in children from young mothers. An adverse effect of prenatal exposure on neurological outcome was also demonstrated in the formula fed group but not in the breast fed group. Despite a higher PCB exposures from breast milk we found at 18 months, 42 months of age, and at 6 years of age a beneficial effect of breast feeding on the quality of movements, in terms of fluency, and on the cognitive development tests. Maternal PCB body load was inversely related to 24-h breast milk volume and milk fat content.

CONCLUSION: These data give evidence that prenatal exposure to PCBs do have subtle negative effects on neurological and cognitive development of the child up to school-age. Human breast milk volume and fat content is adversely affected by the presently encountered PCB levels in W. Europe. Our studies showed evidence that breast feeding counteracts the adverse developmental effects of PCBs and dioxins.