Breast-feeding in a polluted world: uncertain risks, clear benefits

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Résumé : Les effets des biphényles polychlorés ingérés chez les femmes et présents dans leur lait sur la croissance et le développement de l'enfant suscitent une inquiétude particulière dans notre environnement. Les auteurs examinent les résultats d'études de ces effets dans deux populations nord-américaines. Ils débattent aussi les avantages compensatoires de l'allaitement et l'opportunité du dépistage des contaminants environnementaux chez les femmes et le lait maternel.

In 1991 considerable controversy surrounded a report that summarized the rapidly accumulating scientific literature on the effects of chemical contaminants in women and their breast milk on child growth and development.[1] This report, to the International Joint Commission (a Canada–United States body whose responsibilities include the evaluation of progress under the Great Lakes Water Quality Agreement), was interpreted by some as fearmongering for the sake of environmental activism (*Globe and Mail*, Toronto, Sept. 4, 1991: A4; Sept. 21, 1991: D10; Oct. 5, 1991: D10). The final report,[2] revised in response to these criticisms, clearly and correctly explains that despite environmental contamination of breast milk "breast is still best." However, some confusion may remain about the practical consequences of environmental contaminants, especially polychlorinated biphenyls (PCBs), in human milk. In this article we clarify the health risks to children of prenatal and lactational exposure to PCBs and the countervailing benefits of breast-feeding. In addition, we make recommendations for clinical practice and offer general advice for patients considering childbearing and breast-feeding.

Transmission of PCBs from mother to infant

A full review of the global evidence on this subject is beyond the scope of this article and available elsewhere.[3–5] Extensive evidence from Asia of acute exposure to PCBs will not be included, because children had been exposed in utero or through breast-feeding, or both, to very large doses of particular mixtures of organochlorines that suddenly entered the food supply of communities in Taiwan and Japan.[6–8] Virtually all of the women had blood PCB levels that were much higher than those in the general North American population. Furthermore, the exact nature of the contaminants was unknown, and there was a strong indication that many of the toxic effects were attributable to simultaneous high doses of PCBs and PCB "contaminants," particularly dioxin and dibenzofuran. Instead, we focus on recent North American studies of the effects of moderate maternal PCB levels on child health and development.[9–17] These levels are thought to result from ubiquitous environmental contamination and diet, specifically the consumption of fatty freshwater fish.

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Prenatal exposure

Subtle neurodevelopmental and growth deficits, such as decreased birth weight and head circumference,[9] have been attributed to PCBs transferred to infants in utero at ambient North American body-burden levels.[9–17] In one study persistent but minor effects on weight for age were found only in girls until the age of 4 years.[10] Other reported effects were below-average muscle tone and reflexes at birth,[10,14] abnormal visual-recognition memory at 7 months,[14] low overall developmental score at 6 and 12 months,[11] altered psychomotor development at 2 years[17] and short-term memory deficits at 4 years.[17] However, all of these effects were statistically significant only at the group level; none were clinically apparent in an individual child. Since these reports came from two study populations it is worth describing how these populations were selected and their respective characteristics.

The North Carolina study population[9–13] was a representative subset of children of women who had received prenatal care in the southeastern United States; the selection was unrelated to any known PCB exposure such as freshwater-fish consumption. The maternal PCB levels in tissue were therefore thought to represent those in the general childbearing population in that region at that time (the late 1970s and early 1980s). The levels appeared not to correlate with any dietary pattern, including fish consumption. The natural distribution of maternal PCB levels allowed for the comparison of children's outcomes in mother–infant pairs that were "more exposed" and "less exposed." Persistent abnormal development was confined largely to the offspring of women whose PCB levels were above the 95th percentile. PCB levels in North Americans are thought to have been relatively unchanged since the study period and seemed similar across North America, except in native populations and other groups that consumed large amounts of fish and certain contaminated wild game such as marine mammals.[18] Rogan and Gladen[12] therefore reported that the abnormal developmental effects, if truly caused by maternal PCB exposure and transplacental transfer to the fetus, would be expected to occur in 5% of newborns annually in North America.

However, the follow-up of children aged 3 to 5 years in the North Carolina cohort demonstrated no developmental deficits or less-than-average school grades in the children with high prenatal or postnatal PCB exposure.[13] These findings raise questions about the positive correlations in the previous studies and in the Michigan cohort.[14–17] As the investigators in the North Carolina study suggest,[13] the discrepant results over time may merely reflect the relatively large measurement errors inherent in the assessment of neurodevelopment in children. Alternatively, perhaps the many factors that buffet and buffer children in their complex course of development provide opportunities for recovery from "early insults," including PCB exposure — a reassuring interpretation to be sure. Only further follow-up can clarify whether the children with deficits at earlier ages will subsequently have adverse health or developmental outcomes that were not detectable between the ages of 3 and 5 years.

The Michigan study population[14–17] included a group of women chosen specifically as a subpopulation exposed to PCBs through their consumption of fish from the Great Lakes, despite public advisories at the time against such consumption by women of childbearing age. The studies of this cohort have demonstrated substantial differences between such "risk-taking" women and those who did not eat fish: the former had a higher rate of prenatal alcohol consumption, smoking and consumption of over-the-counter drugs, lower prenatal weight and gestational weight gain and a much lower rate of spontaneous delivery. Furthermore, there were only minimal correlations between the consumption of fish and the level of PCBs in the body fluids of the women and their children. Perhaps the study was more an analysis of growth and developmental outcomes among offspring of women who are risk-takers than of women exposed to PCBs or other fish-related contaminants. The multivariate analyses in the Michigan studies may have not adequately controlled for confounding effects such as lifestyle exposures that are known to adversely affect fetal and child development. Some of those exposures (e.g., illicit drug use) were not measured; others may have been underreported by the women.

The PCB levels in both the North Carolina and the Michigan study populations may represent only a proxy measure of the "environmental soup" of persistent lipophilic compounds likely to be toxicologic "fellow travellers" of PCBs. The compounds that accumulate in the general environment and contaminate our food sources may differ from those that accumulate in fatty freshwater fish. This might explain why only some of the findings in the two cohorts were consistent.

Postnatal exposure

In both the North Carolina and the Michigan studies data were analysed for evidence of additional toxic effects correlated with the extra load of PCBs transmitted to the infant through breast milk. In the North Carolina study no effects were attributed to exposure through breast milk.[9,11,13] Thus, the investigators of both study populations suggested that the fetus is much more susceptible to the transfer of PCBs than the infant, even though greater total PCB levels

are transmitted through breast-feeding than through transplacental transfer.[9,11,13,17] However, the media have focused on one report from the Michigan study that demonstrated a marginally significant reduction in "spontaneous activity levels" in a subgroup of five children who were breast-fed beyond the age of 1 year *and* whose mothers had the highest levels of PCBs in their breast milk.[16] In response, Jacobson[19] stated that this finding is almost certainly the result of the confounding effects of high levels of PCBs transferred transplacentally, since the women with the highest PCB levels in their breast milk also had the highest blood levels and body-burden levels. The duration of breast-feeding was not associated with any negative effects. On the contrary, the overall developmental scores rose with increased duration of breast-feeding, an association that was felt to be due largely to the greater stimulation that women who breast-feed appear to give their infants.[19]

Benefits of breast-feeding

Physicians who have not had an opportunity to review the burgeoning literature on the health benefits of breast-feeding should take another look.[20–29] The more that modern biochemical, immunologic, nutritional and epidemiologic methods are applied to the study of the benefits of human breast milk, the more impressive these benefits become. Breast-feeding seems to be particularly protective against some of the common childhood conditions such as eczema, otitis media and iron-deficiency anemia as well as benefitting neurodevelopment in premature infants.[20–23] In addition, recent reviews of the overall reduction in risk of death with breast-feeding suggest that one-third to one-half of current infant deaths in North America are because of a failure to breast-feed *fully* (i.e., to give breast milk exclusively for the first 4 to 6 months of age, then breast milk plus solid food until 12 months).[24,25]

As well, there are substantial health benefits of breast-feeding to the mother and potentially to the whole family.[26–30] Contrary to expert opinions of only a few years ago epidemiologists currently suggest that breast-feeding appears to provide substantial protection against breast cancer[27,28] and osteoporosis.[29] Finally, the average annual cost of formula feeding in the first year of life is between $1275 and $3055 (as of February 1991 [for Saskatchewan]), depending on whether powder, concentrate or ready-to-use formula is used.[30]

Clinical implications

Questions that the public, nursing women and some physicians continue to ask are: Is the contamination of breast milk by PCBs in North America sufficiently hazardous to children that women should refrain from breast-feeding? and Should women have their blood or breast milk routinely screened for PCBs?

The answer to the first question is clear: there is no evidence that PCBs transferred through breast-feeding have adverse health or developmental effects if the level of PCBs in the mother is within the range of levels common in the general North American population.[31] As to the second question a program to screen breast milk for PCBs would not meet well-established epidemiologic criteria for the detection of asymptomatic conditions[32,33] for the following reasons.

- There is no evidence that children will be harmed if they are breast-fed by women who have elevated PCB levels, even if such levels are in the 95th percentile for the general population. Any adverse health effects consistently associated with high maternal PCB levels have been attributed entirely to transplacental transfer, as discussed previously.
- We do not know exactly what medical management befits women with "high" PCB levels in their blood or breast milk: there is little consensus about the level of PCBs (or any other contaminants) that contraindicates breast-feeding, let alone childbearing, and no specific treatment or advice is available to reduce high PCB levels.
- There is no evidence that the benefits of deferring or discontinuing breast-feeding exceed the risks, including those of increased frequency of infection and of nutritional and allergic effects. In addition, one must consider adverse effects in the mother, including "labelling,"[34,35] which can result from being told that one is poisoned and unfit to breast-feed and perhaps even to bear children. (Ironically, one of the few ways a woman can rapidly reduce her body burden of lipophilic toxins is to lactate. Thus arises a bizarre therapeutic option. Should such women pump their breasts and throw away their milk to detoxify themselves? What would be the psychologic and social ramifications?)
- Currently only a handful of laboratories in North America can reliably determine PCB levels. The interpretation of such levels must also consider the substantial biologic variability in breast-milk samples, particularly because a woman's body burden of PCBs is reduced quickly over the first several weeks of lactation. Cholesterol studies have shown that test–retest error, from both laboratory and biologic variability over time, contributes to misclassification in screening programs[33,36,37] and results in more harm to those with false-negative or false-positive results.[32] In addition, most clinicians, even in tertiary care centres, could not be expected to interpret laboratory reports of PCB levels knowledgeably, given the uncertainty of the effects reported in the literature

and the plethora of methods of measurement, particularly for PCB levels in breast milk, which have not been measured in comparable units among studies.

In short, there is no sensible rationale for the mass screening of women for PCB contamination. There may be high-risk circumstances[38] in which testing would be defensible. An example would be case-finding[39] by a knowledgeable clinician using high-quality laboratory equipment in the preconception or prenatal care of a native woman who eats fatty fish, marine mammals or related game (e.g., polar bear).[40–42] However, given the dubious risk–benefit balance for women and infants at any level of risk it would be advisable to obtain fully informed consent from the woman before testing, similar to that obtained before testing for human immunodeficiency virus infection. Consent is especially important if PCB levels are measured before conception, when the results of testing may persuade some women not to bear children. If testing during pregnancy or lactation reveals PCB levels above the 95th percentile for North American women of comparable age, a full discussion should be held with the patient, especially if she is from a native group with high infant morbidity and mortality rates, about the uncertain risks of such levels compared with the clear benefits of breast-feeding. Finally, since a disproportionate fraction of PCB transfer occurs early during breast-feeding, the decision to stop breast-feeding after some months should be guided by personal values.

Conclusions

There is good evidence that subtle fetal and infant health effects result from *prenatal* exposure to PCBs and other lipophilic toxins that may be in the same environmental exposure, metabolic and tissue-storage pathways. However, the neurodevelopmental effects that have been demonstrated in children of women whose PCB levels were in the 95th percentile of the usual range for North Americans are generally insignificant. Furthermore, in children followed up until the age of 5 years, developmental effects seen when they are younger appear to have been temporary.

With respect to *postnatal* exposure to PCBs through breast-feeding, there is no evidence of any adverse effects on child development at ambient North American body-burden levels. However, there is a need for further study of both the North Carolina and the Michigan cohorts and of other heavily exposed populations such as native groups. Several studies are in progress.[43]

Until further evidence is available, physicians should continue to encourage breast-feeding because of its considerable benefits. Since there is currently no medical treatment to lower body levels of chemicals such as PCBs, prevention is better than cure. Consequently women and young children in particular should obey advisories that identify species of fish and game, and their catch locations, season and size, that present a potential toxic hazard if eaten.[44] Finally, physicians can rightly point to PCB body-burden levels as grounds for urgent policies to achieve "zero discharge" of biologically persistent toxins into the environment.

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References

1. Science Advisory Board: *Report to the International Joint Commission of the Great Lakes Science Advisory Board*, International Joint Commission, Windsor, Ont, Sept 1991
2. Science Advisory Board: *Report to the International Joint Commission of the Great Lakes Science Advisory Board*, rev, International Joint Commission, Windsor, Ont, Dec 1991
3. Tilson HA, Jacobson JL, Rogan WJ: Polychlorinated biphenyls and the developing nervous system: cross-species comparisons. *Neurotoxicol Teratol* 1990; 12: 239–248
4. Rogan WJ, Gladen BC, Wilcox AJ: Potential reproductive and postnatal morbidity from exposure to polychlorinated biphenyls: epidemiologic considerations. *Environ Health Perspect* 1985; 60: 233–239
5. Kuratsune M: Yusho, with references to Yu-cheng. In Kimbrough RD, Jensen AA (eds): *Halogenated Biphenyls, Terphenyls, Naphthalenes, Dibenzodioxins and Related Products*, Elsevier, Amsterdam, 1989: 170–192
6. Swain WR: An overview of the scientific basis for concern with polychlorinated biphenyls in the Great Lakes. In D'Itri FM, Kamrin MA (eds): *PCBs: Human and Environmental Hazards*, Ann Arbor Sci Pub, Ann Arbor, Mich, 1983: 11–48
7. Yakushiji T, Watanabe I, Kuwabara K et al: Postnatal transfer of PCBs from exposed mothers to their babies: influence of breastfeeding. *Arch Environ Health* 1984; 39: 368–375
8. Rogan WJ, Gladen BC, Hung K et al: Congenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan. *Science* 1988; 241: 334–336
9. Rogan WJ, Gladen BC, McKinney JD et al: PCBs and DDE in human milk: effects on growth, morbidity and duration of lactation. *Am J Public Health* 1987; 77: 1294–1297
10. Rogan WJ, Gladen BC, McKinney JD et al: Neonatal effects of transplacental exposure to PCBs and DDE. *J Pediatr* 1986; 109: 335–341
11. Gladen BC, Rogan WJ, Hardy P et al: Development after exposure to polychlorinated biphenyls and dichlorodiphenyl dichloroethene transplacentally and through breast milk. *J Pediatr* 1988; 113: 991–995
12. Rogan WJ, Gladen BC: PCBs, DDE and child development at 18 to 24 months. *Ann Epidemiol* 1991; 1: 407–415

13. Gladen BC, Rogan WJ: Effects of perinatal polychlorinated biphenyls and dichlorodiphenyl dichloroethene on later development. *J Pediatr* 1991; 119: 58–63

14. Jacobson SW, Fein GG, Jacobson JL et al: The effect of intrauterine PCB exposure on visual recognition memory. *Child Dev* 1985; 56: 853–860

15. Jacobson JL, Jacobson SW, Fein GG et al: Prenatal exposure to an environmental toxin: a test of the multiple effects model. *Dev Psychol* 1984; 20: 523–532

16. Jacobson JL, Jacobson SW, Humphrey HEB: Effects of exposure to PCBs and related compounds on growth and activity in children. *Neurotoxicol Teratol* 1990; 12: 319–326

17. Jacobson SW, Humphrey HEB: Effects of in utero exposure to polychlorinated biphenyls and related contaminants on cognitive functioning in young children. *J Pediatr* 1990; 116: 32–45

18. Schwartz H: *Trends in Canadian Populations Exposed to Polychlorinated Biphenyls.* Presented at the annual meeting of the Canadian Public Health Association, Yellowknife, NWT, July 8, 1992

19. Jacobson JL: Cited in Daly H, Humphrey HEB: *Final Report of the Workshop on Protocol Development for Determining Human Exposures to Toxic Chemicals, Body Burdens and Neurobehavioral and Developmental Assessment*, Health Committee, Science Advisory Board, International Joint Commission, Windsor, Ont, 1992: 18

20. Cunningham AS, Jelliffe DB, Jelliffe EFP: Breastfeeding and health in the 1980s: a global epidemiologic review. *J Pediatr* 1991; 118: 659–665

21. Lucas A, Brooke OG, Morley R et al: Early diet of preterm infants and development of allergic or atopic disease: randomized prospective study. *BMJ* 1990; 300: 837–840

22. Casey CE, Hambidge KM: Nutritional aspects of human lactation. In Neville MC, Neifert MR (eds): *Lactation: Physiology, Nutrition and Breastfeeding*, Plenum Pr, New York, 1983: 199–248

23. Lucas A, Morley R, Cole TJ et al: Breast milk and subsequent intelligence quotient in children born pre-term. *Lancet* 1992; 339: 261–264

24. Damus K, Pakter J, Krongard E et al: Postnatal medical and epidemiological risk factors for the sudden infant death syndrome. In Harper RM, Hoffman HJ (eds): *Sudden Infant Death Syndrome: Risk Factors and Basic Mechanisms*, PMA Pub, New York, 1988: 187–201

25. Madeley RJ, Hull D, Holland T: Prevention of postneonatal mortality. *Arch Dis Child* 1986; 61: 459–463

26. *The Womanly Art of Breastfeeding*, La Leche League International, Franklin Park, Ill, 1987: 13

27. Layde PM, Webster LA, Boughman AL et al: The independent associations of parity, age at first full term pregnancy, and duration of breastfeeding with the risk of breast cancer. *J Clin Epidemiol* 1989; 42: 963–973

28. Byers T, Graham S, Rzepka T et al: Lactation and breast cancer. Evidence for a negative association in premenopausal women. *Am J Epidemiol* 1985; 121: 661–674

29. Aloia JF, Cohn SH, Vaswani A et al: Risk factors for postmenopausal osteoporosis. *Am J Med* 1985; 78: 95–100

30. Bergerman J, Oleksyn TA: Cited in *INFACT Canada* 1991; summer: 2

31. World Health Organization (Europe): *Assessment of Health Risks in Infants Associated with Exposure to PCBs, PCDDs and PCDFs in Breast Milk. Report of a WHO Working Group* (Environmental Health rep 29), WHO, Copenhagen, 1988

32. Sackett DL: The evaluation of health services. In Last J (ed): *Maxcy-Rosenau Public Health and Preventive Medicine*, 11th ed, ACC, New York, 1980: 1800–1823

33. Toronto Working Group on Cholesterol Policy. Asymptomatic hypercholesterolemia: a clinical policy review. *J Clin Epidemiol* 1990; 43: 1021–1122

34. Haynes RB, Sackett DL, Taylor DW et al: Increased absenteeism from work after detection and labelling of hypertensive patients. *N Engl J Med* 1978; 299: 741–744

35. Lefebvre RC, Hursey KG, Carleton RA: Labelling of participants in high blood pressure screening programs. Implications for blood cholesterol screenings. *Arch Intern Med* 1988; 148: 1993–1997

36. Jonsson GG, Oreberg M, West K: The cholesterol trap: how doctors and patients could avoid overevaluation of a single cholesterol value [C]. *J Clin Epidemiol* 1991; 44: 817–820

37. Natelson BH, Trapp WN, Munsif A et al: Fluctuating serum cholesterol: implications for coronary prevention. *Lancet* 1988; 2: 404–405

38. Sackett D, Haynes RB, Guyatt GH et al: *Clinical Epidemiology — a Basic Science for Clinical Medicine*, 2nd ed, Little, Boston, 1991: 153–170

39. Canadian Task Force on the Periodic Health Examination: The periodic health examination. *Can Med Assoc J* 1979; 121: 1193–1254

40. Dewailly E, Nantel A, Weber JP et al: High levels of PCBs in breast milk of Inuit women from arctic Quebec. *Bull Environ Contam Toxicol* 1989; 43: 641–646

41. Dewailly E, Weber JP, Gingras S et al: Coplanar PCBs in human milk in the Province of Quebec, Canada: Are they more toxic than dioxin for breast fed infants? *Bull Environ Contam Toxicol* 1991; 47: 491–498

42. Dewailly E, Nantel A, Bruneau S et al: Breast milk contamination by PCDDs, PCDFs and PCBs in arctic Quebec: a preliminary assessment. *Chemosphere* 1992; 25: 1245–1249

43. *Report of the Great Lakes Research Consortium*, vol 4 (B), College of Environmental Science and Forestry, State U NY Pr, Syracuse, NY, 1991: 3–4

44. Departments of the Environment, Fisheries and Oceans, and National Health and Welfare: *Toxic Chemicals in the Great Lakes and Associated Effects* (cat EN37-94/1990E), Dept of Supply and Services, Hull, Que, 1991