Review

The Heart of the Matter on Breastmilk and Environmental Chemicals: Essential Points for Healthcare Providers and New Parents

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Abstract

The increasing number of environmental chemicals measured in breastmilk is a consequence of improved analytical capabilities and the increased interest in biomonitoring. It has been generally concluded that the benefits to the infant from breastfeeding outweigh potential risks associated with environmental chemical exposures associated with breastfeeding. However, there have been reports of subtle effects on infants associated with chemicals in breastmilk. Associations between concentrations of chemicals in breastmilk and a biochemical or other change in infants may signal the need for further study or regulatory action, whereas on an individual level, these changes may not be considered adverse. For healthcare providers, this distinction is critical, as many in the field are being asked for nuanced information on risks and benefits associated with breastfeeding, and this information is not readily available. Recognizing the challenge faced by healthcare providers, we have explored and developed a case study on dioxins in breastmilk. The essential conclusion for healthcare providers and new parents is that in studies of breastfed versus formula-fed infants across time, including times when levels of environmental chemicals such as dioxins were higher, beneficial effects associated with breastfeeding have been found. The current evidence does not support altering the World Health Organization recommendations promoting and supporting breastfeeding.

Introduction

The number of investigations into levels of environmental chemicals in breastmilk has grown considerably since the first reports of dichlorodiphenyltrichloroethane (DDT) compounds in milk.1 The increasing number of environmental chemicals measured in breastmilk is a consequence of our improved analytical capabilities and increased interest in biomonitoring. It is axiomatic that exposure to an environmental chemical will lead to some level of that chemical in breastmilk. Of the hundreds of environmental chemicals that may be present in breastmilk, a few have been studied extensively, both for geographic and temporal trends in their concentrations and for their potential effects on the breastfed infant. There are important lessons to be extracted from the history now available to us on studies of those chemicals in breastmilk, lessons that can be used by the healthcare practitioner to counsel patients.

The overarching lesson relates to the critical importance in distinguishing between population effects, which can most effectively be addressed by governmental approaches, and individual effects, which are best addressed by healthcare providers and new parents. Associations between concentrations of a chemical in breastmilk and a biochemical change may signal the need for further study or for regulatory action, whereas on an individual level, these changes may not be considered sufficient to advise against breastfeeding. For healthcare providers, this distinction is critical,
as many are being asked for nuanced information on risks and benefits associated with breastfeeding, and this information is not readily available. Specifically, families, public health officials, and others often seek information from healthcare providers on whether the concentrations of environmental chemicals in human milk make breastfeeding an unsafe activity as compared to formula feeding, or whether the benefits outweigh the potential risks. It is clear that complete knowledge will never be attainable; an examination of overall available evidence on benefits and risks is required in order to make recommendations regarding breastfeeding. Recognizing the challenge faced by healthcare providers, we explore and develop a case study by examining the research on dioxins in breastmilk.

The two principal methods used to obtain information on risk and safety are to compare the dose of a chemical received by the breastfeeding infant to guidance values set by agencies such as the World Health Organization (WHO) or to conduct epidemiological studies following mother–infant pairs over time. Issues of interpretation arise for both approaches used to evaluate benefit and risk. For example, when comparing doses of a chemical to a guidance value, it is legitimate to question whether the guidance value is appropriate for breastfeeding scenarios (relatively short-term exposures at early life stages). On the other hand, epidemiological studies often report associations between perinatal exposures to chemicals and effects in the infants, and these studies may not fully distinguish between transplacental and breastfeeding exposures. While disentangling exposure/effect relationships for pre- and postnatal exposures is difficult, this distinction is essential when considering possible actions that could be taken on a personal level (i.e., whether or not to breastfeed) versus actions required on a governmental level (e.g., crafting regulations for reducing environmental chemical levels). From a societal perspective information related to perinatal exposures is important, but for healthcare providers counseling parents on whether to breastfeed, information related to postnatal exposures is pertinent.

In this paper, we examine the research on dioxins in breastmilk as a case study to highlight the important lessons learned and to discuss the two main approaches to evaluating potential risks associated with environmental chemicals in breastmilk. The approaches include: (1) comparison of infant intake of dioxins via breastfeeding to the tolerable daily intake (TDI) as defined by the WHO and (2) evaluation of epidemiological studies on dioxins in human milk and infant health outcomes. Dioxins were selected for the case study as this group of chemicals has been one of the most extensively studied in terms of exposures and effects in humans, including measurements in breastmilk and effects on infants.

What Are Dioxins?

Dioxins and a related group of compounds called furans are the unintentional by-products of several industrial processes and incineration of municipal and hospital waste. The predominant route of exposure for humans is via diet, especially from animal fats; for infants, the major route of exposure is from breastmilk. There are a possible 75 dioxins and 135 furans, which differ according to the number and location of chlorine atoms. Regulators and scientists have focused on 17 of these compounds—those thought to have the greatest toxicity (congeners with chlorines in the 2-, 3-, 7-, and 8-positions). Rather than estimating exposures to individual dioxins or furans, the following method has been established: the concentration of the individual 17 compounds is multiplied by its toxic equivalency factor (TEF), and the results are summed to derive a toxic equivalency quotient (TEQ). A compound’s TEF is an estimate of the relative potency of each dioxin and furan relative to the most toxic of the congeners: 2,3,7,8-tetrachlorodibenzo-p-dioxin. The 17 dioxins and furans are referred to here as “dioxins.” For some studies, certain polychlorinated biphenyls (PCBs) for which TEFs have been developed are included in the overall TEQ determination. Simply put, a TEQ value (typically in units of ng/kg or parts per trillion) gives an indication of overall exposure to dioxin-like compounds.

Comparison of Infant Exposures to Dioxins in Breastmilk to WHO TDI Values

TDIs are used as guidance values for comparing exposures to chemicals (i.e., dose) with acceptable or tolerable levels as determined by the WHO or other agencies. A TDI of 1–4 pg of TEQ/kg of body weight for dioxins and dioxin-like compounds has been established and has been widely used to place infant exposures to dioxins from breastfeeding into perspective (recent examples include publications by Mato et al. and Polder et al.). The TDI is based on lowest observed adverse effect levels from several laboratory studies with the following responses: in rats, decreased sperm count in offspring, immune suppression in offspring, and increased gestational malformations in offspring; in monkeys, neurobehavioral effects in offspring and endometriosis.

Dioxin concentrations in the lipids of breastfed infants are higher than in formula-fed infants, but these differences abate with time. According to Renwick and Walker, because of the brief time period of breastfeeding and the convergence of dioxin lipid levels in breastfed and formula-fed individuals over time, “intake during lactation would represent an insignificant duration of excess exposure [in excess of the TDI] and the benefits of breastfeeding would not be associated with an increased risk, despite the increased intake.” Thus, while the dioxin TDI provides an approximation of levels of acceptable dioxin intakes, TDIs have shortcomings for evaluating the potential for health risks associated with breastfeeding. Specifically, acceptable daily intakes, reference doses, and cancer slope factors do not necessarily provide useful tools for making public health or individual decisions because these are usually based on chronic toxicity hazards, which are not necessarily germane to infant exposure patterns from human milk. In our view, the available epidemiological studies provide a clearer picture of the potential for adverse health effects associated with dioxins in breastmilk. These studies are reviewed in the following section.

Epidemiological Studies of Dioxins in Breastmilk and Infant Health Outcomes

We focus on three cohorts of mother–infant pairs that have been followed to assess potential health effects associated with postnatal exposures to dioxins: the Rotterdam/Gronin-
gen and Duisburg birth cohorts and a smaller cohort referred to here as the Amsterdam/Zaandam cohort. (Information from four studies from Japan is included where relevant.) A brief description of the characteristics of the three cohorts is given (with Fig. 1 showing the timeline for studies and outcomes examined at various life stages), followed by a synopsis of the study results, with a focus on whether the data support a relationship between postnatal exposure to dioxins via breastfeeding and clinically relevant organ system effects. A general issue in interpretation of the cohort studies is that they often do not provide laboratory reference ranges; “reliable reference ranges are important in the interpretation of laboratory data, and it is incumbent on each laboratory to verify that the ranges they use are appropriate for the patient population they serve.” In the absence of this information, we rely on the study authors’ assessments, or in cases where the authors did not provide comparisons to reference ranges, we use other published reference ranges.

Rotterdam/Groningen cohort

A cohort of 418 mother–infant pairs was recruited from 1990 to 1992 to participate in a prospective longitudinal study to assess health effects of dioxins and PCBs from both prenatal and postnatal (breastfeeding) exposure. Approximately half of the infants were breastfed, and the other half were formula-fed (the number of participants varied in follow-up studies). Infants were healthy and full-term. Breastmilk samples were collected in the second week postpartum, and 200 samples were analyzed for dioxins and PCBs 10 days postpartum (mean dioxin level, approximately 31 ng of TEQ/kg of lipid) (milk was also collected 6 weeks and, if possible, 3 months postpartum).

Amsterdam/Zaandam cohort

Women (n = 38) were recruited from 1990 to 1991 to participate in a study of pre- and postnatal exposure to dioxins (infants were full-term and healthy). Breastmilk samples were collected 3 weeks postpartum. Breastfed infants were not compared to formula-fed infants; rather, mother–infant pairs with breastmilk dioxin TEQs below the group median (8.7–28.0 ng of TEQ/kg of lipid; mean, 18.6 ng of TEQ/kg of lipid) were placed in the “low exposure group,” and the others (29.2–62.7 ng of TEQ/kg of lipid; mean, 37.5 ng of TEQ/kg of lipid) were placed in the “high exposure group.” Mean breastfeeding duration in the “low” and “high” groups was 17.3 (SEM 2.2) and 18.1 (SEM 1.9) weeks, respectively.

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**FIG. 1.** Timeline and end points assessed for the three cohort studies on perinatal exposure to dioxins and infant health: Rotterdam/Groningen cohort (open boxes above timeline), Amsterdam/Zaandam cohort (open boxes below timeline), and Duisburg cohort (shaded boxes below timeline). BMI, body mass index.
**Duisburg cohort**

Mother–infant pairs (infants healthy and full-term, n = 232) were recruited between 2000 and 2002 to assess the health effects of dioxins and PCBs from prenatal exposure (postnatal exposures could be also estimated as dioxins in milk were measured, and duration of lactation was assessed). Breastmilk was collected within 3 weeks postpartum, and dioxin levels were 1.80–34.70 ng of TEQ/kg of lipid (median, 13.10 ng of TEQ/kg of lipid) (with PCBs added to the total, the median value was 26.30 ng of TEQ/kg of lipid).

**Growth**

**Rotterdam/Groningen cohort.** Postnatal exposure to dioxins did not adversely affect growth rate (weight, height, and head circumference) of the breastfed children from birth to 42 months of age.

**Amsterdam/Zaandam cohort.** Up to 6 months of age, no differences in growth rate, liver size, or ratio of liver size to body weight were observed between the high and low exposure infant groups. At 2.5 years of age, no differences were found between exposure groups regarding medical history, physical exam, or growth.

**Neurodevelopment**

**Rotterdam/Groningen cohort.** Infants were assessed for neurological optimality using the Prechtl neurological examination (a neurological examination including assessments of reflexes and muscle tone) between 10 and 21 days postpartum. At the higher concentrations of dioxins in human milk, reflexes and responses were normal, but there was a higher incidence of hypotonia (characterized by the authors as a “minor dysfunction”). Specifically, because of the “very minor character of the deviations,” the authors did not advise against breastfeeding.

Children were assessed at 3, 7, and 18 months of age for mental and psychomotor development with the Bayley Scales of Infant Development and at 31 months with the Hempel neurological exam for toddler age. No significant differences were observed between high and low exposure groups for the mental and psychomotor development scores, and all children had normal Hempel test results. Significantly lower “suboptimality scores” for five test items in the high exposure group were found, and reflexes were significantly higher in the high exposure group. There were no differences in muscle tone between the two groups.

**Duisburg cohort.** Infant exposures to dioxins did not affect neurological, motor, or mental development up to 24 months. The authors concluded that “the current level of exposure has dropped to a degree that further adversity of POP [persistent organic pollutant] exposure on mental and motor development is no longer observable.”

**Thyroid**

**Rotterdam/Groningen cohort.** Maternal and infant thyroid hormone levels were measured. Infants were divided into low and high exposure groups (above and below the median TEQ level in milk samples). Dioxin TEQs in milk were positively significantly correlated with infant plasma thyroid-stimulating hormone (TSH) levels 2 weeks and 3 months postpartum and negatively correlated with plasma free and total levels of thyroxine (T4) 2 weeks postpartum, but it was unclear whether the correlation was due to in utero or lactational exposure; all thyroid levels in all mother–infant pairs were in the normal range.

**Amsterdam/Zaandam cohort.** At 1 and 11 weeks postpartum, mean levels of total T4 (TT4) and T4/T4 binding globulin (TBG) ratios were significantly higher in the high exposure group; at 11 weeks, mean TSH levels were significantly higher in the high exposure group. However, plasma TSH and TT4 levels appeared to be within reported reference ranges. The authors note that the “change in circulating TT4 and TSH concentration does not prove that functional thyroid status has been affected by pre- and postnatal exposure to dioxins.” At 2.5 years of age, there were no significant differences between exposure groups in levels of plasma TT4, total triiodothyronine (T3), free T4, or TBG.

A follow-up when the children reached 7–12 years of age showed no correlation between lactational dioxin exposure and TSH or free T4, and the hormone levels were not considered clinically pathological.

Others have examined the relationship between dioxins in breastmilk and infant thyroid hormone levels. Blood samples from 36 1-year-old infants were analyzed for serum levels of T3, T4, TSH, and TBC. A statistically significant inverse correlation was observed between estimated cumulative levels of dioxin TEQs via breastfeeding (based on analysis of breastmilk at 3 months postpartum [mean TEQ including PCBs of 27.1 ng/kg of lipid], known duration of breastfeeding, and an estimate of daily intake of breastmilk) and levels of serum T3 and T4; no correlation was observed for TSH and TBC. Serum TSH levels generally fell within published reference ranges; serum T4 and T3 levels were within or slightly higher than published reference ranges.
Matsuura et al. collected blood samples from 337 breastfed and 53 bottle-fed infants at 1 year of age for analysis of thyroid hormones. No correlation was found between dioxins in breastmilk (at 30 days postpartum, mean levels of dioxins and PCBs by geographic area were 13.1–29.5 ng of TEQ/kg of lipid) and thyroid hormone levels. There was no difference between breastfed and bottle-fed babies for thyroid function, with levels of thyroid hormones all within normal range, and no evidence of impairment of thyroid function.

Hematology and other laboratory tests

Amsterdam/Zaandam cohort. At 11 weeks of age, alanine aminotransferase (ALT) and aspartate aminotransferase (AST) plasma activities were significantly correlated with estimated dioxin intake but not with measured levels of dioxins in milk lipids. A significant inverse relation was found between cumulative dioxin intake and number of platelets. No relationship was observed between cumulative dioxin intake and plasma γ-glutamyltransferase activity or plasma cholesterol, total and conjugated bilirubin, or white blood cell count. According to Pluim et al., "The clinical significance of our findings is as yet unclear, because AST and ALT plasma activities were in the normal range (normal < 54 U/l) in all but 3 infants, who had slightly elevated AST . . . and ALT . . . plasma activities." Platelet count was significantly inversely associated with cumulative postnatal dioxin intake (but not with absolute milk dioxin TEQs) but was elevated (approximately 80–700 × 109/L) for almost all of the infants (compared to a normal platelet count for adults of 140–450 × 109/L). The authors could not explain this finding. Pluim et al. concluded that background levels of dioxins might have subclinical effects on infants.

At 2.5 years of age, there were no differences in liver-related parameters between high and low exposure groups. Mean AST and ALT were within reference ranges for adults. At 8 years of age, a significant inverse relationship between cumulative postnatal exposure and platelet count was observed, with platelet counts ranging from 248 to 449 × 109/L; while platelet counts for nine of the children were above typical counts for adults, the authors state that this is common and not considered to have clinical relevance.

There was a significant positive association between cumulative postnatal exposure and thrombopoietin concentration, but thrombocyte and thrombopoietin concentrations were “normal.” There were no significant associations between postnatal exposure and red blood cell counts or red blood cell mean volume.

At 7–12 years of age, there was no correlation between lactational dioxin exposure and AST or ALT.

Immune system

Rotterdam/Groningen cohort. Immunologic marker analyses of lymphocytes was performed at 42 months of age. In addition, infectious and allergic disease history was determined by questionnaire, and humoral immunity was determined by antibody levels for mumps, measles, and rubella after the primary vaccination. Dioxin TEQs in milk were significantly correlated with coughing, congestion, and phlegm (odds ratio 1.06, 95% confidence interval 1.00–1.11, p = 0.04). No significant correlations were found between milk dioxin TEQs and antibody levels. White blood cell counts and immunologic marker analyses of the lymphocytes were all within normal ranges and were not correlated with dioxin TEQs.

Amsterdam/Zaandam cohort. At 8 years of age, a significant inverse association was found between allergies (e.g., food allergies, hay fever, dust mite allergy) and postnatal exposure. No association was found between perinatal exposure to dioxins and otitis, pneumonia, or chicken pox. The only immunological parameters significantly associated (positive association) with postnatal exposure were CD4+ (T-helper) cells and CD45RA+ cell count. Because information on background levels of CD4+ in healthy children is sparse and since CD4+ levels are affected by the time of day of blood draw, fatigue and stress levels, infections, and time since recent vaccinations, the significance of the CD4+ results for the clinician is unclear. However, assuming that the CD4+ values are percentages (units not given in the original study), the values are close to reported reference range for healthy urban-dwelling 1–2 year olds. No information on reference ranges for CD45RA+ in children was identified.

Two other studies explored associations between dioxins in breastmilk and infant immunity. Blood samples from 36 1-year-old infants were analyzed for serum levels of lymphocyte subsets. Dioxin levels in breastmilk (mean TEQ in including PCBs was 27.1 ng/kg of lipid) were nonsignificantly positively correlated with infant levels of CD4+ lymphocytes and negatively correlated with CD8+ lymphocytes. The ratio of CD4+/CD8+ T cells was significantly positively associated with total dioxin intake; the ratios fell within a published reference range for healthy adults (no similar ranges were identified for children). Nagayama et al. reported a nonsignificant association between dioxins in breastmilk (median level = 23 ng of TEQ/kg of lipid) and CD4+/CD8+ for 101 infants 7–13 months old. The reported values approximate available reported reference range for healthy children, and the authors note that the “clinical significance . . . is uncertain.”

Respiratory system

Amsterdam/Zaandam cohort. Children 7–12 years old were examined for respiratory effects (forced expiratory volume in 1 second [FEV1]/forced vital capacity [FVC]). The FEV1/FVC ratio ranged from 0.71 to 0.99, with a mean ± SD of 0.90 ± 0.06. A significant inverse relationship between cumulative postnatal exposure and FEV1/FVC was found. No association was observed between postnatal exposure to dioxins and otitis or airway diseases, including bronchitis. The authors reported that the FEV1/FVC results represent mild to moderate “suboptimality” in this group of children. For the clinician, there are two important issues related to these findings that require consideration. First, all of the children had a FEV1/FVC ratio above 0.8 (FEV1/FVC ratios greater than 0.7 are considered normal); thus, the relevance of these findings in a clinical setting is unclear. Second, paternal smoking was not considered as a confounder because of the lack of a “documented link between the lung function of children and smoking fathers”; however, recent studies (e.g., Nuhoglu et al. and Alipour et al.) have demon-
strated significant negative effects from passive smoking on lung function, including FVC, FEV₁, and forced mid-expiratory flow rate.

**Dioxins and Breastmilk Epidemiology Studies in Light of Current Levels of Dioxins in Breastmilk**

The Rotterdam/Groningen and Amsterdam/Zaandam cohorts were recruited from 1990 to 1992, a time when global levels of dioxins were substantially higher. Figure 2 shows the median or mean and maximum levels for these cohorts compared to international breastmilk dioxins data from the time period 1998–2005. It is unlikely that the population background levels observed in the Dutch studies will be found again. Thus, it is possible that observed subclinical effects or associations between postnatal dioxin exposures and subclinical effects in those cohorts would not be found in today’s children. For example, ten Tusscher et al. reported an inverse association between cumulative postnatal dioxin exposures and FEV₁/FVC. Assuming a current representative breastmilk concentration of dioxins of approximately 11 ng of TEQ/L of milk fat, a 1-year breastfeeding duration, and a daily intake of 800 mL milk/day, postnatal cumulative dioxin exposure via breastfeeding would be 82 ng. Seventeen children in the cohort had exposures at or below this value; for these 17 children, whose exposures can be used to represent typical current breastmilk dioxin levels, the reported inverse association between cumulative postnatal dioxin exposure and FEV₁/FVC is no longer observed.

The breastmilk dioxin levels observed in the Duisburg cohort (1.80–34.70 ng of TEQ/kg of lipid; median 13.10 ng of TEQ/kg) more closely mirror the recent international data more closely mirror the recent international data compared to international levels of dioxins in breastmilk from 1998 to 2005. From LaKind.

**Discussion and Conclusions**

In examining the epidemiological data in conjunction with comparisons of infant doses of dioxins to the WHO TDI, demonstrating adverse health effects in breastfed infants exposed to dioxins from breastmilk has been problematic. Reported effects have either been minor and/or transient in nature and were associated with exposure levels unlikely to

![FIG. 2. Comparison of breastmilk dioxin levels for the Rotterdam/Groningen (R/G) (mean and maximum) and Amsterdam/Zaandam (A/Z) (median and maximum) cohorts (collected from 1990 to 1992) and the Duisburg cohort (median and maximum, collected from 2000 to 2002) compared to international levels of dioxins in breastmilk from 1998 to 2005. From LaKind.]

![FIG. 3. Dichotomy in the interpretation of the essential information from available studies for healthcare providers versus regulators and the scientific community. Adapted from LaKind.]

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**Healthcare providers**

- To breastfeed or not to breastfeed?
  - Patient environmental history shows above-background exposures (unlikely as most people won’t know)
    - Yes
    - No
    - Additional history needed
  - In studies of breastfed vs formula fed infants across time, including times when levels of environmental chemicals were higher, beneficial effects associated with breastfeeding have been found.

**Regulators/scientific community**

- Associations found in infants within normal range but statistically significant at population level
  - and/or
  - Effects reported in infants outside normal range
    - Additional study and/or regulation needed

Breastfeed
occur today. Of course, if a large enough number of end points are included in a study, it is statistically likely that an association between levels of a chemical and an outcome will be observed. The question that must be then be asked is: “What is the relevance of the finding?” It is the case, for example, that statistically significant inverse associations between levels of dioxins in breastmilk and infant thyroid hormone levels have been reported, but in each instance, infant thyroid hormone levels tended to be within a normal range. Similarly, while results are often couched in language such as “perinatal effects,” subtle neurological effects reported in epidemiological studies have most often been related to perinatal, rather than breastfeeding, exposures, and it is often difficult to disentangle observations with these two periods of exposure. While from a regulatory perspective it may be important to continue to focus on these outcomes, from the perspective of a healthcare provider seeking to answer questions from patients regarding the safety of breastfeeding, the results indicate that reassurances can be given regarding the benefits of breastfeeding despite the presence of environmental chemicals in milk. We do not seek to diminish the benefits of breastfeeding during the 30-year period (Fig. 4). It is unlikely that we will have, any time soon, extensive studies on the hundreds of other chemicals that can be measured in breastmilk.

It has been generally concluded, based on qualitative analysis, that the known benefits to the infant from breastfeeding outweigh any potential risks associated with background levels of environmental chemicals in breastmilk. In reviewing past studies on breastfed and formula-fed infants, the American Academy of Pediatrics noted the benefits of breastfeeding; while not specifically based on studies of environmental chemicals in breastmilk, the studies included women who most certainly had measurable levels of these chemicals in their milk. A more recent assessment of the literature on breastfeeding in developed countries concluded that “. . . a history of breastfeeding was associated with a reduction in the risk of acute otitis media, non-specific gastroenteritis, severe lower respiratory tract infections, atopic dermatitis, asthma (young children), obesity, type 1 and 2 diabetes, childhood leukemia, sudden infant death syndrome (SIDS), and necrotizing enterocolitis. There was no relationship between breastfeeding in term infants and cognitive performance.” In addition, the WHO has concluded the following on infant risks from dioxins in breastmilk:

Breast-fed infants are exposed to higher intakes of these compounds on a body weight basis, although for a small proportion of their lifespan. However, the consultation noted that in studies of infants, breastfeeding was associated with beneficial effects, in spite of the contaminants present. The subtle effects noted in the studies were found to be associated with transplacental, rather than lactational, exposure. The consultation therefore reiterated conclusions of previous WHO meetings on the health significance of contamination of breast milk with dioxin-like compounds; namely that the current evidence does not support an alteration of WHO recommendations which promote and support breastfeeding.

In our view, the essential point for healthcare providers and new parents regarding breastfeeding and environmental chemicals is: In studies of breastfed versus formula-fed infants across time, including times when levels of environmental chemicals such as dioxins were higher, beneficial effects associated with breastfeeding have been found.

![FIG. 4. Concentrations of dioxins (polychlorinated dibenzo-p-dioxins/dibenzo-p-furans [PCDD/F] WHO-TEQs, parts per trillion [ppt], lipid basis) in human milk from 1975 to 2005. Data represented by circles were compiled in LaKind et al. The data represent areas with both background dioxin levels and those with point sources of dioxins. Reprinted from LaKind.](image-url)
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References

5. Polder A, Gabrielsen GW, Odland JØ, et al. Spatial and temporal changes of chlorinated pesticides, PCBs, dioxins (PCDDs/PCDFs) and brominated flame retardants in human breast milk from Northern Russia. Sci Total Environ 2008;391:41–54.
27. ten Tusscher GW, Guchelaar HJ, Koch J, et al. Perinatal
dioxin exposure, cytochrome P-450 activity, liver functions and thyroid hormones at follow-up after 7–12 years. Chemosphere 2008;70:1865–1872.


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