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State of the Art

Human Breast Milk and Xenoestrogen Exposure: A Possible Impact on Human Health

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Human milk is the best natural and optimal food for neonates with several immunologic, developmental and practical advantages throughout childhood. Although the World Health Organization strongly supports breastfeeding, it recognizes the potential health risks posed by the presence of environmental toxicants in breast milk. Contamination of human milk is widespread and due to decades of inadequately controlled pollution by toxicants, persistent pesticides or chemical solvents. These chemicals tend to degrade slowly in the environment, to bioaccumulate in the food chain and to have long half-lives in humans. Many of these environmental pollutants have estrogen-like activities and, thus they are called environmental estrogen disruptors or xenoestrogens. Certain adverse health and reproductive outcomes are attributed to these chemicals in laboratory animals and in wildlife as well as in humans. Here, we review available data from breast milk monitoring studies suggesting the environmental chemicals that may affect child health through breastfeeding.
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INTRODUCTION

Human milk is without question the best source of nutrition for infants with several immunologic, developmental and practical advantages extending throughout childhood into adulthood. Recognition of the manifold benefits of breast milk (BM) has led to the adoption of breastfeeding policies by numerous health and professional organizations.[1–3] In a recent recommendation, the World Health Organization (WHO) urged its member states to strengthen activities "*to protect, promote and support exclusive breastfeeding for 6 months as a global public health recommendation, and to provide safe and appropriate complementary foods, with continued breastfeeding for up to 2 years of age or beyond*".[3] This emphasis on breastfeeding is motivated by the fact that BM provides the most complete form of nutrition for infants, imparts increased protection from chronic diseases such as asthma and diabetes and improves maternal health through the physiologic responses associated with lactation.[1]

Unfortunately, human BM is not pure. Contamination of human milk is widespread and is the consequence of decades of inadequately controlled pollution of the environment by toxic chemicals. Polychlorinated biphenyls (PCBs), dichlorodiphenyltrichloroethane (DDT) and its metabolites, dioxins, polychlorinated dibenzofurans (PCDFs), polybrominated biphenyls (PBBs), polybrominated diphenylethers (PBDEs), and heavy metals are among the toxic chemicals most often found in BM.[4] These compounds are encountered to varying extents among women in industrially established as well as in developing nations. Some of the highest levels of contaminants are seen among women in agricultural areas of the developing world that are extensively treated with pesticides.[4] Interestingly, women in remote areas, such as the Canadian Inuit, who eat a diet rich in seal, whale and other species high on the marine food chain also accumulate heavy burdens of persistent organic pollutants.[5] For example, methlymercury and PCBs are environmental contaminants that are concentrated in fish and potentially in human BM. In populations where fish is a large part of the diet, maternal consumption can determine the infant's exposure level to these toxins. However, BM pollution and its health effects on further generations are a worldwide focus.[6]

Potential risks associated with breastfeeding also need to be factored into the overall public health assessment when women are encouraged to breast-feed their infants. Breastfeeding for nursing infants, can be a potential source of exposure to toxic chemicals to which the mother has previously been exposed. This may be true for two major reasons. First, BM serves as a food source for this segment of the human population: the diets of many newborns are limited to BM or mother's milk is at least an major nutrient source for suckling infants. Second, breast-fed infants are at the top of the food chain. Therefore, certain chemicals accumulated in the mother's tissues may be transferred to an infant during breastfeeding. This is especially true for environmental lipid-soluble pollutants such as polyhalogenated chemicals, because these chemicals tend to degrade slowly in the environment, to bioaccumulate and bioconcentrate in the food chain and to have

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long half-lives in humans. As the milk fat content is relatively high, breastfeeding potentially causes high-dose exposure of lipid-soluble pollutants. Furthermore, chemicals found in the environment have several mechanisms through which they may disrupt the nervous, endocrine and reproductive systems of humans.[7] Owing to the rapid mental and physical changes that are taking place, neonates can be more susceptible to adverse effects resulting from chemical exposures. For example, antianxiety drugs, antidepressants, neuroleptics, nicotine and silicones have been studied recently by the American Academy of Pediatrics.[8] However, a number of drugs, industrial chemicals, and environmental pollutants are known to be present in human BM.

In this paper, we review available data on BM pollution by environmental contaminants. Specifically, we focused on the breast-feeding pharmacokinetic aspects related to infant exposure of chemical pollutants that have estrogen and antiandrogen activities: environmental estrogen disruptors or xenoestrogens. Estrogen disruptor exposure to the next generations is up-to-date health care. Most xenoestrogens have been considered safe because they exhibit very low acute toxicity at environmental concentrations at which they are usually detected. However, the same chemicals can show hormone activity at concentrations many orders of magnitude below the concentration at which toxicity occurs and within the range of current human and wildlife exposure to these chemicals.

Although scientific evidence to date indicates that the advantages of breast-feeding outweigh any risks from contaminants, it is important to identify contaminant trends, locate disproportionately exposed populations, and take public health measures to decrease and eliminate chemical pollutants from mother's milk.

TOXICITY OF XENOESTROGENS

Whether environmental contaminants with such disrupting properties pose a present danger to humans and wildlife has been controversial ever since the endocrine disruptor theory was first proposed.[8] An endocrine disruptor could be defined as ''*an exogenous substance that changes endocrine function and causes adverse effects at the level of the organism, its progeny and of populations or organisms*''. A number of these chemicals in the environment have estrogenic activity and are referred to as ''xenoestrogens'', as they are produced outside the body. Phytoestrogens are naturally occurring xenoestrogens produced by plants, however, most xenoestrogens are synthetic compounds produced by man.

There are more than 75,000 man-made chemicals in the Toxic Substances Control Act Inventory, but only a few have been tested for endocrine disrupting effects.[9] Recently, Hong et al.[10] used a tree-based model and three structural alerts to evaluate 58,000 chemicals and predict those that have potential to bind estrogen receptors. They predicted that 6903 chemicals were estrogenic through estrogen receptor signaling.[10] Although with different mechanisms, various environmental chemicals such as dioxins, DDT, hexachlorocyclohexane (HCH), alkylphenols, nonylphenol, and octylphenol have all been reported to have estrogen-like activity. For example, HCH does not competitively bind estrogen receptors, but it produces a number of estrogen-like responses: it stimulates proliferation and increases synthesis of progesterone receptors in cultures of human breast cancer cells, and it produces moderate uterotrophic effects in the rodent uterus.[11] In contrast, *o,p′*-DDT competitively binds to the estrogen receptor, and it produces a range of estrogenic responses.[11]

Many xenoestrogens have been considered safe because they exhibit very low acute toxicity. Concentrations at which these compounds are detected usually lie below previously reported no-observed-adverse-effect level (NOAEL), and according to the traditional paradigm of toxicology, exposure should therefore not result in any significant malignant response. However, the same chemicals can show estrogenic activity at concentrations many orders of magnitude below the concentration at which toxicity occurs and within the range of current human and wildlife exposure to these chemicals. In addition, recent reports have suggested nonmonotonic dose–response relationships for natural and synthetic estrogens, where in utero exposure to relatively low concentrations produced measurable effects in adult mice. vom Saal et al.[12] reported that feeding pregnant mice with sub-NOAEL concentrations of estradiol or diethylstilboestrol (DES) resulted in permanently increased prostate sizes in their male offspring, whereas high doses caused significant size reductions. The predicted NOEL for bisphenol A has been estimated at 50 mg/kg/day.[9] However, bisphenol A is active in rodents at 2 to 20 μg/kg/day, which lies near or within the reported ranges of current human exposure to this chemical. Nagel et al.[9] reported that a maternal dose of only 2 μg/kg per day of bisphenol A enlarged the prostate in male mice offspring. This dose is equivalent to a daily dose of 50 μg for a 25 kg child or 150 μg for a 75 kg adult.

On the other hand, environmental pollution is not likely caused by a single compound, but rather a mixture of chemicals and their related metabolites. Using a yeast-based estrogen screening assay, Payne et al.[13] showed that the combined effect of *o,p′*-DDT, genistein, 4-nonylphenol and 4-*n*-octylphenol does not deviate from expected effects of mixtures of these single xenoestrogens. Therefore, BM could carry mixtures of chemical disruptors whose activity is important to collectively determine for combined adverse effects.

BREASTFEEDING AND POLLUTANT PHARMACOKINETICS

Historically, the study of prescription drugs has provided a basis for understanding the governing principles behind transfer of

chemicals through BM.[7] These factors can be separated into two broad categories: maternal and chemical characteristics. Maternal characteristics include the degree of maternal exposure, physiology of the mother and maternal age and parity (number of pregnancies).[7,14] Chemical characteristics refer to aspects of the compound that affect its ability to be taken up in milk, such as the lipid solubility, degree of ionization, molecular weight, and ability to bind to maternal blood and/or milk components.[15]

Environmental chemicals can be absorbed into the bloodstream by three routes: ingestion, inhalation and dermal contact. In general, xenoestrogens such as dioxin, DDT and PCBs are partially lipophilic, which suggests that they are easily absorbed. LaKind et al.[16] assumed that 95% of a pollutant (2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD)) is absorbed. However, the literature that Lorber and Phillips[17] reviewed suggests a lower absorption rate for a dioxin toxic equivalent (TEQ) of 80%. Yet, there is increasing evidence that the absorption is lower as the degree of chlorination increases, particularly for the highest chlorinated congener, octachlorodibenzo-*p*-dioxin, with an absorption measured to be between 2 and 15%.[14] Since TEQ doses are dominated by the lower chlorinated isoforms, low absorption of higher chlorinated congeners may be less critical, suggesting that most chemicals are well absorbed (80 to 90% exposure dose).

Chemical pollutants circulate in the bloodstream either in their free or bound form. Whether free or bound to proteins such as albumin and lipoproteins, pollutants distribute throughout tissue compartments of the body.[18] Ingested chemicals are typically deposited into adipose tissue, from which it partitions into richly perfused tissue (a single compartment composed of the brain, kidney, intestines, liver and spleen) or instantaneously into muscle. As equilibrium states are reached, the chemicals redistribute, and chemicals with high lipid solubility concentrate in tissue with higher fat content, such as adipose tissue in the brain, liver, kidney, and in the case of lactating women, the mammary gland. Patterson et al.[19] reported that average levels of TCDD were 158 times higher in adipose tissue than in serum, but when each matrix was adjusted for its lipid content, the levels of TCDD were comparable in each matrix. Thus, tissue perfusion and the lipid partition coefficient play important roles in the distribution of chemicals in the body. In the case of PBBs, their concentrations in BM were reported to be 0.7 to 0.9 times that of adipose tissue when results from both were reported on a lipid-adjusted basis. In the same study, when results from the latter matrix were reported on a whole weight basis, the lipid-adjusted adipose tissue and BM concentrations were 107 to 119 times that of plasma. However, in the case of sudden weight loss, these chemicals can be released back into circulation, allowing them to be taken up into the fat of the mammary gland.[14]

Analyzing chemicals found in the plasma of US women, Laden et al.[20] determined that the most important predictors of dichlorodiphenylchloroethane (DDE), the major DDT metabolite, and PCBs were age and total cholesterol levels. Previous studies have consistently observed a positive correlation between age and blood levels of DDE and PCBs. This phenomenon is probably a function of both age and a birth cohort effect. Older women had a greater opportunity for high-level exposures to these compounds because they were alive longer during the period when DDT and PCBs were manufactured and used in the US.[20] Furthermore, they have had a longer time to accumulate the metabolites of these compounds in their body.

Milk is synthesized in the mammary alveolar gland; for its synthesis, milk components and their precursors pass through a membrane that separates the blood flowing in capillaries from the alveolar epithelial cell of the breast. However, during this process, certain environmental chemicals present in the blood also can pass through the membrane and become incorporated into BM at concentrations comparable to the chemicals' levels in other fatty compartments in the body.[21]

The aforementioned physical factors together with chemical characteristics, such as the polarity of the compound, determine the ability of a chemical to transfer into milk. For example, the uptake of lipophilic chemical such as benzene into milk is directly related to milk fat content. However, there is no correlation between the uptake of this hydrocarbon and milk protein level. Nonpolar compounds are easily transported across lipid membranes and can be retained in milk fat due to their lipophilic characteristics.[15] The most common mechanism for the passage of environmental chemicals is passive transport, which, in general, allows passage of lipophilic components of molecular weight <800 Da; thus, lipid solubility of a chemical is a primary factor for its incorporation into BM. Factors that affect the lipophilic character of a chemical include its chemical structure and its degree of ionization in the body compartments. Halogens increase the lipophilic nature of a chemical.[15]

The passive transport mechanism for lipid-soluble chemicals is not the only mechanism for chemicals to cross cell membranes. In general, smaller compounds are transferred into mammary tissue more easily than those with high molecular weights (>200 g/mol). Chemicals of high molecular weight (>800 Da) tend not to pass through the membrane and likely do not enter BM.[21] Also, chemicals (e.g. heavy metals) that are highly bound to either plasma proteins or erythrocytes are unlikely to passively diffuse into milk.[22] Overall, the amount of protein-bound chemical that enters milk is of little concern because the protein compartment in the blood is far greater than that in BM.

Other factors that affect the presence of a chemical in BM are its degree of biotransformation (i.e. more water solubility) and its elimination rate. With regard to the elimination rate, chemicals with a slower rate have a long half-life, allowing for more time in the body and hence more time for bioaccumulation in BM. Many halogenated compounds, including the organochlorine insecticides (e.g. DDT and cyclodienes), PCBs, PBBs, PCDDs and PCDFs all

have long biological half-lives and are persistent in the environment and in humans because of their resistance to oxidative degradation and metabolism. For example, the reported biological half-life for TCDD is 7.2 years, where the reported estimates for other dioxins are 3.7 years for 1,2,3,4,6,7,8-heptachlorodibenzo-*p*-dioxin and 15.7 years for 1,2,3,7,8-pentachlorodibenzo-*p*-dioxin.[4]

For many of these long-lived chemicals, lactation is the primary means of excretion. Lipid-soluble chemicals are transported from adipose tissue stores to the lipids in BM and then eliminated from the body during breastfeeding. Thus, during a single lactation period or after several children and lactation periods, the concentration of a persistent chemical tends to decrease, assuming only background exposures to that chemical. This process of decreasing levels of chemicals during the breast-feeding period is known as ''depuration''. The half-life of total PCBs and DDT in human milk has been estimated to be approximately 6 months.[21] Several references have been made in which measurements of BM concentrations of lipophilic compounds (PCBs, DDE/DDT, PCDDs/PCDFs) were shown to decline during the course of lactation.[1]

Lorber and Phillips[17] used a pharmacokinetic model to predict the infant body burden of dioxin-like compounds that results from breastfeeding. As previously suggested by Abraham et al.,[23] the comparison of the mothers' milk from the first to second children was disparate: the first mother had concentrations ranging from 14 to 24 ppt TEQ lipid for the first child, but 13 to 14 ppt TEQ lipid for the second child. The other mother showed a range of 15 to 27 ppt TEQ lipid for the first child, but 13 to 18 ppt TEQ lipid for the second child. Apparently, breastfeeding of the first child resulted in higher body burdens for this infant compared to the second infant and a lower body burden for the mother when the second infant was born.[17] These results compare to the current adult average body burden of 25 ppt TEQ lipid. This is the current average adult tissue concentration derived in the US EPA's draft dioxin reassessment[24] from recent studies of dioxins in blood in background settings of the US. They also found that an infant who had been breast-fed for 1 year had an accumulated dose six times higher than a 1-year-old infant who had not been breast-fed. For a 70-year lifetime, individuals who had been breast-fed had an accumulated dose that was 3 to 18% (i.e. 6 weeks to 2 years of breastfeeding, respectively) higher than individuals who had not been breast-fed.[17] Similarly, Patandin et al.[25] estimated the cumulative PCB–TEQ and dioxin–TEQ intake from birth until 25 years of age. According to their model, breastfeeding for 6 months accounts for 12 to 14% of the dietary exposure until 25 years of age. The daily TEQ intake per kg body weight for infants breast-fed for 6 months is approximately 50 times higher than for adults, which was about the same as reported by the US EPA.[24] For children under 5 years of age, the daily intake per kg BM is three times as high as in an adult.[25]

Therefore, breastfeeding potentially causes high-dose exposure of chemical pollutants that tend to persist in children for extended periods of time.

BM BIOMONITORING STUDIES

Human milk is a convenient specimen for biomonitoring programs because relatively large volumes (50 to 100 ml) can be collected noninvasively. Although extrapolating BM data to the general population are questionable, exposure in the segment of human population (i.e. breast-feeding newborns) obviously is important to monitor. Another reason to monitor BM is that it reflects the amount that resides in the mother. Furthermore, concentrations of lipophilic chemicals in BM indicate the levels of these chemicals in the mother's fat stores during pregnancy and, consequently, provide a dosimeter of prenatal exposure to these chemicals.[15]

One of the earliest reports of the measurement of an environmental chemical in BM was by Laug et al.[26] in 1951. They reported that the BM from 32 women from the general population of Washington, DC, contained 1,1,1-trichloro2,2-bis(4-chlorophenyl)ethane (*p,p′*-DDT) at an average concentration of 0.13 ppm. These authors attributed the primary source of DDT to their diet. Over the years, many more chemicals have been measured in human BM. Other researchers have shown that infant exposure to dioxin-like compounds can be significant by the BM pathway.[16,25,27,28] Ayotte et al.[28] used data on the concentrations of dioxin-like compounds (including dioxin and furan congeners as well as PCBs) in BM of Inuit populations in Nunavik (the Arctic region of Quebec, Canada), with a median concentration of 48 pg dioxin-TEQ/g lipid, to calculate an infant dose of 226 pg TEQ/kg-day. Actually, many chemical pollutants are commonly detected in human milk (Table 1).

BM has not recently been used to a large degree in the US biomonitoring programs. The largest biomonitoring studies involving BM in the US were conducted at Colorado State University. The first, conducted from 1974 to 1976, comprised 1436 nursing women in hospitals; their milk samples were analyzed for selected chlorinated hydrocarbon insecticides and later for PCBs.[29] The second national study, conducted from 1977 to 1983 by the same laboratory, comprised a total of 1842 milk samples, which also were collected from women residing throughout the US and were analyzed for the same organochlorine insecticides and PCBs.[29] Also, in 1980, the US EPA reported qualitative and semiquantitative data on levels of volatile organic compounds in milk samples collected from lactating women in five US cities.[30] Despite these earlier initiatives, milk has not been routinely monitored in recent US national surveys.

Contrary to the US, BM has been more widely used in biomonitoring programs in Europe. For example, Norén and Meironyte[31] in Sweden and Fürst et al.[32] in Germany reported that

Table 1 Environmental Chemicals Detected in BM Monitoring Studies

Chemical class	Mainly reported compounds
Alkylhydroxybenzoate preservatives	Parabens
Alkylphenols	Octylphenol
Bisphenols	Bisphenol A
Dioxins and furans	
DDT-DDE	Dichlorodiphenylchloroethane
Organochlorine cyclodienes	Aldrin; chlordane; dieldrin; heptachlor; hexachlorobenzene; hexachlorocyclohexane
Semi-volatile organohalogens	Polybrominated biphenyls and diphenylethers; polychlorinated biphenyls; dibenzo-*p*-dioxins; dibenzofurans
Heavy metals	Arsenic; cadmium; lead; mercury
Volatile and other organic compounds	Acids; alcohols; aldehydes; alkanes; alkenes; alkynes; benzene; chloroform; cyclic hydrocarbons; epoxides; ethanol; ethers; halogenated hydrocarbons; ketones; methylene chlorine; musk xylenes; nicotine; nitrogen-containing, polycyclic aromatic and sulfur-containing compounds; perchloroethylene; phathalates; styrene; phytoestrogens; toluene; trichloroethylene

the milk levels of PCDDs, PCDFs and PCBs decreased dramatically from the early 1970s to the late 1990s. In contrast, Norén and Meironyte[31] reported a dramatic increase in milk levels of selected PBDE congeners over this period. The WHO European Centre for Environment and Health is conducting its third filed study, which is designed to assess levels and changes in levels of PCDDs, PCDFs and selected PCBs in BM in countries worldwide.[33]

Despite the difficulties in generalizing across studies, there are some consistent predictors of levels of contaminants in BM. Levels of environmental chemicals are influenced by global and local use patterns of the chemical and furthermore by diet, maternal age, parity, and duration of lactation. Heavier local or regional use of chemicals is consistently associated with elevated local or regional levels of residues in BM samples. However, the absence of local use does not mean that contamination is not detectable. Long-range transport and diet has resulted in detectable BM residues in countries where these chemicals were never used.[5] If many different chemicals were detected worldwide in the BM, in general, bans on the production or use of chemical contaminants have been associated with decreasing residues of these chemicals in BM samples over the subsequent decades. Although levels have not declined to zero, there is evidence of downward trends.

Some authors have interestingly determined xenoestrogen exposure through breastfeeding by comparing breast-fed and formula-fed infants. Kreuzer et al.[27] presented data obtained from adipose tissue and liver from three stillborn infants and 17 infants who had died from sudden infant death syndrome. The highest TEQ concentrations were found in the infants who had been breast-fed, with lipid concentrations of 15.9 ppt TEQ (PCDD/PCDF only), compared to formula-fed infants who had concentrations of 4.3 ppt TEQ lipid. All congener concentrations in breast-fed infants were higher than those in formula-fed infants. Patandin et al.[25] investigated PCBs exposure in 105 breast-fed and 102 formula-fed Dutch children. At 3.5 years of age, children in the breast-fed group had sum PCB levels that were nearly four times higher than sum PCB levels measured in the formula-fed group (0.75 vs 0.21 μg/l).[25] Contaminant levels in BM and breast-feeding period are important determinants of the cumulative contaminant intake during breastfeeding. For example, an infant breast-fed for 26 weeks with relatively low TEQ concentration (10th percentile) in BM would have a similar cumulative intake as an infant breast-fed for 8 weeks by a mother with a relatively high TEQ concentration (90th percentile) in BM.[25] Abraham et al.[23] sampled blood from formula-fed and breast-fed infants for dioxins, furans and PCBs. Results showed that the body burden of dioxin-like compounds was more than an order of magnitude higher for breast-fed infants than the formula-fed infants during both time periods. PCDD/PCDF TEQ concentrations ranged from 34.7 ppt lipid (11 months) to 43.9 ppt lipid (25 months) in breast-fed infants compared to 2.7 to 3.3 ppt TEQ lipid for the formula-fed infants. Dioxin-like PCB concentrations were also an order of magnitude different, with the breast-fed infants having a concentration of 31.4 ppt TEQ lipid compared to 2.5 ppt TEQ lipid for the formula-fed infants at 11 months.

Collectively, the above data suggest that xenoestrogen levels in children are the result of exposure through BM and in utero exposure, and that breast-fed infants had elevated TEQ concentrations compared to formula-fed infants. Overall, the influence of dietary pollutant intake after weaning is small compared to the intake during breastfeeding.

CONCLUSIONS

The chemical pollution of human BM requires consideration of several factors beyond the degree and duration of exposure, the effects of depuration and the sampling time during lactation. As shown in Table 2, these include mother's health during pregnancy and/or lactation period, presence and levels of xenobiotics (including environmental chemicals and pharmaceutical agents)

Table 2 Factors Increasing Infant's Xenoestrogen Exposure Through Breastfeeding

Pollutant's factors
High chemical exposure
High lipid solubility of pollutants
High metabolic stability of pollutants
Mother's factors
Maternal diet (e.g. high seafood consumption)
Old maternal age
High serum cholesterol/lipid levels
Change in body mass index
Low number of pregnancies/breastfeeding
Short previous lactation length
Chemical and pharmaceutical agents affecting xenobiotic metabolism

that may alter metabolism, change in body mass index, diet, parity and previous lactation length, number of children being breast-fed at one time and maternal age.[15] Also of importance is the variation of the fat content during lactogenesis and during the course of feeding.[14,21] In addition, infants and children may exhibit unique susceptibilities to the toxic effects of chemicals because they are undergoing rapid tissue growth and development. Infants and children also consume much greater quantities of milk fat and certain foods than do adults on a body weight basis, and thus they may be subjected to proportionately higher levels of exposure to certain chemicals. These exposures occurring earlier in life may predispose infants and children to a greater risk of chronic toxic effects than exposure occurring later in life. Traditional approaches to health risk assessment need to be expanded to encompass those factors and to adequately protect infants and children. Furthermore, it must be recognized that there are limited data on the residue levels of chemicals in milk and food consumption patterns of infants and children that are appropriate for use in risk assessment.

Although some countries, most notably Sweden and Germany, have ongoing BM monitoring programs in place, data from the rest of the world are sporadic. Few data exist for the US and for most developing countries, particularly over the past 2 decades. Many of the studies that have been conducted are small and not necessarily representative of the larger population of the country where sampling was performed. Although much information has been generated on the types of chemicals likely to be found in BM, this database is scattered and incomplete. Data have been collected on only a limited number of chemicals. The fact that most studies have focused on the same panel of persistent organic pollutants is problematic because it limits the ability to detect new or rising trends in contaminants and thereby may impede effective public health responses. In this regard, few data are actually available on the BM pollution by metals, solvents and other chemicals. The prospective epidemiological studies that are needed to assess chronic outcomes that may occur at lower levels of exposure have been undertaken for a few chemical contaminants, most notably PCBs. Few data exist on long-term effects or on interactions among chemicals.

The scientific debate surrounding endocrine disruptors has grown contentious, partly because some suspected endocrine disruptors are economically important chemicals, high in production volume. The public and regulatory concerns led to government regulatory actions and expanded research across Europe, Japan and North America. International efforts to eliminate persistent organic pollutants may help address some of the areas where levels remain high.[4] The good news is that many persistent organic pollutants have significantly decreased in countries that have placed bans on production and use.[6] Conversely, there are other chemicals that have only recently been observed in BM whose levels may be increasing such as PBDEs, naphthalenes and various cosmetic products (e.g. musk xylenes, nitro musks, polycyclic musks, octyl methoxycinnamate and benzophenone), creating a compelling reason to research the health effects of these chemicals.[4]

In a policy statement, the American Academy of Pediatrics[1,7] stated that exclusive breastfeeding is ideal nutrition and is sufficient to support optimal growth and development for 6 months after birth. They recommend that breastfeeding continue for at least 12 months, and thereafter for as long as mutually desired. Similarly, the US EPA,[24] along with several others such as the American Academy of Pediatrics, concluded that the benefits of breastfeeding outweigh any potential risks associated with this practice, and they readily recommend breastfeeding over formula-feeding. Therefore, BM remains the best source of nutrition for babies. However, constant vigilance is needed to keep it as contaminant free as possible because BM pollution may be the cause of future public health and environmental problems.

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