

# Chemical Contaminants in Breast Milk: Time Trends and Regional Variability

Gina M. Solomon<sup>1</sup> and Pilar M. Weiss<sup>2</sup>

<sup>1</sup>Natural Resources Defense Council, San Francisco, California, USA; <sup>2</sup>Division of Environmental Health Sciences, School of Public Health, University of California-Berkeley, Berkeley, California, USA

Research on environmentally related chemical contaminants in breast milk spans several decades and dozens of countries. The ability to use this research as an environmental indicator is limited because of a lack of consistent protocols. Data on xenobiotics in breast milk are influenced by choices in sample selection, sample pooling, analysis, and reporting. In addition, most studies have focused only on a small panel of persistent organic pollutants, despite indications that a wide range of additional chemical contaminants may also enter breast milk. Despite these limitations, however, it is possible to draw some generalizations. In this paper we review available data on levels of organochlorine pesticides, polychlorinated biphenyls (PCBs), polychlorinated dibenzodioxins (PCDDs), polybrominated diphenyl ethers (PBDEs), metals, and solvents in breast milk. Examples drawn from around the world illustrate the available data and the patterns that have appeared in various areas over time. Over the past few decades, levels of the organochlorine pesticides, PCBs, and dioxins have declined in breast milk in countries where these chemicals have been banned or otherwise regulated. In contrast, the levels of PBDEs are rising. Regional differences in levels of xenobiotics in breast milk are related to historical and current local use patterns. Diet is a major factor that influences breast milk levels of persistent organic pollutants, with patterns in fish consumption playing a particularly significant role. Improved global breast milk monitoring programs would allow for more consistent data on trends over time, detection of new xenobiotics in breast milk, and identification of disproportionately exposed populations. *Key words:* breast-feeding, breast milk, chemical contaminants, dioxins, pesticides, pollutants, polychlorinated biphenyls. *Environ Health Perspect* 110:A339–A347 (2002). [Online 13 May 2002] <http://ehpnet1.niehs.nih.gov/docs/2002/110pA339-A347solomon/abstract.html>

Many nonpharmaceutical chemical contaminants, particularly those that are lipophilic and of relatively low molecular weight, can accumulate in breast milk. The potential health effects of these contaminants, also known as xenobiotics, on both mother and child is of great concern, making it important to carefully monitor contaminant levels and trends. Although some countries, most notably Sweden and Germany, have ongoing breast milk monitoring programs in place, data from the rest of the world are spotty. Few data exist for the United States and for most developing countries, particularly over the past two 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 done. Almost all of the studies on xenobiotics in breast milk have focused on the same chemicals: organochlorine pesticides, polychlorinated biphenyls (PCBs), and dioxins. Few data are available on metals, solvents, and other chemicals. The fact that most studies have focused on the same panel of persistent organic pollutants (POPs) is problematic because it limits the ability to detect new or rising trends in contaminants and thereby may impede effective public health responses.

Efforts to compare levels of specific environmental contaminants across time and place are limited by other obstacles. There has historically been no standardized method for conducting breast milk monitoring

studies, with the partial exception of those studies coordinated by the World Health Organization (WHO) (1). A range of issues including donor selection, the timing of sample collection, the use of preservatives in archived samples, and different methods for estimating population averages (pooled vs. single sample detection) can all significantly affect the results of a study and have made comparisons difficult (2,3). Studies may also differ in their data reporting or in their measurement variables. This is problematic because the quantity and presence of different congeners, metabolites, and impurities reflect different exposure scenarios and different stages in metabolism. Some studies report levels only for a subset of chemicals, so it is difficult to determine if gaps are a result of analytic limitations or if the data truly reflect different types of exposure.

The data that do exist suggest that bans and restrictions in recent decades on the use of many of the POPs have led to a decline in levels of these chemicals in breast milk (3–6). Conversely, there are other chemicals that have only recently been tested in breast milk whose levels may be increasing. The polybrominated diphenyl ethers (PBDEs), currently used as flame retardants, are the only set of chemicals known to be on the rise in breast milk (7); nothing can be said about the many chemicals not included in test panels.

All investigations of contaminants in breast milk must be sensitive to the overriding

benefits of breast-feeding. The advantages of breast-feeding have been documented in the neonatal period and extend throughout childhood and into adulthood (8). There are also clear health benefits to the mother (9). Breast-feeding confers nutritional, immunologic, neurologic, and emotional advantages that have been well documented (10). Although the weight of the scientific evidence to date indicates that the advantages of breast-feeding outweigh any risks from contaminants in breast milk, it is important to identify contaminant trends, locate disproportionately exposed populations, and take public health measures to decrease and eliminate xenobiotics from breast milk. A review of data on levels of contaminants in breast milk from women around the world can provide useful information for guiding exposure reduction efforts and for demonstrating the utility of more consistent, routine, breast milk monitoring for a broader spectrum of chemical contaminants.

## Study Design and Data Comparability

Numerous methodologic discrepancies in study design, sample analysis, and reporting make interpretation of the literature on contaminants in breast milk challenging.

Many of the studies are limited by small sample sizes. This problem is compounded by the common practice of pooling samples from study populations so that only an average is available, and variability within the group is not examined. Many studies include data from women of various ages, parity, and duration of breast-feeding. Because levels of many contaminants are associated with age, parity, and duration of lactation, the mixing of breast milk samples from women with these various characteristics makes it more difficult to identify differences related to actual exposure conditions. The selection of study participants may also bias study results. Some studies may have selected women participants based on potentially high exposure to the chemical of interest so

Address correspondence to G.M. Solomon, Environmental Health Program, NRDC, 71 Stevenson Street, Suite 1825, San Francisco, CA 94105-2964 USA. Telephone: (415) 777-0220. Fax: (415) 495-5996. E-mail: [gsolomon@nrdc.org](mailto:gsolomon@nrdc.org)

This article is part of the mini-monograph "Chemical Contaminants in Breast Milk: Impact on Children's Health."

Received 20 December 2001; accepted 26 February 2002.

that the reported values may not represent the general population of the region.

Perhaps the most critical methodologic challenge relates to chemical analysis. Analytic methods have varied widely. For example, it is difficult to compare studies of PCBs in breast milk because of differences in the number of PCB congeners measured, differences in the analytic method used, and differences in reporting the results (11). This issue can also be seen with the choice of dioxin congeners measured. In studies of dioxin levels in breast milk, the congener 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) is almost always measured. However, different researchers may measure different assortments of the remaining dioxins and furans. For coordinated studies, all 17 congeners must be measured so that data are comparable (1,12). Measurement issues are also evident with organochlorine pesticides that may have different metabolites measured in different studies. For example, numerous forms of the pesticide chlordane can be found in breast milk (oxychlordane, *trans*-nonachlor, *cis*-nonachlor, etc.). The quantity and presence of these different metabolites and impurities reflect different exposure scenarios and stages in metabolism. Depending on the study, there may only be levels reported for certain forms of the chemical. It is difficult to determine if these gaps are a result of analytic limitations or if the data truly reflect different types of exposure.

There are also distinct differences in the way breast milk monitoring data are reported. Some studies do not present information on the base population sampled, including occupational exposures, ages, parity, and other relevant variables. Furthermore, the conventions for reporting results have changed over time. For example, unlike many other chemicals whose toxicity is expressed in terms of a measured concentration, toxicity of dioxins and furans is conventionally expressed in terms of toxic equivalencies (TEQs) (13). The most common is WHO's International TEQ, or I-TEQ (13). But some studies use other toxic equivalency calculation schemes. In such cases, it is difficult to compare these with studies using I-TEQs.

Most methodologic problems stem from the lack of a standardized protocol for conducting breast milk monitoring programs. Many scientists have called for the adoption of a consistent method based on the WHO protocol, which addresses some (but not all) of the challenges described above (3,12,14).

## Review of Available Data by Chemical

Despite the difficulties in generalizing across studies, there are some consistent predictors of levels of contaminants in breast milk (4). Levels of POPs are influenced by global and

local use patterns of the chemical and by diet, maternal age, parity, and duration of lactation (15). Heavier local or regional use of POPs is consistently associated with elevated local or regional levels of residues in breast milk samples (16,17). However, the absence of local use does not mean that no contamination is detectable. Long-range transport and diet has resulted in detectable breast milk residues in countries where these chemicals were never used (18).

It is perilous to extrapolate from observed levels of contaminants in breast milk around the world to predict potential health effects or to declare specific levels safe. Although some regulatory agencies have set benchmark levels for some contaminants in breast milk, these benchmarks may not be completely reliable. Different international and national agencies may set different benchmark levels. In addition, some scientists have questioned whether current benchmark levels adequately protect the developing neonate from some of the more newly recognized health effects of organochlorine contaminants, such as endocrine-disrupting effects (4). In general, however, the levels of the organochlorine pesticides in countries where these chemicals have been banned have dropped well below all current benchmark levels. Commonly detected levels of dioxins and polychlorinated biphenyls, on the other hand, are above most regulatory benchmarks (19).

In the absence of poisoning incidents or local exposure, dietary exposure has been shown to be an important predictor of levels of many POPs in breast milk. In Germany, Schade and Heinzow (20) showed that women who ate a healthy diet (low meat consumption and high vegetable and fruit intake) for at least 3 years had much lower levels of hexachlorobenzene (HCB) and hexachlorohexane (HCH) in their breast milk compared to women who ate more than 700 g of meat per week. Dioxin exposure has also been shown to be strongly correlated with diet (21). Vegetarian mothers have been shown to have lower levels of dioxin in their milk compared to women who eat a diet rich in meat (22).

In general, bans on the production or use of POPs have been associated with decreasing residues of these chemicals in breast milk samples over the subsequent decades (5). Although levels have not declined to zero, there is evidence of downward trends. Conversely, continued use of POPs is associated with time-related increases in breast milk contamination. The following sections review some of the data on specific chemicals to illustrate time trends and regional differences in breast milk levels of common contaminants.

**Chlordane.** Chlordane, a mixture of more than 26 compounds, is an organochlorine

cyclodiene pesticide. Chlordane has been used as an agricultural pesticide, on home lawns and gardens, and against termites. Chlordane has been banned in at least 47 countries and severely restricted in an additional 14 (23). Like most POPs, the breakdown of chlordane once it has attached to soil particles or sediment is very slow; in some cases, it has been found in soil up to 20 years after initial treatment (24). Chlordane is rapidly metabolized in organisms into oxychlordane and  $\gamma$ -chlordane or into impurities such as *trans*-nonachlor or *cis*-nonachlor. It is these breakdown products that persist in the tissue of fish, birds, and mammals and that are found in breast milk (25).

Studies examining chlordane metabolites in breast milk have been conducted in at least 14 countries, including Australia, Canada, Israel, Japan, Kazakhstan, Mexico, Russia, Spain, Thailand, the United States, and Scandinavian countries (18,24–28). Data from areas where chlordane was used show significant variability of levels associated with use patterns. For example, in the 1970s in the United States, lactating women in the southern states had an average of 113 ng/g lipid (range 108–118 ng/g) of chlordane in their milk compared to 79 ng/g (range 76–82 ng/g) among women living in other regions, probably due to agricultural use and more intensive home termite control (24). Similarly, in Japan in the 1980s, women living in homes where chlordane was used for termite control had breast milk chlordane levels 4.4 times higher than women whose households used no form of chlordane (27).

Chlordane residues, however, have not been confined to regions where the chemical was used. For example, chlordane was detected in the breast milk of women in Finland in the mid-1980s, even though the chemical was never used in Finland and was heavily restricted in neighboring countries. Exposure has been attributed to bioaccumulation in Baltic fish (18).

Despite the persistence of chlordane in the environment, data from Sweden demonstrate a declining trend in average breast milk residues of chlordane metabolites in the decades since the chemical was banned in most European countries (28,29). The peak concentrations of chlordane reported in the 1970s in Sweden were 4- to 5-fold lower than the contemporaneous average concentrations found in the United States (25).

**Dieldrin and aldrin.** Dieldrin and aldrin are closely related organochlorine insecticides that are extremely persistent in the environment. Both pesticides have been used in agriculture, and dieldrin was also used for vector control, for veterinary purposes, and for termite control (30). In both plants and animals, aldrin quickly converts to dieldrin.

Once present in soil or water, dieldrin breaks down very slowly, does not easily evaporate into the air, and binds to soil particles. Plants take up aldrin and dieldrin residues directly from the soil. In animals, including humans, dieldrin is stored in the fat and leaves the body very slowly (31). Dieldrin and aldrin are the most widely banned and restricted class of pesticides in the world (32). As of 1995, aldrin and dieldrin had been banned or severely restricted in more than 70 countries (23).

Studies evaluating residues of aldrin and dieldrin in breast milk have been conducted in at least 28 countries, including Brazil, France, Great Britain, Greece, Italy, Kenya, Saudi Arabia, Ukraine, and Vietnam. Dieldrin has been found in > 99% of breast milk samples tested in most countries (33). Because dieldrin is lipophilic and breast milk contains a much greater lipid concentration than blood, the level of dieldrin in a woman's milk is generally about six times higher than the level in her blood.

When dieldrin and aldrin first came into widespread international use, detection prevalence in breast milk rose dramatically. As countries have restricted and banned the use of both chemicals, the prevalence of detection has remained high, but levels detected in breast milk have dropped significantly (25,28,34). Some countries have seen a 10-fold decrease in the level of detectable dieldrin in the years following restrictions. Data from Sweden show a clear decrease in average levels of dieldrin detected in breast milk over several decades (Figure 1) (28). Data from Canada, Denmark, Germany, and Japan also show apparent decreases over time (25,35,36). Insufficient studies have been conducted in the United States, making it impossible to reliably document changes in dieldrin residues over time. The few data available suggest that the ban in the United States may have resulted in similar decreases (25,35).

In some countries with ongoing dieldrin use, breast-milk levels relate closely to local use patterns (16,37). In areas where dieldrin is used more heavily, levels in breast milk are often significantly higher. Studies in European countries and in the United States when dieldrin was still in use showed significantly higher concentrations of dieldrin in breast milk in southern areas than in northern

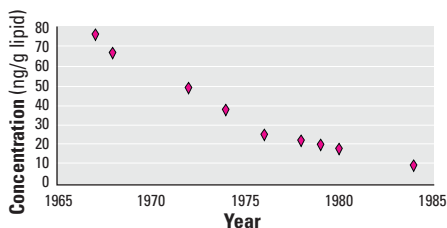


Figure 1. Dieldrin in breast milk in Sweden.

areas. This has been attributed to higher agricultural pesticide use in the southern areas (25). Kenya still uses dieldrin in agriculture, and breast milk of women living in areas with intensive agricultural production has much higher levels of dieldrin than breast milk from women in nonagricultural areas. Figure 2 shows the differences in average dieldrin levels in breast milk during the 1980s around Kenya; the agricultural areas of Loitokitok had the highest levels (16,17). The dieldrin concentrations in Loitokitok are extremely high compared to benchmarks. For example, the U.S. Food and Drug Administration established an action level (the level at which it will consider removing a product from the market) for dieldrin residue in cow's milk of 7.5 ng/g lipid (15). The Kenyan study showed concentrations more than 300 times this level in breast milk. However, it is important to note that this benchmark has not been established as a true safety level for dieldrin. It differs from other benchmarks established by Codex Alimentarius and the WHO (33). There have been no studies that we are aware of evaluating exposures to dieldrin and adverse health effects in breast-fed children.

**DDT.** DDT (dichlorodiphenyltrichloroethane) is a commercial organochlorine insecticide that has been widely used on agricultural crops as well as for vector control (38). DDT and its by-products can persist in soil and sediments for more than 15 years and are known to bioaccumulate in animal tissues. As of 1995, DDT had been banned for all uses in 49 countries and restricted to vector control in 23 (23).

The half-life of DDT in humans is approximately 4 years. DDT's major metabolite, dichlorodiphenylchloroethane (DDE), has a half-life of approximately 6 years (28). The relative proportion of DDT and DDE detected in human tissues can be an indication of the length of time since exposure. In areas where DDT exposure is recent, the DDE/DDT ratio is low, whereas in areas where substantial time has passed since use, the DDE/DDT value is higher. Because DDE is attracted to fat, levels in breast milk are often six to seven times higher in a mother's milk than in her blood (39).

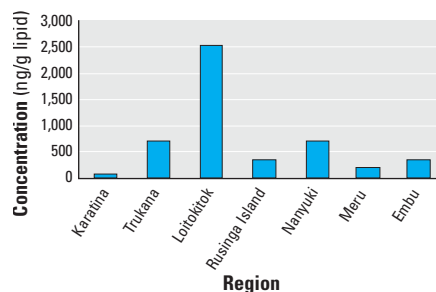


Figure 2. Dieldrin in breast milk in Kenya, 1983–1985.

Although DDT residues in breast milk have been measured in more than 60 countries, only a few nations have comprehensive trend data where multiple studies have been done over time, using large study populations and consistent methods. After the restriction and ban of DDT in some nations, average breast-milk levels decreased substantially. Smith (5) analyzed trend data from around the world and found that the average levels of DDT in breast milk in most countries declined in direct correlation with the length of time since DDT restriction. DDT levels in breast milk in Sweden continuously declined from 1967 through 1997 (Figure 3) (28). The use of DDT was severely restricted in Sweden in 1970 and completely banned in 1975. Germany has also witnessed a rapid decline in average concentrations of DDT in breast milk. Between 1969 and 1995, detectable residue levels decreased by 81%. DDT was banned in Germany in 1972 (20,22,25,36,40,41). Other countries where studies have revealed a downward trend include Canada, Denmark, Norway, Switzerland, Turkey, Yugoslavia, the Czech Republic, Great Britain, Hong Kong, Israel, India, and Japan (18,25,40,42,43).

The difference between areas that currently apply DDT and those that have only the residue of past exposures is particularly evident in data from Zimbabwe and Mexico. DDT was banned for agricultural use in Zimbabwe in 1982 (44), but national averages for DDT in breast milk still show moderately high levels (~6,000 ng/g DDT in lipid). Exposure is generally in the form of DDE, indicating that exposure to DDT was not recent. However, the Kariba region of Zimbabwe, the only region that still actively uses DDT for malaria control, shows much higher levels of total DDT residues (> 25,000 ng/g DDT in lipid) (44). In 1984, WHO established an acceptable daily intake (ADI) level for consumption of DDT in milk. This value of 20 µg/kg/day can be converted into an acceptable level of 5,000–6,000 ng/g DDT in lipid (5). The levels of DDT in some regions of Zimbabwe are above that level.

In Mexico, DDT use has been partially restricted since 1972 and more stringently restricted since 1990. Since that time, overall

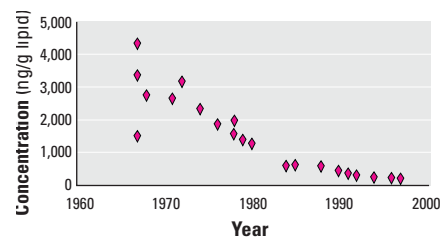


Figure 3. DDT in breast milk in Sweden.

average DDT levels in breast milk appear to have declined (45–48). Despite this downward trend, regional data give cause for concern in those parts of Mexico where DDT continues to be used for malaria control. In the suburban area near Veracruz City, for example, DDT is sprayed at least every 6 months on indoor surfaces and dwellings. Women from suburban areas of Veracruz have higher levels of DDT in their breast milk (average of 10,000 ng/g lipid) than do urban or rural residents of the same area (2,600 and 8,000 ng/g lipid, respectively). In addition, the ratio of DDE/DDT is lower in suburban women, suggesting that these women's residue levels stem from recent, direct exposure. In contrast, the women from the urban and rural areas have high DDE/DDT ratios, suggesting that their breast-milk levels have risen from historical exposures or from exposures through food (49).

The average levels of DDT in breast milk have varied considerably among nations. Figure 4 shows the wide range of levels of DDT found in breast milk in different countries between 1974 and 1976. It was during this time that many industrialized nations banned or began to restrict the use of DDT. At about the same time, use of DDT in developing nations was peaking (18,25,28,34,42,43,45,50–55). Figure 4 reflects data from studies with different designs (as discussed previously). For some of the countries included in the figure, multiple studies may have been conducted in the same time period. Where multiple studies were conducted, the study with the largest study population was included in the figure (25,28,34). Some of the studies were included because no other data for that country exist. This graph illustrates the wide variation that may exist between regions worldwide.

**Heptachlor.** Heptachlor is an organochlorine cyclodiene pesticide that has been used to control termites and as an insecticide on seed grains and food crops. Heptachlor epoxide, the main metabolite of heptachlor, is extremely persistent in soil. In some cases, trace amounts of heptachlor epoxide have been found in soil 14–16 years after application (56). Plants can draw heptachlor epoxide directly from the soil, and the chemical bioaccumulates in animals. Heptachlor has been banned or restricted in more than 60 countries (23,57). However, some of these countries still permit its use for termite and other pest control, and many developing nations still use heptachlor for agricultural purposes (58). Despite the imposition of a ban on use in the United States in 1988, U.S. customs data showed that heptachlor was exported in large quantities through 1994 (59).

Judging from available data, heptachlor epoxide residue levels in breast milk appear

to be decreasing. However, in the time frame for which data are available, the incidence of contamination increased. Early studies often detected heptachlor and heptachlor epoxide in < 10% of samples tested, whereas later studies approached 100% detection with similar analytic methods. As countries have restricted and banned heptachlor, levels detected in breast milk have dropped, often by more than 10-fold (25).

In Alberta, Canada, between 1966 and 1978, the average levels of heptachlor epoxide increased from an average of 2 ng/g lipid to 29 ng/g lipid (35). More striking, however, was the increase in the incidence of detection. From 1966 to 1970, only 5% of collected samples contained detectable levels of heptachlor epoxide. In a 1977–1978 study, the detection incidence had increased to 94% of collected samples (35). The use of heptachlor in Canada was discontinued in 1985 (57). The limited data available suggest that levels in breast milk have probably begun to decrease.

Regional differences have also been reported. In the United States, levels in the Southeast were nearly double the levels in the rest of the country during the period when heptachlor was still used (24). When heptachlor was still used in Belgium, women in the north had breast milk levels 4-fold higher than women in the south, probably related to agricultural use patterns (25). In Spain, a more than a 2-fold difference in heptachlor epoxide levels was found between rural and urban populations, probably due to agricultural use in rural areas (25).

**Hexachlorobenzene.** HCB is a persistent organochlorine chemical that is both a pesticide and an industrial by-product. Its main use is as a fungicide on seed grains (25). HCB is formed as an industrial by-product in chlorination processes, such as wastewater treatment (60). It also forms as a by-product in the manufacturing and production of the wood preservative pentachlorophenol, of chlorinated solvents such as perchloroethylene and carbon tetrachloride, and of various pesticides (25,60). HCB binds strongly to

soil particles as well as to sediment and builds up in plants when it is present in soil (61).

Poisoning incidents in Europe and the United States involving HCB have illustrated the importance of diet as an exposure pathway for breast milk contamination. In an industrial area of Louisiana, cattle were quarantined because of high levels of HCB in their milk and fat. The source of the contamination was thought to be the area where the cattle grazed, which had been contaminated by the disposal of HCB wastes (60). The most notorious example of breast milk contamination by HCB occurred in Turkey in the 1950s. HCB-treated seed wheat intended for agriculture was used for food. Between 1955 and 1959, about 500 people were fatally poisoned by eating bread made with the contaminated seed. More than 4,000 people became ill as a result of the exposure. In some villages, almost all breast-feeding children under the age of 2 years whose mothers had eaten tainted bread died. Locally, this condition was called *pembe yara*. In one mother's breast milk during the incident, the HCB level was 20,000 ng/g in lipid, approximately 2,000 times the average levels of contamination found in breast milk samples around the world (25,60). Follow-up studies 20–30 years after the poisoning found average HCB levels in breast milk still more than 7 times the average for unexposed women in that part of the world (62,63) and 150 times the level allowed in cow's milk (64).

Studies evaluating HCB in breast milk have been conducted in at least 34 countries. Historically, women in areas with less industrialization have had significantly lower levels of HCB in their breast milk. For instance, average HCB levels detected in breast milk in Kenya in the mid-1980s were just 1% of average levels found in Sweden and Germany at a similar time (17). HCB levels in breast milk have declined in some industrialized countries over the past two decades, probably as a result of changes in fungicide use and procedural improvements in industry that have led to a reduction in the generation of HCB

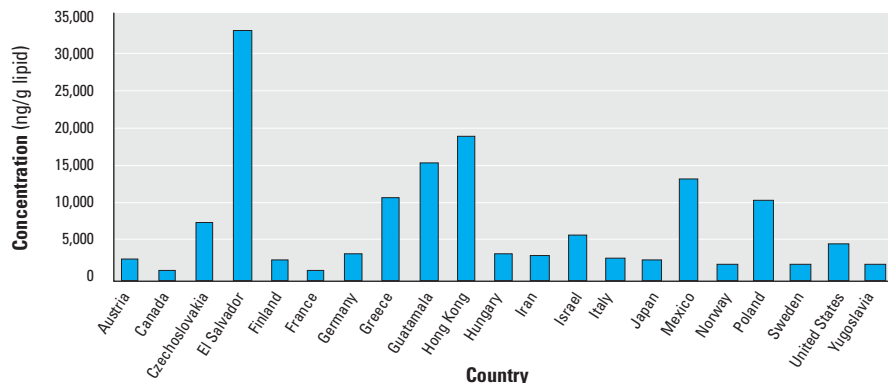


Figure 4. DDT in breast milk around the world, 1974–1976.

by-products (25,28,36). In Czechoslovakia, higher levels of HCB in breast milk were found in industrial areas. These areas (Prague and Kladno) were home to a variety of industries that emitted HCB (65). A study in 1992 found that HCB levels in the breast milk of women living in the Kola Peninsula of Russia were twice as high as in Norway and the Netherlands (66). This area also has much higher levels of industrial pollution than other parts of Europe.

Sweden has witnessed a clear decline in the levels of HCB detected in breast milk (Figure 5). In 1980, Sweden stopped using HCB as a fungicide. In addition, HCB production as an industrial by-product decreased with improvements in industrial technologies (28). Norway experienced a similar decrease, with levels of HCB in breast milk dropping by 65% between the mid-1970s and early 1990s (67). Studies conducted in Germany, Belgium, Canada, Denmark, the Netherlands, and Switzerland suggest a decline in the HCB levels found in breast milk, with a decline of more than 85% in Germany (25).

**Hexachlorocyclohexane.** HCH is an insecticide made up of a mixture of eight isomers. Different isomer forms have different levels of persistence and bioaccumulate in breast milk differently. The  $\gamma$ -isomer of HCH, also known as lindane, is widely used as an insecticide directly applied to the body and scalp to treat head and body lice. The  $\beta$ -isomer of HCH is the most persistent and bioaccumulative form. The  $\alpha$ - and  $\gamma$ -isomers of HCH are converted into the  $\beta$ -isomer in organisms. As a result of this conversion, as much as 90% of HCH detected in human tissues and breast milk is  $\beta$ -HCH (25). HCH is banned or severely restricted in more than 60 countries. Lindane is specifically banned or restricted in 46 countries (23), but its use is often permitted for special uses by exemption. For instance, in the United States, mixed HCH has been banned as an insecticide, but lindane is still allowed as a pharmaceutical for topical application against head lice and scabies, and as a seed treatment.

Studies evaluating HCH contamination of breast milk have been conducted in 41 countries (25). In general, countries that have monitored breast milk for HCH residues

over time have witnessed a steady decrease. Clear downward trends have been reported in the North Rhine Westphalia region of Germany and in Stockholm, Sweden, between 1974 and 1984 (Figure 6) (28,36). Since Japan banned HCH in the 1970s, levels of the pesticide in breast milk have decreased (Figure 7) (25,55).

HCH levels in breast milk are extremely variable and often reflect differences in regional use and exposure patterns. Unusually high levels of HCH in breast milk have been associated with areas of high use. In China and Japan, HCH was commonly used as an insecticide in rice fields, and levels as high as 6,500 ng/g of HCH in lipid have been measured in these countries (25). A 1982 study in Norway, a decade after HCH was banned in that country, found higher levels of the  $\beta$ -isomer of HCH in women who had immigrated from developing countries. Immigrant women had an average level of 433 ng/g  $\beta$ -HCH in their lipid, whereas native Norwegian women had an average of 80 ng/g. The difference was attributed to the likelihood of higher exposures in developing countries (68).

**Dioxins and furans.** Dioxins and furans are two closely related groups of chemical by-products that are produced throughout the world. The dioxin and furan congeners thought to be most toxic to humans are the 7 dioxins and 10 furans known as the 2,3,7,8-congeners. Most studies measuring human exposure to dioxins and furans focus on this group. In breast milk monitoring studies, the term “dioxin” refers to this group of 17 congeners.

Dioxins and furans are listed by several governmental and international agencies as known causes of cancer in humans. Studies have also linked dioxins and furans to reproductive problems, abnormalities in fetal development, immune alterations, and disruption of hormones (19). Unlike other contaminants discussed in this paper where little research into the health effects of low-level exposure is available, there has been considerable work showing effects of dioxin exposure at low levels near the range detected in breast milk (13). Dietary exposure makes up more than 90% of human dioxin intake (21). Because dioxins and furans are so

persistent, lactation is one of the main routes of excretion.

Dioxins and furans have been measured in the breast milk of women from at least 35 countries, including Albania, Cambodia, Croatia, Estonia, the Faeroe Islands, India, Jordan, New Zealand, Pakistan, South Africa, Thailand, Vietnam, most European countries, and the United States (12,13,28,69–79). The general time trend in many countries seems to be toward a slight decrease of dioxin levels in breast milk over the past decade (3). In some countries, the decrease has been quite dramatic, with levels reduced by as much as 50% (69).

Coordinated WHO studies in Europe from 1986 to 1993 showed an average decrease in dioxin levels of approximately 35%, with consistently higher levels in industrial areas (70) (Table 1). Extensive data from Sweden (Figure 8) shows a downward trend in average breast milk levels over 25 years, with a relatively steep decline from the 1970s to the mid-1980s and evidence of a plateau since the mid-1980s (28,74). In many other countries, it has been difficult to make a national assessment of whether levels are going down because of regional variations. For instance, in Croatia, average breast milk levels of dioxin in Krk decreased between 1986 and 1988, while in Zagreb, they increased (12,14). Similarly, findings in Finland and Kazakhstan showed different trends for different regions (12,14,79–81). It is clear that regional variation is important, and different exposure scenarios may have resulted in extremely different levels.

More subtle regional variations have been identified in some studies. For example, in Finland, significant differences in the congener composition of dioxins in breast milk emerged in different regions of the country. The researchers eventually traced the differences to the types of fish consumed in these regions (82). Different species of fish were contaminated with different congener combinations. Similarly, the specific dioxin congeners associated with Agent Orange have been identified in some subpopulations. In Vietnam, concentrations of certain dioxin congeners in breast milk were especially high after intensive aerial spraying of Agent Orange during the Vietnam War (83).

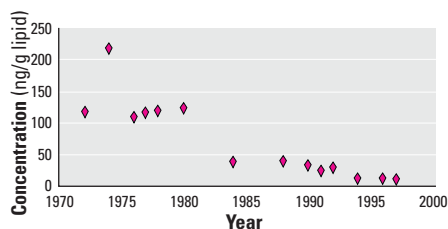


Figure 5. HCB in breast milk in Sweden.

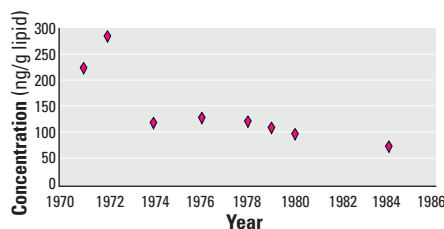


Figure 6.  $\beta$ -HCH in breast milk in Stockholm, Sweden.

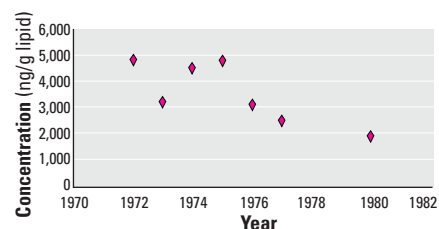


Figure 7.  $\beta$ -HCH in breast milk in Japan.

Especially high levels of dioxin in breast milk as a result of Agent Orange exposure have also been an issue in Kazakhstan (80). In Kazakhstan, women in one geographic region have some of the highest levels in the world of 2,3,7,8-TCDD in their breast milk: Levels in primiparous women in the most exposed area average 53.4 pg/g fat, 10 times higher than U.S. levels. The levels are most likely related to the use of Agent Orange to control weeds in the rice fields. The dioxin contaminants ran off into the lake and accumulated in fish, the dietary staple in the region.

**Polychlorinated biphenyls.** PCBs generally occur as a mixture of several of 209 individual congeners. These persistent chemicals were widely used as flame retardants, in surface coatings, and in electrical equipment such as transformers. The most serious effects of PCBs are on the brain. Low-level PCB exposures, particularly before birth, have been linked to lower IQ, hyperactivity, shortened attention span, and delayed acquisition of reading skills (84,85). PCBs interfere with thyroid hormone, and some researchers believe that this mechanism may explain some of the neurologic effects of PCBs (86). Thyroid hormone is essential for normal growth and development of the brain before birth and throughout infancy (87).

PCBs occur as environmental contaminants around the world. Researchers report that almost all samples of human blood, fat, or breast milk show some detectable level of PCBs (88). Many studies evaluating PCB levels in women have found concentrations in breast milk that are 4–10 times higher than in blood. However, some studies indicate that prenatal exposure (via transplacental transfer) of PCBs that may be more significant to the later health of the child (11,89).

PCBs have been measured in breast milk of women from at least 35 countries, including most of Europe, Greenland, Lithuania, Nigeria, the United States, and Zaire (25,28,65,66,69). Because of the challenges presented by the data measuring PCBs in breast milk, it is difficult to assess trends. Some researchers have speculated that, over the last 25 years, levels may have decreased slightly (88). However, that conclusion is hardly definitive, and the question will most likely remain unanswered until data standardization issues are addressed. In Sweden, where data have been collected following fairly

consistent methods over time, evidence of a downward trend has emerged (Figure 9) (28).

Many researchers have investigated the role of diet in the level of PCBs present in breast milk. In the United States, fish consumption in the Great Lakes area has been associated with a higher body burden of PCBs (90). In Canada, Inuit and fishing populations have higher levels of PCBs in breast milk than do urban populations. Figure 10 shows the difference in some PCB congeners in breast milk levels of Inuit and Caucasian women in Quebec, Canada, in 1989 and 1990 (69). Inuit women, whose diet includes fish and marine mammals, had much higher breast milk levels than urban, Caucasian women. Fish and marine mammal consumption are not the only dietary exposures of concern. In the Czech Republic, the use of a PCB-containing paint in grain silos led to breast milk levels higher than those found in neighboring regions and countries (65).

**Polybrominated diphenyl ethers.** PBDEs are a class of widely used flame retardants. They are added to the plastic material in televisions and computers and are also found in construction materials, furniture, and textiles (7). Unlike the PCBs and many of the organochlorine pesticides, the PBDEs are still widely used throughout the world. The production and use of PBDEs have steadily increased since the 1970s. PBDEs can enter the environment during the production and disposal of materials containing PBDE flame retardants, as well as during the lifetime of PBDE-containing products. PBDEs are not chemically bound to plastics, so they can evaporate into indoor air or the outdoor environment (91). Once released, PBDEs can build up in the environment and in living organisms, binding strongly to sediment and building up in fish and other aquatic organisms (7).

The similarity of the PBDEs to dioxins and PCBs has been a concern because their negative effects on health may prove to be similar (92). In particular, scientists have found indications that the PBDEs may affect hormone function and may be toxic to the developing brain (93). The PBDEs have been associated with non-Hodgkin lymphoma in humans, a variety of cancers in rodents, and disruptions of thyroid hormone balance (92). No restrictions have been placed on the production and use of PBDEs,

but the Swedish government has announced an intention to ban PBDEs in products sold in Sweden, based partly on the detection of these chemicals in breast milk (28,92).

Only a few studies have sought to measure PBDEs in breast milk. Extensive data from Sweden and some limited data from Germany have been collected. In the Swedish study, archived samples collected between 1972 and 1997 were analyzed for the presence of PBDEs to get an overall summed total of PBDEs in milk (7,28). An average for each time period was calculated (Figure 11). The data from Sweden show a logarithmic increase in the quantity of PBDEs detected in women's breast milk.

**Toxic metals.** A number of potentially toxic metals have been reported in breast milk, including lead, mercury, cadmium, and arsenic. Unlike the POPs, metals do not bind to fat and so do not usually accumulate to higher concentrations in breast milk than in blood (94). As a result, infants are likely to be exposed to higher levels before birth than during breast-feeding. Nonetheless, metals in breast milk are important as an additional pathway of exposure and as an indicator of likely prenatal exposures.

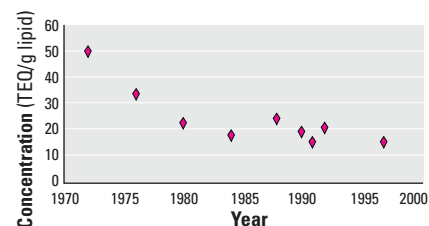
Metals have been detected in breast milk around the world. Twenty-six countries have conducted studies detecting toxic metals, including Bulgaria, Guatemala, Hungary, Iraq, Malaysia, the Philippines, Rumania, and the United States. A WHO study on trace elements in breast milk showed that levels of mercury are extremely variable around the world. Among women who eat a lot of fish, for example, levels of mercury in breast milk may exceed levels in unexposed women by 100-fold (95). Peak measured levels of lead and cadmium worldwide exceed the median by 10-fold, whereas the measured range for arsenic levels is low and very narrow (22).

Several studies have found higher blood lead levels in formula-fed infants than in breast-fed infants (96). This may be a result of contaminated formula cans or formula prepared using tap water with high lead levels. Lead levels in blood and breast milk correlate closely with areas where lead is still used in gasoline, with the highest levels in areas with heavy traffic. In addition, mothers in countries where lead is still used in gasoline, and mothers living near lead smelters have higher

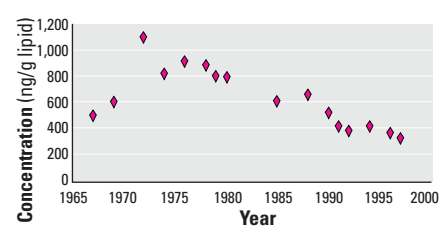
**Table 1.** Dioxins in breast milk in the European Union.

|            | 1988 | 1993 | Change       |
|------------|------|------|--------------|
| Rural      | 28.2 | 17.7 | 37% decrease |
| Urban      | 29.5 | 19.2 | 35% decrease |
| Industrial | 35.9 | 24   | 33% decrease |

Data shown are average concentrations (pg I-TEQ/g fat).



**Figure 8.** Dioxins in breast milk in Sweden.



**Figure 9.** PCBs in breast milk in Sweden.

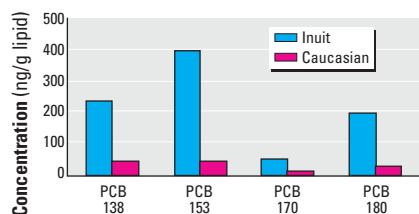
levels of lead in their breast milk due to community contamination (Figure 12) (97).

Much of the lead in breast milk does not come from the mothers' exposures during lactation. Instead, it comes from lead stored in the bones. Calcium extraction from bone is greatest during lactation, and, as a result, lead stored in the mother's bones also enters the blood and breast milk during pregnancy and lactation, posing an exposure risk to the fetus (98). Fortunately, sufficient calcium intake during pregnancy and lactation reduces the extraction of lead from bone (99). Thus women can significantly reduce their child's exposure to lead by getting adequate calcium during pregnancy and lactation.

Breast milk levels of mercury are usually lower than levels of lead. Mercury does not accumulate in breast milk; in fact, the levels in the mother's blood are generally about three times higher than the levels in milk (97). Therefore, prenatal exposure is generally more important than lactational exposure to mercury. Two major forms of mercury can enter breast milk. The most hazardous, methylmercury, does not enter breast milk at high rates because it is attached to red blood cells. But what little does get into breast milk is easily absorbed in the intestine of a nursing infant. The second form, inorganic mercury, enters breast milk easily but is not well absorbed in the infant's gastrointestinal system (100).

In the past, mercury has been responsible for mass poisonings in Minamata, Japan, and in Iraq. In both cases, food contaminated with methylmercury led to illness and death. Some of those affected were breast-feeding children whose mothers had eaten the contaminated food. However, in both of these scenarios, the levels of mercury were far higher than those reported in fish-eating populations today (95).

Cadmium levels in breast milk are significantly associated with cigarette smoking. One German study showed a direct relationship between the number of cigarettes a mother smokes per day and the level of cadmium in her breast milk (101). However, some studies indicate that an infant's exposure to cadmium from soy infant formula is about 20 times higher than the levels generally found in breast milk (102).

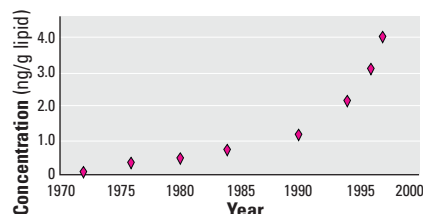


**Figure 10.** Specific PCB congeners in breast milk in Canada (1989–1990). Data from Dewailly et al. (69).

**Solvents.** Organic solvents are ubiquitous in both industrial and household settings. These chemicals are present in paints, varnishes, thinners, dry-cleaning fluids, some glues, degreasers, and gasoline. Solvents include a wide variety of chemicals with varying properties, defined more by their use than by their chemistry or toxicity. In general, organic solvents are highly volatile and readily absorbed through the skin. These chemicals are also common water contaminants. As a result of their widespread presence in the environment, solvents are found in human urine, exhaled breath, blood, and fat (103). Because solvents are relatively short-lived in the body, detection implies recent exposure.

Numerous organic solvents have been detected in breast milk, including benzene, chloroform, methylene chloride, styrene, perchloroethylene, toluene, trichloroethylene, 1,1,1-trichloroethane, and xylene (104). Breast milk levels of these compounds may be higher than blood levels in part because breast tissue does not eliminate solvents as quickly as does blood (104). Perchloroethylene, in particular, is known to concentrate in breast milk to levels about three times higher than levels in blood (105). That said, much of the research on this group of chemicals has been preliminary and is of two types: theoretical models estimating which solvents may get into breast milk, and small monitoring studies (106). The short-lived nature of solvents makes sampling difficult because samples need to be collected and transported in a specialized way and analyzed quickly so they do not evaporate away between the time of collection and the time of analysis. Most countries have not conducted studies of solvents in breast milk and have gathered no data representative of the general population. Due to limited data, it is possible only to conclude that some solvents get into breast milk; no information relevant to the levels of exposure, geographic differences, or time trends are available.

In one case report, a lactating mother who visited her husband daily at a dry cleaning firm had high levels of perchloroethylene in her milk (107). This case does not reflect normal population-level exposure to perchloroethylene; however, it is an important



**Figure 11.** PBDEs in breast milk in Sweden.

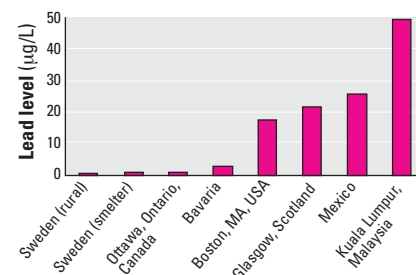
indicator of the potential for exposure to the nursing infant. A modeling study predicted elevated levels of perchloroethylene in the breast milk of women living in apartments in buildings containing dry cleaning businesses (108). In another modeling study, three solvents (perchloroethylene, bromochloroethane, and 1,4-dioxane) were estimated to accumulate in breast milk in excess of the U.S. Environmental Protection Agency's (EPA) health advisory level for drinking water if the mother was exposed at the legally allowable workplace limit (105). In addition, predicted breast milk levels of carbon tetrachloride and trichloroethylene were extremely close to the U.S. EPA health advisory level. These estimates are likely to underestimate potential risk to breast-feeding children of mothers exposed to solvents in the workplace because the U.S. EPA risk values used were for non-cancer health effects in adults, whereas several of the solvents evaluated are carcinogens and the exposure is to neonates.

Solvents are an avoidable source of potential chemical exposures. Although little information has been gathered about these chemicals in breast milk, we do know that many solvents can enter the mother's body and then transfer into her milk. Because use of these chemicals is so widespread, the subject merits further research.

## Summary

Although monitoring of environmental xenobiotics in breast milk has been relatively limited, the available data do provide some useful information about patterns of pollution over time and determinants of exposure. Many of the persistent organic pollutants have significantly decreased in countries that have placed bans on production and use. Exposure varies significantly based on local chemical use and on dietary habits in the population under study. International efforts to eliminate persistent organic pollutants may help address some of the areas where levels remain high.

Breast milk contamination is an important indicator of potential future public health and environmental problems. Increasing levels of the brominated diphenyl ethers and recent reported detections of other chemicals



**Figure 12.** Lead in breast milk by location.

such as naphthalenes and various cosmetic products including musk xylenes, nitro musks, polycyclic musks, octyl methoxycinnamate, and benzophenone-3 (74,109–111) create a compelling reason to research the health effects of these chemicals, increase monitoring efforts, and design health-based strategies for reducing or eliminating exposure.

It is critical to move beyond studies of the same small group of chemicals in a few subjects in limited geographic areas using inconsistent protocols to a more systematic international breast milk monitoring program (3,112). Such a program would use a consistent protocol to monitor for an expanded range of suspected contaminants. The results would be useful to guide research and public health interventions worldwide. It is only with more information from many women and in many locations that we will be able to track trends, identify potential hazards, and protect future generations.

## REFERENCES AND NOTES

- WHO. Levels of PCBs, PCDDs and PCDFs in Human Milk: Protocol for Third Round of Exposure Studies. Bilthoven, Netherlands:World Health Organization European Centre for Environment and Health, 2000.
- Sim M, McNeil J. Monitoring chemical exposure using breast milk: a methodological review. *Am J Epidemiology* 136:1–11 (1992).
- LaKind JS, Berlin CM, Naiman DQ. Infant exposure to chemicals in breast milk in the United States: what we need to learn from a breast milk monitoring program. *Environ Health Perspect* 109:75–88 (2001).
- Solomon G, Weiss P. Healthy Milk, Healthy Baby. New York:Natural Resources Defense Council, 2001. Available: <http://www.nrdc.org/breastmilk> [cited 4 April 2002].
- Smith D. Worldwide trends in DDT levels in human milk. *Int J Epidemiol* 28:179–188 (1999).
- Craan AG, Haines DA. Twenty-five years of surveillance for contaminants in human breast milk. *Arch Environ Contam Toxicol* 35:702–710 (1998).
- Meirionyte D, Noren K, Bergman A. Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972–1997. *J Toxicol Environ Health Part A* 58:329–341 (1999).
- Lawrence RA. Breastfeeding: benefits, risks and alternatives. *Curr Opin Obstet Gynecol* 12:519–524 (2000).
- Labbok MH. Effects of breastfeeding on the mother. *Pediatr Clin North Am* 48:43–58 (2001).
- Oddy WH. Breastfeeding protects against illness and infection in infants and children: a review of the evidence. *Breastfeed Rev* 9:11–18 (2001).
- Dekoning EP, Karmaus W. PCB exposure in utero and via breast milk. A review. *J Expo Anal Environ Epidemiol* 10:285–293 (2000).
- Vrjanheikki EJ, ed. Levels of PCBs, PCDDs and PCDFs in Breast Milk: Results of WHO-Coordinated Interlaboratory Quality Control Studies and Analytical Field Studies. Environmental Health Series 34. Copenhagen:World Health Organization, 1989.
- IARC. Polychlorinated Dibenzo-*para*-Dioxins and Polychlorinated Dibenzofurans. IARC Monogr Eval Carcinog Risks Hum 69 (1997).
- WHO. Levels of PCBs, PCDDs and PCDFs in Human Milk: Second Round of WHO-Coordinated Study. Environmental Health in Europe No. 3. Bilthoven, Netherlands:WHO European Centre for Environment and Health, 1996.
- Rogan WJ, Bagniewska A, Damstra T. Pollutants in breast milk. *N Engl J Med* 302:1450–1453 (1980).
- Ejobi F, Kanja LW, Kyule N, Muller P, Latigo AR. Organochlorine pesticide residues in mother's milk in Uganda. *Bull Environ Contam Toxicol* 56:873–880 (1996).
- Kanja L, Skare JU, Nafstad I, Maitai CK, Lokken P. Organochlorine pesticides in human milk from different areas of Kenya 1983–85. *J Tox Environ Health* 19:449–464 (1986).
- Wickstrom K, Pyysalo H, Siimes M. Levels of chlordane, hexachlorobenzene, PCB and DDT compounds in Finnish human milk in 1982. *Bull Environ Contam Toxicol* 31:251–256 (1983).
- U.S. EPA. Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-*p*-Dioxin (TCDD) and Related Compounds. Washington, DC:U.S. Environmental Protection Agency, 2000.
- Schade G, Heinzow B. Organochlorine pesticides and polychlorinated biphenyls in human milk of mothers living in northern Germany: current extent of contamination, time trend from 1986 to 1997 and factors that influence the levels of contamination. *Sci Total Environ* 215:31–39 (1998).
- Brouwer A, Ahlborg UG, van Leeuwen FXR, Feeley MM. Report of the WHO working group on the assessment of health risks for human infants from exposure to PCDDs, PCDFs and PCBs. *Chemosphere* 37:1627–1643 (1998).
- Somogyi A, Beck H. Nuturing and breast-feeding: exposure to chemicals in breast milk. *Environ Health Perspect* 101(suppl 2):45–52 (1993).
- PANNA. Demise of the Dirty Dozen Chart. San Francisco, CA:Pesticide Action Network North America, 1995.
- Savage E. National study of chlorinated hydrocarbon insecticide residues in human milk, USA. *Am J Epidemiol* 113:413–422 (1981).
- Jensen AA, Slorach SA. Chemical Contaminants in Human Milk. Boca Raton, FL:CRC Press, 1991.
- Sonawane B. Chemical contaminants in human milk: an overview. *Environ Health Perspect* 103(suppl 6):197–205 (1995).
- Taguchi S, Yakushiji T. Influence of termite treatment in the home on the chlordane concentration in human milk. *Arch Environ Contam Toxicol* 17:65–71 (1988).
- Noren K, Meirionyte D. Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20–30 years. *Chemosphere* 40:1111–1123 (2000).
- Noren K. Contemporary and retrospective investigations of human milk in the trend studies of organochlorine contaminants in Sweden. *Sci Total Environ* 139/140:347–355 (1993).
- ATSDR. Public Health Statement for Aldrin and Dieldrin. Atlanta, GA: Agency for Toxic Substances and Disease Registry, 1989.
- ATSDR. ToxFAQs for Aldrin/Dieldrin. Atlanta, GA: Agency for Toxic Substances and Disease Registry, 1993.
- Siedenburg K. Demise of the drins. Global Pesticide Campaigner January (1991). Available: [http://www.igc.org/panna/resources/\\_pestis/PESTIS.burst.121.html](http://www.igc.org/panna/resources/_pestis/PESTIS.burst.121.html) [cited 25 March 2002].
- WHO. Aldrin and Dieldrin. Environmental Health Criteria 91. Geneva:World Health Organization, 1989.
- Mes J, Davies DJ. Presence of polychlorinated biphenyl and organochlorine pesticide residues and the absence of polychlorinated terphenyls in Canadian human milk samples. *Bull Environ Contam Toxicol* 21:381–387 (1979).
- Currie RA, Kadis VM, Breitkreitz WE, Cunningham CB, Bruns GW. Pesticide residues in human milk, Alberta, Canada. *Pestic Monit J* 13:52–55 (1979).
- Furst P, Furst C, Wilmers K. Human milk as a bioindicator for body burden of PCDDs, PCDFs, organochlorine pesticides, and PCBs. *Environ Health Perspect* 102(suppl 1):187–193 (1994).
- Stacey CI, Tatum T. House treatment with organochlorine pesticides and their levels in human milk - Perth, Western Australia. *Bull Environ Contam Toxicol* 35:202–208 (1985).
- ATSDR. ToxFAQs for DDT, DDE, and DDE. Atlanta, GA: Agency for Toxic Substances and Disease Registry, 1995.
- Wolff M. Occupationally derived chemicals in breast milk. *Am J Ind Med* 4:259–281 (1983).
- Mes J, Davies DJ, Turton D, Sun WF. Levels and trends of chlorinated hydrocarbon contaminants in the breast milk of Canadian women. *Food Addit Contam* 3:313–322 (1986).
- Schechter AJ, Furst P, Kruger C, Meemken H-A, Groebel W, Constable JD. Levels of polychlorinated dibenzofurans, dibenzodioxins, PCBs, DDT and DDE, hexachlorobenzene, dieldrin, hexachlorocyclohexane and oxychlordane in human breast milk from the United States, Thailand, Vietnam, and Germany. *Chemosphere* 18:445–454 (1989).
- Prachar V, Veningerova M, Uhnak J. Levels of polychlorinated biphenyls and some other organochlorine compounds in breast milk samples in Bratislava. *Sci Total Environ (suppl Pt 1):237–242* (1993).
- Ip HM, Phillips DJ. Organochlorine chemicals in human breast milk in Hong Kong. *Arch Environ Contam Toxicol* 18:490–494 (1989).
- Chikuni O, Polder A, Skaare JU, Nhachi CFB. An evaluation of DDT and DDT residues in human breast milk in the Kariba Valley of Zimbabwe. *Bull Environ Contam Toxicol* 58:776–778 (1997).
- Albert L, Vega P, Portales A. Organochlorine pesticide residues in human milk samples from Comarca Lagunera, Mexico, 1976. *Pestic Monit J* 15:135–138 (1981).
- Gladden BC, Rogan WJ. DDE and shortened lactation in a northern Mexican town. *Am J Public Health* 85:504–508 (1995).
- Waliszewski SM, Pardo Sedes VT, Chantiti JN, Infanzon RM, Rivera RJ. Organochlorine pesticide residues in human breast milk from tropical areas in Mexico. *Bull Environ Contam Toxicol* 57:22–28 (1996).
- Lopez-Carrillo L, Torres-Arreola L, Torres-Sanchez L, Espinosa-Torres F, Jimenez C, Cebrian M, Waliszewski S, Saldate O. Is DDT use a public health problem in Mexico? *Environ Health Perspect* 104:584–588 (1996).
- Pardio VT, Waliszewski M, Aguirre AA, Coronel H, Burelo GV, Infanzon RM, Rivera J. DDT and its metabolites in human milk collected in Veracruz City and suburban areas (Mexico). *Bull Environ Contam Toxicol* 60:852–857 (1998).
- Brevik EM, Bjerk JE. Organochlorine compounds in Norwegian human fat and milk. *Acta Pharmacol Toxicol* 43:59–63 (1978).
- Bradt PT, Herrenkohl RC. DDT in human milk. What determines the levels? *Sci Total Environ* 6:161–163 (1976).
- de Campos M, Olszyna-Marzys AE. Contamination of human milk with chlorinated pesticides in Guatemala and El Salvador. *Arch Environ Contam Toxicol* 8:43–58 (1979).
- Winter M, Thomas M, Wernick S, Levin S, Farcar MT. Analysis of pesticide residues in 290 samples of Guatemalan mother's milk. *Bull Environ Contam Toxicol* 16:652–657 (1976).
- Polishuk ZW, Ron M, Wassermann M, Cucos S, Wasserman D, Lemesch C. Organochlorine compounds in human blood plasma and milk. *Pestic Monit J* 10:121–129 (1977).
- Yakushiji T, Watanabe I, Kuwabara K, Yoshida S, Koyama K, Kunita N. Levels of polychlorinated biphenyls (PCBs) and organochlorine pesticides in human milk and blood collected in Osaka Prefecture from 1972 to 1977. *Int Arch Occup Environ Health* 43:1–15 (1979).
- EXTOXNET. Pesticide Information Profile. Heptachlor. Corvallis, OR:Oregon State University, 1996. Available: <http://ace.orst.edu/info/extoxnet/pips/heptachl.htm> [cited 4 April 2002].
- WHO. Heptachlor Health and Safety Guide. Health and Safety Guide 14. Geneva:World Health Organization, 1988.
- Noronha F. Persistent organic pollutants pervade Asia. Environment News Service, 1998. Available: <http://ens.lycos.com/ens/nov98/1998L-11-23-03.html> [cited 4 April 2002].
- PANNA. Velsicol Ceases Production of Chlordane and Heptachlor. PANUPS. San Francisco:Pesticide Action Network North America, 1997. Available: [http://www.igc.org/panna/resources/\\_pestis/PESTIS.1997.43.html](http://www.igc.org/panna/resources/_pestis/PESTIS.1997.43.html) [cited 25 March 2002].
- Courtney KD. Hexachlorobenzene (HCB): a review. *Environ Res* 20:225–266 (1979).
- ATSDR. ToxFAQs for Hexachlorobenzene. Atlanta, GA: Agency for Toxic Substances and Disease Registry, 1997.
- Gocmen A, Peters HA, Cripps DJ, Bryan GT, Morris CR. Hexachlorobenzene episode in Turkey. *Biomed Environ Sci* 2:36–43 (1989).
- Cripps DJ, Peters HA, Gocmen A, Dogramaci I. Porphyria turcica due to hexachlorobenzene: a 20 to 30 year follow-up study on 204 patients. *Br J Dermatol* 111:413–422 (1984).
- Peters HA, Gocmen A, Cripps DJ, Bryan GT, Dogramaci I. Epidemiology of hexachlorobenzene-induced porphyria in Turkey: clinical and laboratory follow-up after 25 years. *Arch Neurol* 39:744–749 (1982).
- Schoula R, Hajslova J, Bencko V, Poustka J, Holadova K, Vizek V. Occurrence of persistent organochlorine contaminants in human milk collected in several regions of Czech Republic. *Chemosphere* 33:1485–1494 (1996).
- Polder A, Becher G, Savinova TN, Skaare JU. Dioxins,



- PCBs and some chlorinated pesticides in human milk from the Kola Peninsula, Russia. *Chemosphere* 37:1795–1806 (1998).
67. Johansen HR, Becker G, Polder A, Skaare JU. Congener-specific determination of polychlorinated biphenyls and organochlorine pesticides in human milk from Norwegian mothers living in Oslo. *J Toxicol Environ Health* 42:157–71 (1994).
  68. Skaare JU, Tuveng JM, Sande HA. Organochlorine pesticides and polychlorinated biphenyls in maternal adipose tissue, blood, milk, and cord blood from mothers and their infants living in Norway. *Arch Environ Contam Toxicol* 17:55–63 (1988).
  69. Dewailly É, Ryan JJ, Laliberté C, Bruneau S, Weber J-P, Gingras S, Carrier G. Exposure of remote maritime populations to coplanar PCBs. *Environ Health Perspect* 102(suppl 1):205–209 (1994).
  70. Peterson A. Compilation of EU Dioxin Exposure and Health Data Task 5 - Human Tissue and Milk Levels AEAT/EEQC/0016.5. Oxfordshire, UK:European Commission Environment and UK Department of the Environment, Transport and the Regions, 1999.
  71. Clench-Aas J, Bartonova A, Oehme M, Lindstrom G. PCDD and PCDF in human milk from Scandinavia, with special emphasis on Norway. *J Toxicol Environ Health* 37:73–83 (1992).
  72. Startin JR, Rose M, Offen C. Analysis of PCDDs and PCDFs in human milk from the UK. *Chemosphere* 19:985–988 (1989).
  73. Wearne SJ, Harrison N, Gem MG, Startin JR, Wright C, Kelly M, Robinson C, White S, Hardy D, Edinburgh V. Time trends in human dietary exposure to PCDDs, PCDFs and PCBs in the UK. *Organohalogen Compounds* 31:1–6 (1996).
  74. Lunden A, Noren K. Polychlorinated naphthalenes and other organochlorine contaminants in Swedish human milk, 1972–1992. *Arch Environ Contam Toxicol* 34:414–423 (1998).
  75. Beck H, Dross A, Mathar W. PCDD and PCDF exposure and levels in humans in Germany. *Environ Health Perspect* 102(suppl 1):173–185 (1994).
  76. Furst P, Kruger C, Meemken H-A, Groebel W. PCDD and PCDF levels in human milk - dependence on period of lactation. *Chemosphere* 18:439–444 (1989).
  77. Schecter A, Furst P, Furst C, Papke O, Ball M, Le CD, Hoang TQ, Nguyen TNP, Beim A, Vlasov B, et al. Dioxins, dibenzofurans and selected chlorinated organic compounds in human milk and blood from Cambodia, Germany, Thailand, the U.S.A., the U.S.S.R., and Viet Nam. *Chemosphere* 23:1903–1912(1991).
  78. Hirakawa H, Iida T, Matsueda T, Nakagawa R, Hori T, Nagayama J. Comparison of concentrations of PCDDs, PCDFs, PCBs and other organohalogen compounds in human milk of primiparas and multiparas. *Organohalogen Compounds* 26:197–200 (1995).
  79. Hooper K, Petreas MX, Chuvakova T, Kazbekova G, Druz N, Seminova G, Sharmanov T, Hayward D, She J, Visita P, et al. Analysis of breast milk to assess exposure to chlorinated contaminants in Kazakhstan: levels of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) in agricultural villages of southern Kazakstan. *Environ Health Perspect* 106:797–806 (1998).
  80. Hooper K, Chuvakova T, Kazbekova G, Hayward D, Tulenova A, Petreas MX, Wade TJ, Benedict K, Cheng Y-Y, Grassman J. Analysis of breast milk to assess exposure to chlorinated contaminants in Kazakstan: sources of 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (TCDD) exposures in an agricultural region of southern Kazakstan. *Environ Health Perspect* 107:447–457 (1999).
  81. Petreas M, Hooper K, She J, Visita P, Winkler J, McKinney M, Mok M, Sy F, Garcha J, Gill M, Stephens R, et al. Analysis of human breast milk to assess exposure to chlorinated contaminants in Kazakstan. *Organohalogen Compounds* 30:20–23 (1996).
  82. Vartiainen T, Saarokoski S, Jaakkola JJ, Tuomisto J. PCDD, PCDF, and PCB concentrations in human milk from two areas in Finland. *Chemosphere* 34:2571–2583 (1997).
  83. Lutter C, Iyengar V, Barnes R, Chuvakova T, Kazbekova G, Sharmanov T. Breast milk contamination in Kazakstan: implications for infant feeding. *Chemosphere* 37:1761–1772 (1998).
  84. Lonky E, Reihman J, Darvill T, Mather J, Daly H. Neonatal behavioral assessment scale performance in humans influenced by maternal consumption of environmentally contaminated Lake Ontario fish. *J Great Lakes Res* 22:198–212 (1996).
  85. Jacobson J, Jacobson S. Intellectual impairment in children exposed to polychlorinated biphenyls in utero. *N Engl J Med* 335:783–789 (1996).
  86. Tilson H, Kodavanti P. Neurochemical effects of polychlorinated biphenyls: an overview and identification of research needs. *Neurotoxicology* 18:727–743 (1997).
  87. Haddow J, Palomaki G, Allan W, Williams J, Knight G, Gagnon J, O'Heir C, Mitchell M, Hermos R, Waitsbren S, et al. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. *N Engl J Med* 341:549–555 (1999).
  88. Longecker MP, Rogan WJ, Lucier G. The human health effects of DDT (dichlorodiphenyltrichloroethane) and PCBs (polychlorinated biphenyls) and an overview of organochlorines in public health. *Annual Rev Public Health* 18:211–244 (1997).
  89. Huisman M, Koopman-Esseboom C, Lanting CI, van der Pauw CG, Tuinstra LGMT, Fidler V, Weisglas-Kuperus N, Sauer PJJ, Boersma ER, Touwen BCL. Neurological condition in 18-month-old children perinatally exposed to polychlorinated biphenyls and dioxins. *Early Hum Dev* 43:165–176 (1995).
  90. Falk C, Hanranhan L, Anderson HA, Kanarek MS, Draheim L, Needham L, Patterson D. Body burden levels of dioxin, furans, and PCBs among frequent consumers of great lakes sport fish. *Environ Res* 80:S19–25 (1999).
  91. Hooper K, McDonald TA. The PBDEs: an emerging environmental challenge and another reason for breast-milk monitoring programs. *Environ Health Perspect* 108:387–392 (2000).
  92. Darnerud PO, Eriksen GS, Johannesson T, Larsen PB, Viluksela M. Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. *Environ Health Perspect* 109(suppl 1):49–68 (2001).
  93. Eriksson P, Jakobsson E, Fredriksson A. Brominated flame retardants: a novel class of developmental neurotoxicants in our environment? *Environ Health Perspect* 109:903–908 (2001).
  94. Golding J. Unnatural constituents of breast milk—medication, lifestyle, pollutants, viruses. *Early Hum Dev* 49:S29–S43 (1997).
  95. Grandjean P, Jorgensen PJ, Weihe P. Human milk as a source of methylmercury exposure to infants. *Environ Health Perspect* 102:74–77 (1994).
  96. Rabinowitz M, Leviton A, Needleman H. Lead in milk and infant blood: a dose-response model. *Arch Environ Health* 40:283–286 (1985).
  97. Oskarsson A, Palminger HI, Sundberg J. Exposure to toxic elements via breast milk. *Analyst* 120:765–770 (1995).
  98. Moline J, Carrillo L, Sanchez L, Godbold J, Todd A. Lactation and lead body burden turnover: a pilot study in Mexico. *J Occup Environ Med* 42:1070–1075 (2000).
  99. Gulson B, Mahaffey K, Jameson C, Mizon K, Korsch M, Cameron M, Eisman J. Mobilization of lead from the skeleton during the postnatal period is larger than during pregnancy. *J Lab Clin Med* 131:324–329 (1998).
  100. Oskarsson A, Ohlin I, Schutz A, Lagerkvist B, Skerfving S. Total and inorganic mercury in breast milk and blood in relation to fish consumption and amalgam fillings in lactating women. *Arch Environ Health* 51:234–241 (1996).
  101. Radisch B, Luck W, Nau H. Cadmium concentrations in milk and blood of smoking mothers. *Toxicol Lett* 36:147–152 (1987).
  102. Oskarsson A, Palminger HI, Sundberg J, Petersson GK. Risk assessment in relation to neonatal metal exposure. *Analyst* 123:19–23 (1998).
  103. Lordo RA, Dinh KT, Schwemmerberger JG. Semivolatile organic compounds in adipose tissue: estimated averages for the US population and selected subpopulations. *Am J Public Health* 86:1253–1259 (1996).
  104. Labreche FP, Goldberg MS. Exposure to organic solvents and breast cancer in women: a hypothesis. *Am J Ind Med* 32:1–14 (1997).
  105. Fisher J, Mahle D, Bankston L, Greene R, Gearhart J. Lactational transfer of volatile chemicals in breast milk. *Am Ind Hyg Assoc J* 58:425–431 (1997).
  106. Pellizzari ED, Hartwell TD, Harris BSH, Waddell RD, Whitaker DA, Erickson MD. Purgeable organic compounds in mother's milk. *Bull Environ Contam Toxicol* 28:322–328 (1982).
  107. Bagnell PC, Ellenberger HA. Obstructive jaundice due to a chlorinated hydrocarbon in breast milk. *Can Med Assoc J* 5:1047–1048 (1977).
  108. Schreiber JS. Predicted infant exposure to tetrachloroethene in human breastmilk. *Risk Anal* 13:515–524 (1993).
  109. Schlumpf M, Cotton B, Conscience M, Haller V, Steinmann B, Lichtensteiger W. *In vitro* and *in vivo* estrogenicity of UV screens. *Environ Health Perspect* 109:239–244 (2001).
  110. Kafferlein HU, Angerer J. Trends in the musk xylene concentrations in plasma samples from the general population from 1992/1993 to 1998 and the relevance of dermal uptake. *Int Arch Occup Environ Health* 74:470–476 (2001).
  111. Ott M, Failing K, Lang U, Schubring C, Gent H-J, Georgii S, Brunn H. Contamination of human milk in Middle Hesse, Germany - a cross-sectional study on the changing levels of chlorinated pesticides, PCB congeners and recent levels of nitro musks. *Chemosphere* 38:13–32 (1999).
  112. Hooper K. Breast milk monitoring programs (BMMPs): world-wide early warning system for polyhalogenated POPs and for targeting studies in children's environmental health. *Environ Health Perspect* 107:429–430 (1999).



## Visit *EHP* Career Opportunities Online

- updated weekly
- most ads include e-mail links for instant response

Just point and click. [ehponline.org](http://ehponline.org)