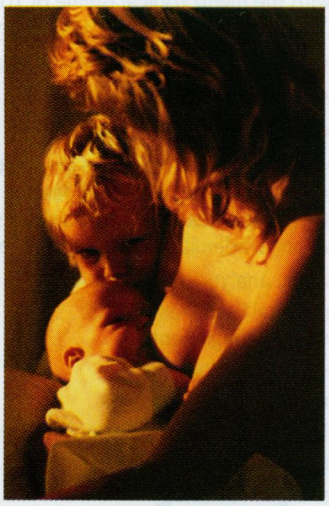
A Contaminant in Mothers' Milk

The Persistent Threat of PBDEs

A class of compounds known as polybrominated diphenyl ethers (PBDEs)—flame-retardant additives used in high-impact plastics, foams, and textiles—has recently sparked concerns among environmental health scientists. PBDEs are part of a superfamily of related toxic compounds known as persistent organic pollutants (POPs). Limited evidence suggests they may be associated with health effects including cancer, thyroid toxicity, and neurodevelopmental problems. In this issue, Kim Hooper, a research scientist with the Hazardous Materials Laboratory in the California Environmental Protection Agency, and Thomas McDonald, a staff toxicologist in the agency's Office of Environmental Health Hazard Assessment, discuss PBDEs and explore what prove to be many parallels between some PBDEs and other members of the POPs family [*EHP* **108:387–392**].



Chemicals latch on to milk. Evidence that toxic PBDEs accumulate in breast milk suggests that they could pose a threat to health.

The POPs family also includes compounds such as dioxins, furans, and polychlorinated biphenyls (PCBs), which have been found in humans, animals, and environmental media all over the world. Hooper and McDonald suggest that the toxicity of PBDEs is likely to be most similar to that of PCBs, causing similar effects.

According to Hooper and McDonald, the weak carbon–bromine bonds of PBDEs render them more susceptible to environmental degradation than the other POPs, most of which contain highly stable carbon–chlorine bonds. Nonetheless, PBDEs are persistent enough to bioaccumulate in fatty tissues as they make their way up the food chain to humans, where they can be passed directly to nursing infants via breast milk.

Of the three major commercial mixtures of PBDEs produced—deca-BDEs, octa-BDEs, and penta-BDEs—it is deca-BDEs (which have the least potential to bioaccumulate) that account for most commercial production. However, in sunlight, deca-BDEs degrade to penta- and tetra-brominated compounds, which bioaccumulate almost as well as PCBs. It is these lesser brominated compounds, particularly the tetra- to hexa-BDEs, that have been identified in Swedish breast milk.

Hooper and McDonald note that Swedish scientists report a doubling of concentrations of PBDEs in human breast milk samples from Sweden every 5 years since the first samples were taken 25 years ago. They warn that large gaps exist in what is known about PBDE toxicity, and suggest that the increasing presence of these compounds in biological tissues could pose a threat to human health. Based on the Swedish data set for PBDEs, Hooper and McDonald suggest that breast-milk monitoring programs are a valuable source of time-trend data that can be used to identify emerging pollutants (such as PBDEs), as well as track population-level changes in the tissue concentrations of known contaminants.

Hooper and McDonald suggest that such programs provide a convenient, noninvasive way to estimate body burdens of POPs in the mother, fetus, and breast-fed child. In addition, breast-milk monitoring programs can be used to identify geographical hot spots for POPs contamination and to identify groups of at-risk individuals that can be followed. Several European countries have operated breast-milk monitoring programs for as long as 30 years. These programs helped to identify PCBs and dioxins as important contaminants in humans. Currently, there are no systematic breast-milk monitoring programs in the United States, where little is known about PBDE concentrations in breast milk or PBDE body burdens. **–Charles W. Schmidt**

Diabetes and Drinking Water

Exploring the Connection to Nitrate

Several recent studies have correlated nitrate in drinking water with the incidence of type 1 diabetes mellitus. Given a sharp increase in type 1 diabetes in the Netherlands, Jan M. S. van Maanen and colleagues attemped to clarify the possible nitrate–diabetes relationship. Their results show no convincing evidence that nitrate in drinking water at concentrations of 25 milligrams per liter (mg/L) or less is a risk factor for the disease, although a link at higher concentrations cannot be excluded [*EHP* **108:461–465**].

The human body transforms nitrate to nitrite. Nitrite may also react with amines in the digestive juices to form *N*-nitroso compounds. *N*-nitroso compounds have been shown to attack pancreatic cells in animals, causing diabetes. Studies in the United Kingdom and United States have linked nitrate in water to type 1 diabetes, while studies in Sweden and Finland have shown a dose–response relationship between type 1 diabetes and foods rich in nitrate, nitrite, and nitrosamines.

In the Netherlands the incidence of type 1 diabetes among children 0–4 years old doubled between 1990 and 1995, and the overall incidence in children aged 0–14 years increased 32% between 1980 and 1995, from 11.1 to 14.6 cases per 100,000. Nitrate concentrations in drinking water are tracked in every postal code in the Netherlands. In their ecological and epidemiological study, the authors sorted by postal code the cases of 1,064 children aged 0–14 years who had been diagnosed with type 1 diabetes between 1993 and 1995. They then compared the incidence of diabetes to nitrate exposure as indicated by the water records.

The study found a correlation between increasing age and the incidence of type 1 diabetes, but no convincing evidence of a link between nitrate exposure and diabetes. Study results do indicate a possible correlation between diabetes risk and nitrate concentrations above 25 mg/L, but the number of children exposed to these concentrations was so small that the results are not statistically significant.

Only 1% of the children in the study were exposed to nitrate concentrations above 25 mg/L, and 18% to concentrations above 10 mg/L, in contrast to a U.K. study where 33% of children were exposed to concentrations above 15 mg/L. The U.K. study linked nitrate concentrations and diabetes at a threshold of 15 mg/L, while the Dutch study did not. The Dutch study was also unable to substantiate a U.S. study that indicated a risk threshold of 10 mg/L.

The authors conclude that more studies are needed to evaluate the possible role of environmental factors in the increase of type 1 diabetes in the Netherlands and to more accurately determine safe concentrations of nitrate in drinking water. According to the authors, the present World Health Organization maximum permissible level of 50 mg/L for nitrate in drinking water may not be adequate to prevent risk of diabetes. **–Kris Freeman**