

FLAME RETARDANTS IN THE BODIES OF PACIFIC NORTHWEST RESIDENTS

A Study on Toxic Body Burdens

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N O R T H W E S T E N V I R O N M E N T W A T C H

1402 Third Avenue, Suite 500, Seattle, Washington 98101-2130

(206) 447-1880, fax (206) 447-2270

www.northwestwatch.org, newfriends@northwestwatch.org

EXECUTIVE SUMMARY

The bodies of Pacific Northwest residents are contaminated by high levels of toxic flame-retardant compounds that are known to cause behavioral aberrations, learning deficits, and other health effects in laboratory animals.

An analysis of breastmilk samples donated by 40 Pacific Northwest mothers—10 each from Montana, Oregon, Washington, and British Columbia—revealed high levels of the flame retardants in every sample tested.

These flame retardants, known as PBDEs (**p**oly**b**rominated **d**iphenyl **e**thers), are commonly added to consumer and household products such as furniture foams, textiles, and consumer electronics. Studies on laboratory animals have shown that PBDEs can impair memory and learning, alter behavior, delay sexual development, and disturb thyroid hormone levels, among other toxic effects. PBDEs are structurally similar to PCBs (**p**oly**c**hlorinated **b**iphenyls), a now-banned class of chemicals that have been linked with a host of developmental delays and other health effects in children and wildlife.

Among the study's findings:

- **Levels of flame retardants were high, compared with other parts of the globe.** The mothers in the study had levels of PBDEs ranging from 6 to 321 parts per billion (ppb), as measured in milk fat, with a median level of 50 ppb. Fifteen of the 40 women had at least 100 ppb of PBDEs in their milk. These levels are comparable to levels found elsewhere in North America, but are 20 to 40 times higher than levels found in Sweden and Japan. PBDE levels in people appear to be rising rapidly in most of the industrialized world, with levels in many countries doubling perhaps every two to five years.
- **Some breastmilk samples contained elevated levels of deca-PBDE.** Deca-PBDE (PBDE-209), the form of PBDE used most widely in commercial applications, was found in 24 of the 40 samples, at concentrations of up to 4 parts per billion measured in milk fat. This level exceeds the *total* concentration of all PBDEs—including deca-, octa- and penta-PBDEs—that is typically found in Japan or northern Europe. Deca-PBDE has not received the same intense regulatory scrutiny as other PBDE formulations in commercial use, in part because it was thought to concentrate less in people. New studies are finding that deca-PBDE may be more toxic than was previously thought, and that deca-PBDE can also break down into other forms of PBDE that are both more harmful and more readily absorbed by people.
- **Exposure is unavoidable.** The bodies of all Pacific Northwest mothers tested contained PBDEs, regardless of their diet, age, or place of residence. This suggests that there is no way to completely avoid contamination from these chemicals. PBDEs have been detected in a wide range of foods in grocery stores, so diet may be a significant source of exposure to the compounds. In

Concentrations of PBDEs in northwesterners were 20 to 40 times the levels found in Europe and Japan

addition, PBDEs are so prevalent in people's homes and workplaces that all Pacific Northwest residents may also be exposed to the compounds by handling consumer products, or by inhaling dust containing trace amounts of crumbling foam or plastic products.

- **Breastfeeding is best for mother and baby.** Despite the presence of contaminants, an extensive body of research demonstrates that breastmilk is the best food for babies, and that breastfeeding is one of the most important contributors to infant health. Infants who are not breastfed do not receive optimal nutrition, important hormones, protective immune factors, and promoters of neurologic development. Among other benefits, breastfeeding reduces infant mortality and may lead to lower rates of obesity and heart disease later in life.

Recommendations for reducing northwesterners' exposure to toxic chemicals include:

- Northwest jurisdictions should ban all forms of PBDEs from commerce, and develop strategies and recommendations for removing products that contain PBDEs from homes and workplaces;
- Northwest jurisdictions should conduct comprehensive screens of human blood and breastmilk for chemical contaminants, both as an early warning system for emerging toxic threats, and as a means to monitor progress in cleaning up existing pollution; and
- The United States and Canada should require industrial chemicals to undergo more rigorous scrutiny for health effects and the potential to collect in the bodies of humans and wildlife before the compounds are used widely in commerce.

R A P I D R I S E O F P B D E S

Epochs in human history are often named for the materials that dominated the commerce of their times. The Stone Age was humanity's first; as technology advanced, brass and iron had their ages too.

Perhaps future archaeologists will label our era the age of plastic. Over the past half century, plastics and other synthetic materials have become ubiquitous in our homes and on our store shelves. Plastics have replaced metals in cars; they have replaced wood and natural fibers in clothes, toys, furniture, and dozens of other products. And they have made possible new categories of goods, most notably the recent proliferation of consumer electronics, such as computers and cell phones.

Like petroleum, from which they are derived, some of these new synthetic materials are quick to combust, and they burn intensely when they do. So, to reduce the risks these materials pose in the event of fire, and to comply with government flammability standards, manufacturers routinely add chemical flame retardants to plastics, foams, and synthetic fabrics.

PBDEs (or polybrominated diphenyl ethers) are one such family of flame retardants. When PBDEs are exposed to heat, they release fire-suppressing bromine atoms that inhibit combustion.¹ PBDEs have proven exceedingly popular: over the past decade, manufacturers have added roughly a billion pounds of PBDEs to consumer electronics, polyurethane furniture foams, industrial textiles, and myriad other products around the globe.²⁻⁴ Tens of millions of pounds of PBDEs now reside in consumer products in the Pacific Northwest alone. And PBDEs are not simply trace additives: up to 30 percent, by weight, of some polymer-based plastics may consist of PBDEs.^{4,5}

In the late 1990s, scientists started to notice an alarming rise in concentrations of PBDEs in the environment. The findings were uniform: PBDE concentrations were increasing rapidly in the blood, fatty tissues, and breastmilk of humans, as well as in fish, wildlife, and the sediments of water bodies.⁶⁻¹⁵ In Sweden, for example, PBDE levels in human breastmilk rose roughly 60-fold between 1972 and 1997.⁶ Closer to home, PBDE concentrations rose more than 10-fold in samples of human breastmilk from Vancouver, British Columbia, between 1992 and 2002,¹⁴ and up to 12-fold in whitefish from British Columbia's portion of the Columbia River system between 1992 and 2000.¹⁵ High PBDE levels have also been detected in fish in Washington State¹⁶ and in Puget Sound orcas.^{17,18} PBDE levels in the environment and people are apparently rising exponentially, doubling every two to five years.

Although PBDE contamination has been detected in most industrialized areas of the globe, North America has by far the highest levels. PBDE concentrations in blood serum, human tissue, and breastmilk samples are 10 to 100 times higher than those found in continental Europe and Asia.¹⁹⁻²⁴ Similarly high levels have been found in North American fish, wildlife, and sewage sludges.^{5,13} Most recently, studies of 47 women in Texas²⁵ and 20 women nationwide²⁶ found that PBDEs were present in all North American mothers tested, at levels that ranged above 1,000 parts per billion—an unprecedented amount and more than 300 times higher than the maximum concentration found in recent Japanese tests.

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North American PBDE levels are high because the United States is the world's largest consumer of PBDEs, particularly those that most readily contaminate human bodies. In recent years, North and South America, led by the United States and Canada, have accounted for half of the world's total PBDE consumption and for 95 percent of the use of the "penta-PBDE" commercial mixture, which is commonly used in polyurethane furniture foams.² Penta-PBDE is the most toxic form of the flame-retardant compounds and the most likely to build up in living things.²⁷

Although scientists are unsure of how, precisely, PBDEs can enter people's bodies, there is growing evidence that the chemicals taint the North American food supply. PBDEs contaminate a wide range of foods from grocery stores, especially foods high in animal fats.²⁸ Likewise, high levels of the compounds have also been found in farmed salmon²⁹ and other fish.³⁰ But PBDEs are so prevalent in people's homes and workplaces that all northwesterners may be exposed to the compounds simply by handling consumer products, or by inhaling dust containing trace amounts of

crumbling foam or plastic products. Recent studies have found that North American house dust samples frequently contain PBDEs, though there is no clear correlation between the levels of PBDEs found in house dust and the levels found in people.³¹

T O X I C E F F E C T S O F P B D E S

At the same time that scientists were becoming aware of the remarkable rise of PBDEs in living things, other researchers were discovering that PBDEs were far more toxic than they had previously believed. Laboratory animals exposed to PBDEs displayed deficits in learning and memory, as well as subtle alterations to their behavior.

Mice given a single dose of PBDEs during a critical period of brain development in infancy did worse on maze tests than did unexposed mice: they could learn a maze once but had difficulty learning a new setup once the maze was changed. And PBDE exposure also changed the behavior of mice when they were placed in new surroundings, making them less active at first but also less likely to settle down and acclimate to new environs. Scientists concluded that PBDE exposure during a critical early period of rapid brain growth in infant mice could cause permanent neurological deficits and behavioral aberrations—effects that worsened as the mice matured.^{32,33}

Other studies have linked PBDEs with effects on hormone function, particularly thyroid hormones. Researchers have found that commercial PBDE products can disrupt thyroid hormone balance in laboratory animals.³⁴ Furthermore, as the liver metabolizes PBDEs, it may turn them into compounds that are particularly likely to interfere with the thyroid system.³⁵ Thyroid hormones are critical to normal brain functioning, and thyroid imbalances in pregnant mothers and fetuses have been linked with IQ deficits in children.²⁷

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PBDEs also affect sexual development by delaying the onset of puberty in both male and female rats and by reducing prostate weights among male rats, among other effects.²⁷ The compounds also have effects on other nervous-system functions,³⁶ including major alterations in the functions of certain neurotransmitters.³⁷

Many of these effects might have been expected, given the structural similarity between PBDEs and another class of compounds known as PCBs (polychlorinated biphenyls). PCBs are oily liquids that were used for decades as insulators and fire retardants in electrical transformers and other industrial applications. The chemical stability that made PCBs excel as industrial fire retardants also made them long-lived in the environment: though banned in the late 1970s, PCBs continue to contaminate marine sediments and build up to toxic levels in fish, marine mammals such as harbor seals and orcas, and people. PCB exposure has been linked with a host of developmental delays and deficits in children.³⁸ The longest-term study of PCB effects on human development showed that developmental and IQ deficits caused by *in utero* exposure to PCBs became worse as children got older and persisted at least through age 11.³⁹

Findings of chemical contaminants in breastmilk samples should not dissuade mothers from breastfeeding. As noted by the US Surgeon General, breastmilk is one of the most important contributors to infant health.⁴¹ Breastfeeding has been shown to reduce the risk of infant mortality,⁴² and breastfed babies may be less likely than bottle-fed infants to develop obesity⁴³ and heart disease^{44,45} later in life.

Despite concerns about chemical contaminants in breastmilk, research suggests that forgoing breastfeeding carries significant risks. For example, low rates and short duration of breastfeeding are associated with a significantly increased likelihood of acute illnesses such as infections of an infant's bloodstream, ears, lungs, urinary system, and gut.^{41,46} In addition, exposure to foods other than human milk in the first few months of life can increase the risk of lifelong autoimmune illnesses such as diabetes⁴⁶⁻⁵¹ and some inflammatory bowel diseases.⁵²⁻⁵⁵ Infants who are not breastfed do not receive optimal nutrition, important hormones, protective immune factors, and promoters of neurologic development. And breastfeeding reduces the instance of anemia and some gynecologic cancers in women, including premenopausal breast cancer.^{41,75}

Formula feeding does not eliminate children's exposure to toxics. Children are exposed to such chemicals regardless of whether they breastfeed, through food and the household environment. And perhaps most significantly, the developing fetus—which is particularly susceptible to the effects of toxics—is exposed to contaminants that cross the placenta.

Indeed, many researchers believe that the greatest concerns about toxic contamination stem from exposures in the womb rather than from exposures through breastfeeding. Chemical exposures before birth have adverse health effects, but common exposures through breastfeeding have not been shown to cause harm. This may be because infants are less vulnerable to the effects of these chemicals than are fetuses, or because breastmilk's beneficial effects on the immune system and brain development protect the infant from harm.⁵⁶

All studies demonstrating the benefits of breastfeeding have been performed on mothers whose milk contained at least some contaminants: For decades, all mothers in the industrialized world who have been tested contained detectable levels of synthetic toxics in their bodies. After toxic chemicals such as DDT and PCBs were banned, levels in breastmilk fell, in some cases substantially, lessening some concerns over contaminant levels.⁵⁷

PCBs and PBDEs are structurally related: both consist of two six-carbon rings that are bonded together, and both are studded with atoms of the halogen family (bromine for PBDEs and chlorine for PCBs). PCBs and PBDEs have similar effects on the nervous and thyroid systems,³³ and recent findings suggest that PCBs and PBDEs may act in concert, affecting some of the same hormone systems in much the same ways.^{37,40} Most troubling, the two pollutants combined may be more potent than either one in isolation: simultaneous exposure to PBDEs and PCBs affected

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behavior in mice far more strongly than did exposure to just one class of compound at a time.⁴⁰ The fact that PCBs and PBDEs should both affect the thyroid system should come as little surprise, given their chemical similarity to thyroid hormones such as thyroxine (see Appendix C).

MONITORING PBDE LEVELS IN PACIFIC NORTHWEST RESIDENTS

Monitoring human milk, particularly if samples are taken soon after birth, provides a useful indicator for exposure levels in early fetal development, the period when humans are most susceptible to toxic effects. After adjusting for differing fat content, PBDE levels are comparable in breastmilk, mother's blood serum, and fetal blood serum.^{21,24} Breastmilk offers other advantages for testing PBDE levels in people. Unlike blood or tissue samples, breastmilk can be collected inexpensively and without invasive medical procedures. It is also high in fat, and PBDEs collect in fat—which makes it possible to run comprehensive tests with small amounts of milk. Breastmilk tests may even be a reliable proxy for PBDE levels from environmental exposures in males of a similar age.⁵⁸

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To help expand understanding of pollutant levels in the bodies of northwesterners—and particularly the contamination levels faced during *in utero* development—Northwest Environment Watch (NEW) undertook a study of breastmilk samples donated by 40 mothers from throughout the Pacific Northwest, 10 each from Washington, Oregon, British Columbia, and Montana. Each of these samples was tested for a broad range of bioaccumulative pollutants, including PBDEs, PCBs, dioxins, and furans. This report presents findings from an analysis of PBDE levels in the 40 samples. This pollutant monitoring project is part of a larger effort by NEW called the Cascadia Scorecard, an index of seven key trends shaping the future of the Northwest.

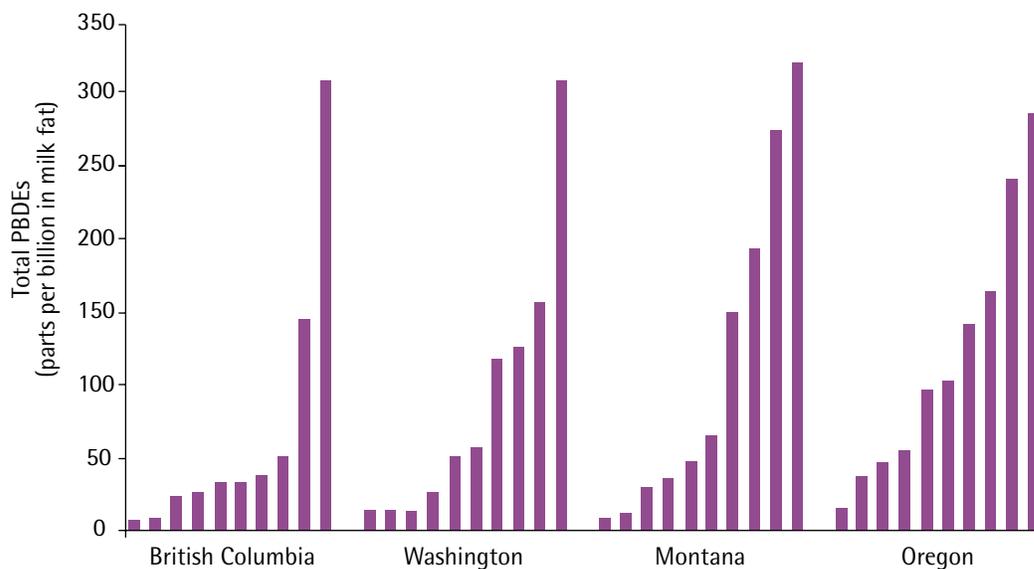
From April through November 2003, NEW and its partner organization, Women's Voices for the Earth in Montana, recruited 40 first-time, breastfeeding mothers from the Pacific Northwest to donate milk samples for chemical analysis. All mothers were healthy and had healthy infants between the ages of two and eight weeks. Mothers were recruited by newspaper advertisements and word of mouth; their demographic characteristics were not necessarily representative of the general population.

Participating mothers hand-expressed two half-cup samples of breastmilk into chemically clean bottles, which were frozen and shipped to the California Environmental Protection Agency's Hazardous Materials Laboratory. The samples were analyzed, using high-resolution gas chromatography/high-resolution mass spectroscopy, for the presence of 12 different congeners, or types, of PBDEs. These 12 include each of the major congeners present in the three PBDE commercial products that are sold in the United States. (See Appendix B for a more complete description of the analytical methods used.)

RESULTS: PBDE LEVELS 20 TO 40 TIMES HIGHER THAN LEVELS IN JAPAN AND SWEDEN

All 40 of the women had PBDEs in their breastmilk, with levels ranging from 6 to 321 parts per billion (expressed as the mass of PBDE per mass of milk fat). The median, or midpoint, concentration of PBDEs was 50 parts per billion. Fifteen of the 40 women had at least 100 parts per billion of PBDEs in their milk (see Figure 1).

Figure 1. All 40 Northwest mothers had PBDEs in their breastmilk, with levels ranging from 6 to 321 parts per billion as measured in milk fat.



Several other studies have shown a rapid rise of PBDE contamination in North Americans.^{13,19-21} Median PBDE levels in North America appear to be 20 to 30 times higher today than they were in the late 1980s (see Figure 2).

The median PBDE levels among Pacific Northwest mothers were roughly on a par with levels detected in two other recent studies in North America. One study of 47 breastmilk samples from Texas women in 2002²⁵ found a median level of 34 parts per billion, somewhat lower than the median of 50 parts per billion found in this study. However, slightly lower numbers might have been expected because some breastmilk donors in the Texas study had been nursing for longer periods of time, and some may not have been first-time mothers, factors that can reduce levels of fat-soluble contaminants in a mother's body.⁵⁹

More directly comparable to this study was an analysis of 20 samples that the Environmental Working Group (EWG) collected from women across the United States. The EWG study collected breastmilk samples in 2002 and early 2003, finding a median PBDE concentration of 58 parts per billion. As with the Pacific Northwest mothers, all mothers in the EWG study were first-time, breastfeeding mothers, with infants between two and eight weeks of age.²⁶

Figure 2. As demonstrated by recent studies, PBDE levels in human tissue and body fluid samples in North America are 20 to 30 times higher today than they were in the late 1980s.

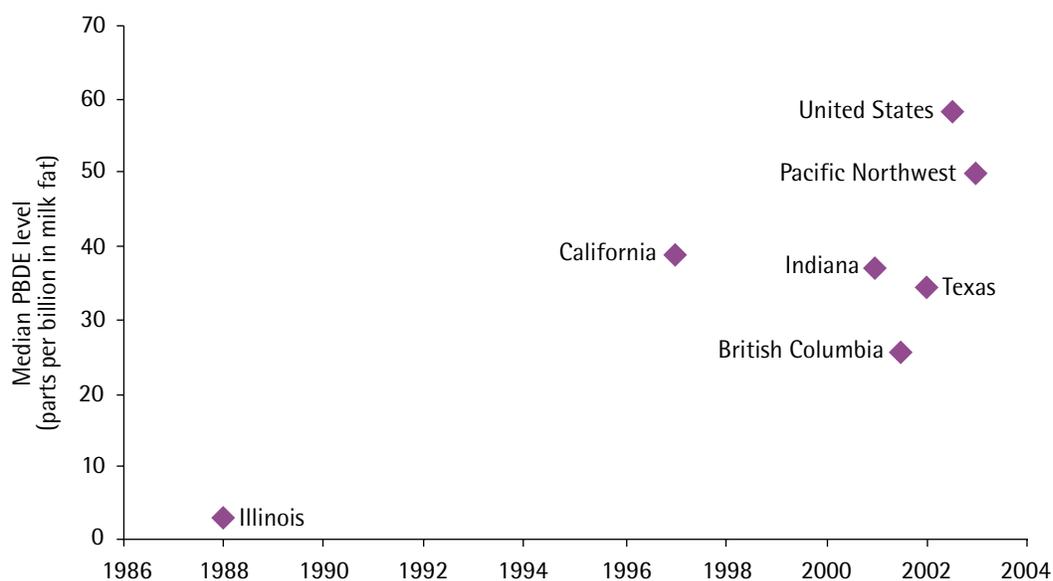


Figure sources: See notes ^{13,20,21,25,26}

Contamination levels varied widely among individuals: at least one sample in each of the four locations contained less than 15 parts per billion PBDEs, and at least one sample in each place contained more than 250 parts per billion (see Table 1). Samples from the British Columbia donors generally contained the lowest levels, with a median of 32 parts per billion, while Oregon donors had the highest levels, with a median of 99 parts per billion. Although the regional differences are not statistically significant, at least one other recent study also found slightly lower levels of PBDEs in BC breastmilk samples than are typically found in the United States.¹⁴

Table 1. Regional results for Pacific Northwest study

PBDE CONTAMINATION (PARTS PER BILLION IN MILK FAT)				
Location	Lowest	Median	Mean	Highest
British Columbia	6.3	32	67	308
Washington	13	53	88	309
Montana	8.7	55	113	321
Oregon	14	99	118	285
All Participants	6.3	50	97	321

While northwesterners' PBDE levels are on a par with the rest of North America, they are much higher than levels found in other parts of the world: 20 times the concentrations found in Sweden²⁴ and more than 40 times those detected in human serum and breastmilk from Japan²³ (see Figure 3). In fact, the lowest contamination level found among the 40 Pacific Northwest mothers (6 parts per billion) was greater than the highest level found in Japan (3 parts per billion).

Figure 3. Levels of PBDEs in the Pacific Northwest are 20 to 40 times higher than levels in Japan and Sweden.

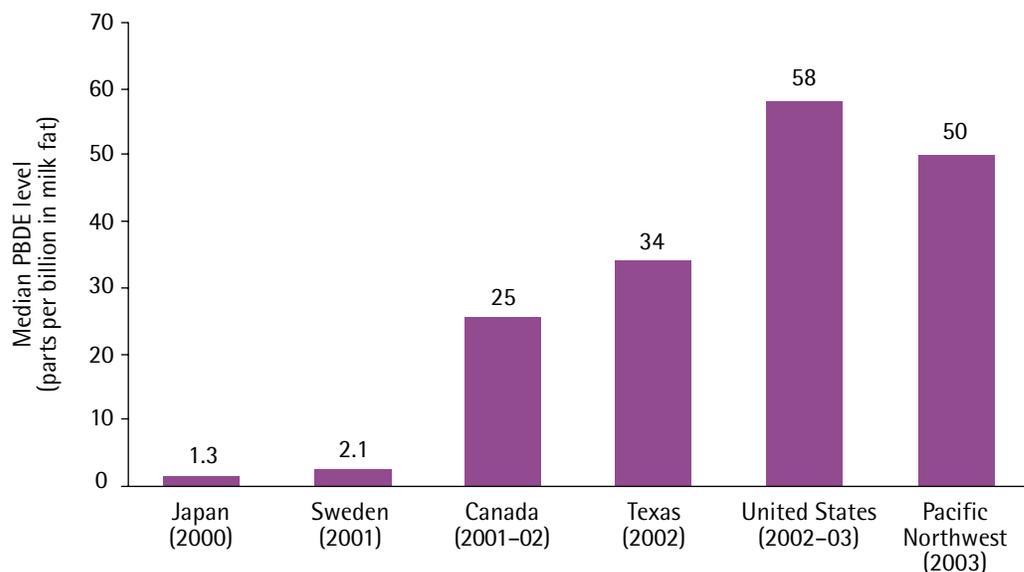


Figure sources: See notes ^{14, 23-26}

Each breastmilk sample in this study was tested for 12 different congeners, or types, of PBDEs (see Appendix A for more complete details), including the major congeners present in the three commercial PBDE products that have been sold in North America. For all mothers in the study, three PBDE congeners—PBDE-47, PBDE-99, and PBDE-100—together accounted for at least three-quarters of the total PBDEs detected.

These three congeners are constituents of the commercial penta-PBDE mixture used most commonly in polyurethane foams. And two of these congeners—PBDE-47 and, in particular, PBDE-99—have been associated with toxic effects in laboratory animals.^{32,33,37,40} In one study, mice found to have as little as 40 parts per billion PBDE-99 in their brain fat during the most critical phase of brain growth developed permanent behavior aberrations.³³ Similarly, PBDE-99 can reduce fertility in male rats⁶⁰ and alter ovary cell structure in female rats⁶¹ at levels of approximately 230 parts per billion—a level comparable to the levels of total PBDEs found among the most exposed northwesterners.⁶²

DECA - PBDE : CONTAMINATION IS WIDESPREAD

Deca-PBDE (PBDE-209) is the form of PBDE used most widely in commercial applications, but it has not received the same intense regulatory scrutiny as penta- and octa-PBDEs, which are being phased out in Europe and removed from production in the United States. Generally, deca-PBDE is found in lower concentrations in people than are the penta- and octa-PBDE formulations.

However, the deca congener was clearly detected in 24 of the 40 samples, at levels as high as 4 parts per billion, a level higher than the total amount of *all* PBDEs—penta-, octa- and deca-PBDE—detected in human serum in a recent Japanese study.²³ The highest levels in the Northwest Environment Watch study were comparable to the levels that have been found in Swedish electronics workers who were occupationally exposed to deca-PBDE in the workplace.⁶³ Levels of the deca congener detected in this study were not correlated with total PBDE levels. This suggests that deca-PBDE may enter people's bodies in different ways, or from different sources, than penta- and octa-PBDEs.

Though deca-PBDE was long believed to be only minimally toxic, there is growing evidence that it can be absorbed into the body, and that it can cause behavioral aberrations in mice that are similar to those caused by penta-PBDE.^{64,65} Furthermore, deca-PBDE can lose bromine atoms when it is released into the environment. Both fish and rats have been found to break down deca-PBDE into lower-brominated PBDE congeners that are more toxic and more easily absorbed by living things.^{66,67} Similarly, deca-PBDE can form more toxic congeners when exposed to ultraviolet light.⁶⁴ Indeed, it is possible that some of the lower-brominated PBDE congeners detected in this study originated as deca-PBDE.

FUTURE DIRECTION OF PBDE POLICY

The evidence from the past several decades is clear: when governments and communities take firm action against toxic chemicals, levels of those chemicals in people decline.

Lead, a potent neurotoxin that can impair intelligence and development, was once a major additive in gasoline and paints—and, as a consequence, was found at high levels in many children. After lead was phased out of such products in the 1970s and 1980s, levels in children declined. Now, despite some hotspots of lead contamination, the average lead levels in US children are well below the levels of previous decades.⁶⁸

Similarly, PCB contamination fell once safeguards were enacted and enforced. After PCBs were removed from commerce, levels declined both in people and in environmental media such as Puget Sound's sediments.⁶⁹ Regulatory actions at many levels of government—often sparked by citizen action—were clearly effective in reducing the threat posed by these compounds.

More recently, the discovery that PBDEs are toxic in animals, and were rapidly increasing in concentration in human bodies, set off regulatory alarm bells through much of Europe, Asia, and North America. Sweden, where the worrisome rise of PBDEs in breastmilk was first detected, was among the earliest to initiate a phase-out of PBDEs. Swedish manufacturers, including Ikea and Volvo, quickly found viable alternatives to the compounds. After PBDEs were removed from the Swedish marketplace, contamination levels in Swedish breastmilk began to decline.⁷⁰

Other governments have followed Sweden's lead. The European Union will phase out penta- and octa-PBDEs by the end of this year, and remove deca-PBDE from consumer electronics by 2006.⁷¹ The US Environmental Protection Agency last year announced an agreement with Great Lakes Chemical, the sole manufacturer of the penta- and octa-PBDE mixtures, to voluntarily withdraw these compounds from the US market by the end of 2004. Just so, the legislatures of California, Maine, Hawaii, and New York have also passed laws to ban some forms of PBDEs. And last month the Washington State Department of Ecology released a draft plan to ban the use of penta- and octa-PBDEs, as well as to eventually ban deca-PBDE in consumer electronics.

These chemical phase-outs, while welcome, leave two problems unaddressed. First, many of the phase-outs allow deca-PBDE to remain on the market for at least some applications. Deca-PBDE is regularly detected in breastmilk samples from North American mothers, and it may lose bromines to produce more toxic forms of PBDEs, both in living things and in the environment.^{66,72}

Second, these bans do nothing to remove the many millions of pounds of PBDEs in furniture, fabrics, computers, and consumer electronics already in people's homes and workplaces. These products may serve as reservoirs for continued contamination for years or even decades to come.⁷³ At present, there are no clear recommendations for what people should do with household and office products that contain PBDEs. Nobody is sure whether removing products such as polyurethane foam padding from underneath carpets will protect homeowners from potential PBDE exposures, or increase potential exposure from handling and breathing crumbling foam.

Despite the shortcomings of the safeguards that have been put into effect, it is encouraging that many lawmakers and government agencies have acted to ban PBDEs once the threats were understood. However, the fact that such steps were necessary should give pause, since it highlights a systemic flaw in how our society treats industrial compounds: we are reactive rather than precautionary, waiting for problems to emerge, for the evidence to become incontrovertible, and for public concerns to build, before we take action on even as obvious a threat as PBDEs.

This problem is not new. Rarely have industrial chemicals been scrutinized before being put on the market. During the 1980s the National Academy of Sciences' National Research Council found that 78 percent of the highest-volume chemicals in commercial use in the United States had not even been subjected to minimal toxicity testing. More than a decade later, no significant improvement was

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seen, and the most basic toxicity testing results could not be found in the public record for nearly 75 percent of high-volume chemicals.⁷⁴

Even now, procedures for introducing new chemicals into commerce provide relatively few safeguards against harmful chemicals. For example, the US Toxic Substances Control Act (TSCA), passed in 1976, established a framework for testing both new and existing chemicals. But the law does not require comprehensive testing for all chemicals, and the US EPA has not been aggressive in exercising the authorities granted to it under that act.

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A precautionary approach would mandate health and safety tests *before* new chemicals are allowed in commerce, and also would require existing chemicals to undergo such tests to remain on the market. The European Union is taking such an approach with a recently enacted program dubbed REACH (for Registration, Evaluation, and Authorization of Chemicals). REACH would transfer the burden of proof for ensuring the safety of industrial chemicals from the public to the manufacturers, and help prevent toxic problems before they arise. Had the REACH approach been in place three decades ago, North Americans would not have such alarming levels of PBDEs in their bodies today.

In hindsight, the risks of PBDEs should have been obvious, as their chemical structure is similar to that of PCBs and some human hormones. But such chemical similarity did not trigger any requirements to take even the most elementary precautions before PBDEs were used in commerce. The result was a cruel irony, and an unnecessary mistake: chemicals that were intended to reduce the risk of fire actually created new and unnecessary risks to human health by sequestering potentially hazardous compounds throughout North American homes and offices.

CONCLUSION AND RECOMMENDATIONS

Breastmilk is not merely a food. It is a symbol of our hope that we can give our children the best possible start in life. And we rightly hold the purity of breastmilk sacrosanct.

But the truth is that mother's milk, like our bodies and the environment that surrounds us, is not pure. As a consequence, we pass on to our children a legacy of our toxic mistakes. Finding contaminants in our bodies and our breastmilk should renew our determination to eliminate the sources of the toxics that now taint us all.

NEW recommends the following:

- **Phase out all PBDEs.** Northwestern jurisdictions, as well as the federal governments of the United States and Canada, should ban PBDEs from commerce. Washington State's proposed ban of penta- and octa-PBDEs, and its phase-out of deca-PBDE in consumer electronics, should be adopted. And these bans should be extended to all other uses of deca-PBDE.

- **Address the problem of PBDEs in people’s homes and offices.** Provincial and state governments throughout the Pacific Northwest should develop strategies and recommendations for helping people remove PBDEs from their homes and workplaces. Citizens concerned about the PBDEs contained in products in their homes should have clear information about the best ways to dispose of those products to minimize the risks to themselves and to the environment.
- **Test people for chemical contamination.** Widespread biomonitoring of human blood and breastmilk for PBDEs and other contaminants would serve as an early warning system to catch emerging toxic problems before they reach epidemic proportions. It would also serve as a gauge of our success in cleaning up old sources of pollution.
- **Formalize the precautionary principle.** The United States and Canada should follow Europe’s lead in adopting safeguards that require new chemicals to be tested for toxicity and the ability to bioaccumulate before they are used widely in commerce, and in requiring testing of older chemicals in order for them to remain on the market.

As noted earlier, evidence suggests that exposure to toxic chemicals is not a choice, and the most effective action is at the societal level. However, parents and consumers concerned about the risks from persistent toxic chemicals may be able to reduce the risks posed by these compounds. NEW recommends:

- **Breastfeeding your child.** Breastfeeding offers significant health benefits to both mother and infant. In addition, breastmilk contains beneficial compounds such as omega-3 fatty acids that are not found in infant formula and that support optimal infant development.
- **Avoiding degraded or crumbling furniture foam** that may contain PBDE fire retardants. Replace or cover couches, stuffed chairs, and automobile seats that have exposed foam.
- **Exercising caution** when removing and replacing the foam padding, which may contain PBDEs, beneath your carpet. Clean up carefully when you are finished. Women of childbearing age should consider not doing this work themselves.
- **Reducing consumption of animal fat.** Like many other persistent pollutants, PBDEs tend to collect in the fats of animals, and the compounds have been detected at particularly high levels in meat and dairy products in US grocery stores. Consider reducing your consumption of foods high in animal fat, and preparing fish and meat by broiling and draining juices, which can reduce levels of contaminants.

**APPENDIX A . NORTHWEST ENVIRONMENT WATCH
STUDY RESULTS BY CONGENER**

PBDE PARTS PER BILLION IN MILK FAT					
PBDE class	PBDE type	Lowest	Median	Mean	Highest
Tri-PBDEs	PBDE-32	0.003 (MDL)	0.1	0.2	1.3
	PBDE-28/32	0.2	1.7	3.8	20
Tetra-PBDEs	PBDE-47	2.6	26	50	201
	PBDE-66	0.01 (MDL)	0.1	0.3	2.8
	PBDE-71	0.01 (MDL)	0.2	0.5	3.4
Penta-PBDEs	PBDE-85	0.004 (MDL)	0.6	1.1	4.7
	PBDE-99	0.8	5.4	10	49
	PBDE-100	0.5	5.2	12	76
Hexa-PBDEs	PBDE-153	0.8 (MDL)	4.8	16	169
	PBDE-154	0.03 (MDL)	0.4	0.8	3.9
Hepta-PBDE	PBDE-183	0.006 (MDL)	0.2	0.3	1.6
Deca-PBDE	PBDE-209	0.05 (MDL)	0.4	0.8	4.3
Sum of all PBDEs tested		6.3	50	97	321

MDL=less than or equal to method detection limit

APPENDIX B: METHODOLOGY

Breastmilk samples were hand-expressed and collected from 40 healthy first-time mothers whose healthy infants were between 2 and 8 weeks of age. Participants were current residents of the Pacific Northwest, with 10 each from the greater metropolitan areas of Missoula, Montana; Portland, Oregon; Seattle, Washington; and Vancouver, British Columbia. Participants represented a convenience sample; no attempt was made to obtain a sample population statistically representative of the population at large, nor of any population subgroup. Samples were collected in two chemically clean glass jars (about 200 mL total) and refrigerated. Samples were frozen prior to shipment to the Hazardous Materials Laboratory of the California Environmental Protection Agency, Berkeley, California, which conducted the chemical analysis.

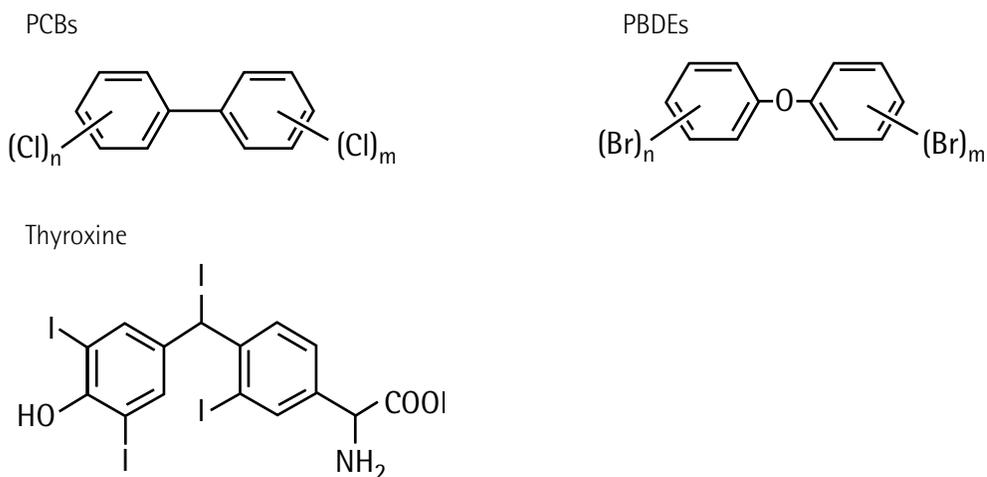
In the laboratory, frozen samples were lyophilized until a stable weight was reached, after approximately 72 hours, and the moisture content determined. Approximately 3.5 g of dried milk samples were spiked with ^{13}C -PBDE recovery standards, and extracted with hexane:methylene chloride:methanol (5:2:1) at an extraction pressure of 1500 psi. Sample extracts were collected in amber glass vials, and aliquots were taken for gravimetric fat determinations. Extracts were reduced to <1 mL by evaporation. Sample cleanup used a mixed silica gel column followed by gel permeation chromatography to remove residual fat.

Target analytes were identified and measured using a high-resolution gas chromatography column/high resolution mass spectroscopy operating in electron impact ionization–selective ion monitoring mode. Analytical results were reported for each of 12 PBDE congeners.

More detailed descriptions of the analytical methods have been published in the scientific literature.^{13,19}

APPENDIX C. CHEMICAL STRUCTURES

Figure 4. PCBs, PBDEs, and Thyroxine (a thyroid hormone) share similar chemical structures.



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ABOUT NORTHWEST ENVIRONMENT WATCH

Northwest Environment Watch (NEW) is a Seattle-based independent nonprofit research and communication center that promotes an environmentally sound economy and way of life in the Pacific Northwest. The Pacific Northwest is a bioregion that includes Washington, Oregon, Idaho, British Columbia, and adjoining parts of Alaska, Montana, and California. NEW's research program focuses on two critical efforts: to monitor the Northwest's progress toward sustainability and to identify the most important reforms for the region to implement.

As part of these efforts, NEW has developed the Cascadia Scorecard, a new gauge of regional progress that monitors seven key trends shaping the future of the Northwest: health, economy, population, energy, sprawl, forests, and pollution. In its regular updates, the Scorecard assesses the Northwest in each area and points to real-world goals for each trend. For more information about NEW and NEW's publications, please see www.northwestwatch.org.

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