Brominated Flame Retardants

A Report to the Joint Standing Committee on Natural Resources, 122nd Maine Legislature

Prepared by:

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March 4, 2005

Senator Scott W. Cowger, Chair
Representative Theodore S. Koffman, Chair
Committee on Natural Resources
122nd Maine State Legislature
Cross Office Building, Room 214
Augusta Maine 04333

Dear Senator Cowger and Representative Koffman:

Enclosed is a report on brominated flame retardants, as required under *An Act to Reduce the Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products*, PL 2003, c. 629.

Brominated flame retardants (BFRs) are added to plastics and foams or applied to textiles to prevent them from catching fire and slow the rate at which they burn. The enclosed report reviews information related to a particular class of BFRs—polybrominated diphenyl ethers or PBDEs. PBDEs have drawn regulatory attention because levels in the environment and humans have increased exponentially since these chemicals were placed in commerce.

There are three commercial PBDE mixtures called penta, octa and deca.. Maine has joined the European Union and several other U.S. states in banning the sale of products containing the penta and octa mixtures, and the chemical industry already has withdrawn them from the marketplace. This report focuses on decaBDE, the only PBDE that currently remains in production.

The report was co-written by staff from the DHHS Bureau of Health and the DEP Bureau of Remediation & Waste Management. I trust you will find the information useful as you weigh the need for regulatory action related to decaBDE.

Please feel free to call me at 287- 2812 if I can be of further assistance.

Sincerely,

Dawn

Dawn R. Gallagher
Commissioner

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I. Executive summary

Polybrominated diphenyl ethers or PBDEs are among more than 175 chemicals that are added or applied to plastics and other petroleum-based materials to increase their resistance to fire. Of all the flame retardant chemicals currently in production, PBDEs in particular have come to the attention of policymakers because levels in the environment, wildlife and human tissue increased exponentially around the world after these chemicals came into wide use during the 1970s.

PBDE levels in human tissue are much higher in the U.S. than in Europe where use of these chemicals has been curtailed. In light of this, and combined with evidence of negative health effects if PBDE levels continue to rise, Maine and four other U.S. states have banned the sale of products containing the "penta" and "octa" PBDE mixtures. Although these bans do not take effect until 2006, the only U.S. manufacturer of these two products already has discontinued further production.

This report reviews information related to decaBDE, the only commercial PBDE mixture still in production. Manufacturers of decaBDE maintain that it has been rigorously tested and found to be safe from both an environmental and human health perspective. Others, including many scientists, have identified data gaps and raised concerns suggesting the safety of decaBDE remains an open question.

To date, the European Union is the only jurisdiction to enact a ban on the use of decaBDE. Its ban on the sale of electronics containing decaBDE is scheduled to take effect July 1, 2006, although the European Commission is expected to reconsider the ban upon the release of an updated risk assessment of decaBDE in early 2005.

In the U.S., the Maine Legislature has expressed its intent to ban the sale of products containing decaBDE beginning January 1, 2008, if a safer, nationally-available alternative is identified. All other states that have considered the matter have called for further study.

Our review of the available data on decaBDE suggests the following:

- DecaBDE is ubiquitous in the environment. It is present in wildlife, sewage sludge, indoor air, house dust, food and human tissue.

- DecaBDE is bioaccumulated and concentrates up the food chain.

- PBDEs are transported globally, appearing in the Arctic in sediments, fish, birds and mammals. DecaBDE is transported on particles in contrast to lighter PBDE molecules, such as those in the pentaBDE mixture, which are transported in the gaseous phase.

- DecaBDE levels in the U.S. are much higher than in Europe where levels appear to have decreased relatively quickly in response to reduced use of PBDE-containing products.

- DecaBDE, like other PBDEs tested, has been reported to produce neurotoxicity when administered to developing rodents. Current data suggest its potency is about the same as other PBDEs that have been tested for toxicity. Additional studies are planned to confirm these findings.

- PBDE levels in humans have not yet reached levels shown to be toxic in rodents, but alarm over the rapid buildup of these levels has led governments and industry to take the precautionary step of ending the use of pentaBDE and octaBDE in new products. The extent to which decaBDE is contributing to current PBDE body burdens is unclear because of incomplete information on the propensity of decaBDE to degrade into lower-brominated PBDEs including penta.

- DecaBDE breaks down in sunlight and is metabolized by living organisms. The breakdown products are not necessarily identified and therefore not included in studies of PBDEs, suggesting that the contribution of decaBDE to PBDE levels in the environment and humans may be underestimated.

- Effective alternatives for achieving flame retardancy appear to be available for all current decaBDE applications. The alternatives generally cost more but do not appear to share the health and environmental safety concerns associated with deca.

- Many manufacturers of consumer electronics already have halted the use of decaBDE in their products or have announced plans to phase out the use of deca. Most of the decaBDE currently produced is used in the outer casings of consumer electronics.

It remains to be seen if the withdrawal of pentaBDE and octaBDE from the marketplace will be enough to reverse the upward trend in PBDE body burdens. To date, the case for banning decaBDE has not been as compelling based on limited study. However, much more information on the fate of this chemical in the environment and its possible health effects is expected to be available well before January 1, 2008, the date by which the Maine Legislature intends to take measures to restrict the sale of products containing decaBDE.

II. Introduction

A. About this report

This report on brominated flame retardants (BFRs) is required under *An Act to Reduce Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products* (the Act).[1]

The full text of the Act appears in Appendix A. Section 1 prohibits the sale of products containing more than 1% of the "penta" or "octa" PBDE mixtures beginning January 1, 2006. Section 2 expresses the Legislature's intent to prohibit the sale of products that contain more than 1% of the "deca" mixture of PBDEs beginning January 1, 2008, if a safer alternative is identified.

To assist the Legislature in determining if restrictions should be placed on other BFRs, the Act requires the Department of Environmental Protection (DEP), with the Department of Human Services, Bureau of Health (BOH), to review relevant information and report annually to the Legislature's Committee on Natural Resources, beginning January 5, 2005.

The reporting requirement extends to all BFRs, defined in the Act to mean "any chemical containing the element bromine that may be added to a plastic, foam or textile to inhibit flame formation." This initial report focuses on emerging information about decaBDE because the main issue before the Legislature is whether to follow through on its express intent to restrict the sale of deca-laden products beginning in 2008.

The information presented here is not a complete survey of all available data on decaBDE. Rather, the document summarizes and cites research studies, risk assessments and other information that, in the judgment of the DEP and BOH, may be useful in the Legislature's deliberations.

B. BFR overview

BFRs and other flame retardants are added to plastics and polyurethane foam to reduce the likelihood that products made of these materials will ignite. Without the addition of flame retardants, these petroleum-based materials could act as an accelerant, causing a fire to spread rapidly. According to the Swedish Rescue Services Agency, the average time from ignition of a structure fire to flashover (when a room bursts into flame) was 15 minutes in 1950. By 1975, that time was down to five minutes, and now fatal conditions can occur after three minutes, all because of the increase of plastics in our homes (Albinson, 2002).

The addition of flame retardants to plastics delays ignition and slows the burn rate, thereby giving building occupants more time to escape a fire and increasing the chances that the fire can be suppressed successfully.

The use of flame retardants is not generally mandated in the U.S. BFR manufacturers state that:

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[1] See PL 2003, c. 629, enacting 38 MRSA § 1609.

> "The U.S. has NO requirement mandating the use of flame retardants, and in fact, lacks any regulation at all with respect to flammability in key areas, such as residential furniture and consumer electronics. Residential furniture, except in California, relies on voluntary measures of the furniture manufacturers. Most consumer electronics, e.g. TVs, computers, stereos, etc., rely on voluntary compliance with UL standards. UL has no statutory mandate."[2]

Nevertheless, flame retardants are widely used in this country, a circumstance apparently driven by the marketplace, which gives strong preference to consumer products that comply with fire safety standards such as those established by Underwriters Laboratory. According to the UL website, "[m]illions of products and their components are tested to UL's rigorous safety standards with the result that consumers live in a safer environment than they would have otherwise."[3]

PBDEs

The class of BFRs known as PBDEs has captured the attention of policymakers because the levels in the environment and humans have increased rapidly since these chemicals came into wide use. "Study after study shows that…PBDEs are traveling throughout the globe instead of staying insider the consumer products they are meant to protect" (Betts, 2004).

The penta and octa PBDE mixtures have garnered the most concern because they appear to be the major contributors to current human body levels. The highest levels of PBDEs in human tissues are found in Canada and the U.S., which is the largest producer and consumer of PBDE products. Levels in Americans are 10 to 70 times higher than levels reported for Europe and Japan.[4] Studies suggest that current levels of human exposure to these chemicals may place some members of the U.S. population at risk of adverse health effects. "The current margin of safety appears low for many individuals" (McDonald, 2004).

PBDEs are manufactured chemicals that do not occur in nature. They are very similar structurally to polychlorinated biphenyls (PCBs), chemicals known to adversely affect the nervous, immune and endocrine systems, and influence metabolism of natural and foreign chemicals in the body. Because bromine atoms in general are more easily lost from the molecule (more "reactive") than chlorine atoms, PBDEs would be expected to undergo various types of degradation and metabolism more readily than PCBs.

PBDEs are comprised of two phenol rings linked by an oxygen (thus the designation as an "ether") (Figure 1). The rings may have a variable number of bromine atoms, from 1-10, in all possible combinations.

Bromine, along with chlorine, fluorine and iodine, are elements in the chemical group known as

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[2] Bromine Science and Environmental Forum, "Flame Retardants Manufacturers Comments of November 11, 2004 on the Draft Washington State PBDE Chemical Action Plan" (October 11, 2004), p. 7.

[3] Underwriters Laboratories Inc., "UL Standards for Safety, http://www.ul.com/info/standard.htm

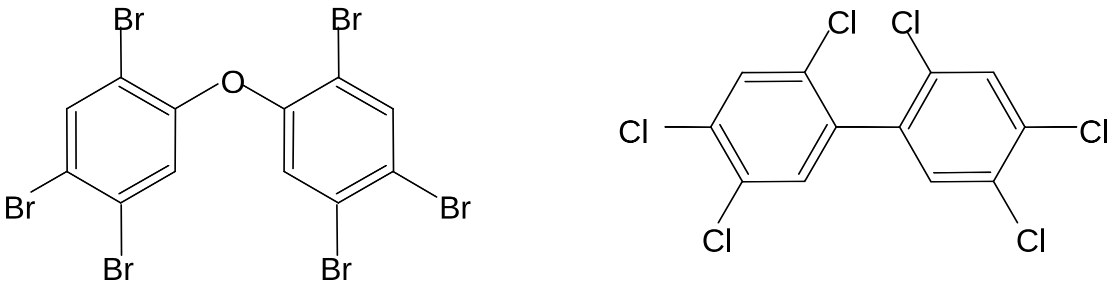
[4] See page 22.

halogens. Of the halogens, only bromine and chlorine are effective as flame retardants. Chlorinated chemicals, however, must be used in higher quantities than brominated chemicals (BFRs) to achieve comparable flame retardancy. The fact that smaller amounts of BFRs are needed means they are less apt to affect the desired properties of the plastics to which they are added. Thus, BFRs often have been the flame retardant of choice when both effectiveness and cost are considered.[5]

Figure 1. Structure of PBDE and PCB

generalized structure of PBDE
There can be up to 10 bromine atoms

generalized structure of PCB
There can be up to 10 chlorine atoms



2,2',4,4',5,5'-hexabromodiphenylether
(PBDE-153)

2,2',4,4',5,5'-hexachlorobiphenyl
(PCB-153)

In light of the concerns about rising PBDE levels, especially in breast milk, the only manufacturer of pentaBDE and octaBDE agreed to withdraw these products from the market at the end of 2004. The other commercial PDBE mixture, decaBDE, appears to be less toxic in its original state and remains in production. The Bromine Science and Environment Foundation (BSEF) maintains that the deca mixture poses no threat to human health, and that the fire safety benefits warrant its continued production and use.

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[5] Bromine Science and Environmental Forum, "What Are Brominated Flame Retardants? What Are They Used For?", www.bsef-site.com

C. DecaBDE production and use

Over 56,000 tons of decaBDE were produced in 2001.[6] This accounted for over 80% of global PBDE production that year. After 2004, when Great Lakes Chemical ceases production of pentaBDE and octaBDE, the deca mixture will account for 100% of PBDE production, as no other companies are known to produce the penta and octa mixtures. Great Lakes Chemical is one of four companies known to produce decaBDE.

Table 1. Companies that produce DecaBDE (Peele, 2004)

Company	Product Name	Country
Albemarle Corporation	SAYTEX 102E	Richmond, Virginia
Dead Sea Bromine (subsidiary Israel Chemicals LTD)	FR 1210	Israel
Great Lakes Chemical Corporation	DE-83R, DE-83	West Lafayette, Indiana
Tosoh Corporation	Flamecut 110R	Japan

DecaBDE is an additive powder suitable for use in numerous polymers and applications. Deca is used solely as a flame retardant and is available at a relatively low cost in comparison to other flame retardants (Leisewitz et al., 2001). Its main use is in high-impact polystyrene (HIPS), which is a hard dense plastic used mainly to make housings for TVs and other electronic equipment. TVs and insulation for wire and cable account for 80% of decaBDE usage.[7] The remainder is used in a backcoating for upholstery textiles in automobiles and furniture.

DecaBDE is used in furniture textiles to meet fire safety standards in Europe.[8] California has similar fire safety standards, but residential furniture upholstery intended for distribution in other parts of the U.S. is not routinely flame retarded according to the bromine industry.[9] DecaBDE is not used in textiles for clothing.[10]

Table 2 on the next page lists applications of decaBDE as published on the website of the bromine industry.

Maine users

The extent to which Maine companies may be using deca-laden materials in manufacturing is unknown. In an attempt to identify Maine users, the DEP arranged for publication of an article in the August 5, 2004, edition of *Impact*, the newsletter of the Maine Chamber of Commerce and

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[6] BSEF, Major Brominated Flame Retardants Estimates, http://www.bsef-site.com.

[7] BSEF *supra* note 2 at 14.

[8] BSEF, "Fact Sheet: Brominated Flame Retardant Deca-BDE" (2004). p. 1.

[9] BSEF *supra* note 2 at 14.

[10] OSPAR Commission, "Certain Brominated Flame Retardants: Polybrominated Diphenylethers, Polybrominated Biphenyls, and Hexabromo Cyclododecane" (2001), p. 8.

Table 2: DecaBDE direct uses and final applications[11]

Direct use	**Final applications**
HIPS (high impact polystyrene)	• TV housings • audio and video equipment • remote controls • mobile phones
Textiles	• upholstery textiles, e.g. sofas, office chairs
PE – polyethylene	Electrical • wire and cables Packaging • squeezable bottles • toys and refrigerated housewares
PP - polypropylene	Electrical • building and communications cables • capacitor films Building and construction • pipes • lamp holders • stadium seats Household • lamp sockets • kitchen hoods
PBT - polybutylene terepthalate	• connectors in electrical and electronic equipment
UPE - Unsaturated polyesters	Building and construction • reinforced plastics Transportation • reinforced plastics
Epoxy resins	• circuit boards in PCs and other electronic equipment
Nylon	• connectors in electrical equipment • circuit breakers • insulated wire

Industry (MCCI). The article described the reasons for banning decaBDE, identified products that typically use this PBDE formulation and invited affected businesses to provide information. The article generated a single response from the among the approximately 2000 MCCI members

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[11] BSEF, "What are brominated flame retardants? What are they used for?," http://www.bsef-site.com, April 24, 2003.

that receive the newsletter.

The response was from Central Maine Power Company (CMP), which reported using control and instrumentation cable that contains decaBDE at its electrical substations.[12] Control cables extend from the substation control house through underground trenches to equipment in the substation yard. Instrumentation cables are located within substation control houses. Deca-containing insulation coats the individual conductors that make up the cable core.

The low response to the newsletter article may reflect the fact that companies fabricating items from plastic or foam feedstock do not know which, if any, flame retardants are added to their materials. Another possible reason for the lack of response is that manufacturers have already switched to feedstock that is PBDE free. Interface Fabrics of Guilford, for example, is known to have taken this step.

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[12] Letter from Gerry Mirabile of the Central Maine Power Company to John James of the Maine Department of Environmental Protection (November 2004).

III. DecaBDE: health effects and environmental contamination

This section of the report provides an overview of new information on the environmental contamination and health effects of PBDEs with a particular focus on decaBDE. Much of the current scientific literature measuring decaBDE in environmental media and biota (including humans) has been published in the last 18 months or so.

This document references a large number of papers presented at the Third International Workshop on Brominated Flame Retardants (2004) and the 24th International Symposium on Halogenated Environmental Organic Pollutants and POPs (2004). A state toxicologist attended this international meeting to gain access to the latest research on these chemicals. Much of this information is not yet published in scientific journals, but is included in the proceedings of the conferences as short (4-page) papers.

Health effects in wildlife are not addressed in this report. However, PBDE levels in wildlife are discussed as an indicator of the temporal trend of PBDEs in the environment.

A. Congener composition of commercial PBDEs

There are 209 theoretically possible variations or “congeners” of PBDEs. Individual congeners are called BDE-1, BDE-2, BDE-3 and so on through BDE-209. However, only a few of the congeners are present in three commercial PBDE mixtures, which are called “penta”, “octa” and “deca” because the molecules in each mixture have on average five, eight or ten bromine atoms. The exact identity and pattern of various congeners in the commercial PBDE mixtures depends on the manufacturer and specific product. Approximate percentages of the main congeners in the commercial mixtures are shown in Table 3.

Table 3: Approximate congener composition of commercial PBDE mixtures

Congener	Commercial Product		
	Penta	Octa	Deca
BDE-47	25%		
BDE-99	50%		
BDE-100	<10%		
BDE-153	<5%	5%	
BDE-154	<5%	5%	
BDE-183		40%	
BDE-203		30-35%	
BDE-208		10%	
BDE-209			98%

Data from Birnbaum and Staskal, 2004; Darnerud *et al.*, 2001; Huber and Ballschiffer, 2001; Hutzinger and Sundstrom, 1976; Sjödin *et al*., 1998; WHO, 1994.

In typical analyses, concentrations of only a handful of known PBDE congeners are analyzed. Further, different studies measure different (and different numbers of) congeners, making comparisons between studies difficult. Any "unknown" PBDE congener, as well as many congeners for which analytical standards are not available, or any metabolic derivative of PBDEs, are not included in analyses; therefore "total PBDEs" will always be an underestimate of PBDEs and PBDE derivatives actually present.

Analysis of PBDEs in environmental media and biota (including human tissue) has until very recently included the congeners most prevalent in the commercial mixtures of penta. There was apparently little recognition that PBDEs may be degraded or metabolized, despite decades of experience with PCBs documenting the fact that congener patterns in various media and humans are substantially different from those of commercial mixtures.

Inclusion of decaBDE in analyses of environmental and biological media is very recent – within the last couple of years. The reason for this may be three-fold. First, analysis of decaBDE is technically difficult, in part because it is rapidly degraded by light (see below). Second, there was a lack of appreciation of the fact that decaBDE is absorbed and metabolized by animals, and may be toxic. Third, laboratory background contamination is a major problem, because there is so much decaBDE in indoor environments. Therefore, the widespread occurrence of decaBDE in environmental media and animals is only recently documented, and the existence and characterization of breakdown and metabolic products of decaBDE remain largely undocumented.

Metabolic products of decaBDE or other PBDEs that are formed by microorganisms, macrobiota, fish, game, farm animals and humans, are therefore unknown and undetected. This reality must be borne in mind when assessing the literature on concentrations and patterns of "deca" or PBDEs in general.

B. Photochemical degradation of decaBDE

DecaBDE is readily debrominated by sunlight, especially in the presence of organic solvents. This has been known for some time (Norris *et al.*, 1973; Watanabe and Tatsukawa, 1987).

The rapid degradation of decaBDE under typical laboratory conditions has been addressed only very recently, within the last couple of years. Analytical procedures involve the use of solvents. In a methanol/water solution, decaBDE is reduced to half its initial concentration in 0.5 hour, whereas the half-life for BDE-47 is 290 hours (Eriksson *et al.*, 2004a). Similar results are obtained in a toluene solution (Hermann *et al.*, 2003; Olsman *et al.*, 2002).

DecaBDE also readily degraded under natural conditions when placed on sand, soil or sediment in natural sunlight (Soderstrom *et al.*, 2004). The half-life of photodegradation was 53 hours in sediment, and 150 to 200 hours in soil. The pattern of debromination was the same regardless of matrix. Debrominated products under natural conditions included nonaBDEs, 4 unknown octas, an unknown hepta, two unknown hexas, the penta congeners BDE-119 and 128, the hexa congeners BDE-140, 153 and 154, and the hepta congeners BDE-183.

The "unknown" compounds are unknown because they do not correspond to peaks in the identification standard. It is important to realize that these compounds are therefore not being looked for in environmental media, food, or human tissues. In addition, some of the identified congeners are not typically included in analyses of various media. It is therefore unknown to what degree "deca" products are present in wildlife or in us.

C. PBDEs in the environment

PBDEs have been found in sediment everywhere they have been looked for in the U.S. (Kohler *et al*., 2003; Zegers *et al*., 2003; Raff and Hites, 2004; Baker *et al*., 2004; Wenning *et al*., 2004; Klosterhaus *et al*., 2004). DecaBDE constituted 97-100% of detected congeners, and levels were very high. Unlike other contaminants, concentrations of PBDEs at 31 locations along the entire Mississippi River system were not associated with large population areas (Raff and Hites, 2004). Levels were higher in spring, presumably as a result of flushing from sources into storm drains.

High concentrations of PBDEs are also found in sewage sludge, with levels in the U.S. 10-100 times those in Europe (Hale *et al*., 2001; Knoth *et al*., 2004; Fabrellas *et al*., 2004). In a recent study in Spain, decaBDE constituted 95-99% of the total PBDEs. DecaBDE also constituted over 90% of PBDEs present in paper biosolid waste, with apparent degradation (or metabolism) over time (Kolic *et al*., 2004).

DecaBDE added to sewage sludge under anaerobic conditions was converted to nona (BDE-203 through 208) and octa metabolites (Gerecke *et al*., 2004). After 114 days, 25% of the decaBDE had been converted.

There is evidence that decaBDE in the effluent of waste water treatment plants is debrominated (La Guardia *et al*., 2004). Nona congeners were detected, as well as a number of lower brominated congeners not present in commercial mixtures.

Over half the sewage sludge produced annually in the U.S. is applied to land as fertilizer (U.S. EPA, 1999). Sludge is treated for odor and pathogens, but not for organohaline content, which is also unregulated. Thus application of sewage sludge may represent a source of exposure to humans and wildlife, through direct contact or uptake by plants. For example, a recent survey of U.S. foods found PBDEs in infant soy formula and beef (see section on food), suggesting that plants can be contaminated with PBDEs.

PBDEs may be found in outdoor air, even at rural locations. In a study in southern Ontario, PBDE levels were about half those of PCBs in early spring (Gouin *et al*., 2002), and decreased following budbreak. Lighter congeners predominated in air; decaBDE was not measured. The authors hypothesized that the decrease in air concentrations following budbreak was the result of sorption to newly emerged foliage, as has been demonstrated for other contaminants. PBDEs were measured in pine needles around Tokyo Bay, and decaBDE accounted for more than 90% of the detected PBDEs based on 10 congeners (Okazawa *et al*., 2004).

A recent study in Europe found that there was a gradient of PBDEs in air samples, with sources near urban areas having higher levels (Jaward *et al*., 2004). In that study, particularly high levels were found in the United Kingdom, presumably because of significant manufacturing activities and higher use compared to other European countries with less stringent fire retardant regulations. BDE-47 and BDE-99 were dominant of the congeners measured; BDE-209 (decaBDE) was not analyzed.

It is becoming recognized that there is a global cycling of PBDEs, as there is for other persistent organic pollutants (POPs). A recent review discussed the deposition into the Arctic (de Wit *et al*., 2004). Air concentrations were higher in some Arctic locations than in Chicago. Soil and sediment contained PBDEs, with levels increasing over time at rates comparable to those in the south. DecaBDE was detected in Arctic sediment, indicating long-range transport. DecaBDE was transported into the Arctic about 10 years later than other congeners, based on its appearance in core samples.

Air transport of decaBDE is apparently the result of sorption to particles (Gouin *et al*., 2004). In contrast, the lighter congeners, dominated by 17, 28, and 47, are in the gas phase. Levels of decaBDE were high during all periods of the year.

D. PBDEs in indoor air and dust

It is currently well established that there may be significant levels of PBDEs in indoor air and dust. In dust collected from vacuum cleaner bags in Germany (Knoth *et al*., 2002, 2003) and the U.S. (Schecter *et al*., submitted; Stapleton *et al*., 2004a, b), decaBDE typically constituted more than 50% of the PBDEs in the sample, and a high proportion of decaBDE was associated with the presence of computers or new carpet. A study of dust samples from homes in several states also revealed that decaBDE was the dominant congener in most homes (Environmental Working Group, 2004a). Levels in the U.S. dust were 50 times higher than those in Germany (Sjödin *et al*., 2004b).

Dust samples were collected from computer casings in seven states, including from the Maine State House and Children's Museum (Computer Take-Back Campaign, 2004). The dominant congener was decaBDE, with the Maine State House sample having the second highest concentration of a total of 16 samples.

Concentrations of PBDEs were compared in indoor and outdoor window film in urban and rural areas of southern Ontario (Butt *et al*., 2004). PBDE concentrations in indoor films were 15-20 times higher than in outdoor films. DecaBDE represented at least 50% of the PBDEs (based on 6 congeners) in most samples in both indoor and outdoor air. For four of 9 outdoor samples, decaBDE comprised 80-90% of total PBDEs.

PBDE levels were measured in indoor and outdoor air in Ottawa, Ontario, Canada (Wilford *et al*., 2004). Indoor air had levels about 50 times higher than outdoor air. DecaBDE was not measured.

E. PBDEs in wildlife

Levels of PBDEs are increasing exponentially in wildlife all over the world: in North America, Europe, and the Arctic. Concentrations are increasing in fish, birds, and mammals from different trophic levels. This congruence of data provides confidence that the observed trends are widespread and reliable, and not the result of local changes in exposure sources or food webs.

Levels in Arctic ringed seals are very high, with a doubling time of 4-5 years (Ikonomou *et al*., 2002). Detectable levels of PBDEs are also found in Arctic frogs, fish, and a variety of birds species and terrestrial mammals (de Wit *et al*., 2004). Levels in animals are highest in regions influenced by atmospheric transport from western Europe and eastern North America.

Levels of PBDEs in ocean fish in San Francisco Bay increased between 1997 and 2002, with a doubling time of 3-4 years (EWG, 2003). San Francisco harbor seals had a doubling time of 1.8 years between 1989 and 1998 (She *et al*., 2002). Columbia River fish had a doubling time of 1.6 years between 1992 and 2000 (Rayne *et al*., 2003). PBDE levels in trout in Lake Ontario increased by 300% between 1978 and 1998 (Luross *et al*., 2000).

Similar increases have been observed in beluga whales (Stern *et al*., 2000). Doubling times of PBDEs are about 3 years in Great Lakes gulls, and 6 years in Swedish birds' eggs (Hites, 2004). Increasing PBDE levels have been documented in multiple other species in various locations in the U.S. (Law *et al*., 2003; Lebeuf *et al*., 2004; Norstrom *et al*., 2002), including marine mammals, birds, and fish in the U.S. In these studies, decaBDE was not analyzed.

F. DecaBDE in wildlife

High levels of PBDEs have been found in peregrine falcons in Sweden (de Wit *et al*., 2004), with detectable levels of decaBDE. Eggs from wild peregrine falcons in Sweden contained concentrations of PBDEs hundreds of times greater than a captive population (Lindberg *et al*., 2004). Wild birds, which fed on either aquatic or terrestrial wild birds, had levels of decaBDE higher than captive birds feeding on chicken.

DecaBDE has been found, at low concentration, in a number of predatory bird species in the UK, including both aquatic and terrestrial predators (peregrine falcon, sparrowhawk, kestrel, barn owl, red kite, harrier, marsh hawk, sea eagle, osprey, golden eagle, and merlin) (de Boer *et al*., 2004). Concentrations of decaBDE in flesh or eggs of a number of species of predatory birds has increased by about a factor of 10 between 1980 and 2002 (de Boer *et al*., 2004). The presence in the terrestrial food chain was particularly unexpected, and levels of decaBDE in the eggs of the aquatic- and terrestrial-feeding falcons were the same. DecaBDE was also detected in herons and crested grebes.

DecaBDE has also been reported in golden eagles, gyrfalcons, and merlins in Norway (Herzke *et al*., 2003), with high total PBDE levels in these birds. In these species, BDE 153 was the dominant congener, indicating a changing congener pattern as PBDEs are passed up the food chain. A change in congener patterns from those in commercial mixtures is to be expected, as a result of differential absorption, metabolism, and elimination by various species. This has been clearly demonstrated with PCBs, for which congener PCB 153 is a major constituent in animal and human tissues.

A study in Virginia rivers of channel catfish, an omnivorous bottom-dwelling species, found PBDEs in 87% of fish (Hale *et al*., 2001b). In addition, very high levels were found in carp. In that study, decaBDE was detected in sludge but not fish. However, appreciation of the challenges inherent in analyzing for deca because of its instability are only recently realized. Therefore, low levels of decaBDE in fish may have been undetected, whereas decaBDE would have been detected in sludge even if a portion degraded, due to the much higher levels in sludge. Alternatively, decaBDE may not have been detectable in the fish as a result of metabolism of decaBDE to lower brominated BDEs (see next section).

These findings demonstrate that contrary to earlier assumptions, decaBDE is bioavailable and concentrates up both aquatic and terrestrial food chains.

G. Metabolism of PBDEs

The lower brominated PBDEs are apparently extensively absorbed following a single oral dose. Over 90% of BDE-47 (tetraBDE) was absorbed, with 86% and 47% percent of a given dose remaining in the body of rats and mice, respectively, after five days (Klasson-Wehler *et al.*, 1996; Orn and Klasson-Wehler, 1998). For BDE-99 (pentaBDE), over 50% of the dose was retained in rats after 72 hours (Hakk *et al.*, 2002), with lipophilic (fatty) tissue preferentially retaining the radiolabeled compound.

In another study with mice dosed with BDE-47, over 80% of a single oral dose was absorbed (Staskal *et al.*, 2004), and 62% was absorbed when applied dermally. Adipose tissue and skin were major tissue depots, as would be expected on the basis of the lipophilicity of PBDEs. Of particular importance is the fact that excretion was biphasic, with an initial short half-life (1-3 days) and a much longer terminal half-life. This suggests the potential for bioaccumulation of this congener.

It appears that the distribution of decaBDE is different for acute versus chronic exposures. About 10% of decaBDE was reported to be absorbed after a single dose in adult male mice, based on fecal excretion and 10% retention in bile (Mörck *et al.*, 2003). DecaBDE was preferentially distributed to plasma- and blood-rich tissues rather than adipose tissue. However, about 65% of the dose was excreted as metabolites, indicating that the 10% absorption may be an underestimate. Metabolites included penta- and heptaBDEs, as well as methoxymetabolites. An early study reported uptakes of 60-70% of decaBDE by chickens (Alumot and Mandel, 1976).

In a 2-year chronic study using a commercial preparation of decaBDE (77.4% deca, 21.8% nona), BDE was preferentially stored in adipose tissue (Kociba *et al.*, 1975a, b). In addition, during a 90-day recovery period with no additional dosing, there was no decrease in levels of brominated compound in adipose tissue following chronic exposure, whereas bromine was cleared from the liver within 10 days. These data suggest that chronic exposure to decaBDE and nonaBDE results in accumulation in fatty tissue, which would then be available for mobilization during pregnancy and lactation. It is also important to note that brain is a very lipid-rich tissue. In these studies, levels in brain were not assessed.

An additional study is relevant to the issue of relative retention in adipose tissue of specific congener classes. Von Meyerinck *et al.* (1990) dosed rats with a single dose of Bromkal 70 (mainly pentaBDE) and killed them after 1-10 weeks. Half-lives in fat for two hexa congeners were 50 and 105 days, and for two penta congeners 42 and 25 days. Half-lives for tetra BDEs were shorter and sex-dependent. This suggests that retention time in fat is greater for more highly brominated congeners, although not necessarily for decaBDE itself.

Distribution of PBDEs into brain has been studied following developmental exposure in mice, in which a single dose of uniformly radiolabeled C14 PBDE was administered on postnatal day (PND) 3, 10 or 19, and distribution of the radiolabel from the administered compounds were measured in mouse tissues. Radiolabel from the penta congener BDE-99 (Eriksson *et al*., 2002) and decaBDE (Viberg *et al*., 2003) show substantial retention in brain. About 5% of the radiolabel from decaBDE was found in brain 24 hours after administration on PND 3 or 10, increasing to about 7 to 10% by PND 7. This suggests redistribution into brain from other tissues. It is unknown the degree to which this represents parent compound or metabolites. (But see section on neurotoxicity.) Most of the radiolabel was found in liver tissue after 24 hours, which decreased by 50% at 7 days.

There is evidence that fish metabolize decaBDE. Trout fed purified decaBDE for up to 120 days absorbed a significant amount, as evidenced by concentrations in muscle and liver (Kierkegaard *et al*., 1999). A number of octa- and hepta-congeners were quantified as homologues but not identified at the level of specific congeners. After exposure for 120 days, levels of individual metabolites in muscle tissue were up to 80 times higher than the parent compound (decaBDE).

Carp are bottom-feeding fish that may have very high tissue levels of PBDEs following environmental exposure (Hale *et al*., 2001). In a metabolic study, carp were fed decaBDE for 60 days at levels simulating the exposure levels carp would encounter in river sediments (Stapleton *et al*., 2004a). No decaBDE was detected in whole fish at the end of exposure, but seven metabolic products were present. The hexaBDEs 154 and 155 were identified. Other congeners were one penta-, three heptas-, and an octa-BDE, which did not correspond to congeners in the standard. Interestingly, the concentration of the penta-BDE was higher 10 days following cessation of exposure than immediately after cessation of exposure, suggesting further metabolism. The fact that no decaBDE was identified suggests that the contribution of decaBDE to PBDEs in the food chain is underestimated. Further, congener 154 is a common constituent of PBDEs in fish tissue, suggesting that metabolism of decaBDE may be a source.

There is evidence that fish and mammals metabolize PBDEs by oxidative as well as debromination pathways. In northern pike dosed with labeled BDE-47, six hydroxy-PBDE metabolites were detected in tissue (Kierkegaard *et al*., 2004). Hydroxy- metabolites have also been identified in rodents following administration of congener 47 (Orn and Klassen-Wehler, 1998) and congener 209 (decaBDE) (Mörck *et al*., 2003).

Hydroxylated and methoxylated BDEs have been identified in several fish species from the Detroit River (Letcher *et al*., 2003, 2004), in Baltic Sea salmon (Marsh *et al*., 2001), herring in Sweden (Asplund *et al*., 2004), whales (van Bavel *et al*., 2001; Lechter *et al*., 2004), birds (Olsson *et al*., 2000; Lechter *et al*., 2004), and seals (Haglund *et al*., 1997). Methoxy- and hydroxy-metabolites are also found in humans (Vitter, 2003). Some of the oxidative metabolites identified may be the result of metabolism of PBDEs, whereas others may result from impurities in other chemicals in the environment.

H. PBDEs in food in the U.S.

PBDEs have been found in the food supply in the U.S., Europe, and Japan, based on market-basket surveys. In addition, PBDE concentrations are high (relative to other foods) in many species of fish that may be caught and consumed by anglers. As is the case for other organohalogen contaminants, PBDEs are found in higher levels in fish at higher versus lower trophic levels (i.e. in predatory fish that feed on other fish).

A number of meat and dairy products, as well as margarine and soy infant formula, were purchased from three major supermarket chains in Dallas, Texas, and analyzed for 13 PBDE congeners (Schecter *et al*., 2004b) (figures 2a and 2b). The highest PBDE level was observed in one of two salmon fillets, followed by catfish, salmon fillet, shark, salmon and catfish. Other high-fat foods were also high in PBDEs: pork sausage, hotdogs, duck, turkey, and cheese, in that order. The congener pattern varied among samples. BDE-47 and BDE-99 predominated in most samples. The decaBDE congener however predominated in one of the two catfish, in a sample of cheese, soy formula and calf liver. This provides evidence that PBDEs generally and decaBDE specifically are present in the food chain, available for human consumption, and may therefore represent an important exposure pathway.

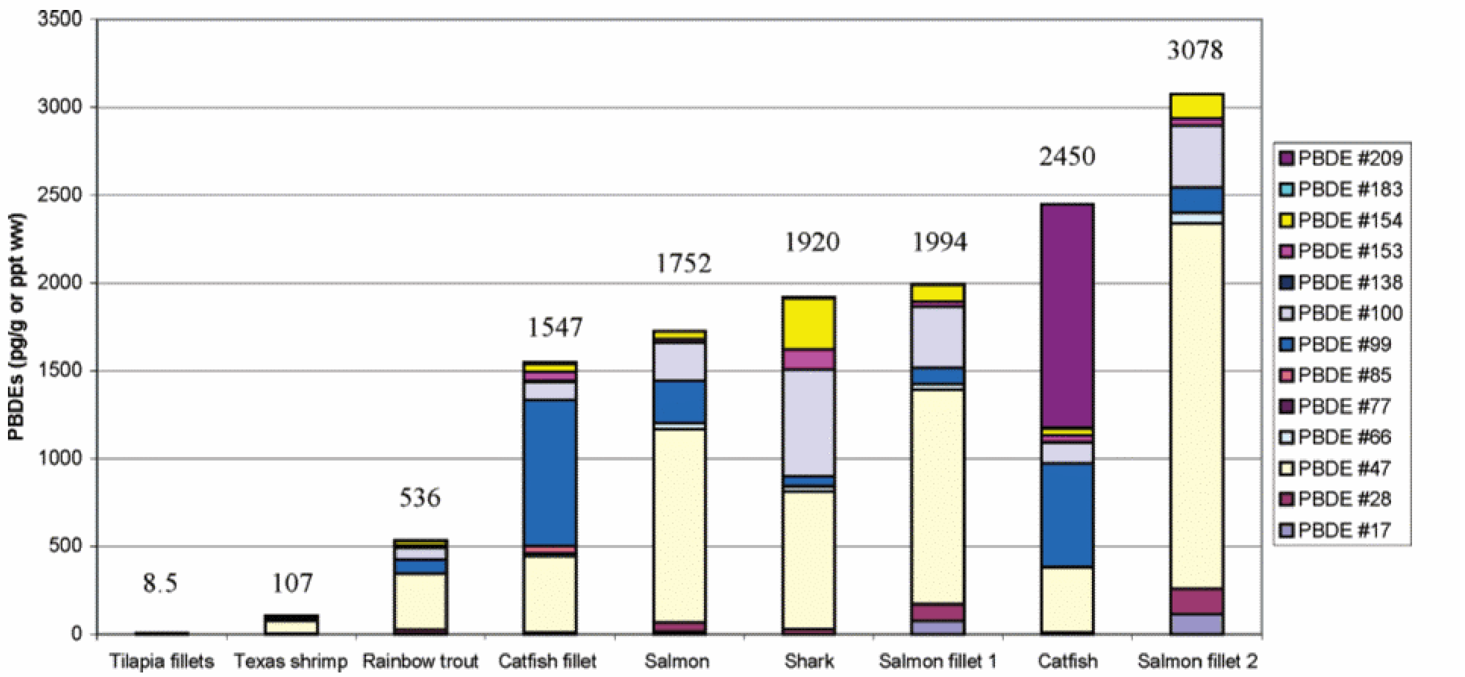
In a market basket survey in California, fish, meat, and fowl products were collected from two cities (Luksemburg *et al*., 2004) (figures 3a and 3b). Highest total levels were found in a swordfish, followed by halibut, Atlantic salmon, catfish and duck. For most of the samples with higher levels of total PBDEs (i.e., > 5000 pg/g), the predominant congeners were tetraBDEs (e.g., BDE 47) and pentaBDEs (e.g., BDEs 99 and 100), consistent with results reported by Schecter *et al*., 2004b. For many of the food samples with total PBDE levels < 500 pg/g, decaBDE was predominant congener. The duck sample had an unusual congener profile dominated by hexaBDEs, in contrast to what was reported by Schecter *et al*., 2004b.

A number of studies have analyzed PBDE levels in various species of fish consumed by humans (see Hites, 2004a for review), including in the U.S. (Rayne *et al*., 2003; Rice *et al*., 2002; Dodder *et al*., 2002; Luross *et al*., 2002; Asplund *et al*., 1999; Hale *et al*., 2001b; Hayward *et al*., 2004; Yan Zhu and Hites, 2004; Manchester-Neesvig *et al*., 2001). Average tissue levels in the U.S. are about 10 times higher than in Europe.

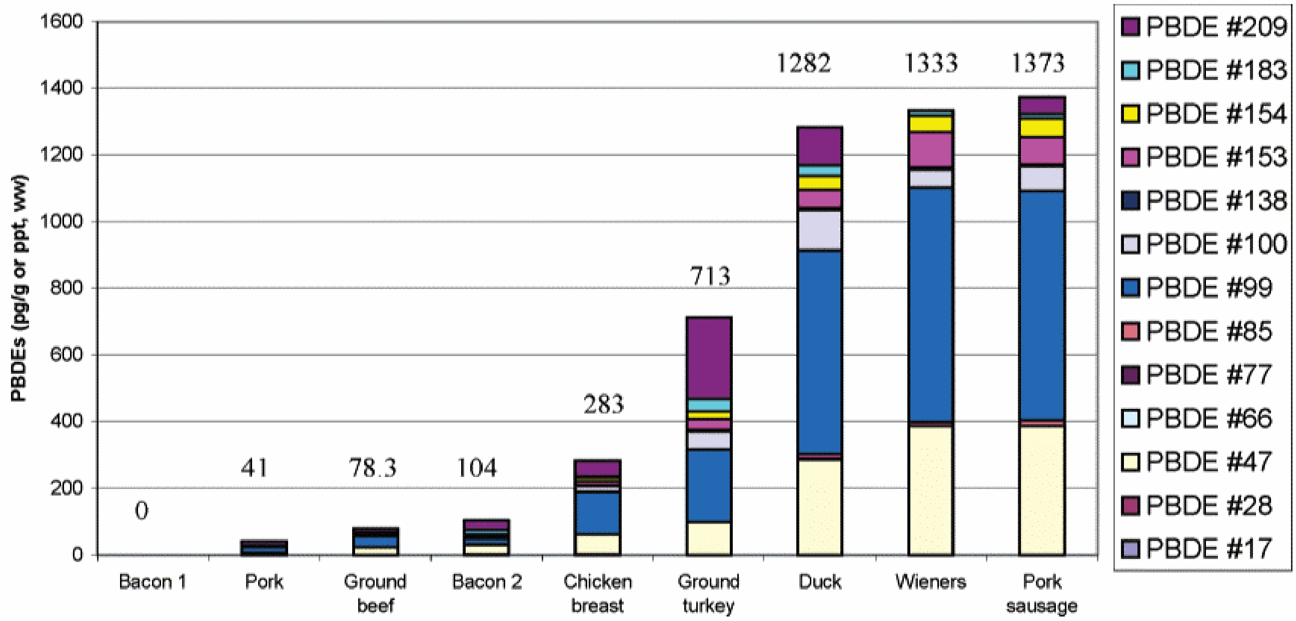
PBDE levels were assessed in fish collected from anglers in San Francisco Bay in 2002 (EWG, 2003). The catch included halibut, striped bass, kingfish, walleye, surfperch, jacksmelt and leopard shark. Levels were higher than levels previously reported for other locations. Levels in halibut and striped bass increased by 2.4-fold to 3.4-fold between 1997 and 2002. DecaBDE was not measured.

Levels of PBDEs in Pacific farm-raised salmon were 15 times higher than in Pacific wild salmon (Easton *et al*., 2002). These findings were replicated in a study of wild and farm-raised salmon samples from around the world (Hites *et al*., 2004). Farmed salmon from North American and Europe contained more PBDEs than those from Chile, consistent with results from analyses of PCBs.

Figure 2a. PBDE levels in various foods in a market-basket survey in Texas
(from Schecter *et al.*, 2004b)

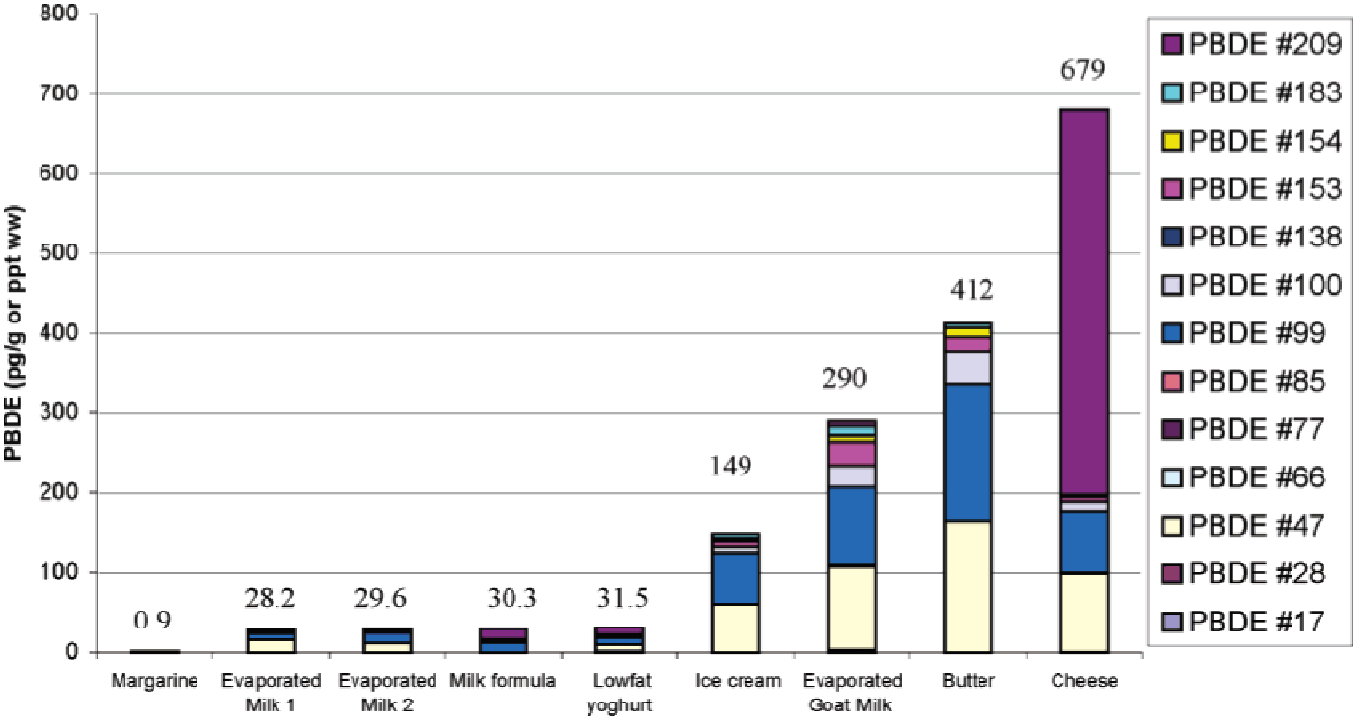


PBDE congener levels and profiles of individual samples of fish products purchased in the United States (pg/g or ppt, ww).

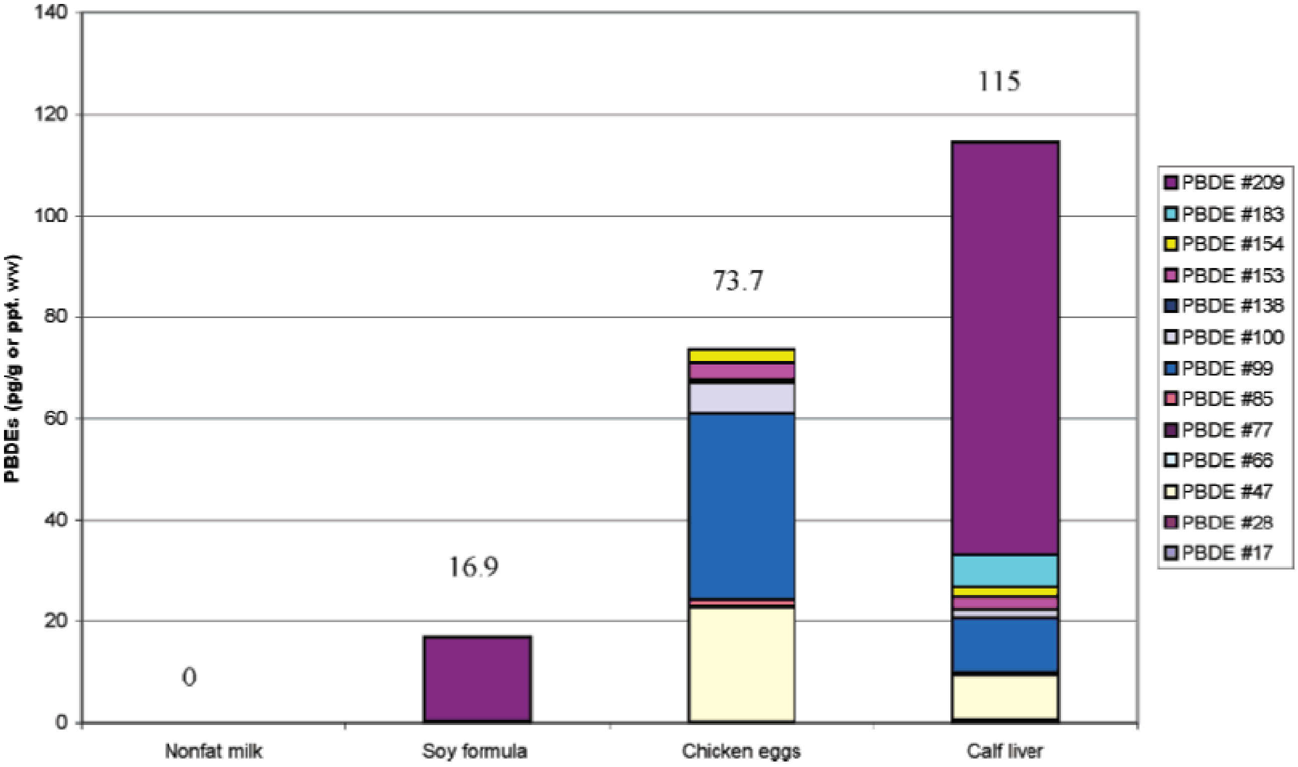


PBDE congener levels and profiles of individual samples of meat products purchased in the United States (pg/g or ppt, ww).

Figure 2b. PBDE levels in various foods in a market-basket survey in Texas
(from Schecter *et al*, 2004b)

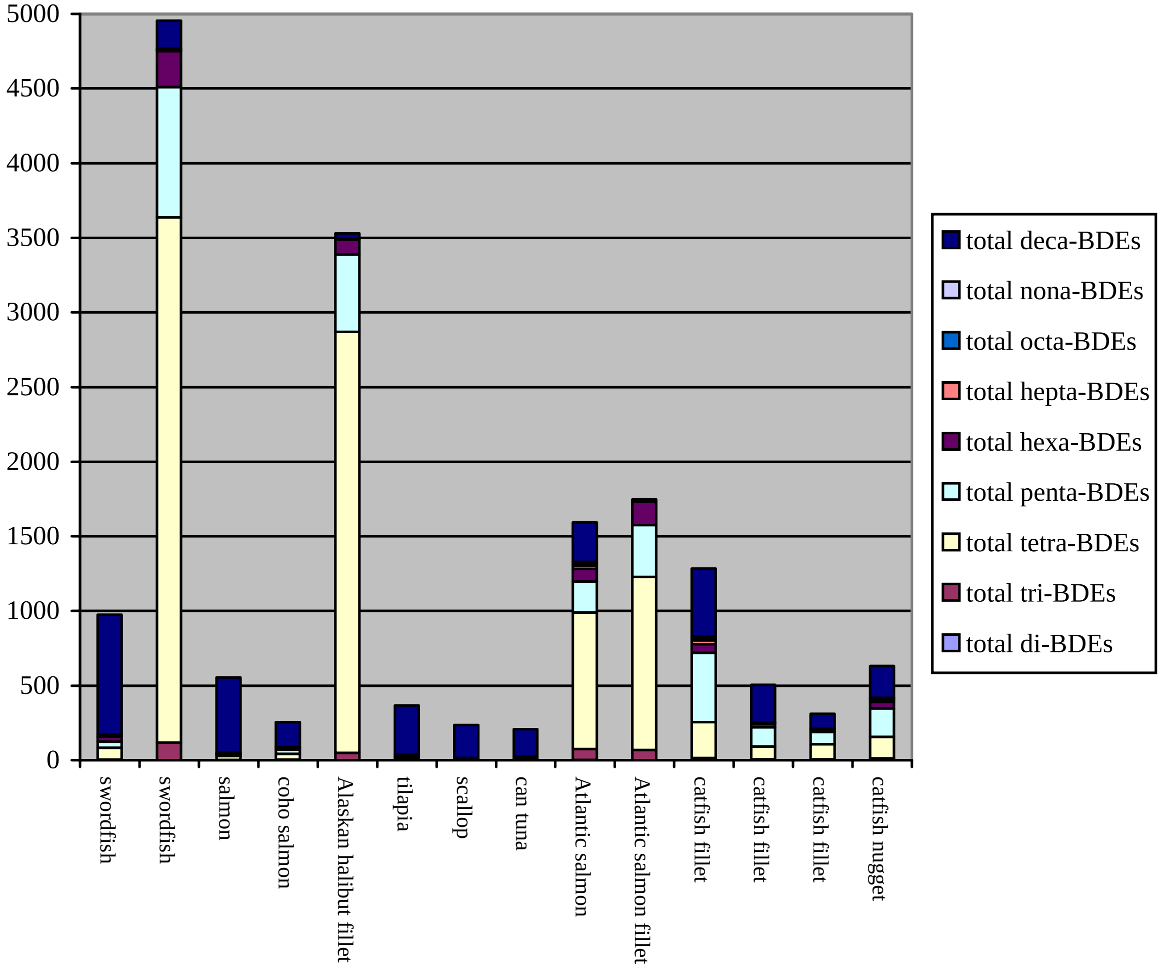


PBDE congener levels and profiles of individual samples of dairy products purchased in the United States (pg/g or ppt, ww).



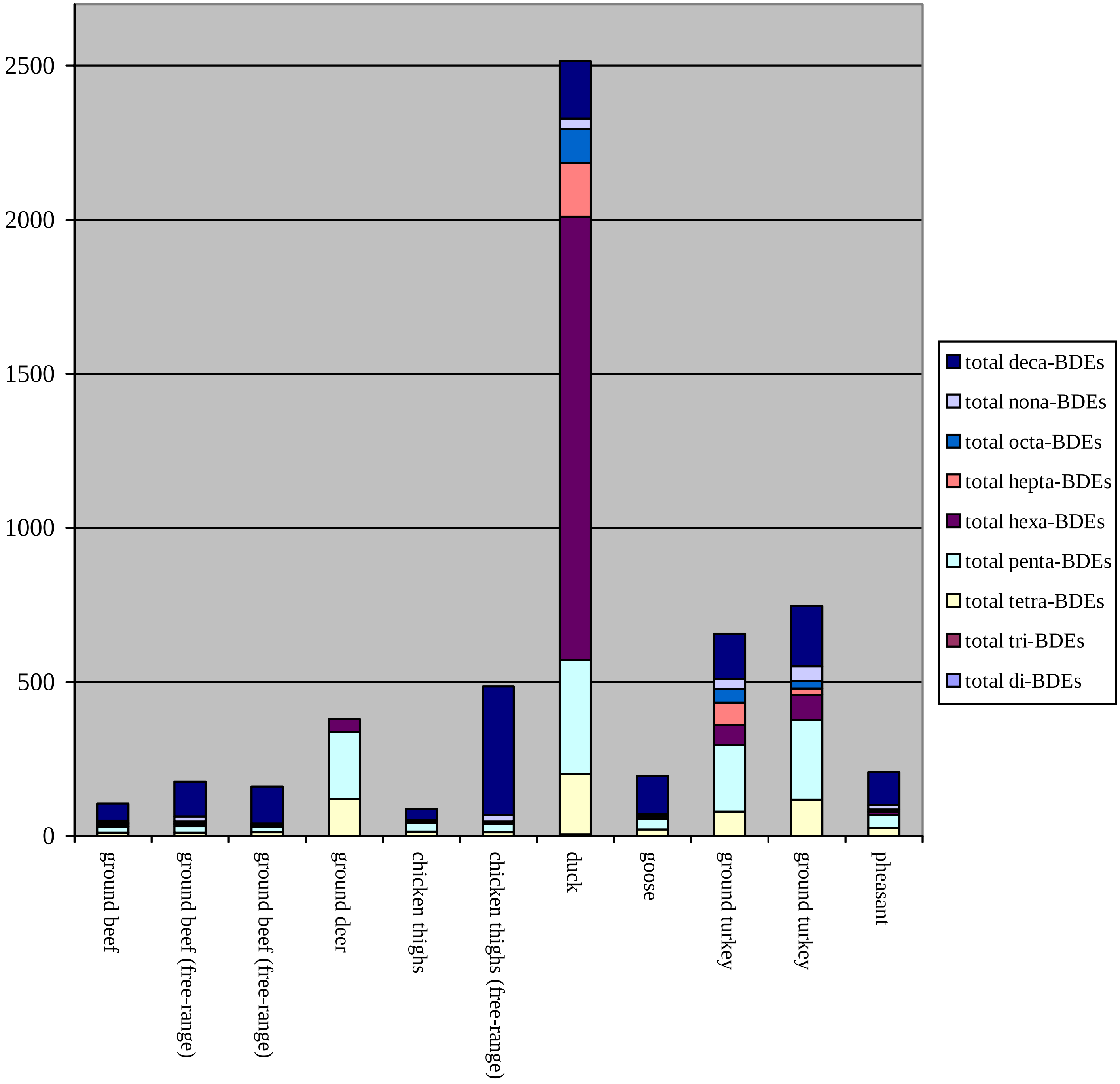
PBDE congener levels and profiles of individual miscellaneous products purchased in the United States (pg/g or ppt, ww).

Figure 3a. Concentrations of PBDEs (pg/g wet weight) in fish products purchased in three food markets in northern California



Data from Luksemburg *et al*., 2004
Figure by Mark Krawec

Figure 3b. Concentrations of PBDEs (pg/g wet weight) in meat and fowl products purchased in three food markets in northern California



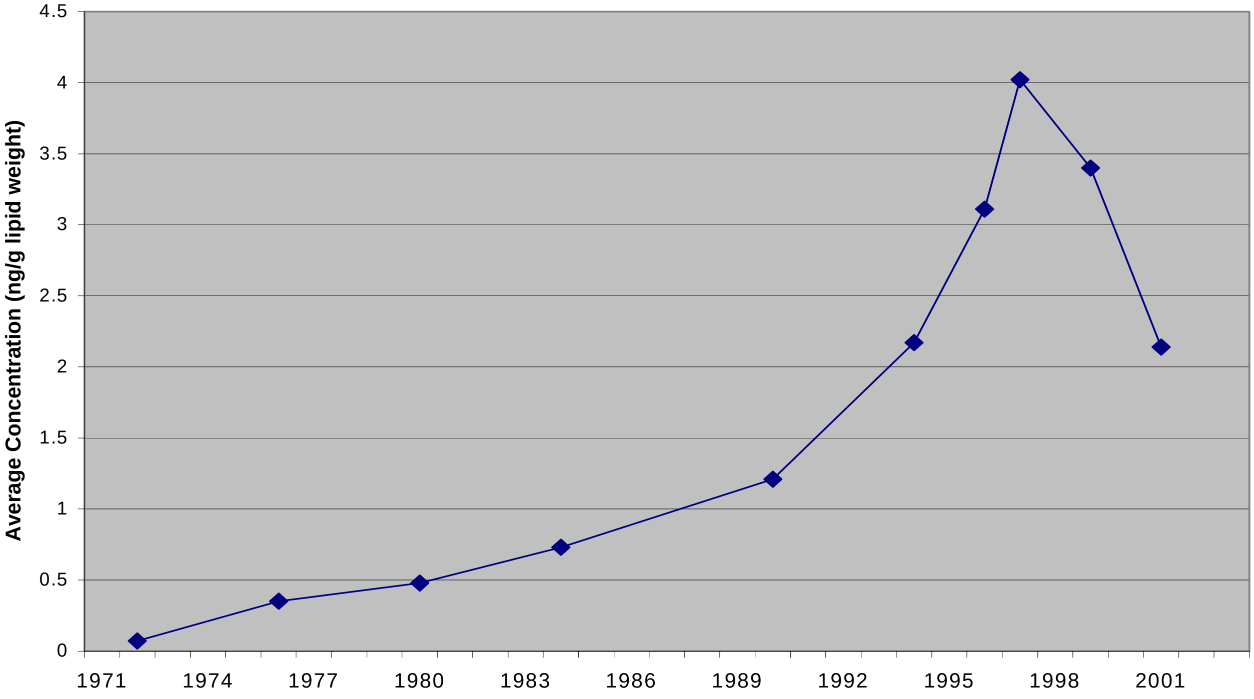
Data from Luksemburg *et al.*, 2004
Figure by Mark Krawec

In a study of fish in Lake Winnipeg, Canada, PBDE levels in fish at various trophic levels were determined (Tomy *et al*., 2004). Predatory fish (perch, walleye) had higher levels than whitefish. DecaBDE was the dominant congener in walleye, perch, and turbot, being very high in perch. BDE-40, a known metabolite of decaBDE in lake trout, was detected in all six species of fish analyzed.

I. PBDEs in human tissue

Levels of PBDEs in human tissues, specifically blood, milk, and fat, have increased exponentially since the 1970s (Norén and Meironyté, 2000; Meironyté *et al*., 1999; Sjödin *et al*., 2004a; Darnerud *et al*., 2002), including the U.S. (Schecter *et al*., 2004a). Currently, the doubling time in the human body is estimated to be 3-5 years (Hites, 2004; Smeds and Saukko, 2003; Polder *et al*., 2004; Schuhmacher *et al*., 2004; Weiss *et al*., 2004; Fangström *et al*., 2004; Ryan and van Oostdam, 2004). PBDE levels in breast milk in women living in Vancouver, B.C. increased 15-fold between 1992 and 2002, a doubling time of 2.6 years (Ryan *et al*., 2002). Levels in Swedish breast milk increased 60-fold between 1972 and 1997, a doubling time of 5 years (Meironyté and Norén, 1999). Currently breast milk levels in Sweden are decreasing (Guvenius *et al*., 2003), presumably as a result of a decrease in the use of PBDE-containing products (Figure 4). The EU has decreased PBDE use by two-thirds in recent years (Madsen *et al*., 2003).

Figure 4. PBDEs in Breast milk from Sweden, 1972-2001



Data from Meironyte and Noren, 1999; Ryan et al., 2002; Guvenius at al., 2003

Source: *Polybrominated Diphenyl Ethers (PBDEs): Background Paper*, Michigan Department of Environmental Quality, 2004

Levels of PBDEs among individuals in North America are 10-70 times higher than in Europe or Japan, as measured in blood, breast milk, or adipose tissue (She *et al*., 2002; Petreas *et al*., 2003; Mazdai *et al*., 2003; Shechter *et al*., 2003; Environmental Working Group, 2004b; Northeast Environment Watch, 2004; Meironyté *et al*., 1999; Thomsen *et al*., 2002; Sjödin *et al*., 2004a; Darnerud *et al*., 2002; Schroeter-Kermani *et al*., 2000; Strandman *et al*., 2000; Thomsen *et al*., 2002; Lind *et al*., 2003; Sjödin *et al*., 2001; Ingelido *et al*., 2004; Vieth *et al*., 2004; Bergman *et al*., 1999; Norén and Meironyté, 2002) (Table 5) (Figure 5).

Table 5. Total PBDE concentrations (ng/g lipid) in contemporary breast milk samples.

Country	Year	Mean	Median	Range	Reference
U.S. (Texas)	2002	73.9	34	6.2 – 419	Schecter *et al*., 2003
U.S.	2002-2003	159	58	9.5 – 1078	EWG, 2003
U.S. (Puget Sound)	2003	63	50	13 – 156	NEW, 2004
U.S. Northwest	2003	48.5	77.5	63 – 309	She *et al*., 2004
Canada	2000-2002	42.8	25.4		Ryan *et al*., 2002
UK	2001-2003	8.9		1 – 63	Kalantzi *et al*., 2004
Faroe Islands (4 congeners)	1987 1994-1995 1998-1999	1.5 3.6 7.2			Fängström *et al*., 2004
Spain	2002	2.2, 2.5			Schuhmacher *et al*., 2004
Germany	2001-2003	2.23	1.78	max 7.25	Vieth *et al*., 2004
Sweden	2000-2001		2.1	0.71 – 8.39	Guvenius *et al*., 2003
Norway (Rogeland)	2003		1.60 – 2.47	1.0 – 10.8	Polder *et al*., 2004
Italy Rome Venice	1998-2000	 4.1 2.3			Ingelido *et al*., 2004
Mexico	2003?	4.4		2.7 – 9.0	López *et al*., 2004

Figure 5. Trends of PBDEs in Human Milk for Sweden, Canada, and the U.S.

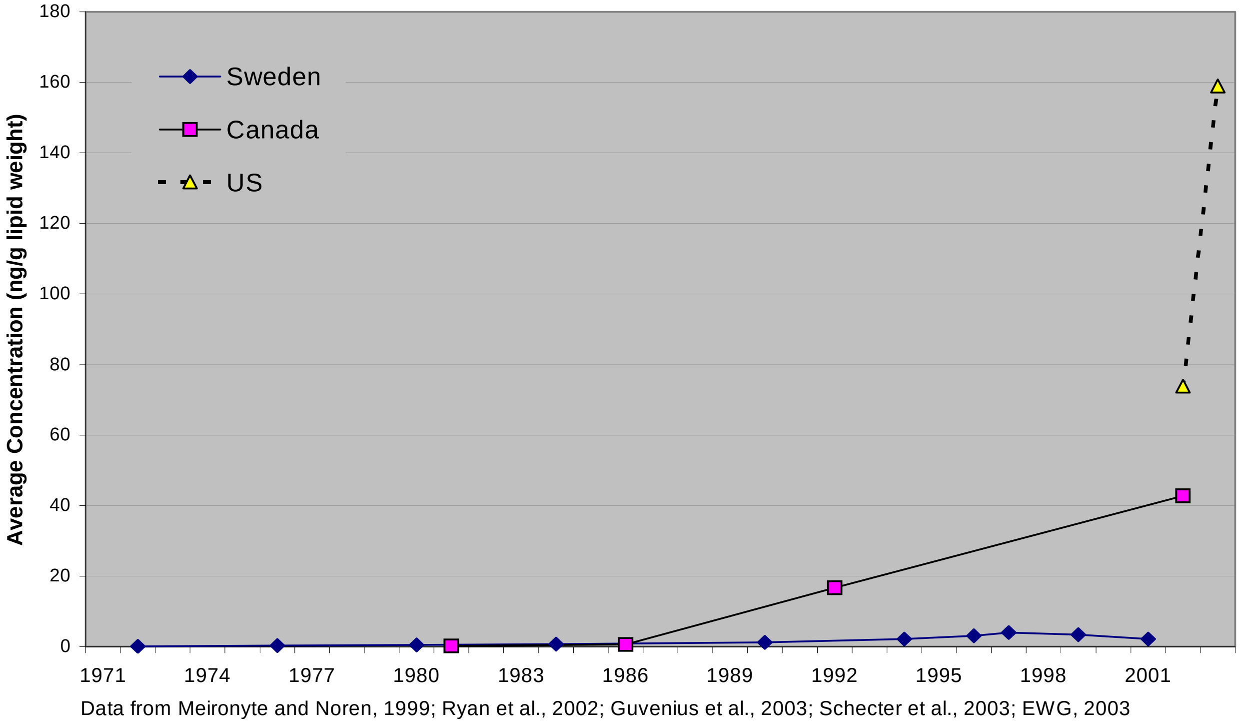


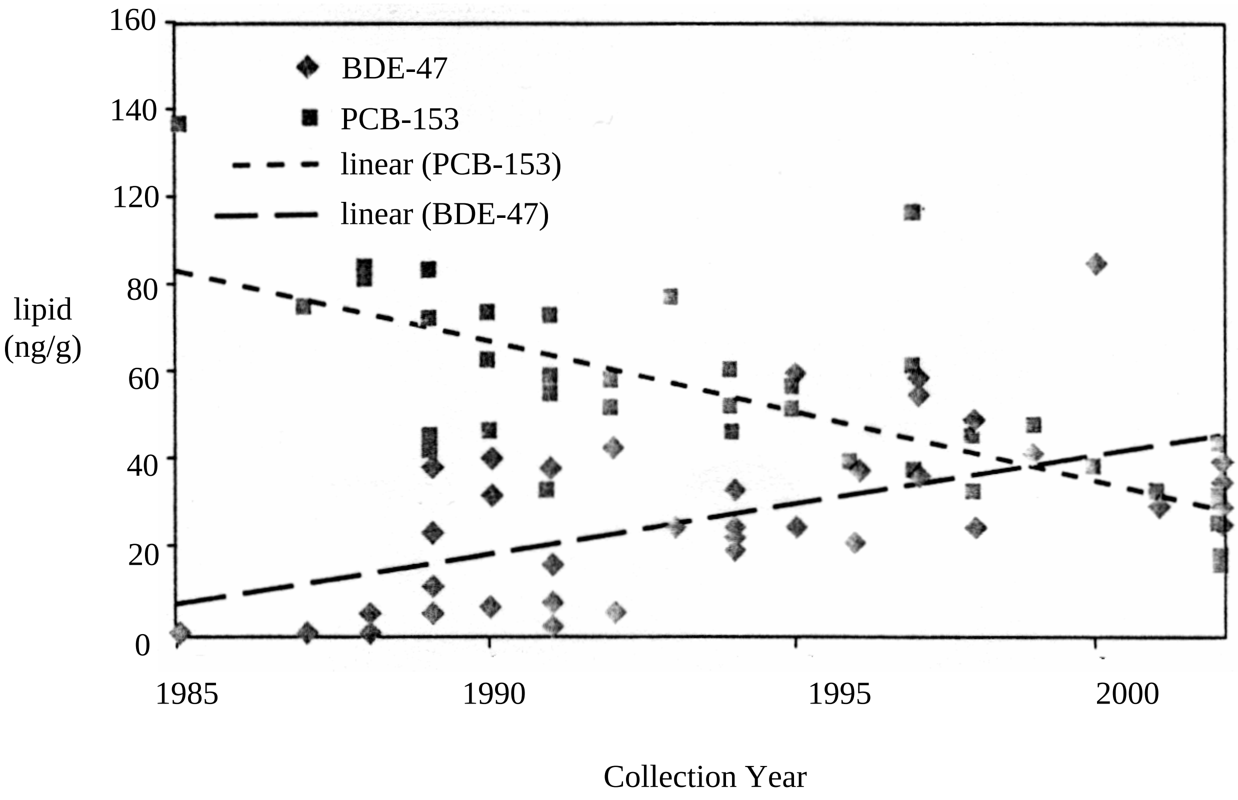
Figure from *Polybrominated diphenyl ethers (PBDEs): Background Paper*, Michigan Department of Environmental Quality, 2004

There is evidence that the pattern of congeners in human tissue is changing from that of a pattern reflective of commercial mixtures. Although congeners BDE-47, BDE-99 and BDE-100 still represent a substantial proportion of total body burden, recent analyses revealed that 153 is the dominant congener in samples from some individuals in various locations around the world. Congener BDE-153 makes up 28% of breast milk PBDEs in German milk (Vieth *et al*., 2004), not very different from BDE-47 (37%) and more than BDE-99 (11%). In a Texas study (Schecter *et al*., 2003), congener BDE-153 made up about 5% of the mean PBDE levels, with BDE-47 predominating. However, for a number of individuals, the concentration of BDE-153 was greater than BDE-99 or BDE-100. In a study of women living on Puget Sound (Northwest Environment Watch, 2004; She *et al*., 2004), concentrations of BDE-153 were generally exceeded only by those of BDE-47. Similarly, a study in the UK reported that breast milk concentrations of BDE-153 were greater than those of BDE-99, BDE-100 or BDE-53 but not BDE-47 (Kalantzi *et al*., 2004).

In a study in the Faroe Islands in the North Atlantic, BDE-153 was the dominant congener in human milk in 1998-1999, increasing by a factor of 6 from 1987 (Fangström *et al*., 2004). In contrast, BDE-99 and BDE-100 were the dominant congeners in 1987, and increased by a factor of 4 in 1998-1999. A food source upon which people depend in the Faroe Islands (marine mammals) is highly contaminated with organochlorines, and body burdens of PCBs are higher than anywhere else measured (Longnecker *et al*., 2003). PBDE levels in the Faroe Islands are not high compared to those in North America, based on breast milk levels. The fact that congener BDE-153 is the dominant congener in this population may result from continuous exposure coupled with the long half-life of BDE-153.

In a U.S. study (Sjödin *et al*., 2004a), PBDEs in serum were measured in a total of 40 blood samples from the southwestern U.S. (1985-2002) and Seattle, Washington (1999-2002). Total PBDEs increased by a factor of seven between 1985-1989 and 1995-1999, a period representing samples from the southeast only. Levels of total PBDEs decreased between 1995-1999 and 2000-2002, but it may be that this apparent decrease represents inclusion of individuals from Seattle. Levels of congeners BDE-47, BDE-85, BDE-99, BDE-100, and BDE-154 all exhibited this apparent decrease after increasing from 1985-98 to 1995-99. However, BDE-153 continued to increase over the entire time period, by a factor of about 9. By 2000, the concentrations of the most prevalent PBDE in this sample (BDE-47) and the most dominant PCB (PCB-153) were equal (Figure 6).

Figure 6. Levels of BDE-47 and PCB-153 in U.S. human blood samples.



Data from Sjödin *et al*., 2004a; figure from *Environmental Science and Technology* 37, p. 384A, 2003.

DecaBDE has only recently been measured in human tissue, particularly breast milk in the U.S. (She *et al*., 2004; Schecter *et al*., 2004; Northeast Environment Watch, 2004; Vieth *et al*., 2004; Ohta *et al*., 2004). DecaBDE was measured in 23 of 47 samples from women in Texas (Schecter *et al*., 2004). Of those 23 women, decaBDE was detected in 7 of them (30%). When detected, decaBDE comprised between 2.1 and 13.2 % of the total PBDEs based on analysis for 13 congeners. In a German study, decaBDE was detected in 25 of 62 (40%) breast milk samples collected from women who were either omnivores (N=37) or vegetarians/vegans (N=25) (Vieth *et* al., 2004). On average, decaBDE made up 7% of total breast milk PBDEs, based on the 7 congeners measured (Vieth *et al*., 2004). In a study of first-time mothers from the Puget Sound area in the Northwest US, 5 of 9 (56%) milk samples contained decaBDE at measurable levels, with decaBDE levels generally contributing less than 1 percent to the total PBDEs based on analysis of 13 congeners.

In a study of Mexican women, the average level of decaBDE in plasma was 9.5 ng/g lipid (López *et al*., 2004), higher than any other congener. DecaBDE was also detected in breast milk, at comparable levels to Swedish breast milk measured in the same study.

A study of 20 women measured PBDEs in breast milk of first-time mothers in 13 states (EWG, 2003). Thirty-two congeners were measured. The dominant congener was BDE-47, followed by BDE-153. In one woman, BDE-153 was the dominant congener. DecaBDE was detected in 80% of samples, with a higher average level than 19 of the 32 congeners.

In a study comparing Swedish electronic dismantlers, hospital cleaners and computer clerks (Sjödin *et al*., 1999), highest levels were found in the electronics dismantlers. It is important to note that decaBDE was found in the blood of all three groups, with higher levels in the dismantlers.

The relative source contribution from various pathways (e.g. food, home environment) are unknown. Vegetarians/vegans have lower milk levels of total PBDEs (based on 9 congeners), congener BDE-47, and congener BDE-153 than omnivores by 15-40% (Vieth *et al*., 2004). The difference in body burden (here measured in breast milk) between vegetarians and omnivores is less than that for PCBs between vegans and omnivores (Schecter *et al*., 2001). This may represent in part the contribution of other pathways in addition to food. It almost certainly also represents the fact that the Schecter *et al*. study compared vegans, whereas the Vieth *et al*. study included other vegetarians in addition to vegans. “Vegetarians” consume dairy products (and sometimes even fish), which can be an important source of exposure. It would be interesting to compare PBDE levels in vegans to omnivores, and include decaBDE in the congeners measured. This would provide important information on food versus inhalation pathways.

The Vieth *et al*. (2004) study also found an orderly decrease in total PBDE levels in breast milk as the number of children breast-fed increased from one to two to three. This is expected based on data from PCBs and lipophilic pesticides. The mother decreases her body stores of these contaminants over successive pregnancies by excreting large quantities into breast milk.

A recent estimation of half-lives in human tissue (Geyer *et al*., 2004), modeled from rat data, indicates that half-lives increase from BDE-47 (tetra) through BDE-100 (penta), BDE-99 (penta), BDE-154 (hexa), and BDE-153 (hexa). The half-life of BDE-153 in adipose tissue is estimated to be between 8-16 years, whereas that of tetraBDE is estimated to be 2-4 years. It is well established that for PCBs, which have been in the environment for 50 more years than PBDEs, the congener pattern in human tissue is very different from that in commercial mixtures that are the ultimate source of exposure, with congeners with long half-lives predominating.

J. Transfer of PBDEs to the fetus and infant

Like other lipophilic compounds, PBDEs readily cross the placenta into the fetus. PBDEs are also found in high concentrations in breast milk. This provides the opportunity for PBDEs to interfere with developmental processes, producing effects not possible in the adult.

In a U.S. study, levels in umbilical cord and maternal serum were comparable on a lipid weight basis, based on six congeners (Mazdai *et al*., 2003). Total PBDEs in fetal blood was 39 ng/g lipid, and in maternal blood 37 ug/g lipid. Total PBDE levels varied 40-fold across the 12 women sampled. Compared to a similar study in Sweden (Guvenius *et al*., 2003), blood levels in U.S. mothers were 20- to 69-fold higher, and fetal blood was 30- to 106-fold higher. DecaBDE was not measured in either study.

A study in Japan compared PBDE levels (congeners 28 + 33, 47, 100, 99 and 153) in placenta, maternal and cord blood, and in breast milk of four women (Hirai *et al*., 2004). On a lipid basis. highest levels were found in breast milk in three of the four, with placenta containing the highest levels in the fourth. BDE-153 had the highest concentrations in 2 of the 4 women in at least one compartment.

A study in the Netherlands measured BDE congeners 47, 99, 100, 153 and 154 in maternal and cord blood (Weiss *et al*., 2004). Levels in the two compartments were comparable on a lipid basis (maternal blood has about 4 times as much fat as fetal blood). In maternal blood, BDE-153 was the dominant congener, whereas in fetal blood, BDE-47 dominated, followed by BDE-153.

The concentrations of PBDEs in placenta and fetal liver were measured in 15 samples collected between 1978 and 2003 in Canada (Doucet *et al*., 2004). Levels in fetal liver increased by about a factor of 10 between 1998 and 2003. The predominant congener was BDE-99, followed by BDE-47. DecaBDE was not measured. Concentrations in liver were much higher than placenta on a lipid basis, and higher than in Canadian human milk (Ryan *et al*., 2002). This suggests that the fetus is accumulating PBDEs.

Levels of PBDEs are three times higher in human breast milk than in maternal blood on a wet weight (rather than lipid adjusted) basis (Guvenius *et al*., 2003). Since PBDEs are lipid soluble, they are concentrated in breast milk because it has a much higher fat content than blood. Therefore the infant is exposed to PBDEs not only *in utero*, but also postnatally. This preferential distribution is also observed with PCBs. It is quite possible that some of the mechanisms of neurotoxicity for PBDEs are similar to those of PCBs, given the structural similarity and the available neurotoxicity data. In epidemiological studies with PCBs, effects on IQ were related to *in utero* exposure (Schantz *et al*., 2003). However, other behavioral effects were related to exposure through breast milk (Vreugdenhil *et al*., 2004; Patandin *et al*., 1999). It is therefore important to consider both prenatal and postnatal exposure to PBDEs.

K. Toxicity of PBDEs

The current data indicate that PBDEs can affect the nervous, endocrine, and immune systems. Individual PBDE congeners as well as commercial mixtures have been assessed for toxicity. PBDEs also produce induction of metabolic enzymes, and produce changes in liver at high doses. DecaBDE is the only congener that has been tested for carcinogenicity (ability to cause cancer). In contrast to the considerable new findings on the presence of PBDEs in environmental media and biota, there is only limited new data on their toxicology. Both existing and new findings are summarized below.

Neurotoxicity

A group of researchers in Sweden studied the behavioral toxicity of PBDEs, as well as PCBs, in two strains of mice during early postnatal development. The postnatal timeframe investigated in the mouse corresponds roughly to the last trimester of pregnancy in humans for most brain areas; thus, this exposure paradigm corresponds to late fetal exposure in humans with regard to developmental stage.

All studies used the same behavioral test: movement in a test chamber that was isolated within a sound-attenuated enclosure. Three types of movement were quantified electronically: horizontal movement around the chamber, vertical movement (rearing – a measure of exploratory behavior), and total cage movement that may also include scratching, grooming, etc. Mice were tested individually for 60 minutes following introduction into the unfamiliar chamber, and movement was recorded in three 20-minute intervals. This allowed assessment of habituation. Normal mice will decrease their activity over time, as they become familiar with the initially novel environment. This habituation is typically considered a simple test of learning, with failure to habituate considered evidence of cognitive impairment. However, failure to habituate may also result from other types of impairment, such as increased arousal or attentional deficits.

Mice were dosed once on postnatal day (PND) 3, 10, or 19. This regimen obviated the opportunity for the chemical to increase in the body over successive doses (exposures), and does not expose the developing mouse to the chemical over the entire period of development. It therefore does not mimic presumed environmental exposure.

All PBDE congeners examined affected behavior on this test. In fact, the pattern of effect was the same for BDE-47, BDE-99, BDE-153, BDE-183 (a heptaBDE), BDE-203 (octaBDE), BDE-206 (nonaBDE) and BDE-209 (decaBDE). Activity was reduced in PBDE-exposed mice relative to controls during the first 20-minute portion of the session, but treated mice failed to habituate over the 60-minute trial period. These effects were the same in the outbred NMRI mouse (Eriksson *et al*., 2001, 2003, 2004b; Viberg *et al*., 2002, 2003a, b) as well as for BDE-99 tested in the inbred C57/B1 mouse (Viberg *et al*., 2004). Mice were tested beginning at two months of age, and were tested at several ages thereafter depending on the study, up to 8 months of age (well into mouse adulthood). In all cases, effects were permanent.

Focusing specifically on the study with BDE-209 (decaBDE) (Viberg *et al*., 2003b), pups were dosed on PND 3 or 19 with 2.22 or 20.1 mg/kg body weight, or on PND 10 with 1.34, 13.4, or 20.1 mg/kg body weight. Pups were tested at 2, 4, or 6 months of age (i.e., a different group of mice at each age). Effects were only observed when pups were dosed on PND 3. Both doses of BDE-209 produced effects, with the higher dose having a greater effect. The higher dose produced a failure to habituate at all three ages, as well as decreased activity during the first 20 minutes. The low dose produced decreased activity during the first 20 minutes on a single measure of activity (not all three) at 2 and 6 months of age. Effects were observed at 0.9 mg/kg with congener BDE-153 (Viberg *et al*., 2003a), at 0.8 mg/kg for BDE-99 (Viberg *et al*., 2004), and 10.5 mg/kg for BDE-47 (Eriksson *et al*., 2001). The relative potency of BDE-209 to these other congeners is unknown, since doses below 2.22 mg/kg have not been examined at PND3. However, the available data suggest that BDE-209 is approximately equipotent.

The fact that effects were only produced following dosing at PND 3 stands in contrast to effects of the lower brominated congeners, in which effects were produced following exposure on PND 3 or 10. The authors postulate that the observed effects were produced by a metabolite or metabolites, since total radioactivity in the brain was about the same 24 hours and 7 days after dosing at either PND 3 or 10. Since the peak of the brain growth spurt in the mouse is about PND 10, it is hypothesized that the radioactivity seen at day 10 after PND dosing on PND 3 constitutes active metabolites, whereas administration at PND 10 is ineffective because only the parent compound is present at the critical window of development. In contrast, for the lower brominated congeners, administration at PND 10 is effective because the parent compound is active, and PND 3 administration is effective because there is still sufficient parent and/or metabolite in the brain at the critical PND 10 period. Interestingly, congener BDE-206 (a nonaBDE) was also only active when administered on PND 3, whereas the BDE-203 (octaBDE) was active when given on PND 3 or 10 (Eriksson *et al*., 2004b). The authors are pursuing speciation of the congeners to test their hypothesis (P. Eriksson to D. Rice, personal communication).

The effects seen on this behavioral paradigm with PBDEs are identical to those produced by PCBs (Eriksson and Fredriksson, 1996a, b). PCBs are neurotoxic in humans, producing deficits in IQ, attention, memory, and social behavior, as well as a number of other behavioral effects (Schantz *et al*., 2003; Rice, in press). This suggests that the effects observed on this paradigm may be predictive of effects in humans.

The Swedish work described above has been criticized because littermates were used as independent observations in the statistical analysis: in other words, they were treated in the statistical analysis as if they were unrelated. The accepted procedure is that the litter (and not the individual) is the unit of statistical analysis. Violation of this principle may result in invalid statistical inference. However, in this case, the effects have been repeated multiple times with multiple PBDE congeners, and in two strains of mice, providing assurance that the results are unlikely to be spurious. In addition, the experimental paradigm has been repeated with congener BDE-99 using the litter as the statistical unit (Eriksson *et al*., 2004), and the results were identical to those from previous experiments that used littermates as the unit. This lends confidence that the effects are real and reproducible.

In a behavioral study assessing the effects of congener BDE-99 on different behavioral endpoints, dams were dosed from gestational day (GD) 6 to PND 21, and offspring were studied on a number of behaviors (Branchi *et al*., 2002). Effects were seen on motor ability and locomotor activity.

Mice dosed with a single dose of BDE-47 on GD 6 were hyperactive at 80 days of age (Kuriyama *et al*., 2004a). There was also evidence that exposed males behaved more like females, which are normally more active, suggesting that the effects may be hormonally mediated (feminization of males).

Congener BDE-153, administered at PND 10, produced deficits in spatial memory in a water maze task at 6 months of age (Viberg *et al*., 2003a).

PBDEs have been found to be neurotoxic in various *in vitro* assays (Mariussen and Fonnum, 2003; Kodavanti and Derr-Yellin, 2002), suggesting possible mechanisms of PBDE-induced toxicity. There is also evidence that PBDEs affect the cholinergic neurotransmitter system (Eriksson *et al*., 2001; Viberg *et al*., 2002, 2003a), a system that is involved in memory and motor function, among others.

The effects of several PBDE congeners have been compared to PCBs for their ability to affect intracellular signaling in a cerebellar (brain) culture system (Kodavanti and Ward, 2004). The signaling pathways studied assessed known mechanisms of neurotoxicity for a number of chemicals, including PCBs. The order of potency was DE-71 (a commercial mixture of tetra-, penta-, and hexa-BDEs) > 47 > 100 > 99. On a molar basis, DE-71 was equipotent with Aroclor 1254, the most widely used commercial PCB mixture. The Swedish group found that BDE 99 and PCB 52 produced effects on behavior when given together but not at the same dose given alone (Eriksson *et al*., 2003). These results suggest that there may be little difference in potency between PBDEs and PCBs for neurotoxicity, and that effects are additive. This implies that body burdens of PCBs and PBDEs in humans may need to be added when assessing risk.

Effects on the endocrine system

PBDEs, like PCBs, affect thyroid function. In the adult, thyroid hormone is important for energy metabolism. However, during fetal and infant development, thyroid hormone is critical to the development of the nervous system. For example, even a small deficit in maternal thyroid hormone, but still within the range that is considered to be clinically “normal”, results in an increase in children with decreased IQs (Haddow *et al*., 1999).

Exposure to mixtures of tetra- through octa- mixtures decreased thyroid hormone in rats during a period of critical brain development (GD 20 to PND 14) (Zhou *et al*., 2001, 2002). DecaBDE was ineffective when given for only four days, with thyroid hormone measured on Day 5. It may be that effects would have been observed if sufficient time had been allowed for metabolism of decaBDE to active compounds.

A single dose of BDE 47 on GD 6 in rats produced decreased thyroid hormone levels in the dams during the period of nursing (Kuriyama *et al*., 2004c). This single dose of BDE 47 also produced induction of specific liver enzymes (i.e. production was increased). Such changes in enzymatic activity can affect the fetus by changing levels of natural hormones or nutrients, or parent compound or metabolites of chemicals or drugs.

In a two-year chronic cancer study (NTP, 1986), decaBDE produced thyroid follicular cell hyperplasia, indicative of disrupted thyroid function. Thyroid hormone levels or other measures of thyroid function were not included in this study.

Female and male rats were exposed to the commercial mixture DE-71 for up to 30 days during the juvenile period (Stoker *et al*., 2004). Serum thyroid hormone levels were decreased in both sexes, with some effects observed in the lowest dose tested (3 mg/kg body weight/day). Specific liver enzymes were induced, and there was an increase in the liver-to-body weight ratio. Seminal vesicle and prostate weights were reduced in males given the highest dose, an effect that is androgen-dependent. Effects on sexual development were also present in the female. The effects on sexual development may be direct effects of PBDEs, or secondary to the effects on thyroid hormones.

The effects of BDE 99 following gestational exposure were studied on sexual and reproductive parameters in rats (Lichtensteiger *et al*., 2004). Sexual behavior was decreased in females. (Males were not assessed). The activity of specific genes involved in sexual and reproductive performance was decreased in both sexes. The adipose tissue levels at 120 days of age overlapped those found in some wildlife, and were only four times higher than levels in humans. A no-effect level was not identified.

A single dose of BDE 99 on GD 6 produced hyperactivity at 36 and 71 days of age in both sexes (Kuriyama *et al*., 2004c). BDE 99 also produced a decrease in the weight of male sexual organs and male sexual performance. Female reproductive capacity was not assessed in that study.

The Xenopus (frog) tail regression model has been used to study the thyroidogenic potency of DE-71, as well as congeners 47 and 99, both of which are major constituents of DE-71 (Balch *et al*., 2004). Tail resorption by tadpoles is one of the last changes in metamorphosis, and is under control of thyroid hormone. DE-71 altered tail resorption, whereas 47 or 99 did not, suggesting that other congeners present in DE-71 are the active agent.

In a series of *in vitro* assays, a number of congeners and their metabolites were potent in binding to thyroid, androgen, and/or estrogen receptors (Hamers *et al*., 2004). DecaBDE was inactive in these assays.

Immune effects

The immune effects of some PBDEs have also been studied, and there is evidence that some congeners are immunotoxic (similar to PCBs). The immunotoxic potential of decaBDE has not been assessed. DE-71 produced a suppression of the ability to mount an immune response in adult mice following subchronic exposure (Fowles *et al*., 1994). Decreased number of splenocytes and decreased production of IgG antibodies in response to a challenge were observed following administration of Bromkal 70-5DE (a commercial mixture of penta- and hexaBDE) or BDE-47 in rats and/or mice (Darnerud *et al*., 1999). PCBs had similar effects in this study. The immunosuppressive potential of tetra-, penta-, or hexaBDEs was assessed in adult mice (Howie *et al*., 1990). All congeners suppressed the splenic plaque-forming cell (PFC) response, with potencies varying among congeners. In an *in vitro* study in human lymphocytes (Fernlof *et al*., 1997), neither PCBs nor PBDEs had an effect on cell proliferation. In contrast, it is well established that PCBs are immunotoxic *in vivo* in humans. The negative results of the *in vitro* study are presumably the result of the fact that PCBs (and perhaps PBDEs) exert their effect through an indirect mechanism.

Carcinogenicity

Only decaBDE has been assessed for carcinogenicity in a chronic bioassay, in a study in rats and mice by the U.S. National Toxicology Program (1986). Female and male B6C3F$_1$ mice were fed a diet containing decaBDE (94-99% pure, no brominated dioxins or furans) for 103 weeks. Administered doses were high—3760 or 7780 mg/kg bw/day for females and 3200 or 6650 mg/kg bw/day for males. However, uptake of decaBDE was low, about 1.3%, so the internal dose was much lower. Non-significant increases in combined hepatocellular adenomas and carcinomas were observed at both doses in males, and combined thyroid follicular cell adenomas and carcinomas in both sexes. In rats, females were dosed with 1200 or 2550 mg/kg bw/day and males with 1120 or 2240 mg/kg bw/day for 103 weeks. A significant dose-dependent increase in the incidence of adenomas of the liver was observed in both sexes, and there was a significant dose-dependent increase in acinar cell adenoma in males. The U.S. EPA classifies decaBDE as a possible human carcinogen.

L. Current research activities in Maine

It is likely that the developing nervous system is particularly vulnerable to PBDEs, including decaBDE. There is only one behavioral study on the developmental neurotoxicity of decaBDE, in mice given a single dose in the early postnatal period. Although the effects of decaBDE are consistent with the effects of other PBDEs and PCBs on the same behavior task, it would be useful to further characterize the behavioral effects produced by decaBDE. In addition, the exposure paradigm for the study performed to date used a single administration, which does not mimic human environmental exposure.

The Bureau of Health is in the process of developing a contract with the University of Southern Maine to perform a number of sophisticated behavioral analyses in mice exposed developmentally (postnatally) to decaBDE. Tests will include learning, attention and activity. Pups will be dosed daily from PND 2 through 20, which better mimics human exposure.

There currently are few Maine-specific data on levels of PBDEs in the environment. A study underway at the Maine Environmental Research Institute in Blue Hill is analyzing tissue PBDE levels in harbor seals and fish. In addition, a few samples of fish, waste water treatment effluent and sludge from sites on the Penobscot River have been analyzed for PBDEs as part of thesis research at the University of Maine. The fish tissue analyzed in this research was procured through the DEP Surface Water Ambient Toxic Monitoring Program (SWAT monitoring program). The preliminary results of the research are reported in Appendix B.

The SWAT monitoring program was established in 1993 to determine the nature, scope and severity of toxic contamination in Maine's surface waters and fisheries. The program must incorporate testing for suspected toxic contamination in biological tissue and sediment, and must collect data sufficient to assess the risk posed by these contaminants to human and ecological health (see 38 MRSA §420-B). PBDEs were added to the program this year. Five bass taken from the Androscoggin River at Lisbon and five from the Penobscot River at Veazie will be analyzed for BDE-47, BDE-99, BDE-100, BDE-153, BDE-154, BDE-183 and BDE-209. The results of these initial analyses likely will be published in March 2005 as part of the 2004 annual report for the SWAT program (B. Mower to J. James, personal communication).

IV. Alternatives to decaBDE

Little new information on alternatives to the use of decaBDE as a flame retardant has emerged in 2004. The principal documents on this topic remain a 1999 Danish study (Lassen *et al.*) and a 2001 German study (Leisewitz *et al.*).

The Lowell Center for Sustainable Production (LCSP) at the University of Massachusetts Lowell expects to publish a report on alternatives to decaBDE early in 2005 (T. Greiner to J. James, personal communication). The U.S. Environmental Protection (EPA) also reportedly plans to evaluate alternatives to the use of decaBDE in textiles as the second phase of a "Design for the Environment" analysis currently being conducted for pentaBDE. EPA is carrying out this analysis in consultation with textile manufacturers, furniture manufacturers, foam manufacturers and flame retardant manufacturers (Peele, 2004).

In the meantime, the 1999 Danish study suggests three possible ways to eliminate the use of decaBDE:

1. Substitute <u>another flame retardant</u> without changing the base polymer (i.e., the type of plastic);
2. Replace the base polymer with <u>another type of polymer</u>; or
3. Replace the base polymer with <u>another type of material</u>, or achieve flame resistance by <u>a different solution</u> (e.g., use of a metal sheet over plastic in contact with current-carrying parts in electronics).

A. Alternatives to the use of decaBDE in HIPS

In the case of decaBDE, the main challenge is to find alternatives for the high impact polystyrene (or HIPS) currently used in the outer casings of electronic equipment, or to find another type flame retardant that can be used in HIPS without compromising its properties. Among the factors to consider in selecting an alternative are:

- The physical and chemical properties of the alternative during manufacturing;
- The physical and chemical properties of the finished product;
- The environmental and health risk of the alternative during manufacturing, use and disposal;
- The price of the alternative; and

The cost of changes in the manufacturing process (e.g., tools and machinery) necessary to accommodate the alternative (Lassen *et al.*, 1999).

The Danish study suggests that feasible alternatives exist for most decaBDE applications, but that the use of the alternatives depend on user willingness to accept cost increases or technical disadvantages. The website of the Great Lakes Chemical Corporation seems to support this, listing numerous deca-free products as suitable for use to flame retard HIPS.[13] According to a bromine industry fact sheet, however, many potential substitutes for decaBDE do not have the mechanical properties, thermal stability and other technical performance requirements needed for particular applications.[14]

Swiss chemical manufacturer Clariant, in comments to the European Commission, writes that phosphorus based flame retardants are not suitable for HIPS. According to the company, however, plastics other than HIPS "can be used for the same applications albeit at slightly higher prices." Computer housing, for example, can be made of co-polymers of polycarbonate and styrenics (PC-ABS) or polyphenylene oxide (PPO) and polystyrene, with phosphorus based flame retardants like aryl phosphates."[15] Clariant adds:

> "…offering a more environmentally friendly product is often not sufficient for market success, even if the price and technical properties are comparable to established flame retardants. Therefore, legally binding requirements together with market pull—manufacturers and consumers of end use products demanding more environmentally friendly solutions—will encourage the development of alternative non-halogen flame retardants. Once they gain a considerable share of the market, economies of scale can materialise and lower the cost of alternatives."

The Computer Take Back Campaign, a national coalition promoting clean production in the computer and electronics industry, reports that manufacturers in Japan and Europe increasingly are switching from HIPS to a PC/ABS polymer mixture[16] that can be flame retarded with organic phosphorus compounds. The coalition further reports that two companies—Bayer AG and GE Plastics—have patented PC/ABS plastic flame retarded with resorcinol-bis-diphenylphosphate (RDP). Two others companies—Dow and BASF—reportedly are developing organic phosphorus flame retardants (McPherson *et al.*, 2004).

B. Alternatives to use of decaBDE in coated wire and cable

A study by the Toxics Use Reduction Institute (TURI) at the University of Massachusetts Lowell reports that decaBDE is one of eight main fire retardants (seven if pentaBDE is excluded) used in coated wire and cable (Harriman *et al.*, 2003). The others include:

- Alumina trihydrate (ATH);
- Antimony trioxide;
- Brominated phthalate ester (in PVC plenum cable);
- Ethylene 1,2 bis(tetrabromophthalimide) (EBTBP or Saytex® BT-93);
- Magnesium hydroxide; and
- Zinc borate.

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[13] Great Lakes Chemical Corporation, "High Impact Polystyrene," www.e1.greatlakes.com.

[14] Bromine Science and Environmental Forum, "Fact Sheet: Why DecaBDE is the Best Fire Safety Option," www.bsef.com.

[15] R. Waltz and A. Beard of the Clariant Corporation, European Commission Stakeholder Consultation on Deca-BDE, June 29, 2004.

[16] A PC/ABS polymer mixture is one that combines the polycarbonate plastics (PC) with acrylonitrile butadiene styrene (ABS).

Some of these alternatives may not meet U.S. fire retardancy standards. For example, TURI notes that the European market for data and communications cable is dominated by products that use aluminum trihydrate and magnesium hydroxide as a flame retardant. These products pass the most stringent fire test mandated in Europe but do not meet the flame retardancy standards of the U.S. National Electrical Code (Harriman *et al*).

On the other hand, TURI found that there have been significant advances in the development of safe, globally acceptable alternatives to decaBDE. No single alternative works in all applications, however. Different solutions have to be sought for different applications. The electrical performance, processability and cost of the available alternatives are potential barriers. The wire and cable industry has taken several approaches to meet this challenge, including switching to materials that do not require flame retardant additives and substituting decaBDE with environmentally safer alternatives (Harriman *et al.*)

The Central Maine Power Company (CMP) reports that it uses control and instrumentation cable containing decaBDE at its electrical substations.[17] Deca-containing insulation coats the individual conductors that make up the cable core. The insulated conductors are cabled together, wrapped in Mylar and then jacketed with steel or plastic such that the deca-containing insulation is encapsulated and isolated from the environment while in use. The cables have a minimum expected useful life of 40 years, at which point they typically are sold to a metals recycler.

CMP reports that cable using a bromine free flame retardant is available from a Canadian supplier but costs about 45% more. CMP does not know the specific type of flame retardant used in this alternative and has not investigated the alternative to determine if it is as effective or as durable as the deca-containing cable CMP currently uses.

C. Alternatives to use of decaBDE in textiles

In April 2000, the National Research Council (NRC) released a report on 16 chemicals (or chemical classes) likely to be used in upholstery fabric to meet a flammability standard under consideration by the U.S. Consumer Product Safety Commission (CPSC). Although the standard under consideration does not stipulate the method of compliance, manufacturers say they would be inclined to use these chemicals to meet the proposed performance requirements (Babich and Thomas, 2001). The chemicals typically are mixed with an acrylic or vinyl polymer that is applied to the back of the fabric. Flame retardant back-coating is used mainly with synthetic fabrics.

Eight of the 16 chemicals evaluated by the NRC were determined to pose little or no health risk to people who may be exposed to them in the home. The eight chemicals deemed safe for use on upholstery fabrics are: hexabromocyclododecane; alumina trihydrate; magnesium hydroxide; zinc borate; ammonium polyphosphates; phosphoric acid; tetrakis hydoxymethyl phosphonium chloride; and decaBDE. Although the safety of decaBDE subsequently has been called into question, the study suggests that at least seven safe alternatives to decaBDE are available to make residential furniture flame resistant.

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[17] Mirabile *supra* n. 12.

It should be pointed out that the NRC did not conclude that the eight other chemicals it examined are unsafe for use in furniture. Rather, the council was unable to rule out the potential for adverse effects given limited data. Further studies may show one or more of these chemicals to be safe. The eight chemicals identified as requiring further analyses are: antimony trioxide; antimony pentoxide and sodium antimonates; calcium and zinc molybdates; organic phosphonates; tris (monochloropropyl) phosphates; tris (1,3-dichloropropyl-2) phosphate; aromatic phosphate plasticizers (tricresyl phosphate); and chlorinated paraffins.

It also should be pointed out that NRC was charged only with the reviewing the toxicological and exposure data on the 16 chemicals. It was not charged with comparing the efficacy and costs of the chemicals.

In April 2001, CSPC staff issued their own assessment of the 16 flame retardant chemicals evaluated by the NRC (Babich and Thomas, 2001). The CSPC conclusions are similar to those of the NRC, and further demonstrate that a number of flame retardant chemicals are available to safely treat upholstery fabrics if decaBDE ultimately is shown to be unsafe and is removed from the market.

In December 2003, the EPA Office of Pollution Prevention & Toxics initiated a joint venture called the Furniture Flame Retardancy Partnership "to better understand fire safety options for the furniture industry."[18] Participants include furniture makers, chemical manufacturers, environmental groups, the American Fire Safety Council and CSPC. The goal of the partnership is to identify and move toward environmentally friendly approaches to meeting fire safety standards for foam and fabric.

EPA formed the partnership in anticipation of final adoption of a flame retardancy standard by the CPSC. The partnership will look not only at the use of chemical flame retardants, but also at the use of inherently flame retardant materials and designs. Cost, functionality, the environment, human health and fire safety will be considered. The partnership expects to make the results of its review of flame retardant options available in 2005-2006.

One promising design alternate under consideration by the partnership is the use of barrier technologies in which fire retardancy standards are achieved by layering a barrier material between the fabric and foam padding. Many types of barrier materials can be used, including fabrics composed of fibers that are inherently flame retardant. Manufacturers of bus and airplane seating have taken this approach, using a blend of natural and synthetic fibers. Mattress and furniture manufactures also are using barrier technology to meet fire safety standards in California (U.S. EPA, 2004).

In September 2004, the U.S. Senate Committee on Commerce, Science and Transportation unanimously approved a bill (S. 1798, 108th Congress) that would require the CSPC, if it has not promulgated a national flame retardancy rule for upholstered furniture by June 1, 2005, to transmit a report to Congress explaining why the rule was not issued. The bill, cosponsored by Senator Snowe of Maine, also calls for the CPSC to create a registry of chemical flame

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[18] U.S. EPA, Furniture Flame Retardancy Partnership, www.epa.gov/oppt/dfe/projects/flameret.

retardants. The registry must include:

- A list of all flame retardant chemicals used in products regulated by the CPSC and a list of studies;
- A list of flame retardant manufacturers; and
- A list of the studies and reports performed by the manufacturers on the human impacts of the chemicals.

The purpose of the registry is to fill gaps in information needed to evaluate the various flame retardant chemicals. A companion bill (H.R. 4233) is still in committee in the U.S. House of Representatives.

As of October 2005, the CPSC was still undecided about the scope of a flame retardancy standard for furniture. At issue is whether the standard should protect against exposure to a small open flame or a smoldering ignition source like cigarettes (K. Vokes to J. James, personal communication).

D. EU stakeholder consultation on decaBDE

The European Commission, in preparation for a possible amendment to its ban on the use of decaBDE in electrical and electronic equipment,[19] solicited responses from stakeholders to the following questions:

Do feasible substitutes to decaBDE currently exist in an industrial or commercial scale?
Do any restrictions apply to such substitutes?
What are the costs and benefits and advantages and disadvantages of such substitutes?
Responses to the solicitation are posted on the Commission website[20] and will be considered by the Lowell Center for Sustainable Production in its forthcoming report on alternatives to decaBDE. Table 4 below, listing alternatives to decaBDE, is reproduced from comments submitted to the Commission by Akzo Nobel Chemicals, a manufacturer of phosphorus-based flame retardants. Akzo Nobel was recently acquired by a U.S. company called Ripplewood Holdings LLC. In its comments, Akzo Noble observes:

> "The influence on the polymer cost of substituting decaBDE is usually to increase cost. However, in the case of HIPS, leading European and Japanese television manufacturers took the decision to change from decaBDE to a non-bromine solution as summarized in [Table 4].
>
> DecaBDE is applicable to a wide range of polymers but alternatives are often restricted to specific polymers. For example there are several different versions of polyamide sold for

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[19] See Directive 2002/95/EC of the European Parliament restricting the use of certain hazardous substances, including PBDEs, in electrical and electronic equipment beginning July 1, 2006. An annex to the directive requires the European Commission to evaluate decaBDE as a matter of priority in order to establish as soon as possible whether the directive should be amended to exempt decaBDE. The commission is required to consult stakeholders before amending the directive.

[20] Http://europa.eu.int/comm/environment/waste/weee_index.htm.

> electrical parts. DecaBDE works in all cases but this is not the case for non-bromine products. Each polyamide needs a different flame retardant product. In some cases, these are available but not yet in the volumes necessary to replace decaBDE completely. On the other hand, deca has limited use in nylons. A wide variety of other halogenated flame retardants other than deca are used depending on application."

Table 4: DecaBDE applications and alternatives

Polymer/Application	**Alternative available**	**Comments**
Polypropylene	Yes	Substitutes have technical problems and require high addition levels, increasing the cost significantly.
High Impact Polystyrene(HIPS)	Yes	HIPS can only be made to an equivalent flammability by blending with PPE(PPO) polymer and using a phosphate ester where Fyrolflex® RDP or Fyrolflex® BDP are the products of choice. This is more expensive than normal HIPS but is currently accepted by the market.
PBT	No	In principle, there are alternatives, but these are not applicable due to high costs and they do not meet the formulation requirements
Polyamide	For some polyamides	Each polyamide needs a different non-bromine flame retardant.
Textile Coating	Yes	Needs reformulation but phosphate ester based products are applicable and implemented in limited volumes.
Thermoset resins(polyester/epoxy)	Yes	

Others responding to the EC's stakeholder consultation include the Clariant Corporation, the American Electronics Association, the European Industry Association, the Japan Business Council in Europe and five Japanese Industry Associations.

Clariant

Portions of Clariant's comments to the EC were excerpted above in the discussion of alternatives to the use of decaBDE in HIPS. Clariant further writes:

> "The bromine industry has come up with substitutes for PBDEs like polybrominated diphenyl ethanes. However, these have been only used reluctantly, seemingly not for technical reasons, but because they are more expensive and less toxicological and environmental data are available."

And on the matter of the costs and benefits of decaBDE substitutes, Clariant writes:

> "Flame retarding polyamide or polyester with [phosphorus based chemicals] will result in compounds which are (at current prices) comparable or only slightly more expensive than compounds with brominated flame retardants like PBDEs…Phosphinates are more expensive on a per kg flame retardant basis, however, realistically the price per volume of flame retarded polymer compound should be compared. The necessary dosage of phosphinates is lower compared to brominated flame retardant systems which usually consist of the brominated flame retardant plus antimony trioxide as a synergist, i.e. phosphinates are more effective…"

As to the fire safety of decaBDE substitutes, Clariant writes:

> "Fire safety of products is defined by flammability tests which have to be met. If the test is passed, then the required level of fire safety is achieved. Only the minimum amount of flame retardants is added to safely pass the required flammability test. The required quantities differ vastly between the various kinds of flame retardants and can range from a few percent to more than 50 percent. The most efficient flame retardant in terms of quantity may not be the optimum solution, because it may be more expensive even in low amounts or it may have negative environmental or health properties. Non-halogenated flame retardants[21] have the advantage that they do not produce toxic halogenated dioxins and furans as well as no corrosive smoke which might damage sensitive equipment and parts of a building. Furthermore, only halogenated flame retardants need antimony compounds as synergists."

American Electronics Association and European Industry Association

These two associations wrote that although "there are potential substitutes for [decaBDE] they have functional disadvantages…These substitutes would need to be added to a much higher percentage thus changing important characteristics of the designed parts."

Japan Business Council of Europe

The council wrote that the cost of available substitutes for decaBDE "seems higher. Halogen free flame retardants in particular are significantly more expensive."

Japan Electronics & Information Technology Industries Association
Communications and Information Network Association of Japan
Japan Business Machine and Information System Industries Association
Japan Electrical Manufacturers Association
Japan Refrigeration and Air Conditioning Industry Association

These five associations noted that there currently is no feasible substitute for the use of decaBDE as a flame retardant binder for filters used in commercial and industrial ventilation systems. The comments note that possible alternatives exist, but their flame-retardant effectiveness has not

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[21] decaBDE is halogenated in that it contains molecules of bromine which is a halogen.

been verified and they cost more. The associations did not give any indication as to the extent of deca usage for this application or whether the industry is making any effort to develop PBDE-free alternatives.

E. Manufacturers who have switched to decaBDE alternatives

A number of electronics manufacturers have ended or are phasing out the use of decaBDE in their products. They include Apple, Brother, Dell, Hewlett Packard, IBM, Intel, Panasonic/Matsushita, Samsung, Sharp, Sony, Toshiba and Xerox (McPherson *et al.*, 2004; Peele, 2004).

Dell, for example, avoids the use of BFRs by using plastics that can be flame retarded with phosphorus-based products and by using design strategies that avoid the need to use any flame retardants. According to the Dell website:

> "Some plastic types cannot be flame-rated with anything other than bromine because reliable alternative technology does not currently exist. As such, we try to avoid these types of plastics where we need flame retardancy."[22]

In January 2004, the NEC Corporation announced that it has developed a biomass-source plastic that is flame resistant without adding BFRs or phosphorous compounds. The bio-plastic meets UL flame resistance standards. It also boasts other properties such as heat resistance, moldability and strength comparable to the petroleum-based polymers currently used in desktop electronics.[23]

The furniture maker IKEA decided to stopping using BFRs in 1999. It accomplished this goal in just three years by changing product design, using naturally less flammable materials, and using phosphorus compounds where flame retardants are still needed (Environmental Working Group, 2003). Magnus Bjork of IKEA North America said the company made the switch when it became clear that BFRs are persistent and bio-accumulative, and share parallels with two banned chemicals—PCBs and DDT (Bjork, 2004). Mr. Bjork identified resistance to change as the biggest obstacle to going bromine free. He said that large companies like IKEA naturally resist changing what they are doing. They are invested in the existing techniques and do not want to take the economic risk of being the first to offer something other than the standard solution to fire safety (Bjork, 2004).

In 1989, German plastics and textile manufacturers voluntarily stopped producing and using decaBDE, suggesting that suitable alternatives have long been available (Lassen *et al.*, 1999; Leisewitz *et al.*, 2001). The use of decaBDE is not prohibited in Germany, however, and the import of deca-laden products appears to have undermined the voluntary agreement according to the German Federal Environmental Agency.

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[22] Dell, Inc., "Dell's Position on Brominated Flame Retardants," http://www1.us.dell.com.

[23] Japan Corporate News Network, press release, January 26, 2004.

V. DecaBDE and the regulatory environment

A. U.S. Environmental Protection Agency

TSCA

EPA is collecting toxicology data on decaBDE but there is not enough evidence to support regulatory action under the Toxic Substances Control Act (TSCA)[24] according to Ken Moss, a policy analyst for the USA EPA's Office of Pollution Prevention. "We can't act on the precautionary principle at this point, as much as some of us would like to" (Betts, 2004; Moss, 2004).

TSCA is the main vehicle for federal action to control toxic substances. Under TSCA, EPA classifies chemicals as "existing" or "new." An existing chemical is one that was listed in EPA's initial TSCA Inventory as being already in commerce at the time TSCA was enacted in 1976. DecaBDE is one of more than 63,000 existing chemicals under TSCA (Environmental Working Group, 2004a).

Section 4 of TSCA authorizes EPA to require companies to conduct testing on the health and environmental effects of existing chemicals, but EPA has not yet exercised this authority for decaBDE. Before the agency can require such testing, it must first show either (1) that the chemical may present an "unreasonable risk" or (2) that it is produced in major quantities and a "substantial" number of humans are being exposed to it. EPA also must show that existing data are insufficient and that testing is necessary.

Section 6 of TSCA gives EPA authority to ban an existing chemical if there is a reasonable basis to conclude that its distribution in commerce "presents or will present an unreasonable risk of injury to health or the environment." The burden is on EPA to prove the chemical presents an unreasonable risk, and EPA has been able to sustain this burden for only a handful of chemicals (e.g. PCBS and lead paint since TSCA was enacted.

Mr. Moss doubts that the EPA will ever be in a position to ban decaBDE under the current TSCA regulatory scheme, which places the burden on the agency to show that decaBDE is unsafe and that the public health benefits of a ban outweigh the costs (Moss, 2004). He points out that EPA was not even able to sustain this burden for asbestos.

Legislation has been introduced to the U.S. Congress that would amend TSCA to:

1. Make it unlawful for any person to manufacture, process or distribute in commerce a product containing more than 1 percent of pentaBDE or octaBDE by mass;
2. Require EPA to determine whether pentaBDE or octaBDE are formed in the environment as a result of chemical degradation of any other material and to identify any such precursors; and

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[24] 15 USC §§ 2601, et seq.

3. If precursors are found, to take action to ensure that products and processes that introduce such precursors are phased out unless EPA finds that to do so would endanger human health and the environment.[25]

The bill was referred to the House Committee on Energy and Commerce on March 30, 2004, and referred to the Subcommittee on Environment and Hazardous Materials on April 8, 2004.[26] The subcommittee has not yet taken up the bill.

VCCEP

Although EPA has not required testing of decaBDE under TSCA, the bromine industry has agreed to review the chemical under EPA's Voluntary Children's Chemical Evaluation Program (VCCEP). Under this program, the EPA asks manufacturers to sponsor evaluation of the chemicals they make. Sponsorship requires the manufacturers to collect or develop health effects and exposure information on decaBDE and then to integrate that information in a risk assessment and a data needs assessment. The assessments then are evaluated by a group of scientific experts using a peer consultation process.

The VCCEP evaluation of decaBDE was carried out by the Brominated Flame Retardant Industry Panel (BFRIP) of the American Chemistry Council. BFRIP released its assessment on December 17, 2002. The VCCEP peer consultants then met to review the manufacturer data and released their report on September 30, 2003 (BFRIP 2003). The report identified several data gaps.

EPA currently is conducting an independent review of the BFRIP assessment and the peer consultant report. The results of EPA's review had not been released as of November 20, 2004, but are expected in the next few months.[27]

PBDE action plan

The EPA is developing a PBDE Action Plan for release in 2005. The plan is being characterized as a blueprint rather than a detailed strategy. It will describe actions being taken under current EPA programs and programs at other federal agencies, and it will identify additional activities that are appropriate and necessary. The plan is expected to include an assessment and evaluation of decaBDE. See Appendix D of this report for a summary of the EPA's current understanding of PBDEs.

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[25] H.R. 4076, 108th Cong., 2d Sess.

[26] Thomas: Legislative Information on the Internet. Lib. Of Congress, December 21, 2004.

[27] U.S. Environmental Protection Agency, Office of Pollution Prevention & Toxics Prevention, "Polybrominated diphenyl ethers (PBDEs)," http://www.epa.gov/oppt/pbde/index-old.htm.

B. European Union

The European Parliament and Council of the European Union have adopted the following directives:

- Directive 2002/95/EC of January 27, 2003 restricts the use of certain hazardous substances in electrical and electronic equipment. This directive is referred to as the "Restrictions on Hazardous Substances" or "RoHS." Among other things, the RoHS requires member states to ensure that new electronic and electrical equipment put on the market after July 1, 2006, does not contain PBDEs.
- Directive 2003/11/EC of February 6, 2003 requires member states to adopt laws that prohibit the marketing or use of any product containing more than 1% by mass of pentaBDE or octaBDE effective August 15, 2004.

There has been some confusion about whether decaBDE is covered by the RoHS, the first of these two directives. Margot Wallström, the European Union's Commissioner for the Environment, attributes the confusion to a discrepancy between the text of the RoHS as approved by the European Parliament and the text as published in the Official Journal of the European Union. Ms. Wallström has opined, based on the clear intent of the legislators, that decaBDE is at present banned by the RoHS and that the ban will be subject to review based on the draft risk assessment recently completed by France and the United Kingdom.[28] She points out, however, that only the European Court of Justice can give a binding interpretation.

In February 2004, France completed an updated risk assessment on the health effects of decaBDE (European Commission, 2004a). The United Kingdom completed an updated risk assessment on the environmental effects of deca in May 2004 (European Commission, 2004b). The updated assessments were conducted in follow-up to a risk assessment published in 2002 that identified a number of areas of uncertainty concerning decaBDE.

Neither France nor the UK identified a clear risk warranting further regulatory controls on decaBDE. France concluded that there is at present no need for further information on the human health effects of decaBDE, or for risk reduction measures beyond those being applied already under the directives noted above. The UK concluded that decaBDE is "likely to be very persistent, but not bioaccumulative nor toxic in the marine environment." The UK did identify areas of uncertainty, however. Specifically, the report says that the risk assessment is complicated by available data on:

- The widespread occurrence of decaBDE in top predators (e.g. birds and mammals, including terrestrial species) and the Arctic;
- The neurotoxic effects and uptake of decaBDE by mammals in laboratory studies; and
- The possibility that decaBDE in the environment degrades into more toxic and accumulative products, such as pentaBDE or other lower brominated diphenyl ethers

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[28] Letter from Margot Wallström to European Parliament members, March 29, 2004.

and dibenzofurans.

In light of this data, the UK assessment report recommends continued monitoring of environmental contamination for both decaBDE and, if possible, its more toxic and bioaccumulative degradation products. The report suggests, at a minimum, that estuarine sediment, bird of prey tissues and sewage sludge samples should be sampled.

A final risk assessment on decaBDE, combining the work by France and the UK, is expected to be issued in 2005, at which point the issue of exempting decaBDE from the RoHS may be brought to a vote. The RoHS, in fact, specifically directs the European Commission to evaluate decaBDE applications as a matter of priority in order to establish as soon as possible whether decaBDE should remain subject to the directive.

Ms. Wallström has said she sees "…outstanding safety concerns related to DecaBDE and … that proportionate precautionary measures are necessary to reduce DecaBDE emissions in the environment. [She] therefore will not propose to the [European] Commission that it lift the ban on DecaBDE currently existing under the RoHS Directive."[29] If the Commission follows her advice, the RoHS ban on the sale of deca-laden electronics and electrical equipment will go into effect on July 1, 2006.

In the meantime, the EU already is moving to fill the data gaps identified in the draft risk assessment reports. Specifically, the following studies will be carried out within the EU program on existing substances:

- A new study to establish whether decaBDE interferes with brain development;
- A study to measure concentrations of decaBDE and its degradation products in the European environment in order to identify a possible trend; and
- A study to measure decaBDE in the blood and breast milk of humans to acquire more knowledge about concentrations and trends.

This work is expected to take some years to deliver results according the UK risk assessment report, and will involve at least 24 different research laboratories across Europe.[30]

In parallel with these studies, the EU's Competent Authorities have reached agreement with industry on a voluntary program to reduce releases of decaBDE within the plastics and textile sectors. This program will involve actions such as:

- Sampling emissions from industrial plants in the supply chain;
- Estimating total emission levels per application;
- Defining tolerable levels of emissions in cooperation with the supply chain and regulatory authorities;

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[29] Margot Wallström, in letter to J. Hontelez, M. Taylor, J. Riss and M. Warhurst, August 24, 2004.

[30] Margot Wallström on behalf of the European Commission in written response to a question posed by European Parliament Member Christopher Huhne, October 9, 2004.

- Assisting the supply chain to implement emission reductions where tolerable levels are being exceeded; and
- Measuring the effectiveness of emission reductions through further sampling of industrial plant emissions and in the environment.[31]

Depending on the success of this voluntary program in reducing emissions, and the results of the further scientific investigations, the European Commission could review the need for more formal risk reduction measures at a later date.

C. Other countries

Canada

The Canadian Environmental Protection Act of 1999 (CEPA 1999) requires the Ministers of Health and the Environment to conduct screening assessments of certain substances to determine whether they pose a risk to human health or the environment. Based on the assessment, the ministers then can propose to take no further action, add the substance to the List of Toxic Substances regulated under CEPA 1999 or add the substance to the Priority Substances List for further study.

In February 2004, Health Canada completed its screening assessment on the human health risk posed by PBDEs, including decaBDE. Based principally on environmental considerations, the assessment report recommends that PBDEs as group be considered "toxic" under CEPA 1999 (Health Canada, 2004). Under CEPA 1999, a substance is toxic if it: (a) has or may have an immediate or long-term harmful effect on the environment or its biological diversity; (b) constitutes or may constitute a danger to the environment; or (c) constitutes or may constitute a danger to human life or health.

Environment Canada has separately released a draft screening assessment report on the environmental risk posed by PBDEs. Environment Canada agreed with its sister agency Health Canada that decaBDE should be considered toxic under CEPA 1999, and further recommended that decaBDE be considered a Track 1 substance under the Toxic Substances Management Policy (Environment Canada, 1999 and 2004). A “Track 1” substance is one that has been determined to be persistent, bioaccumulative, toxic and primarily the result of human activity. Under the policy, Track 1 substances are targeted for virtual elimination from the environment. A Track 1 substance that cannot be managed successfully throughout its life cycle will be targeted for phase-out.

China

China’s Ministry of Information Industry has issued proposed regulations titled “Management Methods for the Prevention and Control of Pollution from Electronics Information Products” (also called the "China RoHS"). The regulations would, among other things, ban the use of

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[31] BSEF, "Product Stewardship for the Sustainable Use of Brominated Flame Retardants, http://www.bsef-site.com/product_stew.

PBDEs in electronic products (Hagen, 2004). The regulations are expected to be signed in early 2005.[32]

Sweden

In November 2004, the Swedish Chemicals Inspectorate issued a report in support of a national ban on decaBDE The report concludes that decaBDE no longer is used by Swedish manufacturers, but may be imported into Sweden in articles. The report further concludes that the use of decaBDE is not required by fire protection standards and that alternative substances or methods are available to ensure fire safety. Finally, the report concludes that a national ban on decaBDE is appropriate based on the precautionary principle, and recommends that Sweden call for a continued ban on decaBDE in the RoHS directive (Swedish Chemicals Inspectorate, 2004).

C. Other states

California

In August 2003, California became the first U.S. state to regulate PBDEs by passing legislation that prohibits the manufacture, processing or distribution of a product containing more than one-tenth of 1% penta or octaBDE beginning January 1, 2008.[33] The bill also required the Senate Office of Research to submit recommendations regarding the further regulation of PBDEs. The recommendations were submitted in a report dated June 2004 (Wiley and McCarthy, 2004).

The report found that human exposure to decaBDE does not appear to be "occurring at a level that is likely to be unsafe for human health or development" and concluded that it would be premature to add decaBDE to the ban. On the other hand, the authors were unable to conclude that decaBDE does not pose a danger to human health. "Rather, the data available does not conclusively show that there is a danger to human health at this time."

The report recommends that California's Office of Environmental Health Hazard Assessment set a reference dose for decaBDE representing the level in human tissue that would be considered unsafe. The report also recommends that the state create a program to monitor PBDE levels in breast milk.

In September 2004, California amended its ban on pentaBDE and octaBDE to change the effective date to June 1, 2006, and to clarify that the ban does not apply to the processing of metallic recyclables containing PBDEs.[34]

Hawaii

In June 2004, Hawaii enacted legislation prohibiting the manufacture, processing or distribution of a product or flame-retarded part of a product containing more than 0.1% by mass of

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[32] American Electronics Association, "International Update", http://aeanet.org/GovernmentAffairs/, viewed January 18, 2005

[33] 2003 Cal. Stat. c. 205, enacting Cal. Health & Safety Code § 108920-23.

[34] 2003 Cal. Stat. c. 641, amending Cal. Health & Safety Code § 108921 and 108922.

pentaBDE or octaBDE.[35] The ban is effective January 1, 2006. As with California's amended law, Hawaii's ban does not apply to the processing of metallic recyclables containing PBDEs.

Michigan

In January 2004, the Michigan Department of Environmental Quality (DEQ) published a white paper summarizing information on PBDEs. The DEQ reports that there are insufficient toxicity and exposure data to conduct a thorough risk assessment, but concludes there is sufficient evidence to support a ban on the commercial use of pentaBDE and octaBDE (Michigan DEQ, 2004).

As for decaBDE, the DEQ says that the scientific evidence is more limited and less convincing because decaBDE does not appear to be as bio-available as the penta and octa PBDE formulations. However, the paper also raises cause for concern, citing recent evidence that decaBDE causes neuro-developmental effects in rodents, may debrominate in carp and is present in humans. The DEQ will continue to monitor the scientific literature and may recommend a ban on decaBDE in the future. The DEQ plans to issue an updated PBDE white paper by the end of 2004.

In the meantime, the Michigan Legislature has joined Maine, California, Hawaii and New York in banning the sale and distribution of products containing penta and octaBDE. Michigan's ban is effective June 1, 2006, and does not apply to replacement parts or the processing of recyclables.[36]

New York

In August 2004, New York also adopted a prohibition on the penta and octaBDE but authorized the Commissioner of Environmental Conservation to waive the prohibition in whole or part upon finding that there is no significant threat to the public health.[37] The bill also establishes a task force to study the risks associated with decaBDE and the availability of safe and effective alternatives to decaBDE. The task force report is due by December 31, 2005.[38]

Washington

The State of Washington's Department of Ecology (Ecology) and Department of Health (Health) issued a PBDE action plan on December 31, 2004 (Peele, 2004). The plan recommends that Ecology and Health, in consultation with stakeholders, develop a proposal to ban the sale of products containing decaBDE. The recommendations are due by December 2005. The stakeholder process will examine:

- Which types of products would be covered by the ban;
- Whether products made from recycled plastic should be exempted;

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[35] Hawaii Rev. Stat., § 332D-1 through 332D-3, as enacted by 2004 Hawaii Sess. Laws, c. 146.
[36] HB 4406 and SB 1458, 92nd Michigan Legislature.
[37] N.Y. Envtl. Conserv. Law §37-0111.
[38] 2004 N.Y. Laws, c. 387.

- The human health, environmental and economic impacts of a ban;
- The availability of alternative fire retardants and the potential impacts of a decaBDE ban on fire safety;
- Impacts on retailers and consumers; and
- Emerging information on decaBDE.[39]

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[39] BSEF, "PBDE CAP Recommendations from the Public Draft compared to Proposed Revised Recommendations, 12/1/04," electronically transmitted to John James by Leann Diehl, December 3, 2004.

VI. Recycling of products containing PBDEs

Although pentaBDE and octaBDE will be taken off the market at the end of 2004, products containing these BFRs remain in use and eventually will end up in the waste stream for recycling or disposal. PentaBDE typically was used in foams for sofas, seat cushions and carpet. OctaBDE typically was used in high-impact plastics, such as in telephones, kitchen appliance casings, computers and automobile trim.

The fact that some penta and octa-laden items may be recycled raises two issues:

- Does Maine law prohibit the sale of products made from recycled material containing pentaBDE and octaBDE?
- Should Maine prohibit the recycling of PBDE-laden products?

These issues are explored below.

A. Is the sale of products made from recycled material containing pentaBDE and octaBDE prohibited under 38 MRSA §1609?

Section 1609, subsection 1, reads as follows:

> Effective January 1, 2006, a person may not sell or offer to sell, or distribute for promotional purposes, a product containing more than 1% of the “penta” or “octa” mixtures of polybrominated diphenyl ethers.

A plain meaning interpretation of this language would prohibit the sale of products containing more than 1% of the penta or octa PBDE mixtures even where the presence of these chemicals is the result of the use of recycled material. Such an interpretation, however, arguably is contrary to the clear purpose of the law, which was to prevent increases in exposure to these two PBDE mixtures by prohibiting the introduction of additional amounts of the chemicals into commerce.

An alternate interpretation, and one that perhaps best effectuates the law's purpose, would only prohibit the sale of new applications of penta and octaBDE. This is consistent with the legislative debate leading to enactment of the ban, and with the Legislature's intent as reflected in its decision to explicitly exempt used products from the ban (see §1609, sub-§3).

There is nothing in the legislative record leading to the enactment of section 1609, or in the wording of this statute, that suggests the need for action to reduce exposure to PBDEs already in commerce. To the contrary, based on the experience in Europe where voluntary bans already are in effect, the DEP and others suggested that prompt action to end new applications of penta and octaBDE would be enough to reverse the trend of increasing PBDE body burdens.

The DEP therefore is inclined to interpret section 1609 such that the ban applies only to the first time the pentaBDE or octaBDE is made available on the market. Under this interpretation, products made of recycled material could be sold after January 1, 2006 if any penta or octa in the product originates from a product that was placed in commerce before January 1, 2006.

B. Should Maine prohibit the sale of products made from recycled materials that contain PBDEs?

PBDEs typically make up 5 to 30% of a product's weight. They are mixed into but not chemically bound to the materials in which they are used. And because they are not chemically bound to these materials, they potentially are released to the environment throughout the life cycle of the product. DecaBDE, for example, has been found in dust on computers, suggesting that continuous off-gassing from HIPS used in the housing of consumer electronics is a source of PBDE contamination (McPherson *et al.*, 2004). PentaBDE, which was used mainly in polyurethane foam, is thought to escape into the environment as the foam becomes brittle and breaks down with age (Wiley and McCarthy, 2004).

The recycling of plastics and foams, as opposed to their landfilling or incineration, may increase the opportunity for PBDE release by extending the time that PBDEs remain in commerce. The recycling process itself also may present a heightened opportunity for release. For example, PBDE concentrations inside and outside an electronics recycling facility in Southern Ontario were higher than in indoor and outdoor ambient levels in Toronto (Butts *et al.*, 2004). Workers in a Swedish electronics recycling factory were found to have PBDE blood levels significantly higher than those found in a control group (Sjödin *et al.*, 1999). Following changes in ventilation and relocation of a shredder outside of the factory, PBDE blood levels in the workers decreased significantly but still were elevated compared to the background population (Thuresson *et al*., 2004).

The bromine industry, on the other hand, has published several papers in support of the proposition that PBDE-laden materials can be recycled or incinerated without leading to any environmental or health problems. In one study, high impact polystyrene (HIPs) flame retarded with decaBDE and antimony trioxide was repeatedly processed. The amount of debromination of the flame retardant and possible formation of dioxins and furans was monitored. No indication of debromination was found, and concentrations of dioxins and furans were below the limits under the German Chemicals Banning Ordinance (Hamm *et al.*, 2001).

Other industry research shows that PBDE-laden material can be safely recycled through thermal processes such as incineration or pyrolysis provided scrubbers are installed to avoid excessive bromine emissions (Vehlow *et al.*, 2002). These studies also show that formation of dioxins and furans from incineration of PBDE waste is insignificant or non-existent in properly run incinerators (Vehlow *et al.*, 2000; Tange and Drohmann, 2002).

The possibility that incineration of PBDEs could contribute to the formation of brominated dioxins and furans, which are possible carcinogens, is a concern identified in numerous reports, including the interim PBDE action plan issued by the Washington State Departments of Ecology and Health in December (Peele, 2004). This plan, while acknowledging that recycling conserves valuable resources, observes that it is unknown if the current system for recycling PBDE-laden materials adequately protects human health and the environment. The plan concludes that additional study is required to assess the need for special handling of PBDE-containing wastes. The plan therefore recommends:

> "[The Department of] Ecology should establish a process, to be completed by July 2006, to

> evaluate and determine appropriate disposal and recycling practices for products containing PBDEs. Ecology will involve appropriate stakeholders in this process, including, but not limited to, local government, private waste haulers and landfill operators, recyclers, manufacturers, environmental advocates, and human health advocates…As part of the evaluation, Ecology will:
>
> 1. Identify known information about potential pathways of PBDEs at end-of-life. Both PBDE releases to the environment and occupational exposure to workers would be examined at waste collection facilities, recycling facilities, waste disposal facilities, manufacturers using PBDEs and service industries such as carpet installers and upholsterers.
> 2. Through a literature search and limited product testing, characterize PBDE content of products along high-priority exposure pathways.
> 3. Establish where monitoring of PBDEs associated with end-of-life, including biosolids, leachate, and incinerator emissions, is warranted and, if so, for what purposes" (Peele, 2004).

C. Current recycling practice

Electronics

In the case of electronics recycling, the PBDE contaminant of concern is the deca used in TV cabinet backs and other cabinetry made of HIPS. Effective January 1, 2006, Maine law will require that TVs and computer monitors be diverted from the municipal waste stream for recycling.[40] The law requires that these electronic devices be sent to a recycling and dismantling facility that processes them in accordance with environmentally-sound management guidelines published by the department.

The Sony Corporation incorporates post consumer recycled plastic containing decaBDE in the manufacture of TV sets. The company has sought confirmation that it will be able to sell these TVs in Maine after January 1, 2008 if the Maine Legislature follows through on its intent to ban the sale of virgin uses of decaBDE. Sony is among the electronics manufacturers that no longer uses virgin decaBDE. The company has chosen to use recycled plastic in part to offset the increased costs associated with discontinuing the use of decaBDE in virgin plastic (D. Smith to J. James, personal communication).

Polyurethane foam

PentaBDE was not used in electronic goods. However, small amounts of penta-containing products are recycled, primarily for the production of polyurethane carpet padding. This recycling generally is limited to scrap that has not reached consumers. There is no significant recycling of post-consumer polyurethane foam such as that used in furniture and mattresses outside of the auto shredding industry (Wiley and McCarthy, 2004).

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[40] See 38 MRSA §§ 1306(4) and 1609 *et seq*.

The American Plastics Council has approached the DEP seeking confirmation that carpet underlay containing pentaBDE from recycled material can be sold in Maine after January 1, 2006, the effective date of the ban on sale of products containing more than 1% penta or octa. Our preliminary opinion is that such sales will be permissible as explained previously on page 48.

Automobile recycling

In the case of automobile recycling, the contaminants of concern are pentaBDE, which was used in seat padding, and octaBDE, which was used in plastic trim. When junked autos are crushed and shredded to make recycled steel, magnets are used to separate the metals from the non-metallic components or "fluff." The plastics and foams containing penta and octa end up in the fluff, which usually is landfilled.

The state legislatures in California, Hawaii and New York have explicitly exempted "the processing of metallic recyclables" from their PBDE laws to avoid any question as to whether automobile recycling is subject to the ban on the manufacture, *processing* and distribution of products containing penta or octa. Similar language is unnecessary in Maine because Maine law bans the sale and not the processing of products containing penta or octa.

VII. Conclusions

Based on review of the studies, risk assessments, findings and rulings discussed in this report, the DEP and BOH conclude as follows:

A. Levels of decaBDE in the environment and in human tissue are increasing. Further information and testing are needed to conclusively determine if this trend poses an unacceptable health threat, but currently available evidence supports the decision of the Maine Legislature to establish a presumptive ban on decaBDE under PL 2003, c. 629, §2.

B. Scientific studies are underway around the world to gather more information on decaBDE. These include studies designed to fill data gaps on the toxicology, degradation and bio-accumulation of decaBDE. Among the numerous reports and assessments expected to be completed or released in 2005 are the following:

- An assessment of decaBDE by the EPA under its the Voluntary Children's Chemical Action program;
- An EPA action plan for PBDE;
- The final version of the European Union's updated risk assessment on decaBDE;
- An evaluation of information related to the toxicology, fate and occurrence of PBDEs in food by an expert committee of the United Nations and World Health Organization;
- A plan for banning the sale of products containing decaBDE in the State of Washington;
- A review of flame retardancy options for residential furniture by EPA Office of Pollution Prevention and Toxics; and
- An evaluation of alternatives to decaBDE by the Lowell Center for Sustainable Production.

C. Safe and effective alternatives for achieving flame retardancy appear to be available for all current decaBDE applications. The alternatives generally cost more. The increased cost is of concern from the standpoint of fire safety because the use of flame retardants generally is not mandated by law in the U.S. In the absence of mandated standards, some manufacturers possibility could respond to a ban on decaBDE by choosing to forego the use of flame retardants or by using flame retardancy methods that are less effective. On the other hand, manufacturer compliance with voluntary fire safety standards developed by Underwriters Laboratory appears to be well established in the U.S.

VIII. Recommendation

The Department of Environment Protection and the Department of Health and Human Services, Bureau of Health, recommend that the presumptive ban on decaBDE under PL 2003, c. 629, §2, stay in place, and that this chemical be further evaluated based on the new scientific findings, reports, assessments and legislative actions expected to be completed and available for review during the coming year.

References

Albinson, B. Alternative ways to achieve fire safety. Swedish Rescue Services Agency, 2002

Alumot, E., and Mandel, E. Intestinal absorption and distribution of decachlorobiphenyl in rats and chicks. Bull. Environ. Contam. Toxicol. 16:746-749, 1976.

Asplund, L., Bignert, A., and Nylund, K. Comparison of spatial and temporal trends of methyoxylated PBDEs, PBDEs, and hexabromocyclododecane in herring along the Swedish coast. Organohalogen Compd. 66:3988-3993, 2004.

Asplund, L., Hornung, M., Peterson, R.E., Turesson, K., and Bergman, Å. Organohalogen Compd. 40:351-354, 1999.

Asplund, L., Malmvärn, A., Marsh, G., Athanasiadou, M., Bergman, Å., and Kautsky, L. Hydroxylated brominated diphenyl ethers in salmon (*Salmo salar*), mussels (*Mytilus edulis*) and the red algae (*Ceramium tenuicorne*) from the Baltic Sea – Natural product in Baltic Sea biota. Organohalogen Compd. 52:67-71, 2001.

Babich, M. and Thomas, T. CPSC Staff Exposure and Risk Assessment of Flame Retardant Chemicals in Residential Upholstered Furniture. U.S. Consumer Product Safety Commission, 2001.

Balch, G., McDowell, S., Reiber, K., Velez-Espino, L., and Metcalfe, C. *In vivo* testing of PBDE flame retardants using the *Xenopus* tail regression model. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 395-398.

Baker, J., Klosterhaus, S., Liebert, D., Stapleton, H. Brominated diphenyl ethers in the sediments, porewater, and biota of the Chesapeake Bay, U.S.A. Organohalogen Compd. 66:3805-3810, 2004.

Bergman, A., Athanasiadou, M., Wehler, E.K., and Sjödin, A. Polybrominated environmental pollutants: Human and wildlife exposures. Organohalogen Compd. 43:89-92, 1999.

Betts, K. PBDEs and the environmental intervention lag time. Environ. Sci. Technol., 40: 386A-387A, 2004.

Bezares-Cruz, J., Jafvert, C., and Hua, I. Solar photodecomposition of decabromodiphenyl ether: products and quantum yield. Environ. Sci. Technol. 38: 4149–4156, 2004.

Birnbaum L.S. and Staskal D.F. Brominated Flame Retardants: Cause for Concern? Environ. Health Perspect., 112: 9-17, 2004.

Bjork, M. Banning Brominated Flame Retardants. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 15-16.

Branchi, I., Alleva, E., and Costa, L.G. Effects of perinatal exposure to a polybrominated diphenyl ether (PBDE 99) on mouse neurobehavioral development. Neurotoxicol. 23:375-384, 2002.

Brominated Flame Retardant Industry Panel (BFRIP). Data summary: decabromodiphenyl ether (a.k.a. decabromodiphenyl oxide, DBDPO). American Chemistry Council, 2002.

Brominated Flame Retardant Industry Panel. Report of the peer consultation meeting on decabromodiphenyl Ether. American Chemistry Council, 2003.

Butt, C.M., Diamond, M.L., Truong, J., Ikonomou, M.G., and ter Schure, A.F.H. Spatial distribution of polybrominated diphenyl ethers in southern Ontario as measured in indoor and outdoor window organic films. Environ. Sci. Technol. 38:724-731, 2004.

Darnerud, P.O., Aune, M., Atuma, S., Becker, W., Bjerselius, R., Cnattingius, S., and Glynn, A. Time trend of polybrominated diphenyl ether (PBDE) levels in breast milk from Uppsala, Sweden, 1996-2001. Organohal. Comp. 58:233-236, 2002.

Darnerud, P.O., Eriksen, G., Johannesson, T., Larsen, P., and Viluksela, M. Polybrominated diphenyl ethers: occurrence, dietary exposure, and toxicology. Environ. Health Perspect. 109:724-731, 2004.49-68, 2001.

Darnerud, P.O., and Thuvander, A. Effects of polybrominated diphenyl ether (PBDE) and polychlorinated biphenyl (PCB) on some immunological parameters after oral exposure in rats and mice. Toxicol. Environ. Chem. 70:229-242, 1999.

de Boer, J., Leslie, H.A., Leonards, P.E.G., Bersuder, P., Morris, S., and Allchin, C.R. Screening and time trend study of decabromodiphenylether and hexabromocyclododecane in birds. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 125-128.

de Wit, C., Alaee, M., and Muir, D. Brominated flame retardants in the Arctic – An overview of spatial and temporal trends. Organohalogen Compd. 66:3811-3816, 2004.

Dodder, N.G., Strandberg, B., and Hites, R.A. Concentrations and spatial variations of polybrominated diphenyl ethers and several organochlorine compounds in fishes from the northeastern United States. Environ. Sci. Technol. 36:146-151, 2002.

Doucet, J., Arnold, D.L., Cooke, G.M., and Goodyer, C.G. Brominated diphenyl ether (BDE) residues in Canadian human fetal liver and placenta. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 53-57.

Easton, M.D., Luszniak, D., and Von der, G.E. Preliminary examination of contaminant loadings in farmed salmon, wild salmon and commercial salmon feed. Chemosphere 46:1053-1074, 2002.

Environment Canada. Canadian Environmental Protection Act 1999 Environmental Screening Assessment Report on Polybrominated Diphenyl Ethers (PBDEs). Draft for public comment, February 2004.

Environment Canada. Toxic Substances Management Policy, 1995.

Environmental Working Group. In the Dust: Toxic fire retardants in American homes. http://www.ewg.org/, 2004a.

Environmental Working Group. Mothers' Milk: Record levels of toxic fire retardants found in American mothers' breast milk. http://www.ewg.org/, 2004b.

Environmental Working Group. Tainted Catch: Toxic fire retardants are building up rapidly in

San Francisco Bay fish – and people. http://www.ewg.org/, 2003.

Eriksson, J., Green, N., Marsh, G., and Bergman, Å. Photochemical decomposition of 15 polybrominated diphenyl ether congeners in methanol/water. Environ. Sci. Technol. 38:3119-3125, 2004a.

Eriksson, P., Fischer, C., and Fredriksson, A. Co-exposure to a polybrominated diphenyl ether (PBDE 99) and an ortho-substituted PCB (PCB 52) enhances developmental neurotoxic effects. Organohalogen Compd. 61:81-83, 2003.

Eriksson, P., and Fredriksson, A. Developmental neurotoxicity of four *ortho*-substituted polychlorinated biphenyls in the neonatal mouse. Environ. Toxicol. Pharmacol. 1:155-165, 1996a.

Eriksson, P., and Fredriksson, A. Neonatal exposure to 2,2´,5,5´-tetrachlorobiphenyl causes increased susceptibility in the cholinergic transmitter system at adult age. Environ. Toxicol. Pharmacol. 1:217-220, 1996b.

Eriksson, P., Jakobsson, E., and Fredriksson, A. Brominated flame retardants: A novel class of developmental neurotoxicants in our environment? Environ. Health Perspect. 109:903-908, 2001.

Eriksson, P., Johansson, N., Viberg, H., Fischer, C., and Fredriksson, A. Comparative developmental neurotoxicity of flame-retardants, polybrominated flame-retardants and organophosphorous compounds, in mice. Organohalogen Compd. 66:3163-3165, 2004b.

Eriksson, P., Viberg, H., Jakobsson, E., Örn, U., and Fredriksson, A. A brominated flame retardant, 2,2´,4,4´,5-pentabromodiphenyl ether: Uptake, retention and induction of neurobehavioural derangement in mice, during a critical phase of neonatal brain development. Toxicol. Sci. 67:98-103, 2002.

European Commission. Risk Assessment Report: Bis(pentabromophenyl ether). European Commission Joint Research Center, 2002.

European Commission. Update of the Risk Assessment of Bis(Pentabromophenyl) Ether (Decabromodiphenyl Ether): Human Health Draft of February 2004. European Commission Joint Research Center, 2004.

European Commission. Update of the Risk Assessment of Bis(Pentabromophenyl) Ether (Decabromodiphenyl Ether): Final Environment Draft of May 2004. European Commission Joint Research Center, 2004.

Fabrellas, B., Larrazabal, D., Martinez, M.A., Eljarrat, E., and Barcelo, D. Presence of polybrominated diphenyl ethers in Spanish sewage sludges: Important contribution of decaBDE. Organohalogen Compd. 66:3755-3760, 2004.

Fängström, B., Strid, A., Athanassiadis, I., Grandjean, P., Weihe, P., and Bergman, Å. A retrospective time-trend study of PBDEs and PCBs in human milk from the Faroe Islands. Organohal. Comp. 66:2829-2833, 2004.

Fernlöf, G., Gadhasson, I., Pödra, K., Darnerud, P.O., and Thuvander, A. Lack of effects of some individual polybrominated diphenyl ether (PBDE) and polychlorinated biphenyl (PCB) on human lymphocyte functions *in vitro*. Toxicol. Lett. 90:189-197, 1997.

Fowles, J.R., Fairbrother, A., Beecher-Steppan, L., and Kerkvliet, N.I. Immunologic and endocrine effects of the flame-retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice. Toxicol. 86:49-61, 1994.

Gerecke, A.C., Schmid, P., Heeb, N.V., Zennegg, M., Hartmann, P.C., Kohler, H.-P. E., and Kohler, M. Formation of lower brominated congeners by anaerobic degradation of decabromodiphenyl ether in sewage sludge. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 235-238.

Geyer, H.J., Schramm, K.-W., Darnerud, P.O., Aune, M., Feicht, E.A., Fried, K.W., Henkelmann, B., Lenoir, D., Schmid, P., and McDonald, T.A. Terminal elimination half-lives of the brominated flame retardants TBBPA, HBCD, and lower brominated PBDEs in humans. Organohal. Comp. 66:3867-3872, 2004.

Gouin, T., Chaemfa, C., Thomas, G.O., Harner, T., and Jones, K.C. Concentrations of decabromodiphenyl ether in air from Southern Ontario: Evidence for particle-bound transport. `4In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 159-162.

Gouin, T., Thomas, G.O., Cousins, I., Barber, J., MacKay, D., and Jones, K.C. Air-surface exchange of polybrominated diphenyl ethers and polychlorinated biphenyls. Environ. Sci. Technol. 36:1426-1434, 2002.

Guvenius, D.M., Aronsson, A., Ekman-Ordeberg, G., Bergman, Å., and Norén, K. Human prenatal and postnatal exposure to polybrominated diphenyl ethers, polychlorinated biphenyls, polychlorobiphenyls, and pentachlorophenol. Environ. Health Perspect. 111:1235-1241, 2003.

Haddow, J.E., Palomaki, G.E., Allan, W.C., Williams, J.R., Knight, G.J., Gagnon, J., O'Heir, C.E., Mitchell, M.L., Hermos, R.J., Waisbren, S.E., Faix, J.D., and Klein, R.Z. Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child. New Engl. J. Med. 341:549-555, 1999.

Hagen, P. Environmental and product-related initiatives: Asia and Latin America. Presentation at 2004 IEEE International Symposium on Electronics and the Environment, Scottsdale, Arizona, May, 2004.

Haglund, P.S., Zook, D.R., Buser, H.R., and Hu, J. Identification and quantification of PBDEs and methoxy-PBDEs in Baltic biota. Environ. Sci. Technol. 31:3281-3287, 1997.

Hakk, H., Larsen, G., and Klasson-Wehler, E. Tissue disposition, excretion and metabolism of 2,2´,4,4´,5-pentabromodiphenyl ether (BDE-99) in the male Sprague-Dawley rat. Xenobiotica 32:369-382, 2002.

Hale, R.C., La Guardia, M.J., Harvey, E.P., Gaylor, M.O., Mainor, T.M., and Duff, W.H. Flame retardants – persistent pollutants in land-applied sludges. Nature 412, 12 July 2001a.

Hale, R.C., La Guardia, M.J., Harvey, E.P., Mainor, T.M., Duff, W.H., and Gaylor, M.O. Polybrominated diphenyl ether flame retardants in Virginia freshwater fishes. Environ. Sci. Technol. 35:4585-4591, 2001b.

Hamers, T., Kamstra, J.H., Sonneveld, E., Murk, A.J., Zegers, B.N., Boon, J.P., and Brouwer, A. *In vitro* screening of the endocrine disrupting potency of brominated flame retardants and their metabolites. Devel. Repro. Toxicity 66:3016-3020, 2004.

Hamm, S., Strikkeling, M., Ranken, P. and Rothenbacher, K. Determination of polybrominated diphenyl ethers and PBDD/Fs during the recycling of high impact polystyrene containing decabromodiphenyl ether and antimony oxide. Chemosphere 44: 1353-1360, 2001

Harriman, E., Greiner, T. and Veleva, V. Environmental Challenges and Materials Restrictions in Coated Wire and Cable. National Fire Protection Research Symposium, 2003.

Hayward, D., Wong, J., and Krynitsky, A. PBDE and PCB levels correlated in wild caught and farm-raised fish fillets in the U.S.A. Organohalogen Compd. 66:3994-3998, 2004.

Health Canada. Screening Assessment Report—Health: Polybrominated diphenyl ethers (PBDEs), 2004.

Hermann, T., Schilling, B., and Päpke, O. Photolysis of PBDEs in solvents by exposure to daylight in a routine laboratory. Organohalogen Compd. 63:361-364, 2003.

Herzke, D., Gabrielsen, G.W., Evenset, A., and Burkow, I.C. Polychlorinated camphenes (toxaphenes), polybrominated diphenylethers and other halogenated organic pollutants in glaucous gull (*Larus hyperboreus*) from Svalbard and Bjørnøya (Bear Island). Environ. Poll. 121:293-300, 2003b.

Hirai, T., Fujimine, Y., Watanabe, S., Nakamura, Y., Shimomura, H., and Nagayama, J. Maternal-infant transfer of polybrominated diphenyl ethers. Organohal. Comp. 66:2451-2456, 2004.

Hites, R.A. Polybrominated diphenyl ethers in the environment and in people: A meta-analysis of concentrations. Environ. Sci. Technol. 38:945-956, 2004a.

Hites, R., Foran, J., Schwager, S., Knuth, B., Hamilton, C., and Carpenter, D. Global assessment of polybrominated diphenyl ethers in farmed and wild salmon. Organohalogen Compd. 66:3873-3876, 2004b.

Holden, A., She, J., Tanner, M., Lunder, S., Sharp, R., and Hooper, K. PBDEs in San Francisco Bay area: Measurements in fish. Organohalogen Compd. 61:255-258, 2003.

Howie, L., Dickerson, R., Davis, D., and Safe, S. Immunosuppressive and monooxygenase induction activities of polychlorinated diphenyl ether congeners in C57BL/6N mice: Quantitative structure-activity relationships. Toxicol. Appl. Pharmacol. 105:254-263, 1990.

Huber, S., and Ballschmiter, K. Characterisation of five technical mixtures of brominated flame retardants. Fresenius J. Anal. Chem. 371:882-890, 2001.

Hutzinger, O., and Sundström, G. Environmental chemistry of flame retardants: The composition of Bromkal 70-5DE, a pentabromodiphenyl ether. Chemosphere 1:3-10, 1976.

Huwe, J. Polybrominated Diphenyl Ethers in Meat Samples Collected from Supermarkets Across the U.S. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F.

Wania), 2004, pp. 41-44.

Ikonomou, M.G., Rayne, S., and Addison, R.F. Exponential increases of the brominated flame retardants, polybrominated diphenyl ethers, in the Canadian Arctic from 1981 to 2000. Environ. Sci. Technol. 36:1886-1892, 2002.

Ingelido, A.M., Di Domenico, A., Ballard, T., De Felip, E., Dellatte, E., Ferri, F., Fulgenzi, A.R., Herrmann, T., Iacovella, N., Miniero, R., Päpke, O., and Porpora, M.G. Levels of polybrominated diphenyl ethers in milk from Italian women living in Rome and Venice. Organohal. Comp. 66:2722-2728, 2004.

Jaward, F.M., Farrar, N.J., Harner, T., Sweetman, A.J., and Jones, K.C. Passive air sampling of PCBs, PBDEs, and organochlorine pesticides across Europe. Environ. Sci. Technol. 38:34-41, 2004.

Kalantzi, O.I., Martin, F.L., Thomas, G.O., Alcock, R.E., Tang, H.R., Drury, S.C., Carmichael, P.L., Nicholson, J.K., and Jones, K.C. Different levels of polybrominated diphenyl ethers (PBDEs) and chlorinated compounds in breast milk from two UK regions. Environ. Health Perspect. 112:1085-1091, 2004.

Kierkegaard, A., Balk, L., Tjärnlund, U., de Wit, C.A., and Jansson, B. Dietary uptake and biological effects of decabromodiphenyl ether in rainbow trout (*Oncorhynus mykiss*). Environ. Sci. Technol. 33:1612-1617, 1999.

Kierkegaard, A., Bignert, A., Sellström, U., Olsson, M., Asplund, L., Jansson, B., and de Wit, C.A. Polybrominated diphenyl ethers (PBDEs) and their methoxylated derivatives in pike from Swedish waters with emphasis on temporal trends, 1967-2000. Environ. Pollution 130:187-198, 2004.

Klasson-Wehler, E., Jakobson, E., and Örn, U. Metabolism of polychlorinated naphthalenes and a tetrabrominated diphenyl ether. Organohalogen Compd. 28:495-499, 1996.

Klosterhaus, S.L., Stapleton, H.M., and Baker, J.E. Polybrominated diphenyl ethers (BDEs) in sediment, pore water, and infaunal invertebrates along a spatial gradient in an urban estuarine river receiving wastewater effluent in Baltimore, Maryland, U.S.A. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 117-119.

Knoth, W., Mann, W., Meyer, R., and Nebhuth, J. Occurrence and fate of PBDE in sewage sludge from municipal waste water treatment plants. Organohalogen Compd. 66:3749-3754, 2004.

Knoth, W., Mann, W., Meyer, R., and Nebhuth, J. Brominated diphenyl ether in indoor dust. Organohalogen Compounds 61:207-210, 2003.

Knoth, W., Mann, W., Meyer, R., and Nebhuth, J. Polybrominated diphenyl ether in house dust. Organohalogen Compd. 58:213-216, 2002.

Kociba, R.J., Frauson, L.O., Humiston, C.G., Norris, N.M., Wade, C.E., Lisowe, R.W., Quast, J.F., Jersey, G.C., and Jewett, G.L. Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. Combust. Toxicol. 2:267-285, 1975a.

Kociba, R.J., Frauson, L.O., Humiston, C.G., Norris, J.M., Wade, C.E., Lisowe, R.W., Quast, J.F., Jersey, G.C., and Jewett, G.L. Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats. Midland, MI: Dow Chemical Co., 1975b.

Kodavanti, P.R., and Derr-Yellin, E.C. Differential effects of polybrominated diphenyl ethers and polychlorinated biphenyls on [3H]arachidonic acid release in rat cerebellar granule neurons. Toxicol. Sci. 68:451-457, 2002.

Kodavanti, P.R.S., and Ward, T.R. Effects of polybrominated diphenyl ether mixtures and congeners on protein kinase C translocation in rat neuronal cultures. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 425-428.

Kohler, M., Zennegg, M., Gerecke, A.C., Schmid, P., and Heeb, N. Increasing concentrations of decabromodiphenyl ether (decaBDE) in Swiss sewage sludge since 1993. Organohalogen Compd. 61:123-126, 2003.

Kolic, T.M., MacPherson, K.M., Reiner, E.J., Ho, T., Kleywegt, S., Payne, M., and Alaee, M. Paper biosolids and manure: An investigative approach regarding levels of brominated diphenyl ethers. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 179-182.

Kuriyama, S.N., Talsness, C.E., and Chahoud, I. Sex-dependent behavioral changes in rat offspring after *in utero* administration of a single low dose PBDE 47. Organohalogen Compd. 66:3893-3900, 2004a.

Kuriyama, S.N., Fidalgo-Neto, A.A., Grande, S.W., Akkoc, Z., de Souza, C.A.M., and Chahoud, I. Thyroid hormone levels and hepatic enzyme activity in lactating dams after gestational exposure to low dose PBDE 47. Organohalogen Compd. 66:3901-3906, 2004b.

Kuriyama, S.N., Talsness, C.E., Grote, K., and Chahoud, I. Developmental exposure to low-dose PBDE 99: 1 – Effects on male fertility and neurobehavior in rat offspring. Environ. Health Perspect. Online at http://dx.doi.org/, doi:10.1289/ehp.7421, November 4, 2004c.

La Guardia, M.J., Hale, R.C., and Harvey, E. Environmental debromination of decabrominated diphenyl ether. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 231-234.

Lassen, C., Løkke S. and Andersen, L. Brominated Flame Retardants: Substance Flow Analysis and Assessment of Alternatives. Danish Environmental Protection Agency, 1999.

Law, R.J., Alaee, M., Allchin, C.R., Boon, J.P., Lebeuf, M., Lepom, P., and Stern, G.A. Levels and trends of polybrominated diphenylethers and other brominated flame retardants in wildlife. Environ. Intl. 29:757-779, 2003.

Lebeuf, M., Gouteux, B., Measures, L., and Trottier, S. Levels and temporal trends (1988-1999) of polybrominated diphenyl ethers in beluga whales (*Delphinapterus leucas*) from the St. Lawrence estuary, Canada. Environ. Sci. Technol. 38:2971-2977, 2004.

Leisewitz, A., Kruse, H. and Schramm, E. Substituting Environmentally Relevant Flame Retardants: Assessment Fundamentals: Results and Summary Overview. German Federal

Environmental Agency, 2001.

Letcher, R.J., D'Sa, I., Valters, K., Marsh, G., Li, H., Bennett, E., and Alaee, M. Polybrominated diphenyl ethers and hydroxylated and methoxylated analogues in Detroit River fish. Organohalogen Compd. 61:29-32, 2003.

Letcher, R.J., Stapleton, H.M., Verreault, J., McKinney, M., Scipione, F., Gabrielsen, G., Chu, S., and Valters, K. New case examples of biotransformation and elimination of polybrominated diphenyl ethers in aquatic and marine wildlife. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 375-378.

Lichtensteiger, W., Faass, O., Ceccatelli, R., and Schlumpf, M. Developmental exposure to PBDE 99 and PCB affects estrogen sensitivity of target genes in rat brain regions and female sexual behavior. Organohalogen Compd. 66:3965-3970, 2004.

Lind, Y., Darnerud, P.O., Atuma, S., Aune, M., Becker, W., Bjerselius, R., Cnattingius, S., and Glynn, A. Polybrominated diphenyl ethers in breast milk from Uppsala County, Sweden. Environ. Res. 93:186-194, 2003.

Lindberg, P., Sellström, U., Häggberg, L., and de Wit, C.A. Higher brominated diphenyl ethers and hexabromocyclododecane found in eggs of peregrine falcons (*Falco peregrinus*) breeding in Sweden. Environ. Sci. Technol. 38:93-96, 2004.

Longnecker, M.P., Wolff, M.S., Gladen, B.C., Brock, J.W., Grandjean, P., Jacobson, J.L., Korrick, S.A., Rogan, W.J., Weisglas-Kuperus, N., Hertz-Picciotto, I., Ayotte, P., Stewart, P., Winneke, G., Charles, M.J., Jacobson, S.W., Dewailly, É., Boersma, E.R., Altshul, L.M., Heinzow, B., Pagano, J.J., and Jensen, A.A. Comparison of polychlorinated biphenyl levels across studies of human neurodevelopment. Environ. Health Perspect. 111:65-70, 2003.

López, D., Athanasiadou, M., Athanassiadis, I., Yáñez Estrada, L., Díaz-Barriga, F., and Bergman, Å. A preliminary study on PBDEs and HBCDD in blood and milk from Mexican women. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 483-487.

Luksemburg, W., Wenning, R., Maier, M., Patterson, A., and Braithwaite, S. Polybrominated diphenyl ethers (PBDE) and polychlorinated dibenzo-p-dioxins (PCDD/F) and biphenyls (PCB) in fish, beef, and fowl purchased in food markets in northern California U.S.A. Organohalogen Compd. 66:3982-3987.

Luross, J.M., Alaee, M., Sergeant, D.B., Cannon, C.M., Whittle, D.M., Solomon, K.R., and Muir, D.C.G. Spatial distribution of polybrominated diphenyl ethers and polybrominated biphenyls in lake trout from the Laurentian Great Lakes. Chemosphere 46:665-672, 2002.

Luross, J.M., Alaee, M., Sergeant, D.B., Whittle, D.M., and Solomon, K.R. Spatial and temporal distribution of polybrominated diphenyl ethers in lake trout form the Great Lakes. Organohalogen Compd. 47:73-76, 2000.

Madsen, T., Lee, S., and Olle, T. Growing Threats – Toxic Flame Retardants and Children's Health. Environment California Research and Policy Center, 2003.

Manchester-Neesvig, J.B., Valters, K., and Sonzogni, W.C. Comparison of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in Lake Michigan salmonids. Environ. Sci. Technol. 35:1072-1077, 2001.

Mariussen, E., and Fonnum, F. The effect of brominated flame retardants on neurotransmitter uptake into rat brain synaptosomes and vesicles. Neurochem. Int. 43:533-542, 2003.

Marsh, G., Athanasiadou, M., Bergman, Å., Jakobsson, E., and Asplund, L. Hydroxylated and methoxylated polybrominated diphenyl ethers in salmon plasma: Synthesis and identification. Organohalogen Compd. 52:62-66, 2001.

Mazdai, A., Dodder, N.G., Abernathy, M.P., Hites, R.A., Bigsby, R.M. Polybrominated diphenyl ethers in maternal and fetal blood samples. Environ. Health Perspect. 111:1249-1252, 2003.

McDonald, T. Distribution of PBDE levels among U.S. women: estimates of daily intake and risk of development effects. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 443-446.

McPherson, A., Thorpe, B and Blake, A. Brominated Flame Retardants in Dust on Computers: The Case for Safer Chemicals and Better Computer Design. Computer Take-Back Campaign, http://www.computertakeback.org/, 2004.

Meironyté, D., and Norén, K. Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972-1997. J. Toxicol. Environ. Health, 58:329-341, 1999.

Michigan Department of Environmental Quality. Polybrominated Diphenyl Ethers (PBDEs): Background Paper, January 2004.

Mörck, A., Hakk, H., Örn, U., and Klasson-Wehler, E. Decabromodiphenyl ether in the rat: Absorption, distribution, metabolism, and excretion. Drug. Metabol. Disp. 31:900-907, 2003.

Moss, K. BFR regulatory update. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 7-9.

National Research Council. Toxicological Risks of Selected Flame-Retardant Chemicals. National Academy Press, 2000.

National Toxicity Program. Toxicology and carcinogenesis studies of decabromodiphenyl oxide (CAS No. 1163-19-5) in R334/N rats and B6C3F1 mice (feed studies). NTP Technical Report Series no. 309, Research Triangle Park, 1986.

Norén, K., and Merionyté, D. Certain organochlorine and organobromine contaminants in Swedish human milk in perspective of past 20-30 years. Chemosphere 40:1111-1123, 2000.

Norris, J.M., Ehrmantraut, J.W., Gibbons, C.L., Kociba, R.J., Schwetz, B.A., Rose, J.Q., Humiston, C.G., Jewett, G.L., Crummett, W.B., Gehring, P.J., Tirsell, J.B., and Brosier, J.S. 1973. Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical. Appl. Polymer Symp. 22:195-

219, 1973.

Norstrom, R.J., Simon, M., Moisey, J., Wakeford, B., and Weseloh, D.V.C. Geographical distribution (2000) and temporal trends (1981-2000) of brominated diphenyl ethers in Great Lakes herring gull eggs. Environ. Sci. Technol. 36:4783-4789, 2002.

North, K. Tracking Polybrominated Diphenyl Ether Releases in a Wastewater Treatment Plant Effluent, Palo Alto, California. Environ. Sci. Technol. 38: 4484-4488, 2004.

Northwest Environment Watch. Flame Retardants in Puget Sound Residents. Seattle, WA: Northwest Environment Watch, February 2004.

Ohta, S., Okumura, T., Nishimura, H., Nakao, T., Shimizu, Y., Ochiai, F., Aozasa, O., and Miyata, H. Levels of PBDEs, TBBPA, TBPs, PCDDs/DFs, PXDDs/DFs and PBDDs/DFs in human milk of nursing women and dairy milk products in Japan. Organohal. Comp. 66:2891-2896, 2004.

Okazawa, T., Hanari, N., Guruge, K., Wzykowska, B., Orlikowska, A., Yamashita, N., and Falandysz, J. Congener-specific data of PBDEs in pine needles. Organohalogen Compd. 66:3774-3778, 2004.

Olsman, H., van Bavel, B., Kalbin, G., Pettersson, A., Stenlund, S., Strid, Å., and Engwall, M. Formation of dioxin-like compounds as photoproducts of decabrominated diphenyl ether (DEBDE) during UV-irradiation. Organohalogen Compd. 58:41-44, 2002.

Olsson, A., Ceder, K., Bergman, Å., and Helander, B. Nestling blood of the white-tailed sea eagle (Haliaeetus albicilla) as an indicator of territorial exposure to organohalogen compounds – an evaluation. Environ. Sci. Technol. 34:2733-2740, 2000.

Orn, U., and Klasson-Wehler, E. Metabolism of 2,2´,4,4´-tetrabromodiphenyl ether in rat and mouse. Xenobiotica 28:199-211, 1998.

Patandin, S., Veenstra, J., Mulder, P.G.H., Sewnaik, A., Sauer, P.J.J., Weisglas-Kuperus, N. Attention and activity in 42-month-old Dutch children with environmental exposure to polychlorinated biphenyls and dioxins. In: Effects of environmental exposure to polychlorinated biphenyls and dioxins on growth and development in young children Ph.D. thesis. Erasmus University, Rotterdam, 1999:123-142.

Peele, C. Washington State PBDE Chemical Action Plan: Interim Plan. December 31, 2004.

Petreas, M., She, J., Brown, F.R., Winkler, J., Windham, G., Rogers, E., Zhao, G., Bhatia, R., and Charles, M.J. High body burdens of 2,2´,4,4´-tetrabromodiphenyl ether (BDE-47) in California women. Environ. Health Perspect. 111:1175-1179, 2003.

Polder, A., Thomsen, C., Becher, G., Skaare, J.U., Løken, K., and Eggesbø, M. The Norwegian Human Milk Study (HUMIS) – Variations in levels of chlorinated pesticides, PCBs and PBDEs in Norwegian breast milk. Organohal. Comp. 66:2476-2482, 2004.

Raff, J., and Hites, R. polybrominated diphenyl ethers in Mississippi River suspended sediment. Organohalogen Compd. 66:3722-3726, 2004.

Rayne, S., Ikonomou, M.G., and Antcliffe, B. Rapidly increasing polybrominated diphenyl ether concentrations in the Columbia River system from 1992 to 2000. Environ. Sci. Toxicol. 37:2847-2854, 2003.

Rice, C.P., Chernyak, S.M., Begnoche, L., Quintal, R., and Hickey, J. Comparisons of PBDE composition and concentration in fish collected from the Detroit River, MI and Des Plaines River, IL. Chemosphere 47:731-737, 2002.

Rice, D.C. Effects of developmental PCB exposure on neuropsychological function in epidemiological studies: Issues and research needs. In: Human Developmental Neurotoxicology, edited by D.C. Bellinger, Marcel Dekker Press, New York.

Ryan, J.J., Patry, B., Mills, P., and Beaudoin, N.G. Recent trends in levels of brominated diphenyl ethers (BDEs) in human milks from Canada. Organohalogen Compd. 58:173-176, 2002.

Ryan, J.J., and van Oostdam, J. Polybrominated diphenyl ethers (PBDEs) in maternal and cord blood plasma of several Northern Canadian populations. Organohal. Comp. 66:2579-2585, 2004.

Schantz, S.L., Widholm, J.J., and Rice, D.C. Effects of PCB exposure on neuropsychological function in children. Environ. Health Perspect. 111:357-376, 2003.

Schecter, A., Cramer, P., Boggess, K., Stanley, J., Päpke, O, Olson, J., Silver, A., and Schmitz, M. Intake of dioxins and related compounds from food in the U.S. population. J. Toxicol. Environ. Health. A. 63:1-18, 2001.

Schecter, A., Päpke, O., Joseph, J.E., and Tung, K.-C. Polybrominated diphenyl ethers (PBDEs) in U.S. computers and domestic carpet vacuuming: Possible sources of human exposure. Submitted.

Schecter, A., Päpke, O., Ryan, J.J., Rosen, R., Tung, K.C., Pavuk, M., Staskal, D., Birnbaum, L., Quynh, H.T., and Constable, J.D. PBDEs in U.S. milk, blood, and food, and temporal trends for PBDEs, PCDDs, and PCBs in U.S. blood. Organohalogen Compd. 66:2834-2840, 2004a.

Schecter, A., Päpke, O., Tung, K.-C., Staskal, D., and Birnbaum, L. Polybrominated diphenyl ethers contamination of United States food. Environmental. Sci. Technol. 38:5306-5311, 2004b.

Schecter, A., Pavuk, M., Päpke, O., Ryan, J.J., Birnbaum, L., and Rosen, R. Polybrominated diphenyl ethers (PBDEs) in U.S. mothers' milk. Environ. Health Perspect. 111:1723-1729, 2003.

Schröter-Kermani, C., Helm, D., Herrmann, T., and Päpke, O. The German environmental specimen bank – application in trend monitoring of polybrominated diphenyl ethers in human blood. Organohalogen Compd. 47:49-52, 2000.

Schuhmacher, M., Kiviranta, H., Vartiainen, T., and Domingo, L.L. Concentrations of polychlorinated biphenyls (PCBs) and polybrominated diphenyl ethers (PBDEs) in breast milk of women from Catalonia, Spain. Organohal. Comp. 66:2560-2566, 2004.

She, J., Holden, A., Sharp, M., Tanner, M., Williams-Derry, C., and Hooper, K. Unusual pattern of polybrominated diphenyl ethers (PBDEs) in U.S. breast milk. Organohal. Comp. 66:3945-3950, 2004.

She, J., Petreas, M., Winkler, J., Visita, P., McKinney, M., and Kopec, D. PBDEs in the San Francisco Bay area: Measurements in harbor seal blubber and human breast adipose tissue. Chemosphere 46:697-707, 2002.

Sjödin, A., Hagmar, L., Klasson-Wehler, E., Kronholm-Diab, K., Jakobsson, E., and Bergman, Å. Flame retardant exposure: Polybrominated diphenyl ethers in blood from Swedish workers. Environ. Health Perspect. 107:643-648, 1999.

Sjödin, A., Jakobsson, E., Kierkegaard, A., Marsh, G., and Sellström, U. Gas chromatographic identification and quantification of polybrominated diphenyl ethers in a commercial product, Bromkal 70-5DE. J. Chromatogr. A. 822:83-89, 1998.

Sjödin, A., Jones, R.S., Focan, J.-F., Lapeza, C., Wang, R.Y., McGahee, E.E. III, Zhang, Y., Turner, W.E., Slazyk, B., Needham, L.L., and Patterson, D.G. Jr. Retrospective time-trend study of polybrominated diphenyl ether and polybrominated and polychlorinated biphenyl levels in human serum from the United States. Environ. Health Perspect. 112:654-658, 2004a.

Sjödin, A., Päpke, O., McGahee, E. III, Jones, R., Focan, J.-F., Pless-Mulloli, T., Toms, L.-M., Wang, R., Zhang, Y., Needham, L., Herrmann, T., and Patterson, D. Jr. Concentration of polybrominated diphenyl ethers (PBDEs) in house hold dust from various countries – inhalation a potential route of human exposure. Organohalogen Compd. 66:3817-3822, 2004b.

Sjödin, A., McGahee, E.E. 3rd, Focant, J.F., Jones, R.S., Lapeza, C.R., Zhang, Y., and Patterson, D.G. Jr. Semiautomated high-throughput extraction and cleanup method for the measurement of polybrominated diphenyl ethers and polybrominated and polychlorinated biphenyls in breast milk. Anal. Chem. 76:4508-4514, 2004c.

Sjödin, A., Patterson, D.G. Jr., and Bergman, Å. Brominated flame retardants in serum from U.S. blood donors. Environ. Sci. Technol. 35:3830-3833, 2001.

Smeds, A., and Saukko, A. Brominated flame retardants and phenolic endocrine disrupters in Finnish human adipose tissue. Chemosphere 53:1123-1130, 2003.

Söderström, G., Sellström, U., de Wit, C.A., and Tysklind, M. Photolytic debromination of decabromodiphenyl ether (BDE 209). Environ. Sci. Technol. 38:127-132, 2004.

Song, W., Ford, J., Li., A., Mills, W., Buckley, D. and Rockne, K. Polybrominated diphenyl ethers in the sediments of the Great Lakes. Environ. Sci. Technol. 38: 3286–3293, 2004.

Stapleton, H.M., Alaee, M., Letcher, R.J., and Baker, J.E. Debromination of the flame retardant decabromodiphenyl ether by juvenile carp (*Cyprinus carpio*) following dietary exposure. Environ. Sci. Technol. 38:112-119, 2004a.

Stapleton, H., Dodder, N., Schantz, M., and Wise, S. Measurement of the flame retardants polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCDD) in house dust. Organohalogen Compd. 66:3740-3744, 2004b.

Staskal, D.F., Diliberto, J.J., Devito, M.J., and Birnbaum, L.S. Toxicokinetics of BDE 47 in female mice: Effect of dose, route of exposure, and time. Toxicol. Sci. e-published ahead of printing, October 27, 2004.

Stern, G.A., and Ikonomou, M.G. Temporal trends of polybrominated diphenyl ethers in SE Baffin beluga: Increasing evidence of long-range atmospheric transport. Organohalogen Compd. 47:81-84, 2000.

Stoker, T.E., Laws, S.C., Krofton, K.M., Hedge, J.M., Ferrell, J.M., and Cooper, R.L. Assessment of DE-71, a commercial polybrominated diphenyl ether (PBDE) mixture, in the EDSP male and female pubertal protocols. Toxicol. Sci. 78:144-155, 2004.

Strandman, T., Koistinen, J., and Vartiainen, T. Polybrominated diphenyl ethers 9PBDEs) in placenta and human milk. Organohalogen Compd. 47:61-64, 2000.

Swedish Chemicals Inspectorate. Decabromodiphenyl ether (decaBDE) - background documentation in support of a national ban: report of a government commission, 2004.

Tange, L., and Drohmann, D. Waste management concept for WEEE plastics containing brominated flame retardants, including bromine recycling and energy recovery. European Brominated Flame Retardant Industry Group, 2002.

Tange, L., and Drohmann, D. WEEE plastics with brominated flame retardants—from legislation to separate treatment. European Brominated Flame Retardant Industry Group, 2003.

ter Schure, A., Larsson, P., Agrell, C. and Boon, J.. Atmospheric Transport of Polybrominated Diphenyl Ethers and Polychlorinated Biphenyls to the Baltic Sea. Environ. Sci. Technol., 38: 1282–1286, 2004.

Thomsen, C., Lundanes, E., and Becher, G. Brominated flame retardants in archived serum samples from Norway: A study on temporal trends and the role of age. Environ. Sci. Technol. 36:1414-1418, 2002.

Thuresson, K., Jakobsson, K., Rothenbacher, K., Herrmann, T., Sjölin, S., Hagmar, L., Päpke, O., and Bergman, Å. Polybrominated diphenyl ethers in blood from Swedish workers – A follow up study in an electronics recycling industry. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 45-48.

Tomy, G., Halldorson, T., Danell, R., Law, K., Stern, G., Gerwutz, S., Whittle, M., Alaee, M., and Marvin, C. Hexabromocyclododecane (HBCD) isomers and brominated diphenyl ether (BDE) congeners in fish from Lake Winnipeg, Manitoba (Canada). In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 213-216.

U.S. Environmental Protection Agency. Biosolids generation, use and disposal in the United States. EPA530-R-99-009. Washington, DC, 1999.

U.S. Environmental Protection Agency. Environmentally Preferable Options for Furniture Fire Safety: Low-Density Foam Volume 1. Draft document, http://www.epa.gov/oppt/dfe/projects/flameret/, September 2004.

van Bavel, B., Dam, M., Tysklind, M., and Lindström, G. Levels of polybrominated diphenyl ethers in marine mammals. Organohalogen Compd. 52:99-103, 2001.

Vehlow, J, Bergfeldt, B., Hunsinger, H., Jay, K., Mark, F., Tange, L, Drohmann, D. and Fisch, H. Recycling of bromine from plastics containing brominated flame retardants in state-of-the-art combustion facilities. Association of Plastics Manufacturers in Europe, 2002.

Vehlow, J., Bergfeldt, B., Jay, K., Seifert, H., Wanke, T., and Mark, F.E. Thermal Treatment of

E+E waste plastics. Waste Management Research 18:131-140, 2000.

Vetter, W. and Jun, W. Non-polar halogenated natural products bioaccumulated in marine samples: II. Brominated and mixed halogenated compounds. Chemosphere 52:423-431, 2003.

Vieth, B., Herrmann, T., Mielke, H., Ostermann, B., Päpke, O., and Rüdiger, T. PBDE levels in human milk: The situation in Germany and potential influencing factors – a controlled study. Organohal. Comp. 66:2643-2648, 2004.

Viberg, H., Fredriksson, A., and Eriksson, P. Neonatal exposure to 2,2´,4,4´,5-pentalbromodiphenyl ether causes increase susceptibility in the cholinergic transmitter system at adult age. Toxicol. Sci. 67:104-107, 2002.

Viberg, H., Fredriksson, A., and Eriksson, P. Neonatal exposure to polybrominated diphenyl ether (PBDE 153) disrupts spontaneous behaviour, impairs learning and memory and decreases hippocampal cholinergic receptors in adult mice. Toxicol. Appl. Pharmacol. 192:95-106, 2003a.

Viberg, H., Fredriksson, A., and Eriksson, P. Investigations of strain and/or gender differences in developmental neurotoxic effects of polybrominated diphenyl ethers in mice. Toxicol. Sci. 81:344-353, 2004.

Viberg, H., Fredriksson, A., Jakobsson, E., Örn, U., and Eriksson, P. Neurobehavioral derangements in adult mice receiving decabrominated diphenyl ether (PBDE 209) during a defined period of neonatal brain development. Toxicol. Sci. 76:112-120, 2003b.

von Meyerinck, L., Hufnagel, B., Schmoldt, A., and Benthe, H.F. Induction of rat liver microsomal cytochrome P-450 by the pentabromodiphenyl ether Bromkal 70 and half-lives of its components in the adipose tissue. Toxicol. 61:259-274, 1990.

Vreugdenhil, H.J.I., Mulder, P.G.H., Emmen, H.H., and Weisglas-Kuperus, N. Effects of perinatal exposure to PCBs on neuropsychological functions in the Rotterdam cohort at 9 years of age. Neuropsychol. 18:185-193, 2004.

Watanabe, I., and Tatsukawa, R. Formation of brominated dibenzofurans from the photolysis of flame retardant decabromodiphenyl ether in hexane solution by UV and sun light. Bull. Environ. Contam. Toxicol. 39:953-959, 1987.

Weiss, J., Meijer, L., Sauer, P., Linderholm, L., Athanassiadis, I., and Bergman, Å. PBDE and HBCDD levels in blood from Dutch mothers and infants – analysis of a Dutch Groningen infant cohort. Organohal. Comp. 66:2677-2682, 2004.

Wenning, R.J., Von Burg, A.M., Pekala, J.M., Maier, M., and Luksemburg, W. Levels of polybrominated diphenyl ethers (PBDEs) in the Hackensack River and Newark Bay, New Jersey U.S.A. In: Third International Workshop on Brominated Flame Retardants (eds. M. Alaee, G. Arsenault, T. Harner, K. MacPherson, G. Pacepavicius, E. Reiner, and F. Wania), 2004, pp. 129-132.

World Health Organization (WHO). Environmental Health Criteria 162: Brominated diphenyl ethers. 1994.

Wiley, K. and McCarthy, B. Polybrominated diphenyl ethers (PBDEs): potential hazards from decaBDE and unresolved issues from AB 302. California Senate Office of Research, June 2004

Wilford, B.H., Harner, T., Zhu, J., Shoeib, M., and Jones, K.C. Passive sampling survey of polybrominated diphenyl ether flame retardants in indoor and outdoor air in Ottawa, Canada: Implications for sources and exposure. Environ. Sci. Technol. in press.

Yan Zhu, L., and Hites, R.A. Temporal trends and spatial distributions of brominated flame retardants in archived fishes from the Great Lakes. Environ. Sci. Technol. 38:2779-2784, 2004.

Zegers, B.N., Lewis, W.E., Booij, K., Smittenberg, R.H., Boer, W., de Boer, J., and Boon, J.P. Levels of polybrominated diphenyl ether flame retardants in sediment cores from western Europe. Environ. Sci. Technol. 37:3803-3807, 2003.

Zhou, T., Ross, D.G., DeVito, M.J., and Crofton, K.M. Effects of short-term *in vivo* exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats. Toxicol. Sci. 61:76-82, 2001.

Zhou, T., Taylor, M.M., DeVito, M.J., and Crofton, K.M. Developmental exposure to brominated diphenyl ethers results in thyroid hormone disruption. Toxicol. Sci. 66:105-116, 2002.

APPENDICES

APPENDIX A

Maine's PBDE legislation: *An Act to Reduce Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products* [PL 2003, c. 629]

APPENDIX B

Thesis paper: *Evaluation of Brominated Organic Compounds in the Penobscot Watershed, Maine*

APPENDIX C

Table of introduced and enacted state and federal PBDE legislation

APPENDIX D

Summary of the U.S. Environmental Protection Agency's understanding of PBDEs

APPENDIX E

Comments of John C. Dean, State Fire Marshal, Maine Department of Public Safety

APPENDIX A

Maine's PBDE legislation

An Act to Reduce Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products [PL 2003, c. 629]

APPROVED

APR 14 '04

BY GOVERNOR

CHAPTER

629

PUBLIC LAW

STATE OF MAINE

IN THE YEAR OF OUR LORD
TWO THOUSAND AND FOUR

H.P. 1312 - L.D. 1790

An Act To Reduce Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products

Be it enacted by the People of the State of Maine as follows:

Sec. 1. 38 MRSA §1609 is enacted to read:

§1609. Restrictions on sale and distribution of brominated flame retardants

1. "Penta" mixture and "octa" mixture of polybrominated diphenyl ethers. Effective January 1, 2006, a person may not sell or offer to sell, or distribute for promotional purposes, a product containing more than 1% of the "penta" or "octa" mixtures of polybrominated diphenyl ethers.

2. Review; report. The department, with the Department of Human Services, Bureau of Health, shall review relevant risk assessments in connection with brominated flame retardants and relevant findings and rulings by the United States Environmental Protection Agency and the European Union. The department shall annually, no later than January 5th, submit a report regarding the regulation of brominated flame retardants to the joint standing committee of the Legislature having jurisdiction over natural resources matters. For purposes of this subsection, "brominated flame retardant" means any chemical containing the element bromine that may be added to a plastic, foam or textile to inhibit flame formation.

3. Application. This section does not apply to the sale of used products.

Sec. 2. "Deca" mixture of polybrominated diphenyl ethers; legislative intent. It is the intent of the Legislature to reduce the risk of the "deca" mixture of polybrominated diphenyl ethers by implementing risk management measures or by prohibiting the sale of products containing more than 1% of the "deca" mixture beginning January 1, 2008 if a safer, nationally available alternative is identified. As part of its annual report pursuant to the Maine Revised Statutes, Title 38, section 1609, subsection 2, the Department of Environmental Protection shall submit a report regarding the nationwide availability of safer alternatives to the "deca" mixture of polybrominated diphenyl ethers to the joint standing committee of the Legislature having jurisdiction over natural resources matters. If, after reviewing the recommendations of the department, the joint standing committee determines that a safer alternative to the "deca" mixture is nationally available, the joint standing committee may report out to any session of any Legislature legislation to implement risk management measures or to enact a prohibition on the sale and distribution of products containing the "deca" mixture.

121st Legislature
Senate of
Maine
Senate District 1

Senator John L. Martin
3 State House Station
Augusta, ME 04333-0003
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June 15, 2004

Mr. Raymond B. Dawson, Chairman
Bromine Science & Environmental Forum
C/o Polymer Chemicals
451 Florida Street
Baton Rouge, LA 70801-1765

RE: LD 1790 – An Act to Reduce Contamination of Breast Milk and the Environment from the Release of Brominated Chemicals in Consumer Products

Dear Ray,

As you know, I appreciate the significant and very important information you provided to the Committee and myself during our deliberation on LD 1790. I know that you expended significant effort to come to Maine on a number of occasions and, from your position, what the Committee approved and the Legislature enacted, was not quite to your liking or that of your colleagues in the industry. I understand your concern, and that of the other members of the Bromine Science and Environmental Forum ("BSEF").

The intent of LD 1790 is to address the very real concern of certain PBDEs being found in human breast milk. LD 1790 addresses this concern by phasing-out the sale or intent for sale or distribution for promotional purposes any product containing the penta or octa mixtures of polybrominated diphenyls effective January 1, 2006. This action is in-line with your industry's voluntary phase out of these materials.

LD 1790 also goes beyond the California law and addresses the deca mixture of polybrominated diphenyl ethers. I know this is the portion of the law that you and others in the industry find troubling. I understand there are some reports that Maine has banned deca. That is not accurate. I would like to clarify that deca remains an acceptable fire retardant for use in the State of Maine. LD 1790 requests that the Department of Environmental Protection include in its annual report to the Legislature's Joint Standing Committee on Natural Resources an update on the nationwide availability of "safer alternatives" to the deca mixture. It is not yet known if such acceptable alternatives will exist and/or if they will meet strong fire safety standards. The Committee will review the annual reports as submitted by the Department to determine if a "safe" alternative that meets the same fire safety requirements is available in all 50 US States. If such alternatives do exist, the Committee may, at that time, report out legislation to implement risk reduction measures or enact a prohibition of the sale of products containing more that 1% of the deca mixture.

Should you have any further questions, please feel free to contact me.

Sincerely,

Senator John L. Martin
Maine State Legislature – *Senate Chair, Joint Standing Committee on Natural Resources*

Fax (207) 287-1585 • TTY (207) 287-1583 • Message Service 1-800-423-6900 • Web Site: http://www.state.me.us/legis/senate

APPENDIX B

EVALUATION OF BROMINATED ORGANIC COMPOUNDS IN THE PENOBSCOT WATERSHED, MAINE - Therese Anderson, EES Ph.D. Candidate, University of Maine (advisor Dr. Jean MacRae)

Brominated Flame Retardants on the Penobscot River.

In recent years, concerns have been rising about the global presence of brominated flame retardants (BFRs) in all areas of the environment. In contrast to the declining levels of polychlorinated biphenyls (PCBs), dioxins and DDT in the environment, levels of BFRs have increased exponentially (1). These compounds are highly lipophilic and readily bioaccumulate in the food chain in a manner similar to dioxins and PCBs.

One class of BFRs is the Brominated Diphenylethers (BDEs). Different degrees of bromination on the diphenyl ether backbone can result in 209 possible congeners, however only a limited number are actually formed due to the chemical directing properties of the ether group. They are commercially produced in mixtures, similar to the Aroclor mixes associated with PCBs. The mixes of concern are the Penta, Octa and Deca formulations. The State of Maine has recently banned the use of the Penta and Octa mixes. The Penta mix is comprised of two major congeners, BDE-47 and BDE-99. These account for over 70% of the total product by weight. BDE-100, BDE-153 and BDE-154 make up the majority of the remaining 30% of the mix. Trace amounts of BDE-17 and BDE-28 are also present. The Octa mix contains predominately BDE-183. BDE-153 and several additional octa and nona substituted BDE are found in minor amounts. The commercial Deca mix is 97% deca with the remainder being nona substituted BDEs. The Deca congener is more difficult to separate and analyze and was not specifically looked for in this study. Trace amounts were found in two wastewater samples.

While the toxicity of these compounds is currently being extensively studied, preliminary work has shown that the pentaBDE mixtures exhibit both dioxin-like Ah receptor mediation and competition with thyroid hormones (T3 andT4) for the transport protein, transthyretin, which could disrupt normal thyroid activity (2, 3). While these hormone effects appear to be lower than exhibited by coplanar PCBs, PBDEs background levels are correspondingly higher and are rising exponentially in North America (1, 4). Many textiles and foams treated with BFRs end up in the solid waste stream and are landfilled or incinerated along with other materials.

The predominant PBDE levels were examined in fish tissue procured for the SWAT/DMP project on the Penobscot River. Separate extractions were performed and the extracts cleaned to maximize the detection of these compounds. Wastewater and sludge samples from Orono Wastewater District were obtained and analyzed. Analysis was performed with low resolution mass spectrometry instead of the high resolution technique outlined in the proposal because the instrument was not available. Also due to the increased costs associated with the low resolution method, the scale of the testing had to be reduced.

Fish samples from sites PBM, PBC, PBV and PBO were analyzed for predominate BFR congeners. Small mouth bass from PBM, PBC and PBM, white suckers from PBV and PBC and eels from PBO were sampled. Wastewater influent and effluent 24-hr composite samples and grab samples of activated sludge were obtained from the Orono wastewater treatment facility. Dewatered biosolids were also obtained and are in process at the time of this report. Results are presented in Table 1. (Concentrations range from noon-detect to 80 ppb in SMB fillets, wet weight, depending on the congener and from non-detect to 500 ppb in whole suckers, wet weight. Wastewater samples ranged from non-detect to 2 ppb. Fish data are in µg/Kg wet weight and wastewater samples are reported on a volume basis. Values lower than the stated detection limits are not reported.

The results for the samples mirror the penta mix composition with BDE-47 and BDE-99 predominating. Totals for some of the congeners decrease as we move down the river but this does not account for all the BDEs found. Since all point sources have yet to be identified this type of analysis cannot be applied to this data set.

These data are consistent with values obtained in previous studies done in both the United States and Europe. Values obtained from the Great Lakes show concentrations for fillets ranging from non-detect to 80 ppb wet weight for congeners other then deca-BDE. Congeners BDE-47 and BDE-99 are the major peaks found after deca. Influent and effluent samples from the Netherlands show concentrations from non-detect to 10 ppb for BDE-47. (5, 6) A target dose for unlimited consumption based on EPA's reference dose for the most toxic mixture, PBDE is 530 ug/kg. Future work includes looking at the fate of BDEs in sludge disposal and attempting to map the major potential point sources in the Penobscot watershed.

1. Ikonomou, M.G., Rayne, S., and Addison R.F., Environ. Sci. Technol. 36:1886-1892, 2002.
2. De Wit, C., Chemosphere 46: 583-624, 2002.
3. Meerts, I.A.T.M., van Janden, J.J., Luijks, E.A.C., van Leeuwen-Bol, I., Marsh, G., Jakobsson, E., Bergman, A., and Brouwer, A., Toxicol. Sci. 56:95-104, 2000.
4. MacDonald, T.A., Chemosphere 46:745-755, 2002.
5. de Boer, J., Wester, P.G., van der Horst, A., and Leonards, P.E.G., Environ. Poll. 122:63-74. 2003.
6. Dodder, N.G., Strandberg, B., and Hites, R>A., Environ. Sci. Technol. 36:146-151. 2002.

Table 1. Poly-Brominated Diphenyl Ethers in Fish and Wastewater Treatment Plants

FISH

Congener	Detection limits	PBV-SMB -1	PBV-SMB -3	PBV-SMB -6	PBV-SMB -7	PBV-SMB -8
Initial weight/volume	grams	20.57	20.76	20.85	20.68	20.66
	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
TriDPE - 17	1.00					
TriDPE - 28	1.00					
TetraDPE - 47	1.00	63.7	47.7	44.1	35.3	18.4
TetraDPE - 71	0.50	1.46	2.89	3.84	2.90	1.45
PentaDPE - 100	0.50	16.5	16.9	6.24	4.84	9.68
PentaDPE - 99	0.50	80.2	62.1	23.5	13.1	43.6
PentaDPE - 85	0.50	0.49				
HexaDPE - 154	0.50	5.83	5.30	2.40	1.93	2.42
HexaDPE - 153	0.50	5.35	5.78	1.92	1.93	1.94
HexaDPE - 138	0.50					
HeptaDPE - 183	5.00					
HeptaDPE - 191	5.00					
DecaDPE - 209	25.00					
TOTAL		**173.6**	**140.7**	**82.0**	**60.0**	**77.4**

Congener	Detection limits	PBC-SMB -8	PBC-SMB -9	PBC-SMB -10	PBC-SMB -11	PBC-SMB -12
Initial weight/volume	grams	20.22	20.93	20.55	19.96	20.4
	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
TriDPE - 17	1.00					
TriDPE - 28	1.00					
TetraDPE - 47	1.00	48.0	72.1	10.2	20.5	3.92
TetraDPE - 71	0.50	1.98	4.30	0.97	1.00	
PentaDPE - 100	0.50	9.40	15.8	6.33	2.51	5.39
PentaDPE - 99	0.50	12.9	33.9	28.7	10.0	5.88
PentaDPE - 85	0.50					
HexaDPE - 154	0.50	5.93	10.5	2.43	6.51	0.98
HexaDPE - 153	0.50	1.48	2.39	2.92	4.01	
HexaDPE - 138	0.50	0.49	1.91		2.51	
HeptaDPE - 183	5.00					
HeptaDPE - 191	5.00					
DecaDPE - 209	25.00					
TOTAL		80.1	140.9	51.6	47.1	16.2

Table 1. Poly-Brominated Diphenyl Ethers in Fish and Wastewater Treatment Plants

FISH

Congener	Detection limits	PBC-WHS -C	PBV-WHS -C	PBM-SMB -7	PBM-SMB -9	PBM-SMB -10
Initial weight/volume	grams	19.99	19.76	20.06	20.17	20.2
	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg	µg/Kg
TriDPE - 17	1.00	1.50	4.55			
TriDPE - 28	1.00	35.5	64.3			
TetraDPE - 47	1.00	430	597	3.49	1.98	
TetraDPE - 71	0.50	4.00	4.55	2.99		
PentaDPE - 100	0.50	279	116	4.99	0.50	1.49
PentaDPE - 99	0.50	3.00	126	12.0	4.46	9.90
PentaDPE - 85	0.50		13.7			
HexaDPE - 154	0.50	60.0	22.3	1.00	0.50	1.98
HexaDPE - 153	0.50	21.5	10.1			1.49
HexaDPE - 138	0.50					3.96
HeptaDPE - 183	5.00					
HeptaDPE - 191	5.00					
DecaDPE - 209	25.00					
TOTAL		**834.9**	**959.0**	**24.4**	**7.4**	**18.8**

Congener	Detection limits	PBO-EEL -C1	PBO-EEL -C1	BLK 1 fish		BLK water
Initial weight/volume	grams	19.99	20.34	20.00		1.00
	µg/Kg	µg/Kg	µg/Kg	µg/Kg		µg/L
TriDPE - 17	1.00					
TriDPE - 28	1.00					
TetraDPE - 47	1.00	114	29.0	0.50		0.08
TetraDPE - 71	0.50	2.50	1.97			
PentaDPE - 100	0.50	58.5	33.4			
PentaDPE - 99	0.50	2.00	4.92			
PentaDPE - 85	0.50	39.5	18.7			
HexaDPE - 154	0.50	1.00	1.97			
HexaDPE - 153	0.50		0.98			
HexaDPE - 138	0.50					
HeptaDPE - 183	5.00					
HeptaDPE - 191	5.00					
DecaDPE - 209	25.00					
TOTAL		217.1	91.0	0.5		0.1

WATER

Congener	detection limits	effluent	effluent	Influent	influent	activated sludge	activated sludge
Initial weight/volume	liters	1.00	1.00	1.00	1.00	1.00	1.00
	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
TriDPE - 17	0.10						
TriDPE - 28	0.10						
TetraDPE - 47	0.10	0.09		1.55	1.73	0.98	0.47
TetraDPE - 71	0.05			0.06	0.08	0.06	0.04
PentaDPE - 100	0.05			0.46	0.42	0.28	0.22
PentaDPE - 99	0.05		0.11	1.59	1.61	0.52	1.15
PentaDPE - 85	0.05					1.33	
HexaDPE - 154	0.05			0.13	0.10		0.10
HexaDPE - 153	0.05			0.12	0.09	0.33	0.14
HexaDPE - 138	0.05						
HeptaDPE - 183	0.50						
HeptaDPE - 191	0.50						
DecaDPE - 209	2.50	3.03			2.66		
TOTAL		**3.1**	**0.1**	**3.9**	**6.7**	**3.5**	**2.1**

APPENDIX C
Introduced and Enacted PBDE Legislation

State	Bill #	Summary	Status
California	AB302; amended by AB2587	Effective June 1, 2006, a person may not manufacture, process or distribute a product, or flame-retarded part of a product, containing more than 0.1% pentaBDE or octaBDE. The prohibition does not apply to processing of metallic recyclables.	Enacted 8/11/03 - 2003 Cal. Stat c. 205 and c. 641
Hawaii	HB2013	Effective January 1. 2006, a person may not manufacture, process or distribute a product, or flame-retarded part of a product containing more than 0.1% by mass of pentaBDE or octaBDE. The prohibition does not apply to processing of metallic recyclables.	Enacted 6/2004 - 2004 Session Laws, c. 146
Maine	LD1790	Effective January 1, 2006, a person may not sell or distribute a product containing more than 1% of pentaBDE or octaBDE. Section 2 of the bill sets forth the Legislature's intent to ban decaBDE beginning January 1, 2008 if a safer, nationally available alternative is identified	Enacted: 4/14/04 - PL 2003, c. 629
Maryland	HB26	Effective October 1, 2008, prohibits a person from manufacturing, processing or distributing products that contain more than one-tenth of 1% of any PBDE. The prohibition does not apply to processing of metallic recyclables.	Did not pass.
Michigan	HB4406 & SB1458 (companion bills)	Effective June 1, 2006, a person may not manufacture, process or distribute a product or material that contains more than 0.1% of pentaBDE or octaBDE. The prohibition does not apply to replacement parts or the processing of recyclables.	Enacted: 12/9/04
Minnesota	HF2232 & SF2193 (companion bills)	Effective January 1, 2006, a person may not manufacture, process or distribute a product, or a flame-retarded part of a product, containing more than 0.1% of pentaBDE or octaBDE by mass. Effective January 1, 2008, a person may not manufacture, process, or distribute a product, or a flame-retarded part of a product, containing more than 0.1% of decaBDE by mass. The prohibition does not apply to the processing of metallic recyclables.	Did not pass.

State	Bill #	Summary	Status
New York	A10050, S7621 (related)	Effective January 1, 2006, a person may not manufacture, process or distribute a product, or flame-retarded part of a product, containing more than 0.1% pentaBDE or octaBDE by mass. The ban does not apply to the resale of products manufactured prior to January 1, 2006. The bill also establishes a Task Force on Flame Retardant Safety to study the risks associated with decaBDE and the availability, safety and effectiveness of alternatives to decaBDE.	Enacted 8/17/04, Chapter 387, Laws of New York, 2004
Washington	HB 1488; SB 5515	Effective January 1, 2006, a person many not manufacture or sell products containing PBDEs including decaBDE. The ban would not apply to: motor vehicles containing components with decaBDE; new products made of recycled materials containing decaBDE; or used products containing PBDEs. The bill also would allow the Department of General Administration to grant exemptions for other products containing decaBDE if the manufacturer shows that: 1) a technically feasible alternative is not available at reasonable cost; or 2) the alternative would pose a greater potential harm to public health or the environment.	Ref. to House Committee on Nat. Res., Ecology & Parks, 1/25/05; Ref. to Senate Committee on Water, Energy & Env., 1/26/05
Federal	HR4076	Effective 2 years after passage, the bill would amend the Toxic Substances Control Act to make it unlawful to manufacture, process or distribute a product containing more than 1 percent of pentaBDE or octaBDE by mass. The bill also would require EPA to determine if pentaBDE or octaBDE are formed in the environment as a result of chemical degradation of another material and to phase out any such precursors.	4/8/04 Referred to Subcommittee on Environment & Haz. Mat.

This table was derived from a table originally compiled by the National Caucus of Environmental Legislators, www.ncel.net

APPENDIX D

Summary of USEPA's Understanding of PBDEs

Polybrominated diphenylethers (PBDEs) are members of a broader class of brominated chemicals used as flame retardants; these are called brominated flame retardants, or BFRs. There are commercial mixtures of PBDEs with different average amounts of bromination: penta-, octa-, and decaBDE. These chemicals are major components of commercial formulations often used as fire retardants in furniture foam (pentaBDE), plastics for TV cabinets, consumer electronics, wire insulation, backcoatings for draperies and upholstery (decaBDE) and plastics for personal computers and small appliances (octaBDE). The value of these chemicals is their ability to slow ignition and rate of fire growth, and as a result increase available escape time in the event of a fire involving the above consumer products.

Although use of these chemicals is intended to save lives and property, there have been unintended consequences. Environmental monitoring programs in Europe, Asia, North America, and the Arctic have detected several PBDEs in human breast milk, fish, aquatic birds and elsewhere in the environment. Tetra- to hexabrominated diphenyl ethers are the PBDEs most frequently detected in wildlife and humans. The exact mechanisms or pathways by which the PBDEs end up in the environment and humans are not known yet, but would include releases from manufacturing or processing of the chemicals into products like plastics or textiles, aging and wear of the end consumer products and direct exposure during use (e.g., from furniture).

EPA is not only interested in responding to monitoring data, however. The Agency continually looks for pollution prevention opportunities; the Pollution Prevention Act of 1990 and EPA's Pollution Prevention Strategy establish that pollution should be prevented or reduced at the source whenever feasible. The Agency has also made protection of children's health a fundamental goal of public health and environmental protection in the United States.

In general, the human health and environmental concerns are higher for the lower brominated mixtures (i.e., pentaBDE and octaBDE), and data suggest that higher brominated forms such as decaBDE can be altered to form more toxicologically active lower brominated forms. The limited toxicity test data that is currently available indicate the potential for adverse effects to humans and environmental organisms, especially for lower brominated mixtures, but existing hazard and exposure information on PBDEs is incomplete. More needs to be understood about the environmental fate and the exposure pathways that lead to PBDE presence in wildlife and people. PBDEs appear to be persistent and bioaccumulative in the environment. EPA believes an improved understanding of potential risks posed by the different PBDE mixtures in their various use applications is needed. EPA is addressing PBDE information needs with a three-pronged approach, which includes:

- Efforts to better understand the environmental properties, exposure pathways and how these chemicals are getting into human tissue;
- Research and detailed testing to determine health and environmental effects; and
- Evaluation of potential substitutes, which includes the analysis of technical performance,

cost-effectiveness and risk-risk tradeoffs related to fire prevention and toxicity.

EPA offices and regions are working with fire safety advocates, industry, environmental and public health groups, other federal agencies, state governments and other national governments to answer the key questions and provide a basis for informed risk reduction decisions, including potential regulatory and voluntary actions. In November 2003, Great Lakes Chemical Corp., the only U.S. manufacturer of pentaBDE and octaBDE, announced a voluntary phase out of production of both those chemicals by the end of 2004. To complement the phase-out, in December 2004, USEPA issued a proposed regulation—a Significant New Use Rule—under the Toxic Substances Control Act to ensure that no new manufacture or import of these chemicals occurs without first being subject to Agency review.

Toxicity

There are commercial mixtures of PBDEs with different average amounts of bromination: penta-, octa- and decaBDE. In general, the human health and environmental concerns are greater for the lower brominated mixtures.

Penta- and OctaBDE

Effects on induction of hepatic enzymes were the basis of the EPA Integrated Risk Information System (IRIS) assessments of commercial pentaBDE and octaBDE mixtures which were completed in 1990. However, although liver enzyme induction was used as the basis for the RfD then, based on current methodology, this endpoint would not now be used as the basis of an RfD given the absence of other negative liver effects or histopathology. An update of the IRIS assessment for PBDEs is in progress. Several recent studies in young laboratory animals (rats and mice) exposed to commercial pentaBDE or to several individual congeners during gestation have shown some evidence of alterations in several behavioral parameters, deficits in learning and memory, and delays in the onset of puberty. Prenatal exposure to octaBDE mixtures in laboratory animals has resulted in reductions in fetal body weight, and delays in ossification - a longer than normal period before hardening of the bones. PentaBDE and octaBDE mixtures and individual congeners have also been shown to disrupt normal thyroid hormone levels in adult rats and mice. This could have possible concerns for developmental neurotoxic effects since it is well-established that disruption of thyroid hormone levels in the pregnant female may affect brain development in the fetus. The National Toxicology Program (an interagency program consisting of relevant toxicology activities of the Centers for Disease Control, Food and Drug Administration and National Institutes of Health) plans to conduct both chronic and subchronic toxicity studies on the commercial pentaBDE mixture, as well as the individual congeners appearing in greatest concentration in the mixture.

DecaBDE

Less is known about the potential toxicity of decaBDE. However, in contrast to penta- and octaBDE, decaBDE is poorly absorbed which may limit its potential toxicity. Some studies have shown thyroid and liver toxicity. Prenatal developmental toxicity studies in animals have been equivocal. A recent study in mice has provided some evidence of behavioral alterations.

The European Commission will be requiring a more complete developmental neurotoxicity study in rodents to help clarify the potential for decaBDE exposure to result in developmental neurotoxicity. In addition, exposure to very high doses of decaBDE has been shown to cause tumors in laboratory animals.

Exposure

PBDEs have been measured in breast milk, adipose tissue and blood serum from human populations in Sweden, Finland, Germany, Japan, Spain, Canada and the United States. PBDE concentrations have steadily increased over 20 years of monitoring conducted in Sweden and Germany. In Sweden, PBDE levels in breast milk had doubled every 5 years between 1972 and 1997, with a decreasing trend since 1997. North American data are limited and additional studies are ongoing to determine relative levels in breast milk and blood serum compared to those found in Europe. However, average levels as measured in 23 human adipose tissue samples and 32 serum samples from among California women and 50 breast milk samples from Canada were higher than PBDE levels measured in Sweden.

Limited monitoring studies have found PBDEs in air, water, sediment, biota and sewage sludge throughout North America. The highest concentrations are generally associated with locations near facilities manufacturing or processing PBDEs. Concentrations of PBDEs are higher in municipal sewage sludge than in other environmental media. Recently reported PBDE concentrations in the United States and Canada are greater than those reported in Europe and Asia.

Different congeners are found at different levels in environmental media and wildlife. Generally the highest measured concentrations are for the tetra (>50%), penta (20-30%), hexa (15-20%) and hepta and octa brominated (< 20%) congeners. Which congeners are found and their relative and absolute concentrations vary from site to site.

Questions and Answers on PBDEs

1. What are PBDEs?

Polybrominated diphenylethers (PBDEs) are members of a broader class of brominated chemicals used as flame retardants; these are called brominated flame retardants, or BFRs. There are three commercial mixtures of PBDEs with differing average amounts of bromination: penta-, octa-, and decaBDE.

2. What are PBDEs used for?

These chemicals are major components of commercial formulations often used as flame retardants in furniture foam (pentaBDE), plastics for TV cabinets, consumer electronics, wire insulation, and backcoatings for draperies and upholstery (decaBDE), and plastics for personal computers and small appliances (octaBDE). The benefit of these chemicals is their ability to slow ignition and rate of fire growth, and as a result increase available escape time in the event of a fire.

3. What are concerns associated with PBDEs?

Although use of flame retardants saves lives and property, there have been unintended consequences. There is growing evidence that PBDEs persist in the environment and accumulate in living organisms, as well as toxicological testing that indicates these chemicals may cause liver toxicity, thyroid toxicity, and neurodevelopmental toxicity. Environmental monitoring programs in Europe, Asia, North America, and the Arctic have found traces of several PBDEs in human breast milk, fish, aquatic birds, and elsewhere in the environment. Particular congeners , tetra- to hexabrominated diphenyl ethers, are the forms most frequently detected in wildlife and humans. The mechanisms or pathways through which PBDEs get into the environment and humans are not known yet, but could include releases from manufacturing or processing of the chemicals into products like plastics or textiles, aging and wear of the end consumer products, and direct exposure during use (e.g., from furniture).

4. What is the Agency doing to better understand the possible risks from exposure to PBDEs?

EPA is currently evaluating a risk assessment and data needs analysis on PBDEs that was developed by industry for the Voluntary Children's Chemical Evaluation Program (VCCEP). This assessment evaluates the potential risks to children and prospective parents from all potential exposure scenarios. EPA will be releasing its views of the assessment, including any further VCCEP data needs, in the next few months.

Directly or through grant mechanisms, EPA has been supporting research aimed at a range of topics related to PBDEs, including measuring PBDE levels in umbilical cord blood from newborn U.S. infants, mothers' blood, house dust, food, breast milk, and children; potential thyroid toxicity and developmental neurotoxicity; and the environmental fate of the PBDEs upon their release during production or after disposal of products that contain these chemicals. EPA's Office of Research and Development, National Center for Environmental Assessment, is enhancing its Integrated Risk Information System (IRIS) database on the PBDEs. IRIS is a database of human health effects that may result from exposure to substances found in the environment. The Agency developed IRIS to provide consistent information on chemical substances for use in risk assessments, decision-making and regulatory activities. The information in IRIS is intended for those without extensive training in toxicology, but with some knowledge of health sciences.

5. How does this action complement the decision by the sole US manufacturer to phase out production by December 31, 2004?

This action builds on the November 3, 2003, announcement by the Great Lakes Chemical Corporation, the only U.S. manufacturer of these chemicals, who agreed to voluntarily phase-out production by December 31, 2004. In 2003, EPA commended Great Lakes Chemical Corporation for taking this responsible action. EPA is concerned that manufacture or import could be reinstated in the future, and thus believes it is necessary to have the opportunity to evaluate any new manufacture or import associated with these chemicals.

6. Why are these chemicals important and are there substitutes?

These chemicals provide a very important benefit because of their ability to save lives and property by slowing ignition and rate of fire growth, and therefore increase available escape time in the event of a fire. However, EPA also believes both the phase out and the Significant New Use rule will further spur the development of safer alternatives.

EPA has been working to ensure that following the phasing out of these two chemicals, acceptable alternatives are available to industry. Such alternatives would need to meet technological requirements of industry users, flame retardancy requirements in US standards, and present lower hazards than the chemicals for which they are substituting. To promote these goals and to explore the safety of alternative flame retardant chemicals, EPA has convened a group of stakeholders in its Furniture Flame Retardancy Partnership, including chemical manufacturers and users, the furniture industry, government agencies, and consumer groups, who will work together to evaluate possible alternatives to PentaBDE.

7. Should consumers discard any products that might contain PentaBDE or Octa?

No, the EPA does not believe that there is a need to remove or replace products that may contain these chemicals. EPA has not concluded that PBDEs pose an unreasonable risk to human health or the environment. However, due to growing concerns, EPA believes that the phase out and the regulatory action taken in this announcement are useful steps to minimize and ultimately help prevent further exposure to these chemicals.

8. What are PBDEs commonly used for?

The PBDEs are major components of commercial formulations often used as fire retardants in furniture foam, plastics for TV cabinets, consumer electronics, wire insulation, and back-coatings for draperies and upholstery, and plastics for personal computers and small appliances. These chemicals slow ignition and rate of fire growth, and, as a result, increase available escape time in the event of a fire involving the above consumer products.

9. How are people exposed to PBDEs?

PBDEs are not chemically bound to plastics, foam, fabrics, or other products in which they are used, making them more likely to leach out of these products. PBDEs may enter the air, water and soil during their manufacture and use in consumer products. The primary route of human exposure is currently unclear.

10. What is the Agency doing to better understand the occurrence of PBDEs in the environment?

EPA is addressing PBDE information needs with a three-pronged approach which includes: 1) efforts to better understand the environmental properties, exposure pathways, and how these chemicals are getting into human tissue; 2) research and detailed testing to determine health and environmental effects from exposure to PBDEs; and 3) evaluation of potential PBDE substitutes, which includes the analysis of technical performance, cost-effectiveness, and risk-risk tradeoffs related to fire prevention and toxicity.

11. What efforts are underway to discourage continued use of the PBDEs?

In November 2003, the Great Lakes Chemical Corporation announced a voluntary phase out of PentaBDE and OctaBDE by the end of 2004. Great Lakes is the only U.S. manufacturer of these PBDEs. To follow up on this voluntary action, EPA is working with chemical manufacturers and end users to facilitate an orderly transition to safer substitutes. The State of California has enacted a law banning use of PentaBDE and OctaBDE by January 2008 (recently changed to June 1, 2006) and other states (including Maine, Hawaii, Washington, and New York) are also considering or have passed similar legislation. In Europe, the European Union enacted a ban on PentaBDE and OctaBDE in all products which took effect on August 15, 2004.

EPA is also working with the fire safety advocates, chemical manufacturers, manufacturers of end products such as furniture or plastics for electronics, environmental and public health groups, other federal agencies, state governments, and other nations to answer key questions and help people make informed decisions based on risk. EPA is considering both regulatory and voluntary actions.

APPENDIX E

STATE OF MAINE
Department of Public Safety
Office of State Fire Marshal
52 State House Station
Augusta, ME 04333-0052

JOHN ELIAS BALDACCI
GOVERNOR

MICHAEL P. CANTARA
COMMISSIONER

JOHN C.DEAN
STATE FIRE MARSHAL

November 5, 2004

John James
DEP-BRWM
17 State House Station
Augusta, ME 04333

Dear John:

Thank you for giving me the opportunity to offer comments on the issue of brominated flame retardants.

The National Association of State Fire Marshals (NASFM) has been very active in researching this issue. As the Vice-President of NASFM I can assure you that we believe that fire safety, health and the environment are equally important to us all.

The bottom line on this issue is that NASFM's policy is to insist on good science here. Chemicals must be evaluated one-by-one. We stand by our policy statement with US EPA that NASFM does not support FR chemicals that pose unreasonable risks, but neither will we tolerate political and commercial attacks on FRs that scientists tell us can be used safely.

NASFM is moving forward with the US EPA and other environmental and health organizations to sort through the questions that are being asked about FRs.

Background

It is hard to separate out the political and commercial noise from the truth in these matters. New Jersey's Chief of Fire Code Enforcement, George Miller, has made it his business to dig deep into these issues. George chairs the International Consortium for Fire Safety, Health and the Environment (ICFSHE), which consists of government authorities and scientists from Belgium, Canada, Denmark, Finland, Germany, the Netherlands, Sweden, and the United Kingdom, as well as senior officials from US EPA, UL, FM Global Research, the American Plastics Council and the California Fire Chiefs representing the US. Scientists from the World Health Organization and the Society of Toxicology participate as well.

The remainder of this letter is from George Miller and I believe we should carefully consider his comments and observations as we proceed here in Maine.

The big issue here is plastics, not FRs. FRs make it possible for manufacturers to use various plastics in products that must meet fire safety requirements. Therefore, FRs are found in products sold for use in the home, health care facilities, schools, offices and retail occupancies, and are commonly used with building materials and virtually all modes of transportation. Where manufacturers use less combustible material, FRs generally are not used.

Like all chemicals, FRs may harm people and the environment. To do so, they must be fairly stable, actually enter biological systems, and then have the ability to cause harm. Most toxicologists seem to agree that if a chemical accumulates to any real extent in the human body, that is a red flag.

The big challenge is to know enough about these and other chemicals to make good choices. Chemical producers who subscribe to the American Chemistry Council's Responsible Care © program, agree to conduct the research necessary to understand the potential health effects of their products. We have a right to expect them to honor that commitment.

For all of the bad press and legislative attention received by the chemicals known as PBDEs, toxicologists consistently tell us they know much more about these FRs than any other category. Two of the three FRs in this category have been voluntarily withdrawn, and the third PBDE, known as deca-BDE or decabrom, has been given an incredibly thorough examination by European environmental agencies, who have recommended its continued use as long as research continues. If one is to be objective, the PBDEs should be considered a major success because policy-makers took the time to dig into the details and make important distinctions. Because of the science, industry and regulators have been able to make important distinctions among similar FRs. They are withdrawing two FRs and have learned that deca-BDE now can be used with confidence with new fire safety standards for furniture, mattresses, electronics, building materials and the rest. It is time to ignore the political and commercial rhetoric and let the science do the talking.

By comparison, little is known about some other important FRs. Last month, the Society of Toxicology -- the world's foremost professional organization of toxicologists -- canceled a workshop on chlorinated phosphate ester and boric acid FRs. The data are so scant on some of these products that the workshop could not be justified. New FRs are under development and others are now under review as new chemicals by US EPA.

The irony here is that the one group of existing FRs for which we have ample data has received the harshest political treatment. A second group of FRs is not supported by much data, and therefore careful scrutiny is not possible. A third group of new FRs -- arguably the best of the lot -- is receiving proper attention but may be withheld from commercial use until they are approved. This strikes us as a triple standard, at a time when one high standard is so badly needed.

Please contact me if I can be of further assistance.

Sincerely,

John C. Dean
State Fire Marshal