GROWING THREATS

Toxic Flame Retardants and Children's Health

Travis Madsen
Susan Lee
Teri Olle

Environment California Research and Policy Center

2003

ACKNOWLEDGEMENTS

The Environment California Research and Policy Center gratefully acknowledges Ake Bergman at the Stockholm University Department of Environmental Chemistry, Linda Birnbaum, Director of the Toxicology Division at the U.S. Environmental Protection Agency, and Tom Webster at Boston University for review and comments on the technical and scientific aspects of the report. The views of these individuals are their own and do not necessarily reflect the opinions of their employers. Additionally, the Center thanks Sonya Lunder at Environmental Working Group, Lena Brook at Clean Water Action / Clean Water Fund, and Jane Williams at California Communities Against Toxics for review of the overall document. The Center is especially indebted to Joel Tickner and graduate students at the Lowell Center for Sustainable Production at the University of Massachusetts for scientific guidance. Additional thanks to the many scientists and government agency staff who provided information and review for this report. Thanks to Susan Rakov, Brad Heavner, Jeremiah Baumann, and Tony Dutzik for editorial assistance.

This report was made possible by the generous support of the California Wellness Foundation.

The authors alone bear responsibility for any factual errors. The recommendations are those of the Environment California Research and Policy Center. The views expressed in this report are those of the authors and do not necessarily express the views of our funders.

For additional copies of this report, send $10 (including shipping) to:

Environment California Research and Policy Center
3435 Wilshire Blvd, Ste 385
Los Angeles, CA 90010

The Environment California Research and Policy Center, the new home of the CALPIRG Education Fund's environmental programs, is a 501 (c)(3) organization that offers an independent, articulate voice on behalf of the public interest in California. Drawing upon 30 years of experience, our professional staff combines independent research and practical ideas to uncover environmental problems, develop pragmatic policy solutions and engage citizens in our work for meaningful results.

For more information about Environment California please contact: (213) 251-3688
Or visit the Environment California website at www.EnvironmentCalifornia.org

Table of Contents

Executive Summary

Brominated flame retardants are chemicals that reduce the spread of fire in a variety of common products from fabrics to plastic. First introduced 30 years ago, they are now widely used despite minimal health testing, and they are rapidly building up inside our bodies. The testing that has been done indicates that brominated flame retardants are toxic to development and the levels found in some mothers and fetuses are rapidly approaching the levels shown to impair learning and behavior in laboratory experiments.

This report presents the latest scientific understanding of these toxic flame retardants in North America, their presence in our bodies and the environment, and their likely effects on children's health.

Toxic flame retardants pose risks to human health and the environment.

Manufacturers of consumer products commonly add flame-retardant chemicals to plastics and other flammable materials to reduce the risk of fire. One class of these chemicals, known as brominated flame retardants, now widely contaminate the environment, are accumulating in the human body, and have the potential to harm human health. The most studied of the brominated flame retardants are the polybrominated diphenyl ethers, or PBDEs. North American industry used 74 million pounds of PBDEs in 1999, accounting for half the world market. These chemicals escape into the environment from common consumer products like home furniture and electronics (including TVs, computers, and others) during manufacture, use, and disposal.

PBDEs are remarkably similar to PCBs, a class of chemicals banned in 1976 because it was found to cause immune suppression, altered sexual development, cancer, delayed brain development, lower IQ, and behavioral problems like hyperactivity in humans. As with PCBs, exposure to PBDEs may be particularly harmful during a critical window of brain development during pregnancy and early childhood.

Levels of toxic flame retardants in people are rising dramatically.

Some types of PBDEs concentrate in the fatty tissues of living organisms. As a result, they bioaccumulate, or build up in the food chain, and now can be found in human blood, fat tissue, and breast milk. Initial studies of PBDE contamination of breast milk indicate U.S. levels are 40 to 60 times higher than levels found in Sweden. Levels of PBDEs in animal and human tissues are growing exponentially, doubling every two to five years. At this rate, tissue levels will increase 100- to 1000-fold every 25 years.

When exposed to sunlight or when ingested by animals, some forms of PBDEs which do not themselves readily bioaccumulate may degrade in the environment into more bioaccumulative compounds. As a result, all commercial PBDE compounds should be considered bioaccumulative for policy purposes.

Levels of toxic flame retardants in people have already reached levels of concern.

Recent research shows that PBDE exposure can interrupt brain development in mice, permanently impairing learning and movement. So far, scientists have not identified "safe" levels of exposure that do not produce damage. Additionally, both PCBs and PBDEs are found in humans, and their effects on brain development may be additive. The most highly exposed people may now have PBDE levels within two-fold of the levels shown to damage mice. If PBDE concentrations in people continue to double every 2.5 years, levels found in the average person will reach this threshold within ten years.

Experience with PCBs shows that failure to act on early warnings can lead to irreversible environmental contamination and damage to health.

Scientists discovered the first indications of systemic harm caused by PCBs as early as 1937. However, PCBs were not banned until 1976, after hundreds of scientific studies documented widespread exposure and actual harm to human health. Further study showed new forms of health impact caused by lower levels of exposure, which continue to be documented decades after the chemicals were phased out.

Phasing out chemicals leads to reduced contamination and exposure levels.

The European Union reduced the use of PBDEs in the late 1990s after finding increasing levels in the breast milk of Swedish mothers and preliminary evidence of toxic effects. Since 1998, concentrations of PBDEs in breast milk of Swedish women have declined steadily. Similarly, PCB levels found in the population began to decline after the U.S. banned the chemical. Reducing exposure prevented further harm to human health.

Safer means of fire-proofing products are widely available.

A variety of furniture, plastic, and electronics manufacturers have already deployed products that meet fire-safety standards without the use of PBDEs. Other strategies for flame-resistance include using inherently non-flammable materials and using alternative flame-retardant chemicals. For example, the furniture company IKEA recently replaced brominated flame retardants in fabrics with less toxic chemicals, and the Toshiba electronics company replaced toxic flame retardants in casings for electronic parts by switching to a non-flammable type of plastic that didn't need any chemical additives.

POLICY RECOMMENDATIONS

The European Union has acted on early warnings of a significant health threat by banning several toxic flame retardants. In early 2003, the European Union officially banned the use of PBDEs and other toxic chemicals in electronics (such as computers and lighting) after mid-2006. A more comprehensive ban on the general marketing and use of several toxic flame retardants in Europe is on track for August 2004.

Phase Out Toxic Flame Retardants

There are still unexplored aspects of the toxicity of brominated flame retardants, and complete study would take many years. However, the evidence indicates that immediate action is warranted in California and the United States. Given the magnitude of the potential threat to public health, the rapidly increasing levels of exposure, and the availability of alternatives, this report recommends immediately phasing out the use of PBDEs and other brominated flame retardants.

Reform U.S. Chemicals Policy

The threat posed by toxic flame retardants demonstrates a national failure to effectively protect public health from toxic chemicals used in industry and placed in consumer products. Tens of thousands of industrial chemicals are on the market with little or no information about potential health impacts. Even where significant evidence of harm to public health exists, inadequate resources and legal authority prevent regulatory agencies from taking protective action.

Chemicals that are untested or known to be hazardous should not be on the market or in widespread use and distribution. U.S. chemicals policy should be reformed to ensure that manufacturers and industrial users provide regulatory agencies and the public with adequate information about their products so that agencies can act to protect public health from potentially dangerous substances before damage is done. The case of toxic flame retardants presents an apt case study of the failings of current policy.

INTRODUCTION

California has the toughest furniture fire safety standards of all U.S. states. These regulations prevent fires and save lives. The U.S. Association of Fire Marshals estimates that if the United States as a whole had flammability standards for furniture as strong as those in California, the number of fires would be reduced by 4,000 per year (or 20%), and fire deaths would be reduced by half, or 400 deaths per year.[1]

Manufacturers of consumer products use flame-retardant chemicals to meet fire safety standards. For the past three decades, one class of chemicals known as brominated flame retardants has been added to products ranging from furniture foam to upholstery fabric to the housings of televisions and other electronics. The use of brominated flame retardants, which contain the toxic chemical element bromine, has created some unanticipated problems. In the emerging case of the polybrominated diphenyl ethers (PBDEs), these problems are becoming all too clear. PBDEs have now spread around the world and are steadily accumulating in the tissues of human beings and other animals. From the breast tissue of women in San Francisco to the blubber of Arctic whales, these toxic chemicals are a much closer part of our lives than their manufacturers ever intended.

Lab research indicates that the toxic flame retardants now found in our bodies have the potential to disrupt the process of brain development in fetuses and infant children. Humans are constantly exposed to a mixture of these chemicals from the first day in the womb. These chemicals may be working together to interrupt normal brain development and produce other toxic effects. At the same time, various studies have found dramatically increasing numbers of children with developmental, learning, and behavior disorders over the last decade, including attention deficit disorder, attention deficit hyperactivity disorder, and autism.[2] While it is usually impossible to connect a single chemical to a broad health trend, the National Academy of Sciences recently estimated that toxic exposures play a role in as many as 1 in 4 cases of developmental disorders.[3] Toxic flame retardants could be joining lead, mercury, and PCBs among the chemicals responsible for harming children's health and development.

Recent concern about PBDEs is eerily reminiscent of the debate over PCBs, (polychlorinated biphenyls) in the 1960s, which led to their ban in the mid 1970s. After incidents of accidental PCB poisoning prompted concern, scientists found that low-level exposure to PCBs was a worldwide problem. After years of study, scientists began to find adverse health effects at PCB levels found in the general population. For example, children born to mothers who had eaten PCB-contaminated fish from the Great Lakes had learning, memory, and behavioral problems. Severe and irreparable damage was found in accidental poisoning victims, including altered reproductive and neural development, immune suppression, and cancer. Even twenty-seven years after these chemicals were banned in the U.S., PCB contamination and exposure persists across the globe today.

Several brominated flame retardants are structurally quite similar to PCBs, and consequently may affect the body in similar ways. As such, brominated flame retardants may have the dubious honor of becoming the modern successor to PCBs.

Fortunately for public health, alternative ways to protect against fire are widely available. Companies are coming up with new ways to design products to be flame-resistant, using inherently non-flammable materials and switching to less toxic chemical additives in their products.

Toxic flame retardants are only one class of many different chemicals in wide use despite inadequate study of health effects and inadequate restrictions on use where health effects are known. Investigating potential hazards and taking regulatory action to protect health when threats are discovered can help lead to a world that is both safe and healthy for our children.

1. Types and Uses of Toxic Flame Retardants

Flame-retardant chemicals are commonly added to plastics and other flammable materials as a fire safety measure. There are hundreds of different types of flame-retardant chemicals on the market today. One class of flame-retardant chemicals, known as brominated flame retardants (BFRs), contain the chemical element bromine. Brominated flame retardants are used in products ranging from the polyurethane foam found in padded furniture to upholstery fabrics to the plastic housings of computers and other electronics. These products–and consequently the chemicals they contain–are a part of every facet of modern life, from the chairs we sit on, to the computers at which we work, to the wiring in our homes.

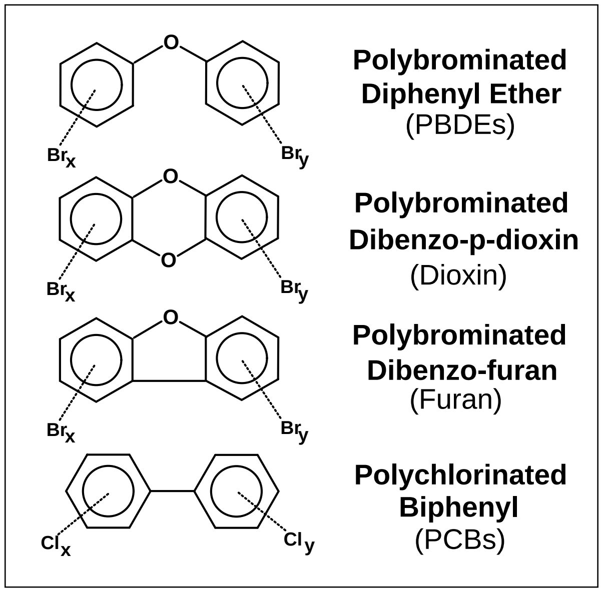
Brominated flame retardants have been commercially available since the 1960s, when the chemical industry found a new use for excess bromine stocks created by the phase out of a bromine-containing pesticide.[4] The demand for these chemicals began to grow in the 1980s as the use of flammable plastics in products increased, accompanied by tougher fire safety standards.[5]

Brominated flame retardants are added to plastics, foams, and textiles at between 5% and 30% of the total product weight.[6] They can be found in a wide variety of consumer products, including computers and other electronic office equipment, televisions, textiles, cushioned furniture in homes, cars, ships, and airplanes, and in insulating foams, cables, and other building materials.[7]

These flame-retardant chemicals are used in two different ways: as additives, or as reactive components that end up chemically bound to the material. Because they are not chemically bound to the product, additive flame retardants can escape to the air or leach out of a product during use or after disposal, contaminating the environment.

The four most-studied types of brominated flame retardants are polybrominated diphenyl ethers (PBDEs), polybrominated biphenyls (PBBs), tetrabromobisphenol-A (TBBPA), and hexabromocyclodecane (HBCD). Although all of these chemicals are of concern, this report mainly focuses on the polybrominated diphenyl ethers, because more information is available about this class. Although PBB production has been discontinued, the other chemicals are still manufactured and used in high volumes across the United States.

Figure 1: Similarities in structure: PBDEs, Dioxins, Furans, and PCBs

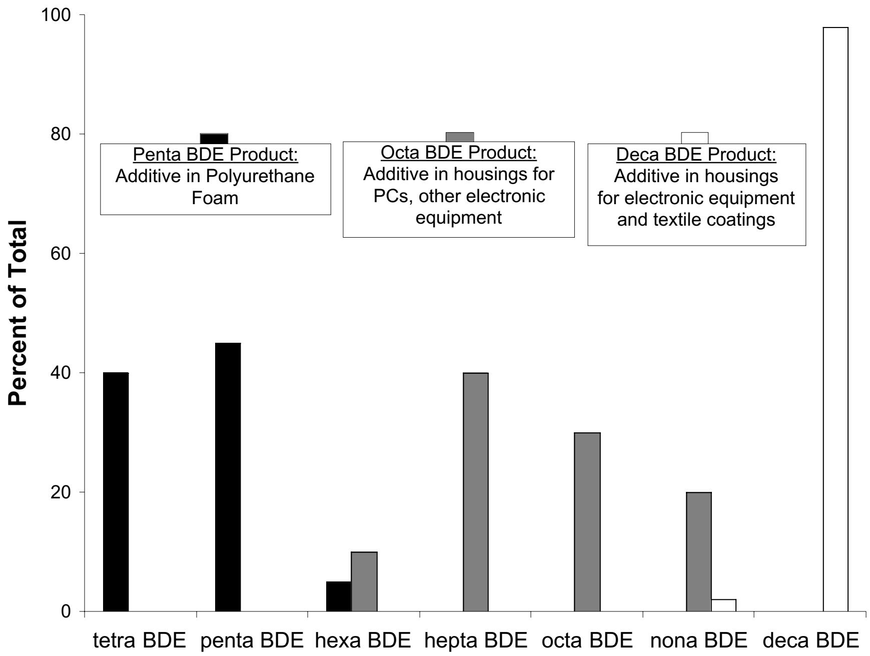


Polybrominated Diphenyl Ethers (PBDEs)

Polybrominated diphenyl ethers are used as additive flame retardants in a wide variety of everyday products, from polyurethane foam in furniture to the high-impact plastics used in computer casings. PBDEs have been continuously produced and used as flame-retardant additives since the 1970s. There are 209 different molecules in the PBDE class, each named according to the number of attached bromine atoms and their geometry. The different molecules are called congeners, and each one is assigned a number.

PBDEs closely resemble polychlorinated biphenyls (PCBs), dioxins, and furans in their structure (see Figure 1), and they are produced commercially

Figure 2: Makeup of commercial PBDE products.



as mixtures, much as PCBs were. Three different mixtures of PBDE are commercially available: Deca, Octa, and Penta BDE. Each product contains a mixture of different molecules with different numbers of bromines attached. (For clarity, the capitalized phrases "Deca, Octa, or Penta BDE product" refer to these commercial mixtures. Lower case tetra, penta, hexa, hepta, octa, nona, and deca BDE refer to individual components of the mixtures, groups of molecules with between four and ten bromines attached. Figure 2 shows the relative makeup of each commercially available mixture.)

Penta BDE

The Penta BDE product is mainly used as an additive in polyurethane foams made in the United States.[8] Polyurethane foams end up in a wide array of upholstered products, ranging from home furniture to seats in airplanes and automobiles.[9]

The Penta BDE product contains a mixture of molecules with 4, 5, or 6 bromines (tetra, penta, and hexa BDEs). These molecules are added to polyurethane foam, and are not chemically bound. As a result, they can escape from the finished product over time. Some of the components of the commercial Penta BDE product are resistant to biodegradation and persist in the environment.[10] They are also quite insoluble in water and concentrate in the fatty tissues of living organisms.[11] Components of the commercial Penta BDE product are found in organisms worldwide. Because of these properties, the European Union recently decided to ban the use of the Penta BDE product in electronic equipment by mid-2006. A separate directive will ban the general marketing and use of the Penta product in Europe by mid-2004.

Octa BDE

The commercial Octa BDE product is used primarily as an additive to a type of plastic known as acrylonitrile-butadiene-styrene (ABS), which is used in housings for office and medical electronics (such as fax machines and computers), the interior and exterior trim of automobiles, telephone handsets, domestic appliance casings (such as food mixers), and others.[12]

The Octa BDE product contains molecules with six to ten bromine atoms attached. Some of the components of the Octa product share similar properties with the Penta product, and have accumulated in a variety of organisms. The European Union recently decided to ban the Octa BDE product in electronic equipment by mid-2006. A separate directive will ban the general marketing and use of the Octa product in Europe by mid-2004.

Deca BDE

Commercial Deca BDE is mainly added to high-impact polystyrene plastic, which is used in a variety of common products including the housings for televisions, computers, stereos and other electronics, recording tape cassettes, and potentially other products subject to fire safety standards including plastic furniture and plastic toys.[13] Deca is also the only PBDE used on upholstery textiles, such as polypropylene.[14] Because it is not chemically bound to the materials in which it is used, it can escape into the environment.

Chemical industry scientists have asserted that the chemicals found in the Deca BDE product are too large to be efficiently taken up by organisms.[15] However, deca BDE has recently been found and quantified not only in peregrine falcons in Europe, but in the blood of workers at electronics recycling plants.[16] Although deca BDE itself does not appear to bioaccumulate as readily as some tetra, penta, and hexa BDEs, lab experiments have demonstrated that it can break down into these molecules when exposed to sunlight, and could be transformed into hexa BDE in fish.[17]

As with the Penta and Octa BDE products, the European Union recently decided to ban the Deca BDE product in electronic equipment by mid-2006.[18] A separate directive may ban the general marketing and use of the Deca product in Europe based on the results of a risk reduction strategy that will be completed in mid-2003.

Polybrominated Biphenyls (PBBs)

Polybrominated biphenyls (PBBs) are nearly identical to polychlorinated biphenyls (PCBs). The only difference is that they contain bromine molecules instead of chlorine.

Nine million U.S. citizens were exposed to PBBs after a flame retardant containing up to 1,000 pounds of PBBs was accidentally mixed with cattle feed in lower Michigan in 1973.[19] PBBs are still found in people in lower Michigan even today, 30 years after the contamination incident. PBB exposure produces health effects similar to PCBs, including hormone disruption, developmental defects, and possibly cancer.[20] Because of the cattle feed contamination incident, industry voluntarily stopped making PBBs in 1976.[21] However, there are currently no official federal guidelines or restrictions on PBB use or exposure.[22]

Tetrabromobisphenol-A (TBBPA) and Hexabromocyclododecane (HBCD)

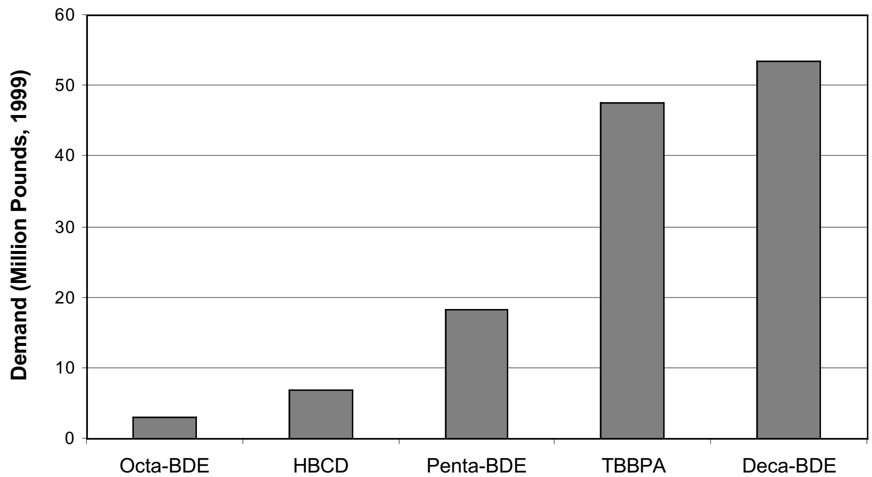
At least two other types of brominated flame retardants are used in high volumes in the U.S. While they have not been studied as thoroughly as the toxic flame retardants discussed above, these chemicals have been associated with potentially negative consequences for public health and the environment.

Tetrabromobisphenol-A (TBBPA) is mainly used in printed circuit boards like those in personal computers and other electronic products, as well as in the plastic housings of office equipment.[23] Sometimes TBBPA is chemically attached to the plastic, but sometimes it is used as an additive and thus can escape from products. It has also been found in the environment and in the food chain.[24] Concern over the ability of TBBPA to form dioxins and disrupt the endocrine system has prompted some electronics manufacturers to seek out alternatives.

Hexabromocyclododecane (HBCD) is mainly used as an additive to the plastic polystyrene, which is used in products like television and computer housings, and in textiles.[25] Although very few studies have looked at the toxicity or environmental levels of this compound, it has been shown to find its way into river sediment near textile manufacturing sites.[26]

Though neither TBBPA nor HBCD have been comprehensively tested for potential to harm human health, some manufacturers are turning to these chemicals as alternatives to the PBDEs. New and emerging information suggests that both of these chemicals can disrupt the endocrine system,

Figure 3: Brominated flame retardant demand in North America, 1999.[32]



and may impair brain development, warranting caution before being used as a replacement for other toxic flame retardants.[27]

Other Potentially Toxic Flame Retardants

Dozens of additional flame-retardant chemicals containing bromine or chlorine are on the market and in use today. Little to no study has been conducted to determine their potential impacts on public health.

USE OF TOXIC FLAME RETARDANTS IS PARTICULARLY HIGH IN NORTH AMERICA

Worldwide, brominated compounds account for about a quarter of all flame retardants produced each year.[28] In 1999, the global industry produced nearly 450 million pounds of brominated flame retardants. TBBPA production accounted for over half of this weight (270 million pounds), commercial Deca BDE represented roughly a quarter (120 million pounds), HBCD accounted for just under 10% (35 million pounds), and commercial Penta and Octa BDE made up 6% (19 million pounds and 8.4 million pounds, respectively).[29] In North America, Deca BDE and TBBPA are the most widely used brominated flame retardants (Figure 3).

Global demand for PBDEs totaled 150 million pounds in 1999, half of which was used by North American industry (74 million pounds). Although much of the world had stopped manufacturing the Penta BDE product by 1999, U.S. manufacturers continue to produce and use this chemical; in fact, 98% of the world's Penta product is used in North America.[31] Figure 4 shows the demand for PBDEs by region.

Two companies in the U.S. carry out actual production of PBDEs: Albemarle Corporation in Magnolia, Arkansas makes the Octa and Deca BDE products, and Great Lakes Chemical Corporation in El Dorado, Arkansas makes the Penta, Octa, and Deca BDE products. Great Lakes Chemical produces the majority of the world's Penta BDE product. Production in Europe has been discontinued.

Globally, the electrical and electronic product industry uses about half of the brominated flame retardants. The building and construction industry, the textile industry, and the transportation industry use the remainder (Figure 5).

Figure 4: World PBDE demand. [34]

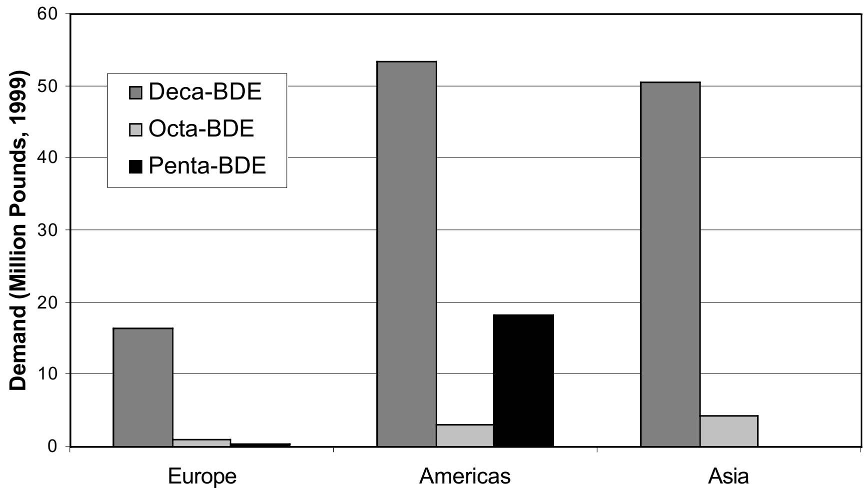
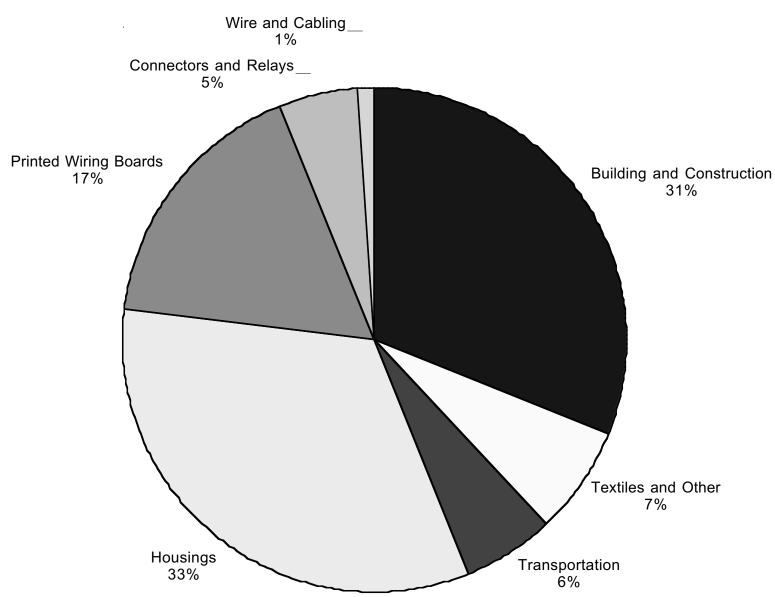


Figure 5: Breakdown of brominated flame retardant use by industry. [33]



2. Contamination of Humans, Animals, and the Environment

California is home to some of the highest observed levels of PBDEs in the world thus far. However, PBDEs have shown up nearly everyplace scientists have looked for them.

TOXIC FLAME RETARDANTS ARE TURNING UP EVERYWHERE, WITH PARTICULARLY HIGH LEVELS IN NORTH AMERICA

Scientists first found PBDEs in the food chain in 1981.[43] Since then, they have found PBDEs in increasing amounts nearly everywhere they have looked.

Scientists have found PBDEs in sediments, sewage sludge, crops, meat products, dairy products, chicken eggs, fish, mammals, human tissue, human breast milk, and human blood plasma.[44] The highly bioaccumulative tetra, penta, and hexa brominated molecules typically are found in humans at the highest levels.[45]

A sampling of places PBDEs have been found:

- Butter, pasta, potatoes, meat, and dairy products.[35]
- Pilot whales from the Atlantic Ocean.[36]
- Ringed seals from the Baltic Sea.[37]
- Beluga whales from Canada.[38]
- Lake Michigan salmon.[39]
- Freshwater fish from the Virginia River.[40]
- Harbor seals in the San Francisco Bay.[41]
- Human breast tissue of California residents.[42]
- Human breast milk in Sweden and North America.[42]

The distribution of PBDEs in the environment is similar to that seen with PCBs and other persistent organic pollutants.[46] However, while levels of PCBs and dioxins have been declining over the past 20 years, PBDE concentrations are rising, especially in North America.[47] Scientists have found increasing levels of PBDEs in sediments, fish, aquatic birds, marine mammals, wildlife, and humans.[48] The levels found in North America have been in the range of 10 to 100 times higher than in Europe, where government is taking action to reduce exposure.[49]

Contamination of Human Tissue and Breast Milk

Observed total levels of PBDEs in human tissues from across the world range from 1 to 500 parts per billion in fat.[50] In general, very few measurements have been taken in the United States. However, the limited data shows levels much higher in the U.S. than in Europe or Japan.

Highest Blood Levels Found in California Women

The highest observed blood plasma levels in the world come from data recently reported by Myrto Petreas at the California EPA.[51] Samples collected in the late 1990s from Laotian women in California show high blood levels of PBDEs. These samples contained tetra BDE at an average level of 50 parts per billion fat, with a range of 10 to over 500 ppb. Because the breast tissue of other non-immigrant women in California fell within a similar range, these levels are probably representative of the California population.

Highest Breast Tissue Levels in California Women

One of the highest PBDE burdens in human fatty tissue found to date came from the San Francisco Bay area. In the late 1990s, breast-fat tissue from women in the area contained 86 parts per billion PBDEs, which is 3 to 25 times higher than comparable samples from Europe.[52] The authors of the study report that "it appears that the levels of PBDEs in the general California population are the highest reported to date."

Highest Breast Milk Levels in U.S. Women

The finding of increasing levels of PBDEs in the breast milk of Swedish mothers was one of the last pieces of evidence prompting the EU to ban the commercial Penta BDE product.[53] Although very few breast milk samples in the United States have been tested for PBDEs, the highest levels found in the world to date come from pooled samples collected in Denver and Austin in the year 2000. In these samples, PBDEs showed up at 200 parts per billion in fat, 66 times higher than comparable measurements in women from Sweden, which were at about 3 ppb (see figure 10).[54] In New York in 1997, PBDEs were found in breast milk at 135 parts per billion fat, 34 times higher than in Sweden.[55] No breast milk samples from California have yet been tested. However, because PBDEs distribute fairly evenly with fat content, the high levels found in the breast tissue of California women suggest PBDE levels in breast milk will be also be high.

Global Contamination of Animal Life

Components of the Penta and Octa BDE products have been found in the fat of animals from such diverse locations as San Francisco Bay and the Northern Arctic.[56] Many PBDE measurements have been taken from marine animals in Europe, but some of the highest levels of PBDEs have been found in animals from the United States:

- The fatty tissue of Lake Michigan salmon and steelhead trout contained PBDE levels between 2,000 and 3,000 parts per billion (ppb).[57]
- In the San Francisco Bay Area in 1998, the blubber of harbor seals showed PBDE levels in the range of 3,000 to 8,000 ppb.[58]
- Even higher levels have been found in Virginia freshwater fish.[59] The fatty tissue of carp found in Virginia's Hyco River at a site near no known manufacturing facilities, contained PBDEs at levels up to 47,000 ppb.
- In comparison, the highest levels in the fat of European freshwater fish, found below a flame retardant manufacturing plant on the River Tees in the United Kingdom, ranged up to 10,000 ppb.[60]

Figure 6: Levels of PBDEs found in the San Francisco Bay area in the late 1990s.[61]

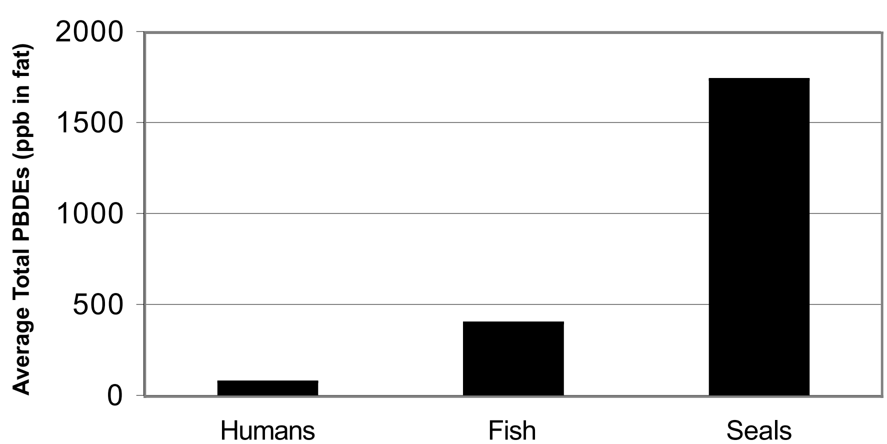
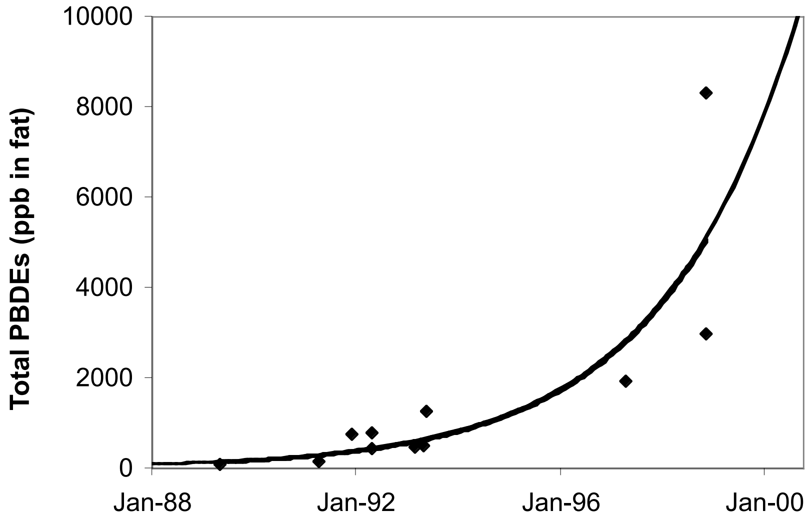


Figure 7: Rising levels of PBDEs in harbor seal blubber from San Francisco Bay.[68]



Widespread Contamination of the Environment

PBDEs have also been found widely in air, sediment, and sewage sludge.

Outdoor and Indoor Air

PBDEs are found at low levels in both outdoor and indoor air. The air above Chicago contains PBDEs at levels 5-10 times higher than rural locations in the Great Lakes area.[62] In this study, types of the chemicals tetra and penta BDE were present in the highest amounts, and deca BDE was also found at measurable levels.

PBDEs have also been found in indoor air in rooms with furniture, televisions, and other electronic equipment at levels similar to or slightly higher than urban outdoor air.[63] Isomers included tetra, penta, and hexa BDEs. [64]

River Sediment

Studies have found many types of toxic flame retardants in river sediments, including PBDEs, TBBPA, and HBCD. In Europe, deca BDE was found in river sediment downstream from textile factories at levels as high as 16,000 parts per billion dry weight.[65] No sediment levels from the U.S. have been measured.

Sewage Sludge

PBDEs are also widespread in sewage sludge. The highest levels detected in the world to date came from sewage sludge destined for land application from sources located in Texas, California, New York, Virginia, and Maryland. Chemical components of the commercial Penta BDE product were found at 1,100 to 2,290 parts per billion dry weight; the narrow range suggests consistent input regardless of region of origin. Concentrations of components of the Deca BDE product fell in a wider range, from 84.8 to 4,890 ppb.[66]

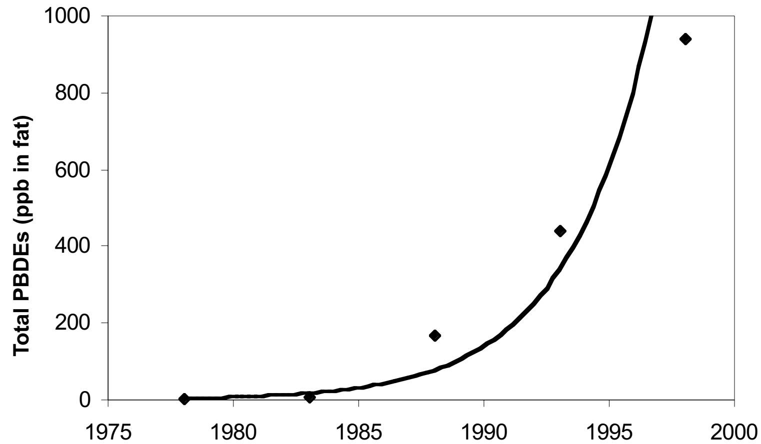
LEVELS OF CONTAMINATION ARE INCREASING EXPONENTIALLY

Bioaccumulative PBDEs are building up rapidly in animals and humans. This trend is most clearly shown in measurements of PBDEs in the blubber of harbor seals in the San Francisco Bay area, in trout from Lake Ontario, and in breast milk from Sweden, Canada, and the United States.

When scientists at the California EPA looked at PBDE levels in archived samples of seal blubber taken from the San Francisco Bay, they found that the total levels of PBDEs in blubber fat increased from 88 parts per billion in 1989 to between 3,000 and 8,300 parts per billion in 1998, a 34-fold increase.[67] At this rate, the level of PBDE contamination in seal blubber is doubling every 1.8 years (see Figure 7).

Scientists at Environment Canada found an increase in PBDE levels of over 200-fold in Lake

Figure 8: Rising Levels of PBDEs in trout from Lake Ontario.[70]



Ontario trout between 1978 and 1998, with levels doubling about every 4 years (Figure 8).[69]

Samples of human breast milk also show an exponential increase in PBDE levels. Sweden is one of very few countries with a breast milk monitoring program to keep track of persistent pollutants in the human body. Samples of milk from Swedish mothers in 1972 had PBDE levels of about 0.072 parts per billion in fat. In 1997, levels had increased about sixty-fold to 4 ppb in fat, doubling every 5 years.[71]

In Europe, concern about the potential health consequences of this trend led to sharply decreased usage of the commercial PBDE products. From 1997 to 1998, the EU cut down on PBDE use by two-thirds, or 180,000 pounds. This reduced usage may be responsible for the fact that PBDE levels in Swedish breast milk have been declining since 1998 (Figure 9).

In sharp contrast, data collected in Canada and the United States show dramatically higher levels of PBDE contamination that are neither leveling off nor declining (Figure 10, next page). Results from women in Vancouver show a ten-fold increase between 1992 and 2002, doubling every 2.5 years. Samples of breast milk taken in the United States, consistent with the breast tissue samples from California, show levels nearly ten times higher than anywhere else in the world.

With continued widespread production and use of PBDEs in North America, tissue concentrations of these chemicals in animals and humans are certain to climb even higher.

Figure 9: Recently declining PBDE levels in Swedish breast milk.[72]

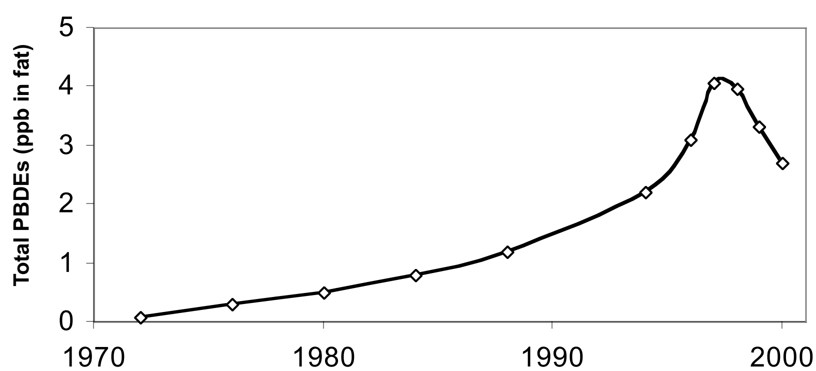
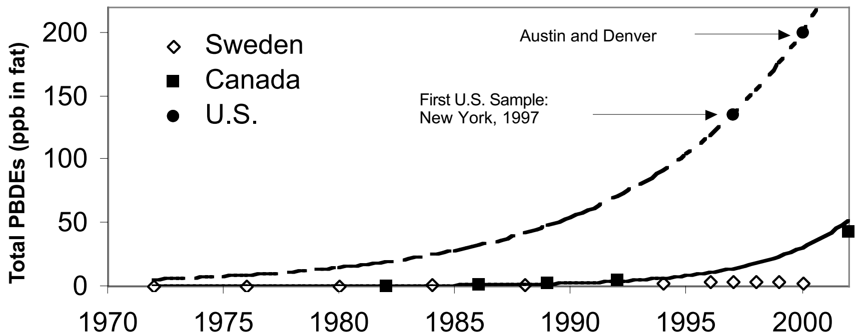


Figure 10. Increasing PBDE levels in human breast milk from Canada and the U.S.[73]



TOXIC FLAME RETARDANTS MAY ESCAPE INTO THE ENVIRONMENT DURING MANUFACTURE, PRODUCT USE, AND DISPOSAL

Toxic flame retardants make their way from manufacturing sites, consumer products, and disposal sites into the food chain. Because the chemicals are oily and very insoluble in water, they likely move around attached to particles in the soil, which can be suspended in water or air. The discovery of PBDEs in the Arctic food chain, far from any flame retardant manufacturing or use, shows that these particles travel to every corner of the world, just as PCBs and other persistent organic pollutants have.[74]

Release During Manufacturing and Industrial Use

The heavy contamination of the River Tees in the U.K. downstream of a Great Lakes Chemical Company factory that produces flame retardants, suggests that PBDEs can escape during their manufacture.[75] Possible routes include release to air from reaction chambers or release to water when containers are rinsed out.

Industrial users of toxic flame retardants–the factories that incorporate them into products–could also release PBDEs into the environment. At a foam manufacturing plant, for example, the Penta BDE product is added at around 10% by weight to foam slabs. PBDE release could occur during the handling of the flame retardant additive, by evaporation from the foam at heated temperatures, or when equipment is cleaned.[76] Waste foam or plastics containing toxic flame retardants may also end up in landfills or other disposal sites, or burned in an incinerator.[77]

Compared to the amount that ends up in consumer products and from there the environment, the PBDE releases during manufacture are probably very small.

Release During Product Use

North American industries used over 18 million pounds of commercial Penta BDE product in 1999.[78] Most of this product was added to polyurethane foams used in furniture, upholstered seating in automobiles, and other products.

The European Chemicals Bureau estimates that 3.9% of the Penta BDE product will escape from foam items over the course of a 10-year life, 2% will be lost at disposal and that 94% will end up incinerated or deposited in a landfill. The Bureau also estimates that 75% of emissions will end up in soil, 0.1% in the air, and 24.9% in surface water and sediment.[79]

These estimates suggest that 700,000 pounds of Penta BDE end up in the environment every year, based on the U.S. use of commercial Penta BDE: 525,000 pounds in soil, 700 pounds in air, and 174,000 pounds in surface water sediment. An additional 17.3 million pounds of Penta BDE product are landfilled or incinerated.

More highly brominated PBDEs like those found in the Deca and Octa BDE products may evaporate from the casings of computers and televisions, especially when the product heats up from use. For example, deca BDE has been found in the blood of people working at a computer disassembly plant, as well as in samples of dust in office buildings.[80]

Release After Product Disposal

The millions of pounds of PBDEs that end up in landfills are likely a major source of these chemicals entering the environment.

Components of the Penta BDE product likely escape into the environment from discarded furniture.[81] Water and sunlight break down foam into small pieces, which could release flame retardants into sediment or introduce them into the food chain through insects. Foam recycling may also hasten the release of flame retardants into the environment. In an experiment in which frogs were kept in an aquarium with crickets and a piece of furniture foam, the frogs accumulated PBDEs in their fatty tissues at extremely high levels (10,000 parts per billion). The authors believed that the crickets ingested small amounts of foam, and then were consumed by the frogs.

Plastic products containing commercial Octa and Deca BDE products may also release these chemicals through decomposition in landfills, especially when exposed to sunlight, which tends to break down plastics more quickly. Recently, a Norwegian study confirmed that PBDEs escape from discarded products and seep out of landfill sites into the environment.[82]

Compounds that Can Appear Safer Degrade into More Bioaccumulative and Toxic Forms

One of the largest debates between proponents phasing out all PBDEs and the chemical industry is over the threat posed by the Deca BDE product. Deca is the most widely manufactured product, but specific types of tetra and penta BDE (found primarily in the Penta BDE product) are the most widely distributed in the environment. However, several studies show that deca BDE, which is less bioaccumulative than other PBDEs, can degrade in simulated environmental conditions into the more bioaccumulative and toxic PBDEs, including types of tetra, penta, hexa, septa, octa, and nona BDE.

Chemical industry scientists argue that deca BDE has a molecular weight and size that renders it incapable of being absorbed by organisms.[83] However, components of the commercial Deca BDE product, including deca BDE, have recently been found in the blood of workers at an electronics recycling facility, as well as in European peregrine falcons, so direct accumulation of deca BDE apparently is possible.[84]

UV light and sunlight can also change deca BDE into other types of PBDEs that accumulate in organisms more readily.[85] This process occurs quickly when the deca BDE is dissolved in an organic solvent, and more slowly when the deca BDE is on silica gel, sand, or sediment. In these experiments, tetra BDE appeared in sediment after 244 hours of sunlight exposure.

Deca and highly brominated PBDEs are often found on particulate matter in outdoor air and in indoor house and office dusts.[86] Deca-containing dusts and particles in outdoor air would be exposed to UV light and sunlight, providing ample opportunity to transform into more bioaccumulative forms. One study also suggests that after ingesting deca BDE, fish may metabolize the chemical into more bioaccumulative forms.[87] In addition, chemical reactions in sediments and soil may change higher-order PBDEs into lower-order ones and combine them with chlorinated organic contaminants to form mixed dioxins and furans.[88]

In 1999, North American industry used 54 million pounds of commercial Deca BDE, the primary component of which is the chemical deca BDE. Decomposition of deca BDE into other types of PBDEs could be a significant source of toxic flame retardant contamination observed in the environment and human tissues. At this point, it is uncertain how much each commercial product contributes to observed contamination.

Toxic Incineration Byproducts

Dioxins and furans are produced when products containing PBDEs are burned in waste incinerators, building fires, landfill fires, or other uncontrolled fires. These substances are among the most potent toxic chemicals known.

Dioxins and furans are so toxic that Germany has strictly limited the use of any substance than can be readily transformed into dioxins. In 1989, German plastic manufacturers pledged to stop using PBDEs because of these concerns.[89]

HUMAN CONTAMINATION MAY BE IMPOSSIBLE TO AVOID

Based on what we know about other persistent pollutants, most PBDE exposure probably comes from food. Mothers accumulate PBDEs in their bodies through this exposure. As a result, from the very beginning of life, fetuses are exposed to the toxic chemicals in their mother's bodies. Ultimately, every exposure to a chemical that accumulates in human tissue is important from a public health perspective.

Because PBDEs are present in products that surround people in their everyday lives, there are many possible routes of exposure. In addition to diet, PBDEs could be entering people through inhalation of contaminated dust in the home or outdoor air, or absorption through the skin from furniture or products.

Our First Exposure: In the Womb

Developing fetuses are exposed to PBDEs by transfer across the placenta from the mother's blood. This may be the most significant route of exposure in terms of public health, because of the potential for the fetus to be exposed during vulnerable periods of brain development.

During pregnancy, a mother's blood transports nutrients to her embryo and removes waste products through transfer across the placenta. Bioaccumulative compounds like PBDEs can move freely across the placenta into the infant's bloodstream.[90]

BREAST MILK AND INFANT HEALTH

Breast feeding is the best source of nourishment for infants and is critical for their health and well-being, despite the presence of PBDEs and other toxic chemicals in human breast milk. Breast milk contains nutrients that are critical for the developing immune system and overall growth. Infant formulas have their own contamination problems and lack these critical nutrients.[93] Breast feeding is also a critical step in forming deep emotional bonds between mother and child which are essential for health.

Several studies have shown that breast feeding can lessen the harmful effect of in-utero exposure to toxins such as PCBs, even if breast milk itself is contaminated with PCBs.[94]

Several studies indicate that PBDEs build up in a developing fetus to the same levels found in the mother. Analysis of samples from 11 Finnish women showed similar PBDE concentrations in breast milk and the placenta.[91] A more recent study from Indiana showed that the levels of PBDEs in the mother and the fetus are practically equal, with less than a 2% difference.[92] The PBDEs act like a drop of dye in a glass of water — they spread out evenly throughout the whole container.

Contamination in Mother's Milk

Toxic chemicals that accumulate in the mother's body can be transferred to newborn infants through breast milk. Fat storage cells in the breast are the primary source of fats for mother's milk. As a mother's body calls on its reserves of fat, the pollutants in these tissues get into the mother's milk, and are subsequently consumed by her baby.

Continued Exposure: A Contaminated Food Supply

Food is likely a major source of PBDE exposure. PBDEs have been found in a wide range of foods, especially those containing animal fat like meat and dairy products. As with other bioaccumulative toxins and metals, studies have shown that PBDE concentrations in people tend to rise as they consume more fish.[95] Food processing may also introduce contamination. PBDEs have been found in processed foods like peanut butter and pasta.[96]

Breathing Contaminated Air

Many types of PBDEs have been found in household air in rooms with electronics, in electronics disassembly plants, and attached to dust particles in European parliament buildings.[97] People may inhale significant amounts of PBDEs by breathing household and workplace dust; children may be exposed to higher levels by accidentally eating dust as well.

Workplace exposure has been shown to lead to elevated blood levels of PBDEs, presumably through dust inhalation.[98] Computer disassembly workers in Sweden had PBDEs in their blood at 26 parts per billion in fat, while office workers showed levels 5 times lower. A control group of cleaning workers with no occupational exposure showed the lowest levels at 3.3-4.1 parts per billion in fat.

WHY ARE PBDE LEVELS IN CALIFORNIA AND THE U.S. SO HIGH?

Levels of PBDEs found in sewage sludge, food, animals, and humans in North America are higher than those found in Europe. This likely occurs for two reasons.

First, North American industries use higher volumes of PBDEs than much of the rest of the world. North American industries use almost all of the world's commercial Penta BDE, and nearly half of the world's commercial Deca BDE. In contrast, European manufacturers no longer make Penta BDE at all and the use of all PBDEs in Europe has declined by at least two thirds since 1998.

Second, California has the most stringent fire retardancy standards for furniture of all U.S. states.[99] While these standards do not mandate the use of a specific flame retardant, Penta BDE may be widely used to meet these standards at higher levels than may be used in consumer products destined for other markets.[100] So in addition to higher overall use, products destined for California may have higher per-product amounts of PBDEs.

Places that Process PBDEs in California

Foam manufacturers with factories, offices, and distribution warehouses in California are listed in Table 1. PBDE levels may be elevated in the vicinity of some of these sites.[101]

Additionally, there are 144 factories in the US that process more than 1,000 pounds of Deca BDE product; 5 of these are based in California.[103] No other information about facilities that use PBDEs has been reported, although factories that manufacture plastics for use in computers, televisions, and other electronic equipment may possess significant volumes of toxic flame retardants.

Table 1: Companies in California that manufacture and distribute foam products, which likely contain Penta BDE.[99]

Company	Locations in California
E. R. Carpenter, Inc.	Riverside, Lathrop
Crain Industries	San Leandro, Compton
Foamex Products, L.P.	San Bernadino, Orange, La Mirada, Ontario, Hayward, West Sacramento
Future Foam, Inc.	Fullerton
Hickory Springs of California, Inc.	Commerce, Sacramento, Hayward

3. Toxic Flame Retardants Pose A Significant Threat to Children's Health

Evidence to date indicates that the health effects of PBDE exposure are likely very similar to those of PCBs (polychlorinated biphenyls), including interference with thyroid hormone function, disruption of brain development in fetuses and infant children, and possibly cancer.[104] Recent research shows that PBDE exposure can interrupt brain development in mice, permanently impairing learning and movement. So far, no lower limit of exposure that does not cause these effects has been identified. Levels of PBDEs building up in some mothers and fetuses are approaching the levels shown to impair learning and behavior in these experiments, and their effects may be additive with those of PCBs.

TOXIC FLAME RETARDANTS CAN IMPAIR DEVELOPMENT

Chemical Exposure Can Harm Fetal and Infant Health

Exposure to toxic flame retardants is likely to be most harmful to fetuses and infant children. Children are likely to receive higher doses than adults because children drink more water, breathe more air, and eat more food relative to their body weight.

Additionally, developing children undergo rapid and complex changes within a relatively short period of time. Many important developmental processes happen in the womb and during the first few years of life, from organ development to physical growth. Toxic flame retardants like the PBDEs can readily cross sensitive membranes, including the placenta that protects and nourishes the developing fetus, allowing exposure to sensitive organs at the most sensitive times.

Human development happens in a series of stages, each of which is a critical window during which exposure to a developmental toxin can have serious effects. For example, the human brain grows most rapidly from the third trimester of fetal development through the second year of life.[105] This period is known as the brain growth spurt, when specialized nervous system tissue develops under the influence of the thyroid hormone system.[106] Exposure to chemicals that disrupt thyroid hormone balance during this period can permanently disrupt brain development. Exposure to toxins like PBDEs can affect the developing nervous system in other ways as well, including changes in cellular signaling mechanisms–one of the foundations of the way human bodies function.

Exposure to toxic chemicals can interfere with development and create functional or structural problems later in life. Although neurological effects can be very significant in their impact on a child's life, they can be hard to detect. The window of time during which a key developmental process is most vulnerable may only last for a few days. Exposure before or after this period may not produce the same results, and harmful effects are often not readily apparent until much later in a child's life.

Some Toxic Flame Retardants Can Disrupt the Thyroid Hormone System

Thyroid hormone function is very important in brain development. Disruptions in thyroid levels as early as week eight in the womb through the second year of life can disrupt normal brain development and impair the intelligence and psychomotor skills of children.[107]

All of the PBDE products disrupt thyroid hormone balance. While the commercial Deca product is much less potent than the commercial Penta or Octa BDE products, Deca can degrade under simulated environmental conditions to form components of commercial Octa and Penta BDE (See Chapter 2).

There are at least two ways in which PBDEs can affect thyroid hormone function. First, like dioxins, PBDEs can activate liver enzymes that lower thyroid hormone levels.[108] Second, other liver detoxification enzymes can change some PBDEs into hydroxylated PBDEs, metabolites that closely resemble the structure of the thyroid hormones.[109] These metabolites mimic thyroid hormones and bind to the thyroid hormone transport protein, preventing the proper function of the system.[110]

As a result of these processes, PBDE exposure produces depressed thyroid hormone levels and physical changes in the thyroid gland.[111] Depressed thyroid hormone levels have been shown to occur in mice when exposed to commercial penta BDE at single doses as low as 0.8 milligrams per kilogram of body weight.[112] These effects appear to be additive with the effects of PCBs and dioxins on thyroid hormone levels.[113]

Some scientists think that the ability of PBDEs to mimic thyroid hormone and attach to thyroid binding protein also may help PBDEs reach the fetal brain cavity. Thyroid hormone binding protein transports thyroid hormone from mother to fetus and across a membrane that protects the developing brain. By binding to transport proteins, PBDEs not only reduce the amount of thyroid hormone reaching the brain, but may also find their way to the fetal brain and concentrate there.[114]

Damage to the Developing Nervous System: Impaired Intelligence and Motor Skills

Several studies provide evidence that PBDE exposure damages neural development. Mice exposed to PBDEs as newborns have learning and movement problems that worsen as the animals grow older, an effect similar to that seen with PCBs.[115]

Lab studies on mice show effects occurring after a single oral dose of 0.8 milligrams of PBDEs per kilogram of body weight on the 10th day of life, during the period of mouse brain growth analogous to the human brain growth spurt. These effects have been demonstrated with both the commercial Penta BDE product and a specific penta BDE molecule often found in humans. Evidence also indicates that PBDEs may affect nerve impulse transmission and disrupt communication systems inside cells that could prevent the cell from functioning properly.[116]

To date, the effects of PBDEs on neural development are known only for mice. Human brains are far more sophisticated organs than those of mice. The results seen in mice are relatively crude compared to the subtle effects very small PBDE exposures could cause in humans.

Human Exposure Levels in the U.S. Are Approaching Those Seen to Cause Developmental Damage in Mice

Levels of PBDEs in Americans are rising rapidly and approaching the levels shown to impair learning and behavior in mice. No lower limit of exposure has yet been identified that does not cause this effect. Therefore, toxic flame retardant exposure could already be having a significant impact on human health in some parts of the population.

- A mouse given a dose of 0.8 milligrams of PBDEs per kilogram of weight on the tenth day of life will show developmental damage which grows more severe as time passes, including abnormal behavior and impaired learning skills.[117]

- Assuming that the mouse absorbs 100% of the dose, it will have PBDEs in its body at 800 parts per billion.

- Assuming that the mouse is 15% fat and all the PBDEs end up in fat, fat levels of PBDEs will be 5,300 parts per billion.[118] We can compare observed human levels of PBDEs to this value to estimate the potential for harm.

- Blood levels of PBDEs found in California women in the late 1990s ranged from 10 to 500 parts per billion in fat, averaging 50 ppb.[119] Breast fat levels of PBDEs found in California women in the late 1990s ranged from 17 to 462 parts per billion, averaging 86 ppb.[120]

- Assuming that these levels are doubling every 2.5 years, like they are in Canadian human breast milk, in 2003 levels in California may be in the range of 50 to 2,600 ppb, with an average of approximately 450 ppb. This correlates with what we would expect given the pooled breast milk sample from Denver and Austin in 2000, which showed PBDE levels of 200 ppb.[121]

- PBDEs distribute evenly in the human body, like a drop of ink in a glass of water. As a result, a fetus will have the same level of PBDE contamination in its tissues as its mother.[122]

- If PBDE levels continue to double every 2.5 years, within ten years the average person may have a similar level of PBDEs in their body as that already shown to cause developmental damage in mice. Highly exposed people may already have PBDE levels within two-fold of this threshold.

- Although no scientific studies have yet documented harm in the human population, we cannot rule out the possibility that it is happening even now:

  - No "safe" PBDE exposure levels have been identified to date. Further study could very well identify health effects at lower levels of exposure.

  - The effects of PBDEs may be additive with those of PCBs. Background levels of PCBs have been shown to affect thyroid hormone levels in people as recently as the early 1990s. Both chemicals can be found in people today.

  - The human brain is a much more sophisticated organ than the brain of a mouse, and it could be more sensitive to PBDE exposure. In addition, certain people might be inherently vulnerable to exposure.

  - PBDE levels vary by about 50 fold in the population, which means that a significant fraction of the population will have very high tissue levels of PBDEs.

In other words, children at the upper range of exposure today probably have PBDEs in their bodies near the levels known to cause brain damage in lab animals. Because we haven't found any "safe" level in lab experiments, and because PBDEs and PCBs could be additive in their effects, these children may already be suffering damage. Levels are increasing rapidly, and within ten years children with average exposure may have PBDEs in their bodies above the levels known to cause brain damage in lab experiments..

EMERGING HEALTH CONCERNS

Cancer-Causing Potential

The U.S. National Toxicology Program classifies PBDEs as "reasonably anticipated to be human carcinogens."[123]

The bioaccumulative tetra, penta, and hexa BDE molecules have not been tested for carcinogenicity, but based on their similarities to PCBs, there is reason to suspect that they could cause cancer. Tetra and penta BDEs, if they prove to be carcinogenic like the PCBs, may present a greater risk of cancer than the Deca BDE product because they are more likely to enter the body and stay there longer. One study suggests a positive association between the risk of Non-Hodgkin's lymphoma and tissue levels of tetra BDE in humans.[124]

Deca BDE is the only product that has been tested for carcinogenicity, and only a few studies have been done. Exposure to the Deca product produced some evidence of carcinogenicity in male and female rats and equivocal evidence in male mice.

Deca exposure also created tumors in the liver and pancreas at relatively high doses (2.5% - 5% of the diet).[125] However, uptake of Deca BDE is low compared to other PBDEs; only roughly 1/1000 of the given dose was absorbed by the rodents. This result indicates that Deca BDE could potentially be a carcinogen even at very low levels in tissue.

Immune System Impairment

Limited studies to date suggest that the Penta BDE product can impair the immune response in exposed rodents.[126] These effects could be due to the contamination of commercial Penta BDE with dioxins and furans. Similar effects have been seen with PCBs.

Transformation Products in the Body: Toxicity Unknown

When considering the health effects of PBDE exposure, the products of transformation in the body and the products of environmental degradation must also be considered. For example, the liver modifies PBDEs to hydroxy PBDEs, which may have much stronger effects on thyroid function and higher estrogen hormone activity than the original commercial products. Hydroxy PBDEs may also be present in food. Additionally, environmental exposure can modify PBDEs to methoxy PBDEs, which have been found in some environmental samples at levels higher than the major PBDEs. Their toxicity is unknown.[127]

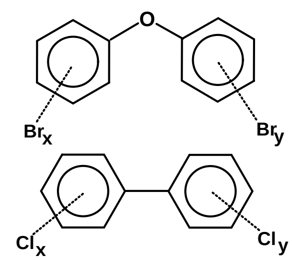
Burning Toxic Flame Retardants Creates Dioxins and Furans

Dioxins and furans are among the most toxic and dangerous compounds humans have ever created. All types of PBDEs form significant amounts of dioxins and furans when they burn. In fact, mere exposure of Penta BDE to light has produced furans. Almost no studies have looked for combustion byproducts of PBDEs in the environment.

Dioxins and furans can cause health effects at levels much lower than PBDEs. The fact that PBDEs can form these toxic compounds has been a major motivation for companies looking for alternative flame retardants to make their products safer.

4. Lessons Learned for Protecting Public Health: The Legacy of PCBs

The legacy of damage to the environment and human health caused by polychlorinated biphenyls (PCBs) provides a compelling reason for taking action to prevent damage to public health from toxic flame retardants. PCBs were introduced, mass-produced, and globally distributed before their effects were known. It took decades for scientists to notice widespread human exposure. Tragically, the problem was allowed to reach the point where harm to human health was inevitable. Victims of accidental poisonings and regular people with background levels of exposure provide ample documentation of a preventable public health disaster. Our experience with PBDEs could very well be headed in the same direction.



Polybrominated Diphenyl Ether (PBDEs)

Polychlorinated Biphenyl (PCBs)

PARALLELS BETWEEN TOXIC FLAME RETARDANTS AND PCBS

PCBs are a family of chemicals that are similar in structure to PBDEs. Both chemicals are persistent, bioaccumulative, and toxic: they do not degrade easily in the environment; they can be transported across long distances in the air; they accumulate in the fatty tissues of living organisms; and they have a variety of toxic effects (see Table 2).

Both PCBs and PBDEs are not readily biodegradable, and therefore persist in the environment for a long time.[128] Penta BDE molecules deposited in sediments a few decades ago are still present in measurable levels.[129] PCB contamination of the Hudson River by discharges from General Electric manufacturing facilities remains a problem more than thirty years after the discharges occurred. Both PCBs and PBDEs have traveled to the Arctic environment through the atmosphere. One study even found that tetra BDE levels in arctic seals were higher than levels of the PCB generally found at the highest levels in organisms.[130]

Both PCBs and PBDEs accumulate in the fatty tissues of living organisms. The most bioaccumulative PBDEs (types of tetra BDE, penta BDE and hexa BDE) are taken up by fish as efficiently or more efficiently than PCBs.[131]

Both PCBs and PBDEs have toxic properties. In animal experiments so far, these two classes of chemicals have been shown to cause chronic damage to organs, changes in thyroid hormone levels, affect signaling in nerve cells, and cause spatial learning impairment and irregular movement in mice exposed early in life.

Table 2: Comparing PCBs and PBDEs

Attribute	PCBs	PBDEs
Persists in the environment	+	+
Transported long distances in the air	+	+
Bioaccumulates	+	+
Disrupts thyroid hormone levels	+	+
Causes problems with neurological development in animal experiments	+	+
Exposure causes neurological damage in humans	+	NOT TESTED
Recognized probable carcinogen[126]	+	NOT TESTED
Used in products found in the home, like televisions and furniture	-	+
Production banned in the U.S.	+	-

THE HISTORY AND LEGACY OF PCBS[133]

A New Wonder Chemical

The Swann Chemical plant in Anniston, Alabama began producing a group of new chemical compounds in 1927. The chemicals were remarkably stable; low heat would not degrade them; they would not burn; and they did not conduct electricity. The manufacturers began to market these new chemicals as ideal insulators for electric products. These new chemicals were polychlorinated biphenyls (PCBs) (see Table 3).

Monsanto Corporation took over the operation in Anniston about a decade later. Clients including General Electric and Westinghouse developed a range of products that relied heavily on PCBs, including electrical transformers, fluorescent lights, and other products that needed insulation or protection from bursting into flames.

Illness in Workers

Beginning in the 1930s, some workers at plants producing and using PCBs began to suffer from a severe skin condition known as chloracne. Chloracne and associated systemic damage caused by exposure to PCBs and related compounds killed three workers at Halowax Corporation in New York; autopsies revealed extensive liver damage. In 1937 at the request of Halowax, a Harvard University Researcher investigated and discovered that rats exposed to these chemicals also suffered severe liver damage.[134]

PCBs in the Food Chain

The first warning about widespread PCB contamination of the environment came in 1966 from Soren Jensen, a Swedish scientist. While looking for DDT, he found PCBs in fish and fish-eating birds. Word began to spread in the inner circles of Monsanto, but it took Jensen two years of work to conclusively identify the chemical and expand the number of species tested, and his work was not published until 1969.

PCBs leapt onto the public stage in 1968 after a tragic accidental poisoning incident in which 1,800 Japanese people consumed rice oil heavily contaminated with PCBs. Exposed people became very sick, with symptoms ranging from nausea to chloracne;

Table 3: Timeline — PCBs and PBDEs[143]

Year	PCBs	Year	PBDEs
1927	Commercial production began	1970s	Commercial production began
1930s	First evidence of health impacts: Chemical industry workers get chloracne, study links PCB exposure and liver disease in rodents.	1980	First evidence of health impacts: Researchers note that flame retardants and their byproducts may have "considerable toxicological problems," and document liver changes after PBDE exposure.
1966	First evidence of bioaccumulation — fish (Sweden)	1981	First evidence of bioaccumulation - fish (Sweden)
1969	Found in the food chain in the U.S.	1990s	Found in the food chain in the U.S.
1968	Accidental poisoning causes severe health problems in 1,800 people in Japan, first evidence of toxicity to fetuses.	1994	Ability of Penta BDE to mimic hormones first discovered.
1970	Peak U.S. Production of PCBs, 85 million pounds per year.	?	Peak U.S. Production of PBDEs — currently unknown.
1970s	Scientists studying reproductive problems in wildlife from DDTidentify PCBs as an additional culprit.	1997	Exponential increase found in milk from Swedish women, Europe begins to reduce use.
1976	The U.S. Congress bans PCBs with the Toxic Substances Control Act. Production stops one year later.	2003	European Union bans PBDEs in electronics.
1990	Scientists discover health problems in children exposed to "background levels" of PCBs.	1998-2002	PBDEs shown to irreparably alter brain development in mice.
1990s	Scientists show that children exposed to PCBs are more likely to have learning disabilities.	2002-	

exposed women suffered from miscarriages or gave birth to babies with lower birth weights at higher rates than normal. The incident graphically demonstrated that PCBs harm human health.

Widespread contamination of the food chain in the U.S. was demonstrated in 1969 by Dr. Robert Riseborough of the University of California at Berkeley, who happened upon it in his work with peregrine falcons. PCBs were getting into rivers and streams and moving up the food chain, exposing humans through the fish and other food they were eating.

Representative William Fitz-Ryan from New York made the first congressional proposal for a total ban on PCBs in 1970. However, the chemical industry muddled the issue by commissioning fraudulent studies and using inherent uncertainties in the science to argue that without conclusive proof, action couldn't be taken. Internal chemical industry documents revealed decades later that Monsanto was aware of PCB pollution and significant health dangers of PCBs well before the public became aware.[135] Even worse, the industry knowingly put out false or grossly inadequate scientific studies. Dr. Paul Wright, a toxicologist at Industrial Bio-Testing Co., who had worked at Monsanto both before and after his tenure at Bio-Testing, was eventually convicted of multiple counts of fraud in federal court after directing studies commissioned by Monsanto that falsely concluded PCBs were not carcinogenic.[136] However, to this day, Monsanto representatives still deny that their products caused any harm.

Production Banned

Finally, in response to growing evidence of harm to human health, the U.S. Congress banned the manufacture of PCBs with the passage of the Toxic Substances Control Act in 1976. This is the only chemical that Congress has ever taken direct action to ban. Manufacture of PCBs ceased in 1977, more than forty years after exposure was first linked to harm to human health. By then, millions of tons of PCBs had already been manufactured and distributed in electrical transformers and other products across the country.

At the time of the ban, scientists did not yet understand the full scope of problems with persistent and bioaccumulative chemicals. Only one study had linked PCB exposure to thyroid hormone changes, and the critical role of this hormone in human development was not completely understood. Scientists were just discovering that chemicals like DDT and dioxin could harm health. Understanding came slowly; the U.S. Department of Health and Human Services did not recognize PCBs as a probable human carcinogen until 1991.

Scientists Discover that Small Doses Disrupt Infant Development

Another accidental PCB poisoning occurred in Taiwan in 1978, when 1,800 people consumed PCB contaminated rice oil. The rice oil was being heated in a machine that used PCBs as a heat-exchange fluid. When PCBs leaked out through pinholes in the heat-exchange coils, they contaminated the rice oil, which was then distributed by merchants across wide areas. As the children of poisoning victims grew, they showed signs of irreparable damage associated with developmental toxins: immune suppression, altered sexual development, delayed brain development and increased behavioral abnormalities like hyperactivity and behavioral problems at school.[137]

Further studies found observable health effects at lower levels of exposure with more sensitive tests and different timing of exposure. Studies in North Carolina, Michigan, upstate New York, and the Netherlands all yielded similar results: as the level of PCB exposure before birth rose, the mental and physical abilities of infants after birth declined. Even at very low levels, prenatal PCB exposure contributed to hyperactivity and attention problems later in childhood.[138]

These discoveries lead scientists to conclude that persistent, bioaccumulative chemicals like PCBs can have their most significant effects in fetuses and developing infants.

Thirty Years Later, PCB Exposure is Still A Problem

Scientists have continued to find health effects in people with no occupational or accidental PCB exposure as recently as 1994. These studies found that the levels of PCBs in the general population were still high enough to affect thyroid hormone balance in mothers and their nursing infants.[139] Reduced thyroid levels in the first few weeks of life for pre-term and low birth weight babies are associated with increased risk of neurological disorders, including the need for special education by age nine.[140]

PCB contamination remains a well-known problem in places like the Hudson River in New York, where General Electric dumped PCBs from their factories for decades, and Anniston, Alabama, where the first PCB factory dumped its waste. In 2001, the U.S. Environmental Protection Agency ordered G.E. to pay $500 million to dredge hundreds of thousands of pounds of PCBs from the upper Hudson river.

Today in 2003, PCBs are still found in significant amounts in many people.[141] Although levels have declined since the 1976 ban, widespread contamination persists. The PCB problem may be getting worse, thanks to PBDEs. In contrast to declining PCB levels, PBDE levels are rising rapidly. Because PBDEs and PCBs both affect thyroid hormone levels, these two toxic chemicals may be working together to harm human health, extending the legacy of PCB contamination well into the future.[142]

Lessons Learned from the PCB experience:

1) **Failure to act on early warnings leads to irreversible harm.**

   Scientists had the first hints that PCBs were harmful to human health in the 1930s. However, use of this persistent and bioaccumulative chemical continued for decades, resulting in irreversible contamination of humans and the environment. This contamination reached the point of causing harm to humans and wildlife by the time action was debated.

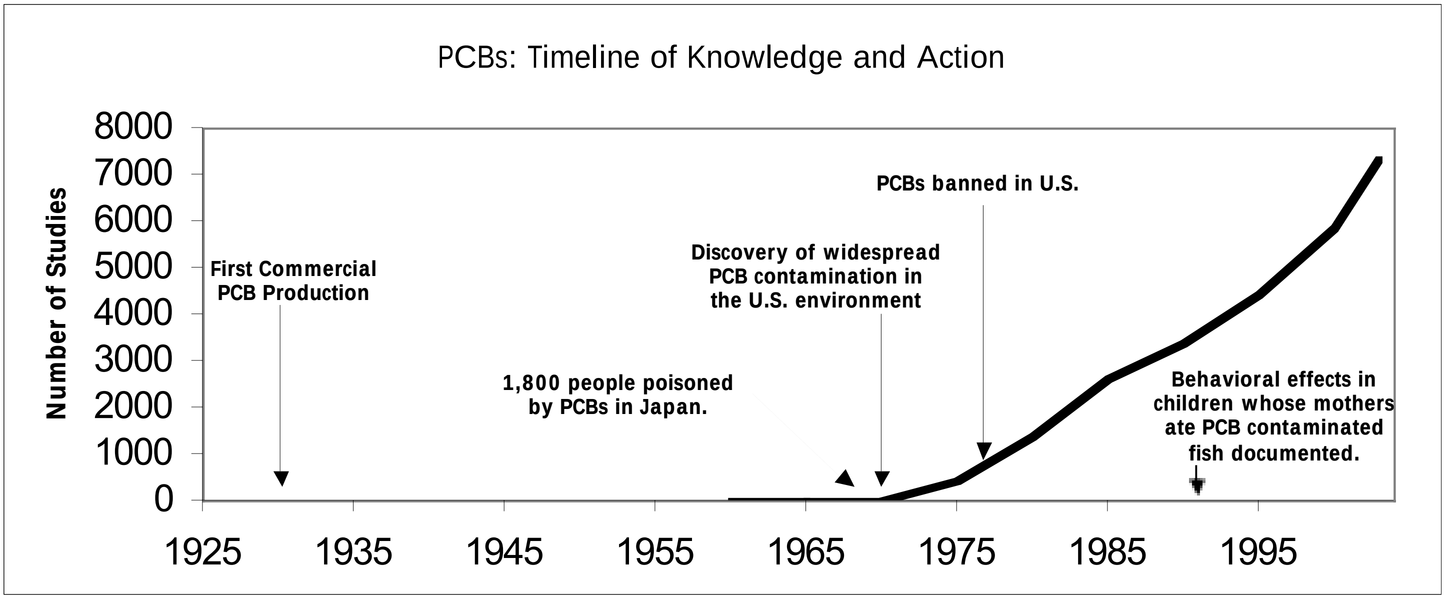
2) **Further study only identified new types of health impacts caused by lower levels of exposure.**

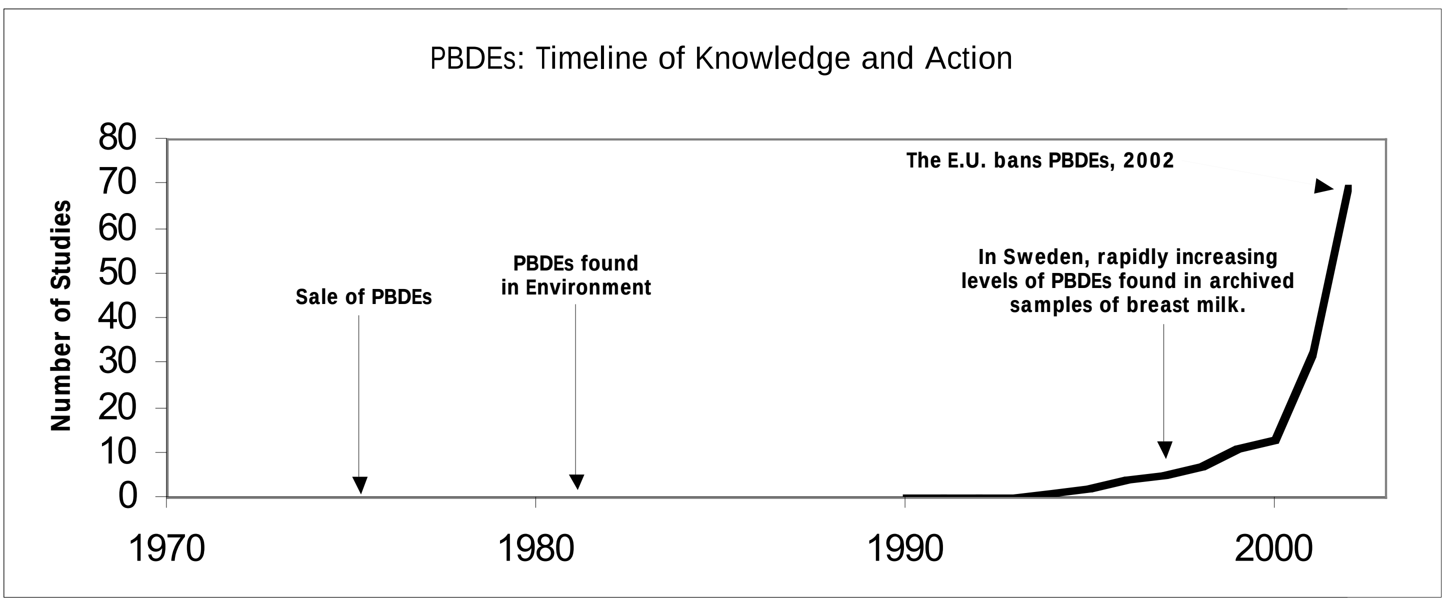
   As scientists studied PCBs further, the evidence that they could harm health only became stronger. Current knowledge of PCBs required more than 7,000 scientific studies, many decades of work by thousands of people, and uncounted millions of dollars spent. Despite all of this work, the chemical industry continues to insist that PCBs have never been linked to some health effects.

3) **Phasing out the use of chemicals based on clear evidence of a health threat can reduce exposure and prevent further damage.**

   Phasing out the use of PCBs resulted in a decline in contamination found in people, preventing the damage from becoming any worse.

Figure 11 : The progress of scientific knowledge about PCBs and PBDEs and key events in their histories.[144]





The Threat to Public Health Will Grow with Increasing Contamination

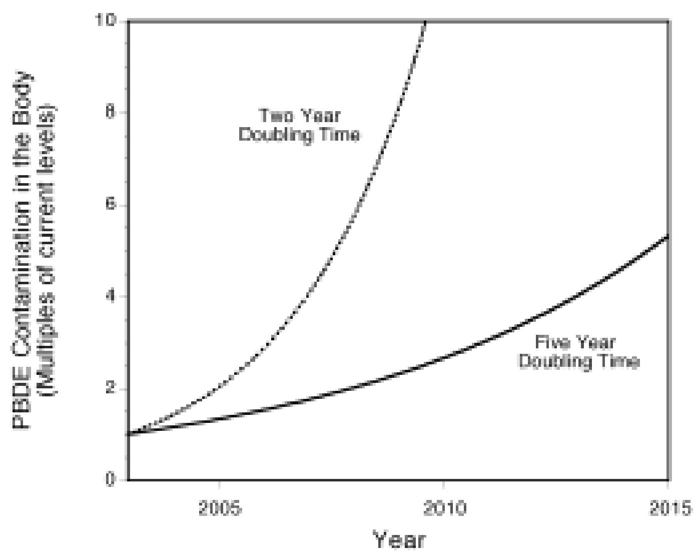
Harm to public health will become even more certain if levels of PBDEs continue to increase exponentially. Samples taken from organisms ranging from trout in Lake Ontario to seals in San Francisco Bay to human breast milk in Canada show that the levels of PBDEs are doubling every 2 to 5 years (Figure 12).

Levels in fish and marine mammals are much higher than in humans at this point. But as more PBDEs in the environment work their way into the food chain, human exposures will increase.

As PBDE levels rise, it becomes more likely that exposure will result in visible damage to children, including altered behavior patterns, learning abilities, memory capacities, intelligence, and other potential effects (Figure 11). At a doubling rate of 5 years, PBDE exposures will increase100-fold in 25 years. At a doubling rate of every two years, as observed in harbor seals in San Francisco Bay, exposures will increase more than 1000-fold in 25 years.

Action can be taken to halt this troubling trend and begin reducing human exposure to PBDEs. In the late 1990s, Sweden sharply cut back on the use and import of the PBDE products after finding evidence of their accumulation in breast milk. From 1997 to 1998, the European Union as a whole cut down on PBDE use by two thirds, or 180,000 pounds. Likely a direct result of reduced use, PBDE levels in Swedish breast milk have been declining since 1998 (See Figure 8).

Figure 12: Rise in PBDE levels with a doubling time between two and five years.



5. Products Can Be Made Fire-Safe in Less Toxic Ways

There are several ways to make products that are both flame-retardant and non-toxic. First, products can be designed from the start to reduce the chances of a catastrophic fire and eliminate components that pose risks to public health. Second, inherently flame-retardant materials that do not require flame-retardant additives can replace highly flammable plastics. Third, less toxic flame-retardant additives can replace toxic brominated flame retardants (BFRs) where products cannot be made less flammable. A few currently available replacement flame retardants and materials are summarized in Appendix 1.

Many companies, especially those in the electronics industry, have already begun the use of alternative designs, materials, and flame-retardant additives, or are hard at work exploring the possibility of phasing out BFRs. Responsible companies are using flame retardants that pose fewer environmental health risks while working to minimize costs, meet flammability standards, and maintain appropriate physical properties of plastics and other materials.

DESIGN FOR FIRE PREVENTION

Products can be designed to be inherently less vulnerable to catching fire. For example, flammable components can be isolated from heat sources or protected with non-flammable coverings. Flammable materials can be replaced with non-flammable ones. Changes in design can happen at any level. A product can be replaced by a different product that accomplishes the same task, or new type of solution altogether. The creativity of the engineer is the only limit to this approach.

For example, in furniture manufacturing, a polyurethane foam mattress could be replaced by one made of coiled springs. In electronics, products can be built with materials of increased thickness and thus less flammability, flammable parts can be physically separated from heat generating parts, or protective metal shields can be placed around plastic in contact with current carrying components to prevent the spread of flames.

INHERENTLY NON-FLAMMABLE MATERIALS[145]

Some materials are inherently able to resist flame. From natural materials like wool and leather to some types of synthetic plastics, manufacturers can replace flammable components with naturally flame resistant materials to eliminate the need for toxic additives.

Wool and leather are naturally flame-resistant materials that can be produced without many of the harmful environmental effects of plastic, which comes from petroleum products. Wool fibers and leather are difficult to ignite and burn slowly. A couch covered in rayon is much more likely to catch fire than one covered with wool or leather.

Some plastics are inherently flame-retardant and do not require chemical additives like brominated flame retardants. For example, plastics that contain sulfur, such as polyphenylene sulphide, have inherent flame resistance.[146] Also, experimental programs are underway to develop new low-flammability materials which produce little smoke and only non-corrosive gas, such as preceramic polymers. These materials hold promise to make toxic flame retardants less necessary, and also to make smoke inhalation less deadly during accidental fires.[147]

Some of these alternative materials have already been used in place of materials containing brominated flame retardants. For example, the Toshiba electronics company used polyphenylene sulfide to replace BFR-containing casings for electronic parts in its products.[148] This replacement material is available at the same cost as the BFR-containing material.[149]

Some furniture manufacturers are considering textiles made from alternative materials like aramide blends or carbonized fibers which are inherently flame retardant.

LESS TOXIC FLAME-RETARDANT CHEMICALS

Brominated flame retardants can also be replaced by alternative chemicals which may have less harmful properties. A wide spectrum of possibilities exist, including the 200+ different commercial flame-retardant chemicals in use today, as well as compounds currently under development.

The best alternative flame retardants have the following properties:

- no acute or chronic effects on human health or development, or the environment
- minimum release during production, from product use, and during disposal (either in landfills or incinerators)
- do not interfere with re-usability or recyclability of products and are biodegradable into non-hazardous byproducts.
- able to suppress the formation of smoke and hazardous fumes during fires
- do not adversely affect product function or longevity

Industries seek flame retardants that have reasonable costs and maintain the appropriate physical properties of the materials they use. Consumers desire products without ingredients that contaminate people and the environment and harm human health. These two considerations can and must be met simultaneously.

Generally, the best types of alternative flame retardants are halogen-free. Halogenated chemicals, those that contain the chemical elements fluorine, chlorine, bromine, or iodine, are generally more likely to bioaccumulate and have toxic effects. For example, some companies have been looking to other brominated flame retardants like TBBPA and chlorinated parraffins to replace PBDEs. Unfortunately, evidence suggests that both TBBPA and chlorinated parraffins cause toxic effects, including disrupting hormone systems. Non-halogenated alternatives will have environmental problems as well, but generally on a much lower scale. For example, phosphorous compounds could contribute to water pollution.

Non-Halogenated Alternatives

There are three categories of non-halogenated chemical flame retardants: organophosphorus compounds, inorganic compounds, and nitrogen containing compounds (see Appendix).

The German Environmental Protection Agency selected red phosphorous, ammonium polyphosphate, and aluminum trihydroxide as the least environmentally problematic alternatives.[150] Red phosphorus can technically be used in a variety of polymers to meet even the toughest fire safety standards, although it may not work for all applications.[151]

The Scandinavian furniture company IKEA chose to remove all brominated flame retardants from its products in the late 1990s.[152] In fabrics, IKEA replaced halogenated flame retardants with a coating made of less toxic alternatives (organic phosphorous and nitrogen containing compounds). For polyurethane foam, the company replaced brominated flame retardants with alternatives to the Penta BDE product (melamine and chlorinated parraffins). Because of public health concerns with halogenated chemicals, IKEA is currently working with suppliers to eliminate the chlorinated paraffins and move to completely non-halogenated foams. Their products are able to meet California flame retardancy standards.[153]

The RTP Company in Minnesota is one company active in developing alternatives. According to promotional materials, the company currently produces eight different non-halogenated flame retardants, as well as non-flammable plastics. The company states that in many cases, non-halogenated flame retardants have “lower cost per volume and reduced part weight” than brominated flame retardants, and can be used in computer housings, business equipment, appliances, telecommunications, and building components.[154]

PRODUCTS WITH AVAILABLE ALTERNATIVES

A wide variety of halogen-free products are already in use. In general, these products are similar in cost or slightly more expensive than their brominated counterparts. [155] In situations where alternatives are more expensive, their prices will undoubtedly decline as demand increases.

Printed Circuit Boards

Printed circuit boards are presently coated with TBBPA or PBDEs to increase their fire safety. However, alternatives using halogen free epoxy-based laminates are being produced at only slightly higher cost in Germany and Denmark.[156]

Motorola found that some types of halogen-free laminates are able to meet fire safety standards and even have better electric and mechanical properties that make them more desirable for use in their products. The cost and availability of these products were also able to meet the needs of the company.[157]

Housings for Consumer and Medical Electronics

Traditionally, brominated flame retardants have been used in the housings for televisions, computer monitors, and other electronics. Several alternative casing materials are available today, in grades that meet U.S. fire retardancy requirements for TV and computer monitor backplates.[158]

Switches, Sockets, and Lighting

The plastics used in switches and sockets in direct contact with current have been traditionally protected with brominated flame retardants. However, halogen-free polyamide products are available for most applications.[159] Some alternative sockets are made with porcelain and bakelite, a phenol-based compound. Plastic cover parts in lighting can be replaced with metal parts or halogen-free plastics. One Danish company produces lighting with all halogen-free compounds.[160]

Wires and Cables, etc.

Industry is developing bromine-free products in wiring to minimize the formation of toxic gases by fire and to make wire more recyclable. One producer expected bromine-free rubber cables to be on the market by 2000.[161] Other types of cables made from polyethylene use aluminum trihydroxide as a flame retardant.

Textiles

A wide range of alternative methods to protect fabrics from flame are in use today, including using inherently flame resistant materials like wool, down, or leather.[162] Some synthetic fibers such as aramide (a type of Kevlar) or carbonized fiber are also flame retardant. Alternative flame retardant coatings using reactive phosphorous compounds are also available.

Furniture

Alternatives to toxic flame retardants are in use in foams and furniture upholstery today. Denmark has completely phased out the use of brominated compounds in flexible foams, replacing them with ammonium polyphosphates and reactive phosphorus compounds.[163] Additionally, simply increasing foam density meets some flammability requirements.

Carpets

Aluminum trihydroxide is widely used to make carpets flame-resistant in Denmark, a system that can meet all known requirements at a price comparable to that of brominated flame retardants.[164]

Table 4: Examples of Manufacturers Phasing out Toxic Flame Retardant Use

Company	What they are doing to reduce Brominated Flame Retardant Use
Apple	Most Apple products contain no PBDEs in plastic parts weighing more than 25 grams.[165]
Ericsson	PBBs and PBDEs have been totally banned from the products of this Swedish cellular phone company. The company expected 80% of its printed wiring boards to be halogen free in 2002.[166]
IBM	IBM produces the Intellistation, using 100% recycled plastic containing no halogenated flame retardants.[167]
IKEA	IKEA has totally phased out the use of BFRs in its products, including furniture, and is working steadily toward being completely halogen free.[168]
Intel	Intel has replaced BFRs in most plastics, and completely replaced PBBs and PBDEs.[169]
Motorola	Motorola produces one phone that is BFR free, and has successfully replaced BFRs in laminated circuit boards.[170]
NEC	NEC produces a plastic called NuCycle which is halogen free and phosphorous free. It is used in producing casings for their products and contains recycled polycarbonate.[171]
Panasonic	In 1999, Panasonic produced a television without halogenated flame retardants in wires, the casing, or in a number of the circuit boards. Products which use some halogen free plastics include PCs, air conditioners, televisions, and washing machines.[172]
Phillips	Phillips Consumer Electronics has a list of banned substances that include PBBs and PBDEs. Products are evaluated against this list before introduction.[173]
Sony	Sony's green management plan calls for the full elimination of BFRs from its products by 2003.[174]

6. Policy Recommendations

Evidence to date indicates that PBDEs pose a risk to public health in California and the U.S. as a whole. PBDE levels are rising exponentially and exposure may interfere with normal brain development.

Our current state of knowledge about widespread PBDE exposures closely parallels the debate about PCBs 30 years ago. PCB levels in human breast milk are now slowly declining, 27 years after the 1976 manufacturing ban. Years of research have confirmed that PCBs are highly toxic. Unfortunately, PCB exposures were allowed to climb high enough to cause measurable effects in human children before action was taken. The same outcome from PBDEs may still be avoidable.

THE EUROPEAN UNION HAS ALREADY ACTED TO PROTECT PUBLIC HEALTH

In September of 2001, the European Union adopted a directive on managing waste from electrical and electronic equipment. The plan is designed to divert electrical products from landfills, to promote recycling, and to eliminate lead, mercury, cadmium, hexavalent chromium, PBB and PBDEs from consumer electronics. The directive, officially adopted in February 2003, bans the use of the Penta, Octa, and Deca BDE products in consumer electronics beginning in mid-2006.[175]

Additionally, the European Union has issued a separate rule which fully bans the marketing and use of the Penta and Octa BDE products in all sectors beginning in mid-2004. The final text of this rule was published in the official journal of the European Union in February 2003.[176] Deca BDE may be added to the comprehensive marketing and use ban, depending on the results of a risk reduction strategy currently being assembled by the European Chemicals Bureau.[177]

The E.U. acted on initial signs of a significant threat to human health and the environment despite incomplete toxicology data for the chemicals. When the European Union completed a risk assessment of the Penta BDE product in August of 2000, member states noted the uncertainties surrounding the risk for infants exposed through breast milk.[178] Instead of waiting for years of scientific studies to resolve those uncertainties, the member states voted to take risk reduction measures without delay. The European Parliament insisted that Octa and Deca BDE be regulated alongside Penta BDE, rather than wait for further study while exposures increased exponentially.[179]

Germany originally began the phase-out effort in the 1980s with a Dioxin directive, which prompted German chemical manufacturers to voluntarily stop manufacturing PBBs and PBDEs in 1989 in order to prevent the continued creation of brominated dioxins.[180] Because Sweden regularly monitors breast milk for the presence of contaminants, they were able to detect the problem and prompt Europe to limit exposure by to cutting the use of PBDEs by two-thirds in 1997. This step may have led to a decline in PBDE levels in the breast milk of Swedish mothers since 1998 (see figure 8). Community-based breast milk monitoring programs such as this are an excellent tool for citizens to evaluate their chemical exposures and encourage a timely response.

E.U. member countries continue to take positions on chemical regulation that protect public health. For example, in January 2003, the Dutch State Council rejected a permit for the production of a new brominated flame retardant because the manufacturer was not able to provide enough evidence that the product was safe. This is the first time a court has placed the burden of proof on a company to show that their chemical will not create environmental harm.[181]

POLICY RECOMMENDATIONS

1. Phase out Penta, Octa, and Deca BDE

The most effective way to reduce the risk of harm to infant children from PBDE exposure is to stop manufacture and use of all the commercial PBDE mixtures. PBDE exposures pose a risk to the neurological health of current and future generations. The potential for harm from the use of these chemicals in California and the United States is even greater than in Europe. What is more, these exposure levels are climbing exponentially, making harmful health effects more likely with every passing year.

In order to protect the health of children, we must stop the flow of PBDEs into the food chain. The only way to achieve this goal is to stop making and using them. The lesson of PCBs tells us that if PBDEs were banned today, levels of exposure would start to decline. Still, the legacy of contamination and exposure would remain for decades and further study could prove PBDEs to be more harmful than current evidence already indicates. The case for immediate action should be clear.

The manufacture and use of Penta BDE product, whose ingredients appear at the highest levels in living organisms, should cease immediately. Evidence that components of the Octa and Deca BDE products can degrade in the environment into more bioaccumulative and toxic forms suggests that their manufacture and use should be phased out as well.

2. Phase Out Other Brominated Flame Retardants

Initial evidence shows that other brominated flame retardant chemicals that are used in high volumes, like HBCD and TBBPA, are likely to damage human health and the environment. Studies have shown indications that they are persistent and could disrupt hormone systems. More studies should be conducted as soon as possible, and if those studies back up initial findings, regulatory agencies should take early action to phase them out.

3. Reform U.S. Toxic Chemicals Policy

PBDE use skyrocketed before regulatory agencies in the United States noticed the possibility of a problem. The threat posed by toxic flame retardants demonstrates the general failure of chemical regulation in the United States to effectively protect public health from toxic chemicals used in industry and placed in consumer products. U.S. chemical companies hold licenses to make at least 80,000 chemicals for commercial use, with about 2,000 new ones introduced every year. Approximately 85% of these chemicals are missing even basic information on potential health effects.[182] For more than a thousand chemicals where significant evidence of harm to public health exists, inadequate resources and legal authority prevent regulatory agencies from taking protective action.

Chemicals that are untested or known to be hazardous should not be on the market or in widespread use and distribution. U.S. chemicals policy should be reformed to ensure that manufacturers and industrial users provide regulatory agencies and the public with adequate information about their products, or remove them from the market in favor of safer alternatives. U.S. chemicals policy should also be revised so that where there is evidence of serious or irreversible health impacts, agencies can restrict or prohibit the manufacture and use of the chemical, to protect public health from potentially dangerous substances before further damage is done.

Appendix: Replacement Flame-Retardant Chemicals and Materials

Table A-1: Some Currently Available Alternative Flame-Retardant Chemicals and Materials[183]

Materials traditionally treated with BFRs	Main Application	Alternative Flame Retardants	Alternative Materials
Epoxy	Printed circuit boards	Nitrogen and phosphorus compounds; ammonium polyphosphate and aluminum trihydroxide	Polyphenylene Sulfide
Phenolic Resins	Printed circuit boards	Nitrogen and phosphorus compounds; aluminum trihydroxide	
Acrylonitrile-Butadiene-Styrene (ABS)	Casings of electronic products	PC / ABS blends or PPE / PS blends with organic phosphorus compounds	
High Impact Polystyrene (HIPS)	Casings of electronic products, wiring parts	Organic phosphorus compounds	Polyethylene with magnesium hydroxide
PBT / PET	Switches, sockets, parts of electric machines	Alternatives experimental	Polyamide, polyketone, ceramics or self-extinguishing plastics for some applications.
Polyamide	Parts of electronic equipment	Magnesium hydroxide, red phosphorus, melamine cyanurate, melamine polyphosphate	
Polycarbonate (PC)	Parts of electronic equipment	Organic phosphorous compounds	
Polypropylene	Roofing foils	Ammonium polyphosphate	
Rigid polyurethane foam	Insulation of cold storage rooms	Ammonium polyphosphate and red phosphorous	Mineral wool or other solutions for some applications
Soft polyurethane foam	Cushioned furniture	Ammonium polyphosphate, melamine, reactive phosphorous polyols	
Cotton textiles	Furniture	Ammonium polyphosphate, diammonium phosphate	
Synthetic textiles	Furniture and protective clothing	Reactive phosphorus compounds	

Table A-2: Common Non-Halogenated Flame Retardants

Organophosphorus Compounds	Inorganic Compounds	Nitrogen-Containing Compounds
Triphenyl phosphate	Aluminum trihydroxide	Melamine
Tricresyl phosphate	Magnesium hydroxide	
Resorcinol bis(diphenylphosphate)	Ammonium polyphosphate	
Phosphonic Acid	Red phosphorous	
	Zinc borate	

NOTES

1. National Association of State Fire Marshals, "California Upholstered Furniture Fire Losses vs. the Rest of the U.S.," viewed at www.firemarshals.org/issues/home/upholstery_fires.html on 4 December 2002.

2. L.M. Robison et al, *National trends in the prevalence of attention-deficit/hyperactivity disorder and the prescribing of methylphenidate among school-age children: 1990-1995.* Clin. Pediatr. (Phila) 38:209-17, 1999; L.M. Robison et al, *Is attention deficit hyperactivity disorder increasing among girls in the US? Trends in diagnosis and the prescribing of stimulants.* CNS Drugs 16:129-37, 2002; J.M. Zito et al, *Trends in the prescribing of psychotropic medications to preschoolers.* Journal of the American Medical Association 283:1025-30, 2000.

3. National Academy of Sciences, National Research Council, Committee on Developmental Toxicology, *Scientific Frontiers in Developmental Toxicology and Risk Assessment*, National Academy Press, 1 June 2000; B. Weiss and PJ Landrigan, *The developing brain and the Environment*. Environmental Health Perspectives 107, June 2000; National Academy of Sciences, National Research Council, Committee on Developmental Toxicology, *Evaluating Chemical and Other Agent Exposures for Reproductive and Developmental Toxicity*, National Academy Press, 2001; National Environmental Trust, Physicians for Social Responsibility and the Learning Disabilities Association of America, *Polluting Our Future: Chemical Pollution in the U.S. that Affects Child Development and Learning* September 2000.

4. Environmental Finance Center, EPA Region 9, *Brominated Flame Retardants Background and Issues.* viewed at www.greenstart.org, 21 June 2002; John Karl, "High Levels of Chemical Flame Retardant Found in Lake Michigan Salmon," *Great Lakes Online*Wisconsin Sea Grant*, 14 February 2001.

5. Environmental Finance Center, EPA Region 9, *Brominated Flame Retardants Background and Issues.* viewed at www.greenstart.org, 21 June 2002.

6. World Health Organization, International Program on Chemical Safety, *Brominated Diphenyl Ethers*, World Health Criteria #162, 1994.

7. National Chemicals Inspectorate (Sweden), "KEMI Proposes a Prohibition of Flame Retardants," 15 March 1999; Bromine Science and Environmental Forum, *An Introduction to Brominated Flame Retardants*, 19 October 2000.

8. Robert Hale et al, *Potential Role of Fire Retardant-Treated Polyurethane Foam as a Source of Brominated Diphenyl Ethers in the U.S. Environment.* Chemosphere 46, 729-735, 2002.

9. World Health Organization, International Program on Chemical Safety, *Brominated Diphenyl Ethers*, World Health Criteria #162, 1994; European Chemicals Bureau, *European Union Risk Assessment: Diphenyl Ether, Pentabromo Derivative*, August 2000.

10. The specific congeners most often found in the environment are BDE-47, BDE-99, BDE-100, and BDE-153.

11. European Chemicals Bureau, *European Union Risk Assessment Report: diphenyl ether, pentabromo derivative*. August 2000.

12. Richard Wenning, Environ International Corporation, *Uncertainties and Data Needs in Risk Assessment of Three Commercial Polybrominated Diphenyl Ethers*, Chemosphere 46, 779-796, 2002; The British Plastics Federation, "Acrylonitrile-Butadiene-Styrene," (*factsheet*), viewed at www.bpf.co.uk on 20 February 2003.

13. Richard Wenning, Environ International Corporation, *Uncertainties and Data Needs in Risk Assessment of Three Commercial Polybrominated Diphenyl Ethers*, Chemosphere 46, 779-796, 2002; Dow Chemical Company, "Technical Library: High Impact Polystyrene Resins," (*factsheet*), viewed at www.dow.com on 20 February 2003.

14. Richard Wenning, Environ International Corporation, *Uncertainties and Data Needs in Risk Assessment of Three Commercial Polybrominated Diphenyl Ethers*, Chemosphere 46, 779-796, 2002.

15. M.L. Hardy, *The toxicology of the three commercial polybrominated diphenyl oxide (ether) flame retardants.* Chemosphere 46, 757-77, 2002; M.L. Hardy, *A comparison of the properties of the major commercial PBDPO/PBDE products to those of major PBB and PCB products.* Chemosphere 46, 717-28, 2002.

16. Cynthia deWit, *Evaluation of the TSRI Workshop on Brominated Flame Retardants, August 23-24, 2001.* in *Impact of Polybrominated Diphenyl Ethers to Canadian Environment and Health of Canadians,* TSRI Project #237, Final Report, 2002; A. Sjodin et al. *Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers*. Environmental Health Perspectives 107(8), 643-648, 1999; A. Sjodin et al, *Flame Retardants in Indoor Air at an Electronics Recycling Plant and at Other Work Environments.* Environmental Science and Technology, 35(3), 448-454, 2001.

17. Eriksson et al, *Photo decomposition of brominated diphenyl ethers in methanol/water*. Poster at the BFR 2001 conference, Stockholm, BFR abstract book page 203-206, May 2001; S. Ohta et al, Organohalogen Compounds 52 (2001) 321-323; U. Sellstrom, *et al, Photolytic debromination of decabromodiphenyl ether (DeBDE).* Organohalogen Compounds 35, 447-450, 1998; I. Watanabe, *et al, Formation of brominated dibenzofurans from the photolysis of flame retardant decabromodiphenyl ether in hexane solution by UV and sunlight.* Bulletin of

Environmental Contaminant Toxicology 39, 953-959, 1987; C.P. Rice et al, *Comparisons of PBDE composition and concentration in fish collected from the Detroit River, MI and the Des Plaines River, IL.* Chemosphere 49, 731-737, 2002.

18. The European Parliament, *Directive of the European Parliament and of the Council on the restriction of the use of certain hazardous substances in electrical and electronic equipment.* Document PE-CONS 3662/02, Brussels, 8 Nov. 2002.

19. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, *Toxicological profile for polybrominated biphenyls and polybrominated diphenyl ethers — draft for public comment.* Atlanta, GA, 2002.

20. Cynthia de Wit, Institute of Applied Environmental Research at Stockholm University, *An overview of brominated flame retardants in the environment.* Chemosphere 46, 583-624, 2002.

21. Agency for Toxic Substances and Disease Registry, "Polybrominated Biphenyls and Polybrominated Diphenyl Ethers (PBBs and PBDEs)" (factsheet), October 23, 2002.

22. Ibid.

23. Bromine Science and Environmental Forum, *An Introduction to Brominated Flame Retardants*, 19 October 2000.

24. Umweltbundesamt (Germany's Federal Environment Agency), *Substituting Environmentally Relevant Flame Retardants: Assessment Fundamentals*. ISSN 0722-186X, March 2001.

25. Ibid.

26. Ibid.

27. Tom McDonald, CalEPA, *Brominated Flame Retardants (BFR) Primer,* presented at the EFC9 Brominated Flame Retardants and Electronics Conference and Roundtable, San Francisco, 24 September 2002; Linda Birnbaum, U.S. Environmental Protection Agency, personal communication, 4 Feb. 2003.

28. Jon Karl, GlobalTechnoScan.com, "Chemical Flame Retardant Found in Salmon," 27 February 2002.

29. See Note 23.

30. Ibid.

31. Ibid.

32. Ibid.

33. Ibid.

34. Jake Ryan, Environment Canada, *Levels and trends of polybrominated diphenyl ethers (PBDEs) in human milks, and food exposure in Canada.* Canada TSRI Project #237, Final Report, 2002.

35. G. Lindstrom et al, *Identification of 19 PBDEs in long-finned pilot whales from the Atlantic*, Arch. Environ. Contam. Toxicol. 36, 355, 1999.

36. P.S. Haglund et al, *Identification and quantification of polybrominated diphenyl ethers and methoxy-polybrominated diphenyl ethers in Baltic biota,* Environmental Science and Technology 31, 3281-3287, 1997.

37. G.A. Stern and M.G. Ikonomou, *Temporal trends of polybrominated diphenyl ethers in SE Baffin Beluga: increasing evidence of long range atmospheric transport.* Organohalogen Compounds 47, 81-84, 2000.

38. Jon Manchester et al, University of Wisconsin, *Comparison of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in Lake Michigan salmonids.* Environmental Science and Technology 35, 1072-77, 2001.

39. Robert C. Hale, Mark J. La Guardia, Ellen P. Harvey, T. Matteson Mainor, William H. Duff, and Michael O. Gaylor, The College of William and Mary Institute for Marine Science, *Polybrominated Diphenyl Ether Flame Retardants in Virginia Freshwater Fishes*, Environmental Science and Technology 35, 4585 -4591, 31 October 2001.

40. Jianwen She et al, California Department of Toxics Substances Control, *PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue.* Chemosphere 46, 697-707, 2002.

41. Ibid.

42. D. Meironyte; K. Noren, A. Bergman, *Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972-1997.* Journal of Toxicology and Environmental Health Part A, 58(6), 329-41, 26 Nov 1999.; Noren K., Meironyte D., *Certain Organochlorine and Organobromine Contaminants in Swedish Human Milk in Perspective of Past 20-30 Years.* Chemosphere 40, 1111-1123, 2000; J. Ryan and B. Patry, Health Canada, *Determination of brominated diphenyl ethers (BDEs) and levels in Canadian human milks.* Organochlorine Compounds 47, 57-60, 2000; O. Papke, *et al Determination of PBDEs in human milk from the United States: Comparison of results from three laboratories.* Organohalogen Compounds 52, 197-200, 2001.

43. O. Andersson and A. Wartanien, *Polybrominated aromatic pollutants in fish in Sweden*, Chemosphere 10, 347-350, 1981.

44. See Note 20.

45. Ibid.

46. Olaf Paepke and Peter Fuerst, Ergo Forschungsgesellschaft mbH and Chemisches Landes- und Staatliches Veterinäruntersuchungsamt, *Background Contamination of Humans with Dioxins, Dioxin-like PCBs and other POPs*. The Handbook of Environmental Chemistry, Volume 3, Part O, Chapter 10, 2003.

47. Ibid.

48. Kim Hooper and Thomas McDonald, CalEPA, *The PBDEs: an emerging environmental challenge and another reason for breast-milk monitoring programs.* Environmental Health Perspectives 108, 387-392, 2000.

49. Cynthia de Wit, Institute of Applied Environmental Research at Stockholm University, *An overview of brominated flame retardants in the environment.* Chemosphere 46, 583-624, 2002; Jianwen She, *et. al,* California Department of Toxics Substances Control, *PBDEs in the San Francisco Bay Area: measurements in harbor seal blubber and human breast adipose tissue.* Chemosphere 46, 697-707, 2002.; Robert Hale, *et al* Virginia Institute of Marine Science, *Persistent Pollutants in Land Applied Sludges.* Nature 412, 140-141, 2001.

50. Thomas McDonald, California EPA, *A perspective on the potential health risks of PBDEs,* Chemosphere 46, 745-755, 2002.

51. Myrto Petreas, CalEPA, *PBDEs in California Humans and Wildlife,* presented at the Environmental Finance Center Region 9 Conference on BFRs, San Francisco, 24 Sept. 2002.

52. See Note 40.

53. Kellyn Betts, "Rapidly Rising PBDE levels in North America," Environmental Science and Technology Science News, 7 Dec. 2001; D. Meironyte; K. Noren, A. Bergman, *Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972-1997.* Journal of Toxicology and Environmental Health Part A, 58(6), 329-41, 26 Nov 1999.

54. O. Papke, *et al Determination of PBDEs in human milk from the United States: Comparison of results from three laboratories.* Organohalogen Compounds 52, 197-200, 2001.

55. Ibid.

56. See Note 20.

57. L. Asplund et al, *Levels of polybrominated diphenyl ethers (PBDEs) in fish from the Great Lakes and Baltic Sea,* Organohalogen Compounds 40, 351-354, 1999; Jon Manchester et al, University of Wisconsin, *Comparison of polybrominated diphenyl ethers (PBDEs) and polychlorinated biphenyls (PCBs) in Lake Michigan salmonids.* Environmental Science and Technology 35, 1072-77, 2001.

58. See Note 40.

59. Robert C. Hale, Mark J. La Guardia, Ellen P. Harvey, T. Matteson Mainor, William H. Duff, and Michael O. Gaylor, The College of William and Mary Institute for Marine Science, *Polybrominated Diphenyl Ether Flame Retardants in Virginia Freshwater Fishes*, Environmental Science and Technology 35, 4585 -4591, 31 October 2001.

60. C.R. Allchin et al, *Polybrominated diphenylethers in sediments and biota downstream of potential sources in the UK.* Environmental Pollution 105, 197-207, 1999.

61. Myrto Petreas, CalEPA, *PBDEs in California Humans and Wildlife*, presented at the Environmental Finance Center Region 9 Conference on BFRs, San Francisco, 24 Sept. 2002.

62. B. Strandberg et al, *Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air.* Environmental Science and Technology 35, 1078-1083, 2001.

63. See Note 11.

64. Ibid.

65. See Note 20.

66. Robert Hale et al, Virginia Institute of Marine Science, *Persistent pollutants in land-applied sludges*. Nature 412, 140-141, 2001.

67. See Note 40.

68. Ibid.

69. J.M. Luross et al, Environment Canada, *Spatial and temporal distribution of polybrominated diphenyl ethers in lake trout from the Great Lakes.* Organohalogen Compounds 47, 73-76, 2000.

70. Ibid.

71.. D. Meironyte; K. Noren, A. Bergman, *Analysis of polybrominated diphenyl ethers in Swedish human milk. A time-related trend study, 1972-1997.* Journal of Toxicology and Environmental Health Part A, 58(6), 329-41, 26 Nov 1999.; Noren K., Meironyte D., *Certain Organochlorine and Organobromine Contaminants in Swedish Human Milk in Perspective of Past 20-30 Years.* Chemosphere 40, 1111-1123, 2000.

72. Noren K., Meironyte D., *Certain Organochlorine and Organobromine Contaminants in Swedish Human Milk in Perspective of Past 20-30 Years.* Chemosphere 40, 1111-1123, 2000.

73. Canadian Milk Bank: J. Ryan and B. Patry, Health Canada, *Determination of brominated diphenyl ethers (BDEs) and levels in Canadian human milks.* Organochlorine Compounds 47, 57-60, 2000; Sweden Milk Bank:

Noren K., Meironyte D., *Certain Organochlorine and Organobromine Contaminants in Swedish Human Milk in Perspective of Past 20-30 Years.* Chemosphere 40, 1111-1123, 2000; Preliminary U.S. Data: O. Papke, *et al Determination of PBDEs in human milk from the United States: Comparison of results from three laboratories.* Organohalogen Compounds 52, 197-200, 2001; Figure based on Mehran Alaee, Environment Canada, *Rapidly Rising PBDE levels in North America*, Environmental Science and Technology Science News, 7 Dec. 2001.

74. Michael G. Ikonomou, Sierra Rayne, and Richard F. Addison, Canada Department of Fisheries and Oceans, *Exponential Increases of the Brominated Flame Retardants, Polybrominated Diphenyl Ethers, in the Canadian Arctic from 1981 to 2000;* Environ. Sci. Technol. 36, 1886 -1892, 3 April 2002.

75. Dr A. Michael Warhurst, "Brominated Flame Retardants," viewed at website.lineone.net/~mwarhurst/bfr.html, 21 June 2002.

76. See Note 11.

77. Ibid.

78. See Note 23.

79. See Note 11.

80. A. Sjodin et al. *Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers***.** Environmental Health Perspectives 107(8), 643-648, 1999; A. Sjodin et al, *Flame Retardants in Indoor Air at an Electronics Recycling Plant and at Other Work Environments.* Environmental Science and Technology, 35(3), 448-454, 2001; Cynthia de Wit, Institute of Applied Environmental Research at Stockholm University, An overview of brominated flame retardants in the environment. Chemosphere 46, 583-624, 2002.

81. Robert Hale et al, College of William and Mary, *Potential role of fire retardant-treated polyurethane foam as a source of brominated diphenyl ethers to the US environment*, Chemosphere 46, 729-735, 2002.

82. Environment Daily, "Norway detects 'firsts' on retardant seepage." Issue No. 1364, 16 Jan. 2003.

83. Marcia Hardy, Albemarle Corporation, *The Toxicology of the Three Commercial Polybrominated Diphenyl Oxide (Ether) Flame Retardants.* Chemosphere 46, 757-777, 2002.

84. Cynthia deWit, *Evaluation of the TSRI Workshop on Brominated Flame Retardants, August 23-24, 2001.* in *Impact of Polybrominated Diphenyl Ethers to Canadian Environment and Health of Canadians,* TSRI Project #237, Final Report, 2002; A. Sjodin et al. *Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers***.** Environmental Health Perspectives 107(8), 643-648, 1999; A. Sjodin et al, *Flame Retardants in Indoor Air at an Electronics Recycling Plant and at Other Work Environments.* Environmental Science and Technology, 35(3), 448-454, 2001.

85. Eriksson et al, *Photo decomposition of brominated diphenyl ethers in methanol/water*. Poster at the BFR 2001 conference, Stockholm, BFR abstract book page 203-206, May 2001; S. Ohta et al, Organohalogen Compounds 52 (2001) 321-323; U. Sellstrom, *et al, Photolytic debromination of decabromodiphenyl ether (DeBDE).* Organohalogen Compounds 35, 447-450, 1998; I. Watanabe, *et al, Formation of brominated dibenzofurans from the photolysis of flame retardant decabromodiphenyl ether in hexane solution by UV and sunlight.* Bulletin of Environmental Contaminant Toxicology 39, 953-959, 1987.

86. B. Strandberg et al. *Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air.* Environmental Science and Technology 35, 1078-1083, 2001; D. Santillo et al. *The presence of brominated flame retardants and organotin compounds in dusts collected from Parliament buildings from eight countries.* Greenpeace Research Laboratories, Exeter, U.K., GRL-TN-03-2001, 2001.

87. C.P. Rice et al, *Comparisons of PBDE composition and concentration in fish collected from the Detroit River, MI and the Des Plaines River, IL.* Chemosphere 49, 731-737, 2002.

88. I. Watanabe, M. Kawano, and R. Tatsukawa, *Polybrominated and Mixed Polybromo/Chlorinated dibenzo-p-dioxins and —dibenzofurans in the Japanese Environment*, Organohalogen Compounds 24, 337-340, 1995.

89. Environment News Service, "EU lawmakers vote broad fire retardant ban," 6 Sep. 2002.

90. A. Lione, *Polychlorinated Biphenyls and Reproduction.* Reproductive Toxicology 2, 83-89, 1988.

91. T. Strandman, J. Koistinen, and T. Variainen, *Polybrominated diphenyl ethers (PBDEs) in placenta and human milk.* Organohalogen Compounds 47, 61-64, 2000.

92. A. Madzai, N. Dodder, M. Abernathy, R. Hites, and R. Bigsby, *Polybrominated Diphenyl Ethers in Maternal and Fetal Blood.* Environmental Health Perspectives, (in press as of 2003).

93. National Resources Defense Council, *Healthy Milk, Healthy Baby: Chemical Pollution and Mother's Milk.* 22 May 2001.

94. M. Juisman et al. *Neurological condition in 18-month old children perinatally exposed to PCBs and dioxins.* Early Human Development 43, 165-176, 1995; C. Lanting et al., *Breastfeeding and neurological outcome at 42 months.* Acta Pediatr. 87, 1224-1229, 1998.

95. A. Sjödin et al, Stockholm University, *Influence of the consumption of fatty Baltic sea fish on plasma levels of halogenated environmental contaminants in Latvian and Swedish men*. Environmental Health Perspectives 108, 1035. November 2000.

96. Jake Ryan, Environment Canada, *Levels and trends of polybrominated diphenyl ethers (PBDEs) in human milks, and food exposure in Canada.* Canada TSRI Project #237, Final Report, 2002.

97. European Chemicals Bureau, *European Union Risk Assessment Report: diphenyl ether, pentabromo derivative*. August 2000; B. Strandberg et al. *Concentrations and spatial variations of polybrominated diphenyl ethers and other organohalogen compounds in Great Lakes air.* Environmental Science and Technology 35, 1078-1083, 2001; D. Santillo et al. *The presence of brominated flame retardants and organotin compounds in dusts collected from Parliament buildings from eight countries.* Greenpeace Research Laboratories, Exeter, U.K., GRL-TN-03-2001, 2001.

98. A. Sjodin et al., *Flame retardant exposure: polybrominated diphenyl ethers in blood from Swedish workers.* Environmental Health Perspectives 107, 643-648, 1999.

99. National Association of State Fire Marshals, "California Technical Bulletin 117," viewed at www.firemarshals.org/issues/home/upholstery_fires.html on 4 Dec. 2002

100. See Note 50.

101. The European Chemicals Bureau predicts higher PBDE concentrations localized near factories. See Note 11.

102. California Bureau of Home Furnishings and Thermal Insulation, Research and Development Department, personal correspondence, 17 April 2001.

103. Agency for Toxic Substances and Disease Registry (ATSDR), *Toxicological profile for polybrominated biphenyls and polybrominated diphenyl ethers — Draft for public comment. Chapter 5 — Production, import, use, and disposal*. Atlanta, GA: U.S. Department of Health and Human Services, September 2002.

104. See Note 50.

105. G. Koren, *Maternal-Fetal Toxicology (2nd Ed.)*, Marcel Dekker, Inc. 1994.

106. S.P. Porterfield, C.E. Hendrich, *The role of thyroid hormones in prenatal and neonatal neurological development-current perspectives.* Endocrinol Rev 14:94-106, 1993.

107. V.J. Pop et al, *Low maternal free thyroxine concentrations during early pregnancy are associated with impaired psychomotor development in infancy.* Clinical Endocrinology 50, 149-155, 1999; J.E. Haddow et al, *Maternal thyroid deficiency during pregnancy and subsequent neuropsychological development of the child*. New England Journal of Medicine 341, 549-555, 1999; G. Morreale de Escobar et al, *Is neuropsychological development related to maternal hypothyroidism or to maternal hypothyroxinemia?* J. Clin. Endocrinol. Metab. 85, 3975-3987, 2000; K. Howdeshell, *A model of the development of the brain as a construct of the thyroid hormone system.* Environmental Health Perspectives, 110, 337-348, 2002.

108. Zhou et al, *Effects of short term in vivo exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats.* Toxicological Science 61, 76-82, 2001.

109. U. Oern and E. Klasson-Whehler. *Metabolism of 2,2',4,4'-tetrabromodiphenyl ether in rat and mouse.* Xenobiotica 28, 199-211, 1998;.

110. Kim Hooper and Thomas McDonald, CalEPA, *The PBDEs: an emerging environmental challenge and another reason for breast-milk monitoring programs.* Environmental Health Perspectives 108, 387-392, 2000; P.O. Darnerud et al, *Polybrominated diphenyl ether occurrence, dietary exposure and toxicology*. Environmental Health Perspectives 109 supplement 1, 49-68, 2001; Zhou et al, *Effects of short term in vivo exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats.* Toxicological Science 61, 76-82, 2001.

111. Zhou et al, *Effects of short term in vivo exposure to polybrominated diphenyl ethers on thyroid hormones and hepatic enzyme activities in weanling rats.* Toxicological Science 61, 76-82, 2001.

112. J.R. Fowles et al, *Immunologic and Endocrine Effects of the Flame-Retardant Pentabromodiphenyl Ether (DE-71) in C57BL/6J Mice,* Toxicology 86, 49-61, 1994.

113. S. Hallgren and P.O. Darnerud, *Effects of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), and chlorinated paraffins (CPs) on thyroid hormone levels and enzyme activities in rats.* Organohalogen Compounds 35, 391-394, 1998.

114. Reviewed in Note 50.

115. P Eriksson et al. *Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment?* Environ Health Perspect 109, 903-8, 2001; P Eriksson et al. *A brominated flame retardant, 2,2',4,4',5-pentabromodiphenyl ether: uptake, retention, and induction of neurobehavioral alterations in mice during a critical phase of neonatal brain development.* Toxicol Sci 67, 98-103, 2002; H Viberg et al, *Neonatal exposure to the brominated flame retardant 2,2',4,4',5- pentabromodiphenyl ether causes altered susceptibility in the cholinergic transmitter system in the adult mouse.* Toxicol Sci 67, 104-7, 2002; H. Viberg, A. Fredriksson, and E. Jakobsson, *Developmental neurotoxic*

effects of 2,2,4,4,5-pentabromodiphenyl ether in the neonatal mouse. Toxicologist 54, 1360, 2000; H. Viberg, A. Fredriksson, E. Jakobsson, U. Ohrn, and P. Eriksson, *Brominated flame retardant: Uptake, retention, and developmental neurotoxic effects of decabromodiphenyl ether in the neonatal mouse.* Toxicologist 61, 1034, 2001; I. Branchi et al. *Effects of perinatal exposure to a polybrominated diphenyl ether (PBDE 99) on mouse neurobehavioural development*. Neurotoxicology 23, 375-84, 2002; J.L. Jacobson., S.W. Jacobson, H.B. Humphrey, *Effects of in Utero Exposure to Polychlorinated-Biphenyls and Related Contaminants on Cognitive-Functioning in Young Children.* Journal of Pediatrics, 116:38-45, 1990.

116. Thomas McDonald, CalEPA, *A perspective on the potential health risks of PBDEs.* Chemosphere 46, 745-755, 2002; P. Kodavanti and E. Derr-Yellin, *Differential effects of polybrominated diphenyl ethers and polychlorinated biphenyls on [3H]arachidonic acid release in rat cerebellar granule neurons*, Toxicological Science 68, 451-7, 2002.

117. See Note 115.

118. An average mouse is 10% to 20% fat: J. Friedman, Rockefeller University, "Body Weight Regulated by Newly Discovered Hormone," (*News Release*), News from the Rockefeller University, 27 July 1995.

119. See Note 61.

120. See Note 40.

121. See Note 54.

122. A. Madzai, N. Dodder, M. Abernathy, R. Hites, and R. Bigsby, *Polybrominated Diphenyl Ethers in Maternal and Fetal Blood.* Environmental Health Perspectives, (in press as of 2003).

123. Agency for Toxic Substances and Disease Registry (ATSDR), *Draft for Public Comment — Toxicological Profile for Polybrominated Biphenyls and Polybrominated Diphenyl Ethers (PBBs and PBDEs).* Chapter 8 — Regulations and Advisories, September 2002.

124. L. Hardell et al, *Concentrations of the flame retardant 2,2',4,4'-tetrabrominated diphenyl ether in human adipose tissue in Swedish persons and the risk for Non-Hodgkin lymphoma.* Oncology Research 10, 429-432, 1998.

125. See Note 50.

126. P.O. Darnerud, A. Thuvander, *Studies on Immunologial Effects of Polybrominated Diphenyl Ethers (PBDE) and Polychlorinated biphenyls (PCB) Exposures in Rats and Mice*, Organohalogen Compounds, 35:415-418, 1998; J.R.Fowles et al, *Immunologic and endocrine effects of the flame retardant pentabromodiphenyl ether (DE-71) in C57BL/6J mice.* Toxicology 86, 49-61.

127. See Note 50.

128. S. Ayris, S. Harrad, *The fate and persistence of polychlorinated biphenyls in soil.* J Environ Monit 4:395-401, 1 August 1999.

129. K. Nylund et al, Analysis of some polyhalogenated organic pollutants in sediments and sewage sludge. Chemosphere, 24: 1721-1730, 1992; B.N. Zegers et al, Levels of Some Polybrominated Diphenyl Ether (PBDE) Flame Retardants in Dated Sediment Cores. Organohalogen Compounds, 47: 229-232, 2000.

130. M. Alaee et al, Distribution of Polybrominated Diphenyl Ethers in the Canadian Environment. Organohalogen compounds, 40: 347-350, 1999. M. Alaee et al, Determination of Flame Retardants in Environmental Matrices. Poster, 48th ASMS conference, 2000.

131. The most bioaccumulative PBDEs are two specific types of tetra and penta BDE molecules known as BDE-47 and BDE-53; Bioaccumulation data: S. Burreau et al, *Dietary uptake in pike (Esox lucius) of some polychlorinated biphenyls, polychlorinated naphtalenes and polybrominated diphenyl ethers administered in natural diet.* Environmental Toxicology and Chemistry, 16: 2508-2513, 1997; K. Gustafsson et al. *Bioaccumulation Kinetics of Brominated Flame Retardants (Polybrominated Diphenyl Ethers) in Blue Mussel (Mytilus edulis).* Environmental Toxicology and Chemistry, 18: 1218-1224, 1999; P. Haglund et al, *Identification and Quantification of Polybrominated Diphenyl Ethers and Methoxy-Polybrominated Diphenyl Ethers in Baltic Biota.* Environmental Science and Technology, 31: 3281-3287, 1997; T. Strandman et al, T., *Levels of Some Polybrominated Diphenyl Ethers (PBDEs) in Fish and Human Adipose Tissue in Finland.* Organohalogen Compounds, 40: 355-358, 1999.

132. PCBs were not officially recognized as probable human carcinogens by the U.S. Department of Health and Human Services until 1991.

133. This section is based on: Janna Koppe and Jane Keys, European Environment Agency, *Late lessons from early warnings: the precautionary principle 1896-2000; Chapter 6: PCBs and the precautionary principle.* Environmental Issue Report 22, 10 January 2002; Eric Francis, "Conspiracy of Silence: The story of how three corporate giants — Monsanto, G.E., and Westinghouse — covered their toxic trail." *Sierra Magazine*, September/October 1994; and Environmental Working Group, *Chemical Industry Archives: Poisoned by PCBs.* viewed at www.chemicalindustryarchives.org/dirtysecrets on 10 Feb 2003.

134. Cecil Drinker et al. *The problem of possible systemic effects from certain chlorinated hydrocarbons.* Journal of Industrial Hygiene and Toxicology 19, 283-311, September 1937.

135. Environmental Working Group, *Chemical Industry Archives: Poisoned by PCBs.* viewed at

www.chemicalindustryarchives.org/dirtysecrets on 10 Feb 2003.

136. Dan Fagin, Marianne Lavell, and the Center for Public Integrity, *Toxic Deception*. (Seacaucus, N.J.: Carol Publishing Group), 1996; Eric Francis, "Conspiracy of Silence: The story of how three corporate giants — Monsanto, G.E., and Westinghouse — covered their toxic trail." *Sierra Magazine*, September/October 1994

137. W.J. Rogan, *et al*, *Congenital poisoning by polychlorinated biphenyls and their contaminants in Taiwan.* Science 241, 334-338, 1988; Y.C. Chen, Y.L. Guo, and W.J. Rogan, *Cognitive Development of Yu-Cheng (Oil-Disease) Children Prenatally Exposed to Heat Degraded PCBs*. Journal of the American Medical Association 268, 3213-8, 1992.

138. J.L. Jacobson and S.W. Jacobson, *Effects of in utero exposure to PCBs and related contaminants on cognitive functioning in young children.* Journal of Pediatrics 116, 38-45, 1990; J.L. Jacobson and S.W. Jacobson, *Intellectual impairment in children exposed to polychlorinated biphenyls in utero.* New England Journal of Medicine 335, 783-789, 1996.

139. C. Koopman-Esseboom, *et al*, *Effects of dioxins and polychlorinated biphenyls on thyroid hormone status of pregnant women and their infants.* Pediatric Research 36, 468-473, 1994.

140. A.L. den Ouden et al, *The relation between neonatal thyroxine levels and neurodevelopmental outcome at age 5 and 9 years in a national cohort of very preterm and/or very low birth weight infants.* Pediatric Research 39, 142-145, 1996.

141. U.S. Centers for Disease Control and Prevention, *Second National Report on Human Exposure to Environmental Chemicals*, 31 January 2003.

142. S. Hallgren and P. Darnerud, *Effects of polybrominated diphenyl ethers (PBDEs), polychlorinated biphenyls (PCBs), and chlorinated paraffins (CPs), on thyroid hormone levels and enzyme activities in rats.* Organohalogen Compounds 35, 391-394, 1998.

143. Fox River Watch, *The History of PCBs: When Were Health Problems Detected?*, viewed at www.foxriverwatch.com on 20 February 2003; PBDE information comes from a survey of the literature in the National Library of Medicine as of February 2003.

144. The number of studies published was determined using the National Library of Medicine's NLM Gateway database.

145. Listing in this section does not imply endorsement by Environment California, nor does it imply that we have fully investigated any potentially adverse environmental or health problems that may be caused by these alternatives.

146. Other inherently flame retardant polymers include polyphenylene oxide, polyamide-imides, polyphenylene, isophthalimides, polysulphone, polyaryletherketone, and polyethersulfone.

147. Danish Environmental Protection Agency, *Brominated Flame Retardants: Substance Flow Analysis and Assessment of Alternatives,* Environmental Project # 494 1999.

148. Toshiba Corporation, "Toshiba Develops Environmentally Sophisticated Package for Transistor ICs," (press release), 27 April 1998.

149. See Note 147.

150. See Note 24.

151. See Note 147.

152. Bjorn Frithiof, IKEA, personal communication, 5 Dec. 2002.

153. Ibid.

154. RTP Company, "RTP Company Introduces Non-Halogenated Flame Retardant Compounds," (*press release*), viewed at www.rtpcompany.com on 20 February 2003; RTP Company, "Engineers Develop Eco-Friendly, Flame Retardant, Reinforced Polyester Compound," (*press release*), viewed at www.rtpcompany.com on 20 February 2003

155. See Note 147.

156. Ibid.

157. Steve Scheifers, Motorola, *Bromine Free Alternatives in Electronic Products.* presented at the EFC9 Brominated Flame Retardants and Electronics Conference and Roundtable, San Francisco, 24 September 2002.

158. Alternatives include: Acrylonitrile-Butadiene-Styrene / Polycarbonate blends with triphenyl phosphate, Polystyrene containing organic phosphorous, Polystyrene / polyphenylene blends with organic phosphorous compounds, Polycarbonate with organic phosphorous compounds; See Note 147.

159. See Note 147.

160. The company uses polyamides in plastic cover parts.

161. See Note 147.

162. Ibid.

163. Ibid.

164. Ibid.

165. Apple Computer, "Environmental Attributes in Current Apple Products," viewed at www.apple.com on 20 Jan. 2003.

166. Bette K. Fishbein, Inform Inc., *Waste in the Wireless World: The Challenge of Cell Phones*, May 2002.

167. IBM annual environmental report cited by the Silicon Valley Toxics Coalition, "Halogen Elimination," viewed at www.svtc.org on 20 Jan. 2003.

168. See Note 152.

169. Greg Clemons and Todd Brady, Intel Corporation, *Going BFR Free,* presented at the EFC9 Brominated Flame Retardants and Electronics Conference and Roundtable, San Francisco, 24 September 2002; Electronic Industries Alliance, *Assessing End of Life Electronics Through Design: A compendium of Design-for-Environment Efforts of EIA Members*, viewed at www.eia.org on 20 Jan. 2003.

170. Bette K. Fishbein, Inform Inc., *Waste in the Wireless World: The Challenge of Cell Phones*, May 2002; Steve Scheifers, Motorola, *Bromine Free Alternatives in Electronic Products.* presented at the EFC9 Brominated Flame Retardants and Electronics Conference and Roundtable, San Francisco, 24 September 2002.

171. NEC annual environmental report cited by the Silicon Valley Toxics Coalition, "Halogen Elimination," viewed at www.svtc.org on 20 Jan. 2003.

172. Matsushita Electric, *Environmental Data Files: Halogen Free Plastics*, viewed at www.matsushita.co.jp/environment on 20 Jan. 2003. The full extent of halogenated compound replacement is not clear from the report.

173. Electronic Industries Alliance, *Assessing End of Life Electronics Through Design: A compendium of Design-for-Environment Efforts of EIA Members*, viewed at www.eia.org on 20 Jan. 2003.

174. Sony Corporation, *Green Management Plan*, viewed at www.sony.com on 13 Jan. 2003.

175. The European Parliament and the European Council, "Directive 2002/96/EC of 27 January 2003 on waste electrical and electronic equipment," *The Official Journal of the European Union*, 13 February 2003.

176. Europe's Environmental News Service, "Flame Retardant Controls Confirmed," *Environment Daily* Issue 1388, 19 February 2003.

177. Environmental News Service, "EU Lawmakers Vote Broad Fire Retardant Ban," 6 September 2001.

178. European Chemicals Bureau, *European Union Risk Assessment: Diphenyl Ether, Pentabromo Derivative*, August 2000; The European Parliament, Report on the joint text approved by the Conciliation Committee for a European Parliament and Council directive amending for the 24th time Council Directive 76/769/EEC, viewed at europa.eu.int/comm/enterprise/chemicals/markrestr/ on 10 Feb 2003.

179. The European Parliament, Report on the joint text approved by the Conciliation Committee for a European Parliament and Council directive amending for the 24th time Council Directive 76/769/EEC, viewed at europa.eu.int/comm/enterprise/chemicals/markrestr/ on 10 Feb 2003.

180. Environmental News Service, "EU Lawmakers Vote Broad Fire Retardant Ban," 6 September 2001.

181. Greenpeace, "Ban on New Brominated Flame Retardant in the Netherlands Lets Everyone Breathe Easier." (Press Release) 30 Jan. 2003.

182. Ted Schettler et al, Greater Boston Physicians for Social Responsibility, *In Harm's Way: Toxic Threats to Child Development*, May 2000.

183. This table was put together by the Danish EPA: See Note 147. This table is by no means exhaustive. Many more alternate materials exist than are listed in the table, which only includes alternative materials where no alternative flame retardants are commercially available in that product, or where manufacturers are currently replacing BFRs with alternative materials.