

# **BODY OF EVIDENCE**

**New Science in the Debate Over Toxic Flame  
Retardants and Our Health**

**Yana Kucher  
Meghan Purvis**

**February 2004**

**U.S. PIRG Education Fund  
Environment California Research & Policy Center**

# Acknowledgments

---

© Environment California Research & Policy Center

The authors would like to acknowledge Travis Madsen of the State Public Interest Research Groups (PIRGs) and Alison Cassady of U.S. PIRG Education Fund for coordinating the research and distribution of this report, as well as their endless editorial support. Thanks also to Anna Aurilio and Teresa Olle for their guidance, editorial assistance and review. We also acknowledge the staff of Environment California Research & Policy Center for their work on a 2003 report entitled *Growing Threats: Toxic Flame Retardants and Children's Health*, which provided a solid foundation for this study.

In addition, we would like to thank those who provided technical and editorial review, including Dr. Linda Birnbaum of U.S. Environmental Protection Agency; Dr. Robert Hale of the Virginia Institute of Marine Science; Dr. Thomas McDonald of Cal/EPA's Office of Environmental and Health Hazard Assessment; Dr. Kim Hooper of the Department of Toxic Substances Control at Cal/EPA; Steve Gurney, M.S., of the Environmental Health Strategy Center; and Dr. Joel Tickner of the Lowell Center for Sustainable Production.

Environment California Research & Policy Center's Toxics and Environmental Health Program is grateful to the California Wellness Foundation, CS Fund, and individual contributors for their support.

The authors alone are responsible for any factual errors. The recommendations are those of Environment California Research & Policy Center. The views expressed in this report are those of the authors and do not necessarily reflect the views of our funders, those who provided editorial and technical review, or their employers.

Environment California Research & Policy Center, the new home of 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 additional copies of this report, send a check for \$25 made payable to:

Environment California Research & Policy Center  
369 Broadway #200  
San Francisco, CA 94133  
415-622-0086  
info@environmentcalifornia.org

For more information about Environment California, visit our Web site at <http://www.environmentcalifornia.org>.

# Table of Contents

---

EXECUTIVE SUMMARY .....	1
INTRODUCTION.....	3
TOXIC FLAME RETARDANTS THREATEN CHILDREN'S HEALTH.....	5
TOXIC FLAME RETARDANTS ARE ROUTINELY ADDED TO CONSUMER PRODUCTS.....	6
RISING LEVELS OF PBDES IN PEOPLE LEAVE LITTLE MARGIN OF SAFETY .....	7
TOXIC FLAME RETARDANTS THREATEN CHILDREN'S HEALTH .....	8
HOW ARE TOXIC FLAME RETARDANTS GETTING INSIDE PEOPLE? .....	9
CALIFORNIA AND THE EUROPEAN UNION HAVE BANNED CERTAIN FLAME RETARDANTS.....	13
THE DANGERS OF DECA: A WOLF IN SHEEP'S CLOTHING .....	14
DECA IS THE MOST WIDELY USED PBDE .....	14
DECA BREAKS DOWN INTO MORE TOXIC FORMS.....	15
DECA ITSELF BUILDS UP IN THE BODY .....	18
DECA ITSELF MAY DISRUPT NEUROLOGICAL DEVELOPMENT .....	20
DECA AND ITS BREAKDOWN PRODUCTS MAY HAVE ADDITIONAL HEALTH EFFECTS .....	21
DECA: NOT NECESSARY FOR FIRE SAFETY.....	22
REPLACING DECA .....	22
COMPANIES REPLACING DECA WITH VIABLE ALTERNATIVES.....	23
WHY WE FACE THIS CRISIS: FAILURE OF U.S. AND STATE TOXICS POLICY .....	24
TOXIC CHEMICALS MISSED BY REGULATION.....	24
CURRENT LAW LEAVES EPA WITH LITTLE POWER TO PROTECT PUBLIC HEALTH.....	25
SWIFT GOVERNMENT ACTION CAN PREVENT FUTURE HARM .....	25
POLICY FINDINGS.....	27
PHASE OUT TOXIC FLAME RETARDANTS .....	27
REFORM CHEMICAL REGULATION.....	28
APPENDIX A: MAJOR MANUFACTURERS OF TOXIC FLAME RETARDANTS IN THE U.S.....	29
APPENDIX B: FACILITIES THAT PRODUCE, PROCESS OR USE IN THE U.S. ....	30
END NOTES.....	31

# Executive Summary

---

New evidence indicates that the chemical flame retardant decabromodiphenyl ether (Deca) may threaten the health of Americans.

Manufacturers of common household products add Deca to plastics or fabrics to make them resist the spread of fire. A growing body of evidence shows that exposure to Deca may cause adverse health effects in developing children, including damage to the nervous system and impaired motor skills. New research also indicates Deca can break down into the types of flame retardants recently banned in the European Union and California because of their bio-accumulative and toxic properties.

Unfortunately, the story of Deca is not unique. Deca is one of many potentially hazardous chemicals that are in widespread use, due to a failed national policy that presumes chemicals are safe until proven beyond a doubt to cause harm.

## **Toxic flame retardants are commonly added to household products.**

Deca is the most heavily used member of a class of flame retardants known as polybrominated diphenyl ethers, or PBDEs. There are three main types of commercially used PBDEs: Penta, Octa, and Deca. Deca is added to products used in the home, in travel, and in the workplace, including televisions, stereos, computers, hair dryers, toasters, draperies, and upholstery fabrics. These materials contain as much as 5-30 percent Deca by weight. In 2001 alone, North American industry used 49 million pounds of Deca, accounting for almost half the world market.

## **The European Union and California banned Penta and Octa flame retardants because they pose a threat to human health.**

The European Union has developed a policy banning the use of all PBDEs (Penta, Octa, and Deca) in consumer electronics beginning in mid-2006 and banning the marketing and use of the

Penta and Octa products in all sectors beginning in mid-2004. In 2003, the state of California followed suit, banning use and distribution of Penta and Octa. A few months later, the largest U.S. manufacturer of these two chemicals announced a national phase-out of their production.

Numerous laboratory studies point to potential health effects from exposure to Penta and Octa flame retardants:

- Infant mice exposed to these toxic flame retardants suffer disrupted brain development, permanently impairing learning and movement.
- Components of Penta and Octa are rapidly building up inside people. American women's breast milk and breast tissue contain some of the highest levels of PBDEs found in any population in the world.
- Human contamination levels leave little margin of safety. PBDEs found in some mothers and fetuses are rapidly approaching the levels shown to impair learning and behavior in lab testing.

## **Contrary to industry claims, Deca also poses a threat to human health.**

Deca escapes into the environment because it is not chemically bound to products to which it is added. Within the home, Deca has been found in household dust and as a film coating the surfaces of windows. It also escapes from products in landfills to spread through air and water.

- *Deca decomposes into forms that are more toxic and more easily absorbed by the body.* Although Deca itself is less easily absorbed by the body than other PBDEs, lab experiments have demonstrated that Deca can break down and convert to more dangerous forms, including the Penta and Octa scientists have

found rapidly accumulating in our bodies. New evidence also indicates that Deca decomposes in sunlight and ultraviolet light and within the bodies of animals.

- *Deca itself has been found in animals and humans.* The chemical industry has asserted that the Deca molecule is too large to be efficiently taken up by organisms. However, Deca has been found in peregrine falcons, in workers at electronics recycling plants, in regular citizens in the U.K., and in the breast milk of mothers in the United States. One recent study of American women's breast milk found levels of Deca in 16 of 20 women tested. A study from the University of Texas found a maximum level of Deca 40 times higher than industry's estimated maximum body burden for women who disassemble Deca-containing computers for a living.
- *Deca itself may be neurotoxic.* Recent research also has revealed that Deca exhibits some of the same toxic properties as Penta and Octa. When infant lab animals are exposed to Deca during a key period of development, they develop permanent damage to their nervous systems, resulting in impaired motor skills. This damage worsens with age.

**Safer means of fire-proofing products are widely available.**

Leaders in the furniture, plastic, and electronics industries already have manufactured products that meet fire-safety standards without the use of Deca. Strategies for flame-resistance include using better product design, inherently non-flammable materials, or alternative flame-retardant chemicals. For example, Ericsson, which manufactures cell phones and other electronics, has banned Deca and other PBDEs from its products and applications and found replacements at

comparable cost.

**U.S. chemicals policy compromises public health.**

In the U.S. alone, tens of thousands of industrial chemicals are on the market with little or no information about potential health impacts. Where significant evidence of harm to public health exists, inadequate resources and legal authority prevent regulatory agencies from taking protective action.

**Recommendations**

*Phase Out Toxic Flame Retardants*

Despite remaining data gaps about the hazards of Deca, the U.S. and California should take action based on current evidence. Given the scientific studies showing that Deca accumulates in humans, breaks down into more hazardous chemicals, and potentially harms brain development, the United States and California should phase out the use of Deca and other brominated flame retardants—especially given the availability of viable alternatives.

*Reform Chemicals Policy*

U.S. and California chemicals policy should 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. Chemicals that are untested or known to be hazardous should not be allowed on the market or in widespread use and distribution. In addition, the costs of developing analytical methods and testing for chemicals' safety should fall to the manufacturers who stand to profit from the product. In the absence of adequate data, the U.S. and California must take measures to prevent exposure to chemicals when there is evidence of potential harm.

# Introduction

---

Despite advances in modern medicine, many chronic diseases are on the rise. Recently, the National Cancer Institute found that cancer rates have increased over the past decade, despite past reports of declining or unchanging rates.<sup>1</sup> Cognitive development experts report that learning disabilities have risen 191% between 1977 and 1994, while the California Department of Developmental Services reports an apparent 210% increase in rates of autism over the last decade.<sup>2</sup> Additionally, it is reported that one in eight children is born prematurely; nationwide, the rate of premature births appears to have jumped 29 percent since 1981.<sup>3</sup>

The apparent increase in chronic illnesses in the country coincides with an explosion of industrial chemical synthesis and use. Modern industry has created more than 75,000 chemicals, used in manufacturing processes and incorporated in everyday consumer products. The modern human lives in an environment filled with complex mixtures of these chemicals, affecting human health and development in untold ways. Most of these chemicals have not been studied sufficiently to prove lack of harm. The health effects of almost half of the major industrial chemicals have not been studied at all.<sup>4</sup>

While it is virtually impossible to connect a single chemical to a broad health trend, the National Academy of Sciences estimates that chemical exposures play a role in at least 1 in 4 cases of developmental disorders.<sup>5</sup>

How did we get into this situation? First, current policy does not require that chemicals already on the market be tested sufficiently for health effects, particularly chronic effects, nor does it require any action if harm is found. American chemical regulation apparently takes an “innocent until proven guilty” approach, allowing widespread exposure to toxic chemicals before they have been

tested for safety. The burden of proving harm remains on those who suffer the harm—the public. Second, the government probably needs to do more to regulate the use and release of toxic chemicals and to identify new ones. According to the Toxic Release Inventory, industry discharges tens of millions of pounds of chemicals into California’s environment every year. Additionally, manufacturers include millions of pounds of these industrial chemicals in consumer products, from computers to shower curtains to light bulbs.

Meanwhile, an ongoing chemical industry campaign understandably attempts to convince the public that chemicals are safe and are tested for safety before being placed on the market.<sup>6</sup>

The case of PBDEs illustrates the shortcomings of federal and state chemical regulatory policies. Introduced as a fire safety product without adequate health testing, the toxic flame retardants known as polybrominated diphenyl ethers (PBDEs) have now spread to every corner of the globe and are building up in human bodies. New research in lab animals links exposure to these chemicals during a critical window of brain development to neurological impairment and reproductive damage. The European Union and California have imposed new restrictions on the marketing and use of these chemicals, and one U.S. manufacturer has pledged to phase out two types of toxic flame retardants by 2005.

However, the toxic flame retardant story is not over. Industry continues to assert that the third and most heavily used type of PBDE, decabromodiphenyl ether (Deca), is safe. This report catalogs the emerging evidence that this third chemical may pose a threat to children’s health.

There is a lesson to be learned here. It seems unwise that chemicals are “presumed innocent”

and used widely when there is inadequate study of their health effects, persistence, or bio-accumulative qualities.

Alternative models of chemicals policies do exist. Several Scandinavian countries have based chemical regulation on prevention, requiring thorough testing. The European Union recently introduced a draft policy known as REACH (Registration, Evaluation, and Authorization of

schools, and our homes. Investigating potential hazards and taking action to protect health when threats are discovered can lead to a world that is both safe and healthy for our children.

Kim Hooper, PhD  
Hazardous Materials Laboratory  
Department of Toxic Substances Control  
Cal/EPA<sup>a</sup>

Chemicals) that will require safety testing of thousands of chemicals that are already on the market.

We must do more to remove unnecessary health risks from our workplaces, our communities, our

---

<sup>a</sup> Affiliation included for identification purposes only. Content and opinion do not necessarily reflect the views of the California Environmental Protection Agency.

# Toxic Flame Retardants Threaten Children's Health

---

In the modern era, dangerous chemicals have regularly been produced and widely distributed before scientists could discover hazards to human health and the environment. The problem arises from inadequate testing and regulation of industrial chemicals. Dozens of examples exist, from DDT, a pesticide that nearly caused the extinction of bald eagles, to PCBs, an industrial insulating chemical that caused developmental problems in exposed humans. Both of these chemicals have become global contaminants, persisting in our environment despite being banned in the United States. Both can still be found in our bodies to this day.

Over the last five years, scientists have uncovered yet another emerging threat to human health. The central figure in this new story is a group of chemicals known as polybrominated diphenyl ethers (PBDEs), or toxic flame retardants. Widely used in foams, fabrics, and plastics to delay the spread of fire, these chemicals can now be found practically everywhere scientists look.

Despite the claims of the chemical industry, evidence continues to accumulate that PBDEs threaten human health:

- Industry stated that flame retardants would not escape from treated products into the environment. Scientists have found them in rapidly increasing amounts in all parts of the world, from the blubber of harbor seals and polar bears to the blood and breast milk of humans. In particular, women's breast milk and breast tissue in America contain some of the highest levels of PBDEs found anywhere.

- Industry assured the public that the chemicals were non-toxic, yet scientific studies have shown that exposure to toxic flame retardants during critical windows of development can interrupt brain development in mice, permanently impairing learning and movement. Toxic flame retardants also have been linked to disruption of thyroid function, cancer, immune system harm, and reproductive system damage.
- Contamination levels in humans have grown rapidly to the point where little margin of safety exists. Flame retardants found in some American mothers and fetuses are approaching the levels shown to impair learning and behavior in laboratory studies. Some subset of the population likely already carries PBDEs at levels that could be harmful to fetal development.

PBDEs are persistent, bio-accumulative, and harmful. They persist for long periods of time both in the environment and in our bodies and travel through a variety of media, including air and water. They bio-accumulate, meaning they find their way into the bodies of humans. These chemicals are also toxic and may be harmful to human and ecological health. Chemicals that demonstrate these properties, known as persistent bio-accumulative toxicants (PBTs) or persistent organic pollutants (POPs), can leave a toxic legacy for generations to come.

## Toxic Flame Retardants are Routinely Added to Consumer Products

Table 1: Common Uses of Toxic Flame Retardants

Type	Added to:	Found in (partial listing):	Quantity Used in N. America in 2001
<b>Deca</b>	High-impact plastics and textiles	Casings for electronic equipment, small electrical parts, fabric backings and coatings, rubber cables, paints	49 million pounds
<b>Octa</b>	Plastics	Casings for electronic equipment and small electronic parts in office equipment	3 million pounds
<b>Penta</b>	Polyurethane foam and other materials	Primarily used in polyurethane foam in furniture and mattresses. Also used in small quantity in carpet padding, packaging, fabric backings and coatings, imitation wood, paints, sound-insulating panels, plastic electrical parts	14.2 million pounds

SOURCE: Janet Raloff, "New PCBs?," *Science News*, 25 October 2003; Bromine Science and Environmental Forum (BSEF), "Major Brominated Flame Retardants Volume Estimates," available at [www.bsef-site.com](http://www.bsef-site.com), 21 January 2003.

Manufacturers of common household products routinely add PBDEs to plastics, fabrics, and foam in order to delay the spread of fire and improve product safety.

The chemical industry produces and sells three different mixtures of PBDEs: Deca, Octa, and Penta. Each product contains a mixture of molecules with different numbers of bromines attached. The Penta mixture is mostly made up of the lower brominated molecules – molecules with between four and six bromine atoms attached. Octa has mostly six to eight bromine atoms attached, and Deca is almost exclusively made up of the molecules with ten bromines.<sup>b</sup>

In 2001, more than 66 million pounds of PBDEs were used in North America.<sup>7</sup> Table 1 lists common types of materials and products that can contain these flame retardants.

Although flame resistant products certainly save lives and help avoid injury from fire, PBDEs may have other severe health consequences.

<sup>b</sup> For clarity, the capitalized words "Deca," "Octa," or "Penta" refer to the 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. In this report, we use "toxic flame retardants" to refer to the PBDE class of chemicals. In addition, there are at least 35 other types of potentially toxic chemicals containing bromine used in fire-safety applications, as well as other halogenated chemicals (those that contain chlorine, fluorine, or iodine) that also could be dangerous to human health and the environment.

## Rising Levels of PBDEs in People Leave Little Margin of Safety

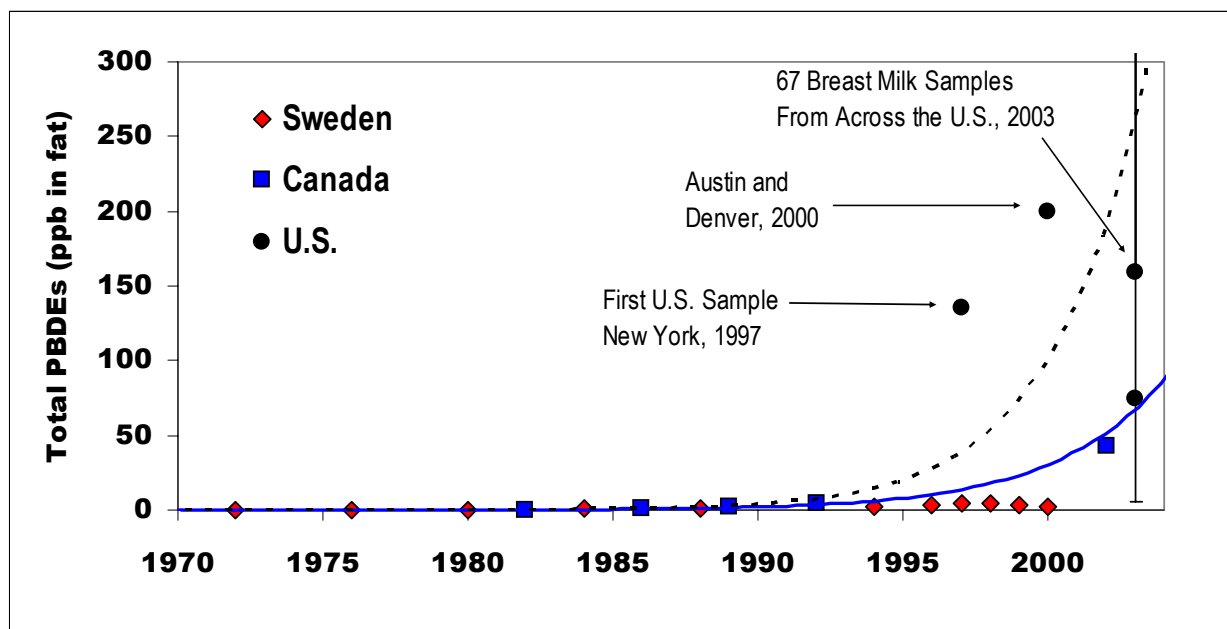
In the last few years, scientists have discovered that PBDEs are rapidly building up in our bodies. Recent studies in the United States have found the highest human contamination levels yet recorded. Contamination levels in the breast tissue of California women and in the breast milk of women throughout America are up to 75 times higher than those found in European countries (Figure A).<sup>8</sup>

Levels found in some mothers and fetuses are rapidly approaching the levels shown to impair learning and behavior in laboratory experiments.<sup>9,10,11</sup> Studies of maternal and umbilical cord blood show that levels of PBDEs are the same, meaning

that newborns may be exposed *in utero* to levels approaching those found to cause damage.<sup>12</sup>

In addition, Canadian studies have found PBDE levels in humans doubling every 2.5 years.<sup>13</sup> Therefore, there may already be segments of the U.S. population with body burdens of PBDEs that in laboratory testing cause developmental damage. Given the increasing levels of PBDEs in humans, we can safely assume that within three years, many more segments of the population will have PBDEs in their blood at levels that could cause developmental damage.

**Figure A:** This chart shows the trend in average levels of PBDEs in breast milk from Sweden, Canada, and the United States. A 2003 study recorded average PBDE levels of 159 parts per billion in fat, although two participants exceeded 700 parts per billion, the highest levels yet recorded in humans.



SOURCE: Data up to 2003: Mehran Alaei, Environment Canada, "Rapidly Rising PBDE Levels in North America," *Environmental Science and Technology*, Science News, 7 December 2001. The lower point in 2003: A. Schechter, et al., "Congener Specific Measurement of Polybrominated Diphenyl Ethers in 47 Individual Milk Samples From Nursing Mothers in the U.S.A.," *Organohalogen Compounds* 61, 13-16, 2003. The upper point in 2003: Lunder, Sonya and Renee Sharp, Environmental Working Group, *Mothers' Milk: Record levels of toxic fire retardants found in American mothers' breast milk*, September 2003; this study showed a range of levels from 9.5 parts per billion (ppb) in fat to 1,078 ppb in fat.

## Toxic Flame Retardants Threaten Children's Health

---

Exposure to toxic PBDEs may be particularly harmful during a critical window of brain development in utero and early childhood. Recent research shows that these chemicals can interrupt brain development in mice, permanently impairing learning and behavior patterns, and may cause reproductive system damage. In addition, there is limited evidence that PBDEs may cause cancer and harm the immune system. Unlike some toxic chemicals, PBDEs have low acute toxicity, meaning that short-term exposure has not been shown to cause effects such as rashes or vomiting. However, subtle damage to the developing brain may have permanent and devastating effects.

### **Damage to Neurological Development Affecting Learning and Memory**

When infant mice are exposed to PBDEs during a key window of their growth, they develop irreversible deficits in memory and learning. These effects worsen as the animals grow older.<sup>14</sup>

Scientists suggest that these neuro-developmental effects could be a result of disruption of the thyroid hormone system. The thyroid hormone system is instrumental in normal brain development. Exposure to certain chemicals at an early age can disrupt thyroid levels, inhibiting normal brain development and impairing the intelligence and behavior of children.<sup>15,16</sup>

PBDE exposure produces lowered thyroid hormone levels and physical changes in the thyroid gland of mice when exposed to Penta at a single dose as low as 0.8 milligrams per kilogram of body weight.<sup>17,18</sup> These effects on thyroid hormone levels appear to be additive with the effects of related environmental contaminants known as polychlorinated biphenyls (PCBs) and dioxins.<sup>19</sup> This means that various chemicals could be working together in the body to produce greater effects. PBDEs also may affect nerve impulse transmission

and disrupt communication systems inside cells, which could prevent the cell from functioning properly.<sup>20</sup>

### **Reproductive System Damage**

Studies presented for the first time in 2003 point to yet another potential health consequence of PBDE exposure: irreparable damage to developing reproductive systems. These studies show that PBDE exposure can delay onset of puberty in both males and females and impair development of reproductive organs in laboratory animals.<sup>21</sup>

One study found that pregnant rats exposed to a single dose of Penta produced offspring with structural changes in their ovaries.<sup>22</sup> Another study showed that adult male rats exposed to a single low dose of Penta while in the womb had significantly decreased sperm counts.<sup>23</sup>

### **Possible Links to Cancer**

The U.S. National Toxicology Program found that high levels of Deca exposure created tumors in the liver, thyroid and pancreas in laboratory animals.<sup>24</sup> Penta and Octa have not been tested for carcinogenicity. One study suggests a positive association between the risk of Non-Hodgkin's lymphoma and tissue levels of tetra BDE, yet another type of PBDE, in humans.<sup>25</sup>

### **Immune System Impairment**

Conflicting research studies present an unclear picture of the potential effects of PBDEs on immune systems. Limited studies to date suggest that the Penta BDE product may impair the immune response in exposed rodents.<sup>26</sup> Contamination of commercial Penta with brominated dioxins and furans could explain this result,<sup>27</sup> as dioxins and furans have been linked to immune system impairment. Similar effects have been seen with PCBs. However, other scientists have found a lack of immune system effects from Penta exposure.<sup>28</sup>

## How are Toxic Flame Retardants Getting Inside People?

---

Flame retardants are used in common products, such as couches and computers, which are found in the home or office and are often disposed of in landfills or incinerators. Scientists need to conduct more research into exact routes of human exposure. However, there is evidence that flame retardants can escape from products into the home or work environment or enter the food chain after disposal, ultimately ending up inside our bodies.

### **Toxic Flame Retardants Escape from Products into Air and Water**

Because PBDEs are not chemically bound to the materials in which they are used, they can escape into the environment. Because the chemicals have extremely low solubility in water, they likely move around attached to dust particles, which can be suspended and transported in air or water. The discovery of PBDEs in the Arctic food chain, far from any site of manufacture or use of flame retardants, shows that these PBDE-containing dust particles travel to every corner of the globe, similar to PCBs and DDT.

More highly brominated PBDEs like those found in the Deca and Octa products may evaporate from the casings of computers and televisions, especially when the product heats up from use. This has been evidenced by high levels of Deca found in dust samples from office buildings and in the film on the surfaces of household windows.<sup>29</sup>

Toxic flame retardants also are released during manufacturing, as evidenced by the heavy contamination of the River Tees in the U.K., downstream from a Great Lakes Chemical Company factory that produces the flame retardants.<sup>30</sup>

A Norwegian study recently confirmed that PBDEs escape from discarded products and seep out of landfills into the environment.<sup>31</sup> Plastic products in landfills containing Octa and Deca may release these chemicals through decomposition, especially

when exposed to sunlight.

High levels of PBDEs have been found in water coming out of wastewater treatment plants, and PBDEs are also widespread in sewage sludge, which can contaminate water and the food supply.<sup>32,33</sup> Scientists tested sewage sludge from Texas, California, New York, Virginia, and Maryland, finding concentrations of Deca at levels ranging from 84.8 parts per billion to 4,890 parts per billion—the highest levels ever recorded.<sup>34</sup> This sewage sludge was to become fertilizer for farmland.

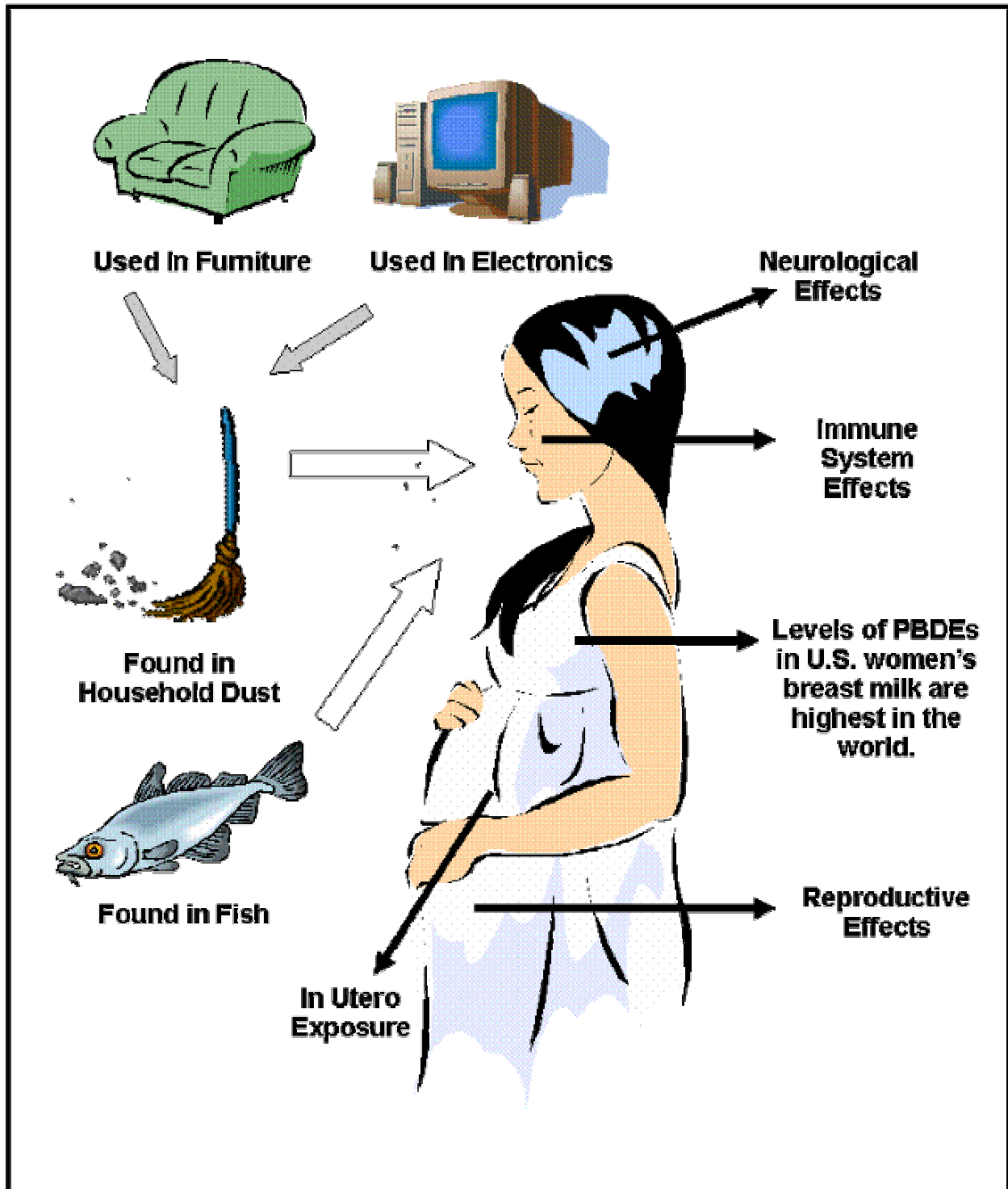
### **Toxic Flame Retardants Enter People through Inhaled Dust and Through Food**

Once PBDEs enter the environment, some inevitably find their way into our bodies, where they accumulate in fatty tissues. As the chemicals escape from products, they attach themselves to household dust that can be inhaled or ingested. Other potential routes of exposure include contamination of the food supply or absorption through the skin from furniture, products, or other surfaces.

#### *Contaminated Indoor and Outdoor Air*

PBDEs are found at low levels in both outdoor and indoor air. The air above Chicago contains PBDEs, including Deca, at levels 5-10 times higher than rural locations in the Great Lakes area.<sup>35</sup> Workers can be exposed to Deca and other PBDEs via inhalation of contaminated air in workplaces. Many types of PBDEs have been found in household air in rooms with electronics and in workplace air in electronics disassembly plants.<sup>36</sup>

Figure B: Routes of Exposure to PBDEs and Health Effects Found in Laboratory Tests



### *Inhalation or Ingestion of Household Dust*

High concentrations of PBDEs in household dust suggest that inhalation, ingestion, or skin contact may be a significant form of human exposure to some PBDEs in the home and workplace.

In 2003, Greenpeace published a study that looked at a variety of chemicals in household dust in European countries. Deca was the most widely found brominated flame retardant, at levels from 3.8 to 19.9 parts per million (ppm), levels significantly higher than in a 2001 study in Parliament buildings. In household samples from Finland and Denmark, where Deca is being phased out, the levels were between 10 and 100 times lower than those levels found in the U.K.<sup>37</sup>

A 2003 study of indoor air and house dust samples from 120 homes in Cape Cod found many different types of chemicals that are used in products such as plastics, detergents, furniture, electronics, pesticides, and cosmetics. Although the authors of the study did not look for Deca, they did find other types of PBDEs, suggesting household dust as a possible method of human exposure.<sup>38</sup>

Deca also has been found in high levels in the film that builds up on the inside and outside surfaces of household windows, in both rural and urban homes. Levels were significantly higher in the urban locations, and were higher on the inside surfaces of the windows.<sup>39</sup>

### *Contaminated Food Supply*

Food may be a major source of PBDE exposure. PBDEs have been found in a wide range of foods, especially those containing animal fat such as dairy products. As with other bio-accumulative toxics and metals, limited studies have shown that PBDE concentrations in people tend to rise as they consume more fish.<sup>40</sup>

### **Exposure in the Womb**

Women accumulate PBDEs in their bodies throughout their lifetime. During pregnancy, a mother's blood transports nutrients to her embryo through transfer across the placenta.<sup>41</sup> A number of bio-accumulative compounds like PBDEs have been shown to move freely across the placenta into the infant's bloodstream, as though the placenta were transparent to the chemicals.<sup>42</sup>

Because a number of PBDEs easily cross the placenta, fetuses may be exposed to these toxic chemicals during vulnerable periods of brain development.

Several studies indicate that PBDEs build up in a developing fetus to the same levels found in the mother. Analysis of samples from eleven Finnish women showed similar PBDE concentrations in breast milk and the placenta. A more recent study of mothers from Indiana showed that the levels of PBDEs in the mother and the fetus are practically equal, with less than a two percent difference.<sup>43</sup>

### **Contamination in Mothers' Milk**

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 milk. As a mother's body calls on its reserves of fat, the pollutants in these tissues are mobilized along with the fat and go into the mother's milk, where the infant subsequently consumes them. Despite these dangers, studies that look at the adverse effects of breastfeeding due to chemical exposure conclude that exposure through breast feeding is often less of a threat than

*in utero* exposures (See Box).

One 1996 study by Wayne State University tracked 11-year-old children that had been born to mothers who had eaten PCB-contaminated fish during pregnancy, and found that they experienced neurological delays, lower IQ scores, poorer reading comprehension, and memory and attention issues.<sup>44</sup> However, the study also found that exposure from breastfeeding "did not seem to cause any further harm to the children's mental abilities."<sup>45</sup>

### **- Benefits of Breastfeeding -**

*"To breastfeed or not to breastfeed is not the question...*

*The message needs to be that we need to move immediately to divorce our economy from any toxic chemical known to accumulate in mothers' milk." – Sandra Steingraber, PhD.<sup>46</sup>*

Breast milk is by far the healthiest food for an infant and provides irreplaceable nutrients that aid in proper development. The American Academy of Pediatrics strongly recommends breastfeeding despite potential exposure to toxic chemicals, as breast milk helps protect infants against certain diseases and infections. In addition, several studies point to the improvement of cognitive development in children who breastfeed.<sup>47</sup>

Formula may not be free of toxic chemicals. Formula can be contaminated with chemicals such as lead or pesticides.<sup>48</sup> Furthermore, formula mixed with water that contains contaminants could cause diarrhea and other illness.<sup>49</sup> Finally, formula is often fed to infants with plastic bottles, which may contain other dangerous chemicals that leach from the bottle, such as the endocrine-disruptor

bisphenol-A.<sup>50</sup>

In addition to providing incomparable benefits to an infant's health, breastfeeding also benefits the health of the nursing mother, helps to protect the environment, reduces national health care costs, and may save the family approximately \$400 in the first year of life in formula costs.<sup>51</sup>

For all of these reasons, breastfeeding should be encouraged as the best way for mothers to feed their children. Instead of reacting to information about dangerous chemicals in breast milk by stopping breastfeeding, individuals should join with organizations and efforts that work to eliminate the use of toxic chemicals, thereby ensuring the safety of both breast milk and children.

## California and the European Union Have Banned Certain Flame Retardants

---

In the past year, the European Union and the state of California have taken significant action to protect the public from toxic flame retardants.

In September of 2001, the European Union developed a directive on managing waste from electrical and electronic equipment. The directive, adopted in 2003, bans the use of all PBDEs in consumer electronics beginning in mid-2006.<sup>52</sup>

Additionally, the European Union has issued a separate rule that fully bans the marketing and use of Penta and Octa in all sectors beginning in mid-2004.<sup>53</sup> Deca may be added to this comprehensive ban, depending on the results of a risk reduction strategy currently being assembled by the European Chemicals Bureau.<sup>54</sup> The European Union released the first section of its Draft Risk Assessment Report, analyzing the environmental impacts of Deca, in late 2003. Although this segment reaches no conclusion as to the human toxicity of Deca, it does state that “there is a need for further information and/or testing.”<sup>55</sup> There are two more steps in this process, however, expected in March 2004 and May 2004.

The European Union acted on initial signs of a threat to human health and the environment despite incomplete toxicology data for the chemicals. When the EU completed a risk assessment of Penta in August of 2000, member states noted uncertainties surrounding the risk for infants exposed through breast milk.<sup>56</sup> Instead of waiting for years of scientific studies to resolve those uncertainties, member states voted to take risk reduction measures without delay. The European Parliament insisted that Octa and Deca be regulated alongside Penta, rather than wait for study while exposures increased exponentially.<sup>57</sup>

The state of California followed suit in the summer of 2003, when it acted to ban the use and

manufacture of Penta and Octa beginning in 2008.

In addition, the Environmental Protection Agency (EPA) reached an agreement with Great Lakes Chemical Manufacturers to cease production of Penta and Octa by 2005, following Great Lakes’ development of a substitute chemical.<sup>58</sup> It is unclear whether this new chemical, “Firemaster 550,” will prove to be safe for human health, although Great Lakes claims EPA has provided a “favorable environmental assessment.”<sup>59</sup> EPA has stated that this chemical will not present the same problems as Penta and Octa, and does not plan to redo its initial analysis of Firemaster 550, which assumed a much lower use of this chemical.<sup>60</sup> EPA will not issue a recall for products that currently contain Penta and Octa. Because Great Lakes is not the sole manufacturer of Octa, and Penta may still be imported, this phaseout may not have the widespread effect that some claim it will.

Unfortunately, regulatory action has been halted on Deca, the third type of PBDE. At the time of the debates in California and Europe on banning flame retardants, scientists had produced few studies about Deca’s breakdown, absorption, and toxicity. This allowed the chemical industry to portray Deca as safe, arguing that the large size of the molecule makes it difficult for the body to absorb.<sup>61</sup>

Growing concern over toxic flame retardants, however, has prompted a flurry of new studies. While the chemical industry is working to ensure that Deca remains on the market, mounting evidence indicates that Deca in fact may not be safe. New data indicates that Deca is absorbed by the body, can be broken down to less brominated PBDEs, and can cause neurodevelopmental effects similar in nature to those caused by prototypical PBDEs. In other words, in several significant ways, Deca may not be so different from the other PBDEs.

# The Dangers of Deca: A Wolf in Sheep's Clothing

---

Despite the attention focused on PBDEs and Europe and California's regulatory action on Penta and Octa, Deca continues to be manufactured and incorporated in a variety of common household products and plastics.

At first, industry scientists claimed that all flame retardants were safe to use, and that the benefits of fire safety outweighed any possible risks. Slowly industry scientists have come to acknowledge some of the potential risks from certain flame retardant chemicals. Nevertheless, they currently assert that the dangers of PBDEs to health and the

environment are limited to the Penta and Octa formulations, that Deca does not share these dangerous properties, and is therefore safe to use.<sup>62</sup>

However, new research is beginning to indicate otherwise. Deca has been shown to break down into more toxic and more bio-accumulative chemical forms that make up the two banned PBDE products. Deca has been linked to neurological damage in mice, and has been found in the bodies of animals and the blood and breast milk of European and American women.<sup>63, 64, 65, 66</sup>

## Deca is the Most Widely Used PBDE

---

The chemical flame retardant Deca (decabromodiphenyl ether) amounts for approximately 83% of the PBDEs used worldwide.<sup>67</sup> North American industry alone used approximately 49 million pounds of Deca in 2001, almost half the world market. European use of Deca has recently increased after the phaseout of Penta and Octa.

Deca is added to many products used in the home, in travel, and in the workplace, including televisions, stereos, cassettes, computers, hair dryers, toasters, draperies, and upholstery fabrics used in office furniture and seats in cars, buses, trains, and airplanes (Table 2). These materials contain as much as 5-30% Deca by weight.

Table 2: Common uses of Deca flame retardant

Types of Products	Examples	Proportion of Deca Usage
Electrical & electronic equipment	CPU housings, wire & cable, TV cabinets	~80%
Upholstery textiles	Auto upholstery, Upholstered furniture	~20%

SOURCE: American Chemistry Council's Brominated Flame Retardant Industry Panel for the Voluntary Children's Chemical Evaluation Program (VCCEP), *Report of the Peer Consultation Meeting On Decabromodiphenyl Ether*, Organized by Toxicology Excellence for Risk Assessment, September 2003.

## Deca Breaks Down into More Toxic Forms

An industry-supported panel presenting at the 2003 Dioxin Conference in Boston asserted that “DBDPO [Deca], because of its large molecular size, low oral bioavailability, and rapid elimination, is expected to partition only minimally into breast milk....”<sup>68</sup> Until about a year ago, we had little evidence of Deca’s accumulation in the human body.

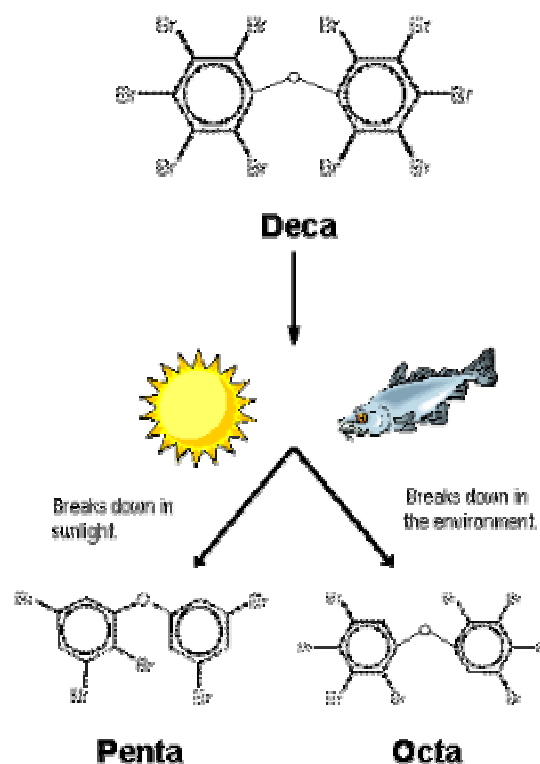
However, new studies have documented that the large molecules of Deca can break down to form smaller molecules. These breakdown products have fewer bromines attached and are therefore easier for the body to absorb. Recent evidence suggests that breakdown of Deca may even form molecules that make up the Penta and Octa mixtures, those banned in California and the European Union. Several studies have documented the breakdown process in laboratory settings and in the bodies of animals.

### Breakdown in the Bodies of Fish

Research by the University of Maryland's Center for Environmental Science provides persuasive evidence that fish exposed to Deca can metabolize it into the less fully brominated compounds associated with the Penta and Octa flame retardant formulations.

Scientist Heather Stapleton and her colleagues exposed juvenile carp to food spiked solely with pure Deca.<sup>c</sup> After 60 days, no detectable traces of Deca were observed in the fish. However, researchers identified concentrations of the less brominated molecules that make up the Penta and

**Figure 1:** Deca breaks down into components of Penta and Octa in sunlight as well as in the bodies of fish.



Octa formulations in the fish.<sup>d</sup> Concentrations of components of Penta continued to increase up to 10 days after the exposure ended. Additionally, levels of the higher brominated Octa immediately began to decrease in concentration as the exposure ended, indicating that the large molecules may have continued to break down. The results suggested that Deca was breaking down to form hepta and octa (which contain seven and eight bromine atoms, respectively), which in turn proceeded to break down to form penta and hexa (which have five and six bromine atoms and are components of the commercial product Penta).<sup>69</sup> Stapleton found levels of Penta components above the levels found in the food fed to the fish, showing that these components must have come from

<sup>c</sup> There are 209 different congeners of PBDEs. BDE 209 is the primary component of the commercial Deca formulation, making up more than 97% of the Deca product. Most research on health effects and bio-accumulation of PBDEs is congener-specific.

<sup>d</sup> Scientists found levels of penta-, hexa-, hepta-, and octa-BDEs in the exposed fish.

breakdown of Deca.<sup>70</sup> Although the human body can take up Deca in its original form, these breakdown molecules are smaller than the parent Deca and therefore more easily absorbed by the body.

This research confirmed earlier studies of rainbow trout that also showed Deca breaking down into smaller forms.<sup>71</sup> Deca also may break down into other substances that are much more toxic, including hydroxylated compounds (which are more water soluble) or fully debrominated diphenyl ethers (which are more easily absorbed).<sup>72</sup>

### **Breakdown in the Environment**

Another set of studies point to the breakdown of Deca as a possible source of the components of Penta and Octa in the environment. Chemists Mark La Guardia and Robert Hale at the Virginia Institute of Marine Science recently found high levels of flame retardants in the bodies of minnows and sunfish in a river that flows through other bodies of water into Lake Gaston, near the Virginia border.

They discovered that a wastewater treatment plant just below the border was releasing large quantities of Deca BDE into the adjacent stream. The stream contained 50 parts per billion of Deca, which may be the highest number ever reported in a body of water. Thus, wastewater treatment plants are another source of release of Deca into the environment, one that had been previously been ignored. High levels of Deca and smaller quantities of the other PBDEs were found in soil and sediments as far as 6.7 miles away from the treatment plant. Downstream from the plant, the bodies of fish also contained levels of Deca, showing that Deca is bio-available, or able to be taken up by the body.<sup>73</sup>

Importantly, the downstream fish also contained high concentrations of the lower brominated PBDEs that make up Penta and Octa, suggesting that Deca may have broken down into the more bioavailable and toxic forms. In the water discharged from the treatment plant, the level of Deca was roughly 12,000 ng/L, much higher than

the most common components of Penta at about 12 ng/L and 8 ng/L.<sup>e</sup> In other words, levels of Deca in the water were 1000 times higher than levels of Penta. The opposite was true for downstream fish; sunfish had levels of Deca close to 500 parts per billion (ppb), whereas concentrations of two Penta components were 6500 ppb and 7200 ppb.<sup>f</sup> Because the levels of Penta and Octa components in the water were very low, the findings suggest the high levels of Penta and Octa in the fish may result from the breakdown of Deca.

The scientists also found two forms of octa present in the fish that are not present in commercial PBDE mixtures, again supporting the idea that they are breakdown products of Deca. It is unclear whether this breakdown is happening due to UV light exposure, microbial activity, or another process.<sup>74</sup>

---

<sup>e</sup> The components of Penta most commonly found in living things are PBDE congeners 99 and 47.

<sup>f</sup> Fish tissue levels were 6500 ppb of BDE 47 and more than 7200 ppb of BDE 99, two major components of Penta, compared with under 500 ppb of BDE 209, the primary component of Deca.

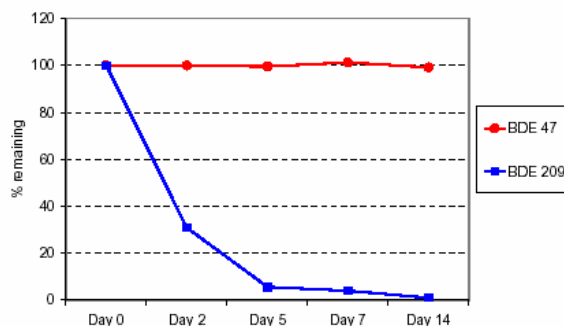
### Breakdown in Sunlight/Ultraviolet Light

For several years, scientists have been studying the breakdown of Deca when exposed to sunlight or ultraviolet light in an effort to explain the discrepancy between the large amount of Deca released into our environment and the relatively small amount present in living things.

Numerous studies have shown that when exposed to sunlight or ultraviolet light, Deca breaks down into the lighter, more toxic forms of PBDE, including some of those that make up the more bio-accumulative Penta and Octa formulations.<sup>75</sup> Deca and highly brominated PBDEs are often found on particulate matter in outdoor air and in indoor house and office dusts.<sup>76</sup> Deca-containing dusts and particles in outdoor air would be exposed to UV light and sunlight, providing ample opportunity to transform into the more bio-accumulative forms.

In laboratory conditions, Deca breaks down into the lighter forms so quickly that scientists have even had difficulty analyzing Deca samples and have resorted to keeping the samples in brown glass vials, rather than the usual clear, and even wrapping aluminum foil around the vials to prevent breakdown.<sup>77</sup> In one study, after five days in a clear glass vial exposed to daylight, only six percent of the original Deca sample remained, compared with 94 percent remaining when the sample was kept in a brown glass vial. After 14 days in the clear vial, only one percent of the original Deca remained. However, the quantity of a major component of Penta slightly increased in the sample over the same time period (Figure D).<sup>78</sup>

**Figure D:** Storage of PBDE solution in clear glass, daylight exposure over 14 days. BDE 209 is the main component of Deca (making up more than 97% of the product). BDE 47 is one of the main components of Penta, one of the forms most commonly found building up in the human body and in animals.



*SOURCE:* T. Herrmann, B. Schilling, and O. Papke, "Photolysis of PBDEs in Solvents by Exposure to Daylight in a Routine Laboratory," *Organohalogen Compounds* 63, 361-364, 2003

Other studies have shown a variety of half-lives for Deca, ranging from 15 minutes to 200 hours depending on the substance in which it is carried.<sup>79</sup>

Although these studies were conducted in a laboratory setting, scientists have noted Deca's breakdown in substances where Deca is likely to be found in the environment, such as sand, sediment, and soil.<sup>80</sup> The various studies agree that Deca can break down to form the lower brominated molecules – substances that are more easily taken up by the body and hence present a health concern.

Therefore, the use of Deca may be responsible for some of the contamination scientists observe in organisms across the planet.

## Deca Itself Builds Up in the Body

---

In addition to breaking down into more dangerous forms, new studies show that Deca shares some of the dangerous inherent properties of Penta and Octa. For years, due to the large size of the molecules, many believed that Deca could not be absorbed by the body and therefore had no measurable effects on wildlife or humans.<sup>81</sup> One of the main reasons that the California ban on two types of PBDEs (Penta and Octa) excluded Deca is that there was no evidence at the time that Deca bio-accumulates in the general populace.

However, groundbreaking new studies are rapidly disproving previous beliefs. Recent studies have shown that despite its larger and more cumbersome size, animals as diverse as peregrine falcons, laboratory rodents, fish, and humans can absorb Deca. Although the concentrations are typically much smaller than those of Penta and Octa, Deca has been found in human blood and breast milk, in the bodies of electronics workers as well as in those not occupationally exposed.

### Animal Evidence

The first evidence of Deca's absorption by living things was found in the eggs of peregrine falcons in Sweden. Peregrine falcons are near the top of the terrestrial food chain. In the past, they have been endangered due to high body concentrations of the now-banned pesticides DDT, dieldrin, and aldrin, as well as with mercury, all of which affect reproductive success and survival. This makes them an appropriate species to assess for presence of this emerging class of PBDE chemicals.

Scientists analyzed 21 eggs, representing females from northern Sweden, southwestern Sweden, and a captive breeding population. Deca was found in 18 of the 21 eggs analyzed (ranging from 28 ppb to as high as 430 ppb). Eggs from the wild populations had significantly higher Deca concentrations than the captive population, which were fed chickens. The study provides evidence

that Deca is present in the environment and may bio-accumulate.<sup>82</sup>

Scientists have since documented uptake by various species of fish, mice, and rats, all of which are capable of absorbing this chemical.<sup>83,84</sup>

### Human Evidence from Occupational Exposure

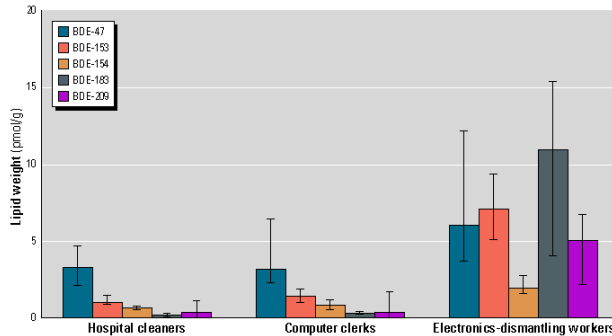
Recent studies show that Deca is far more bio-available than previously thought.<sup>85</sup> In the past several years, scientists have found the first measured occurrences of Deca in human blood and breast milk. At highest risk may be those exposed to the chemical in the workplace.

The chemical industry often argues that Deca is safe, citing low levels of human exposure as evidence.<sup>86</sup> Industry has created a model approximating a "reasonable estimate" (0.02 ppb, or parts per billion) and an "upper estimate" (0.2 ppb) for exposures of women who disassemble computers for a living, insisting that these levels are low enough to not cause concern.<sup>87</sup>

However, Swedish studies have found levels of Deca in the blood of workers at a Stockholm area electronics dismantling plant much higher than industry's estimates. Of 59 workers tested, 45 had detectable levels of Deca in their blood. Electronics dismantling workers had a median of 1.53 ppb, with a high of 6.81 ppb, levels much higher than those of computer clerks and hospital cleaners (Figure E).

Interestingly, electronics dismantling workers also had higher levels of components of Penta and Octa in their blood than the two other categories of workers. This discrepancy did not appear to be related to age or fish consumption.<sup>88</sup> Again, this may point to Deca in the work environment breaking down into the components of Penta and Octa.

**Figure E:** A comparison of blood levels of PBDEs among hospital cleaners, computer clerks, and electronics-dismantling workers. The primary component of Deca, BDE 209, is shown in the right-most column of each set.



*SOURCE:* Kristina Jakobsson, Kaj Thuresson, Lars Rylander, Andreas Sjödin, Lars Hagmar, Ake Bergman, “Exposure to Polybrominated Diphenyl Ethers and Tetrabromobisphenol A among Computer Technicians,” *Chemosphere*, 46(5), 709-716, February 2002.

### Human Evidence from General Exposure

Several years later, World Wildlife Fund tested the blood of United Kingdom residents who did not work in computer factories and found Deca in the blood of the general population, at levels similar to those observed in Swedish electronics workers and at much higher levels than industry’s estimated “worst case scenario.”<sup>89</sup>

Recent studies of American women’s breast milk have replicated those findings. Of 23 women tested for Deca in a Texas study in 2003, eight had detectable levels of Deca in their blood. The highest level found was 8.2 parts per billion (ppb) in lipid (fat), more than 40 times the level of industry’s worst case estimate for computer workers. The average level of the women tested was 0.92 ppb, almost five times higher than industry’s worst-case estimate.<sup>90</sup> Another U.S. study found Deca in the breast milk of 16 of 20 women, with an average of 0.24 ppb and a high of 1.2 ppb.<sup>91</sup>

Measuring PBDE levels in breast milk provides a useful indicator of PBDE exposure for the developing fetus. Levels of PBDEs measured in a woman’s breast milk are similar to levels in her infant’s umbilical cord blood. Finding Deca and related flame retardants in American women’s breast milk indicates that babies in the U.S. are being exposed to these chemicals in the womb, during their most vulnerable stages of development.

## Deca Itself May Disrupt Neurological Development

---

Deca appears to share some of the toxic qualities of other PBDEs and PCBs. Studies on laboratory mice have shown neurotoxic effects from exposure to Deca, and a few studies also have indicated that Deca may be carcinogenic. Because we know that Deca can be absorbed into women's blood and breast milk, one of the biggest concerns is the effect that exposure can have on a pregnant woman's developing fetus.

### **Prenatal Exposures May Cause the Most Harm**

Infants exposed to chemicals while in the womb are particularly vulnerable to their effects, since their bodies and brains are still at critical stages of development where the slightest disruption may cause permanent damage.

Human development happens in a series of stages, each of which is a critical window during which exposure to a developmental toxicant 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.<sup>92</sup> This period is known as the brain growth spurt, when specialized nervous system tissue develops under the influence of the thyroid hormone system.<sup>93</sup> Exposure to chemicals such as PBDEs that disrupt thyroid hormone balance during this period can permanently disrupt brain development. Such toxicants 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.

### **Neurological and Developmental Impairment from Exposure to Deca**

Preliminary study shows that exposure to Deca can impair neurological development, an effect similar to that of Penta, Octa, and PCBs.

The brain growth spurt occurs in mice during the first three weeks after birth. In a study of Deca's effects on development, newborn mice were

exposed to Deca on their third, tenth, and nineteenth postnatal day. In all three cases, Deca reached the brain within 24 hours, although for mice exposed on their nineteenth day, the uptake was much lower. The amount of Deca reaching the brain for those exposed on the third or tenth day doubled within the first week after exposure. This shows that Deca can be absorbed and can find its way to the brain during the critical brain growth spurt.<sup>94</sup>

Mice exposed to Deca on their third day of infancy showed neurological impairment that was permanent and worsened with age. Exposure on the tenth and nineteenth day did not result in the same effects. This could be either because the effects were from a breakdown product of Deca, or because of the amount of time it took for enough of the chemical to reach the brain. The studies show that Deca can remain active within the body for as long as a week, and that a *single dose* can interfere with the vital brain development that occurs at an early age. These effects follow a similar pattern to those caused by exposure to PCBs.<sup>95</sup> In addition, PBDEs can cooperate with PCBs in the body to produce greater effects.<sup>96</sup>

As mentioned earlier, Deca may break down to form some of the smaller, more bio-accumulative chemicals that make up the Penta and Octa formulations. These chemicals may have more potent effects on infant thyroid function than Deca would have directly and could lead to impaired intelligence and motor skills of children.<sup>97</sup>

To date, the effects of PBDEs on neural development are known only for mice. The results seen in mice are relatively crude compared to the subtle effects PBDE exposures could cause in humans – human brains could be more sensitive to PBDE exposure. In addition, certain populations, such as developing children or electronics workers, may be more vulnerable to its effects.

## Deca and Its Breakdown Products May Have Additional Health Effects

---

### Products of PBDEs' Transformation in the Body: Toxicity Unknown

In addition to Deca degrading to form lighter PBDE congeners, PBDEs can undergo other breakdown processes that may result in more hazardous forms. For example, the liver modifies PBDEs to hydroxy-PBDEs, which more closely resemble the thyroid hormone and may have additional effects on thyroid function and estrogen hormone activity than the original commercial products. Hydroxy-PBDEs also may be present in food. Additionally, PBDEs can be transformed to methoxy-PBDEs in the environment. These methoxy-PBDE metabolites are found in some environmental samples at levels higher than the parent PBDEs. Their toxicity is still unknown.<sup>98</sup>

### Cancer

Deca is one of the only PBDEs that has been tested for carcinogenicity. Tests on laboratory animals exposed to Deca resulted in tumors of the liver, thyroid, and pancreas. The doses of Deca administered were high (2.5-5 percent of the diet), but uptake of Deca was low compared to other PBDEs--only roughly 1/1000 of the given dose was absorbed by the rodents.<sup>99</sup> This result indicates that Deca BDE could act as a carcinogen even at low levels in tissue, but more study is needed to determine whether the same effects would be produced with low doses of Deca. Unfortunately, few studies on these effects have been conducted.

### Burning Toxic Flame Retardants Creates Cancer-Causing Dioxins and Furans

Although toxic flame retardants are used to slow the spread of fire, they do not prevent fire altogether. When they do burn, whether in house fires or in incinerators, all types of PBDEs form

brominated dioxins and furans.<sup>100</sup> Dioxins and furans are among the most toxic and dangerous compounds known. Some components of Penta, which may be formed by Deca's breakdown, can produce brominated furans from mere exposure to light.<sup>101</sup> Few studies have looked for combustion byproducts of PBDEs in the environment.

Dioxins and furans can cause health effects at levels much lower than PBDEs. This has motivated several companies to find an alternative to brominated flame retardants. The electronics company Motorola, for example, has developed a line of bromine-free products.<sup>102</sup>

### More study on health effects of Deca is needed

Study on Deca's toxic properties is certainly not complete. The European Union is currently conducting a risk assessment of the chemical, and preliminary results have prompted a call for more studies. Although results of safety tests so far have been mixed, several important studies indicate that Deca may be dangerous to our health. Again, this highlights the problem that these studies are being called for after years of widespread use of this chemical, as well as widespread exposure.

Our experience with persistent chemicals of the past such as DDT and PCBs has shown what happens when we wait to gather conclusive evidence of a chemical's harm instead of acting on mounting evidence. By the time the chemicals were regulated, they had spread across the globe and left a path of damage from which we have yet to recover. We should not continue down the same path with PBDEs and Deca.

## Deca: Not Necessary for Fire Safety

---

Manufacturers can make products that are both flame-retardant and non-toxic. First, manufacturers can design products 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, such as metal, leather, and wool, do not require flame-retardant additives and can replace highly flammable plastics and fabrics. Third, less toxic flame retardant additives can replace brominated flame retardants where manufacturers cannot make products inherently less flammable.

Many companies, especially those in the electronics industry, already have incorporated alternative designs, materials, and flame-retardant additives or are hard at work exploring substitutions for Deca and other toxic flame retardants. Responsible companies are using materials and other chemical additives that pose fewer environmental health risks while minimizing costs, meeting flammability standards, and maintaining appropriate physical properties of plastics and other materials.

### Replacing Deca

---

When companies consider alternatives to Deca and other brominated flame retardants, they look for flame retardants that have reasonable costs and maintain the appropriate physical properties of the materials they use. Consumers look for products that do not contain chemicals that are toxic to the environment and human health. Both of these considerations can be met simultaneously.

The best types of alternative flame retardants are halogen-free chemicals, or those that do not contain fluorine, chlorine, bromine, or iodine. Halogenated chemicals, those that contain fluorine, chlorine, bromine, or iodine, are generally more likely to bio-accumulate and have toxic effects.

The best alternative flame retardants have the following properties:

- Able to suppress the formation of flames, smoke and hazardous fumes during fires;
- No acute or chronic effects on human health and development or the

environment;

- Minimum release during production, product use, and after disposal (either in landfills or incinerators);
- Do not interfere with re-usability or recyclability of the product;
- Are biodegradable into non-hazardous byproducts; and
- Do not adversely affect product function or longevity.

Unfortunately, many of the alternatives to Deca and other brominated flame retardants are halogenated chemicals, such as chlorinated paraffins. Chlorinated paraffins cause toxic effects, including hormone disruption, and do not pose a viable and safe alternative to brominated flame retardants.<sup>103</sup> Industry must develop non-halogenated flame retardant replacements for Deca and test the potential replacement chemicals for any possible negative health effects before introducing them as substitutes.

## Companies Replacing Deca with Viable Alternatives

---

Many companies have found viable alternatives to Deca and other PBDEs. A few of the electronics companies that already produce some PBDE-free products include Apple, Dell, IBM, Motorola, Panasonic, Phillips and Sony. In addition, Ericsson, Intel, Phillips, Sony and Toshiba have announced a complete ban of PBDEs by 2006.<sup>104</sup> The furniture company IKEA is a leader in replacing PBDEs in its products.

### Toshiba

In April of 1998, Toshiba announced a new casing for transistors that does not require the use of Deca or other brominated flame retardants. The new casing is made of a type of plastic designed to withstand high temperatures, polyphenylene sulfide (PPS). Transistors are usually made of epoxy, which melt at high temperatures. This was the first thermoplastic casing for transistors in the semiconductor industry.<sup>105</sup>

### Intel

Intel Corporation does not use any PBDEs in its products. The company also ensures that its suppliers do not include PBDEs in the raw materials that go into Intel's products.<sup>106</sup>

### Ericsson

Ericsson has banned PBDEs from its products and all applications due to European action and has banned chlorinated paraffins, a common alternative to Deca.<sup>107</sup> Ericsson also keeps a list of substances it is "observing" for possible phase-out and legislative action, including all other

halogenated flame retardants. In August of 2003, Ericsson announced a line of converters that are halogen free.<sup>108</sup> In addition to being free from PBDEs and other halogenated flame retardants, these converters are about 25 percent cheaper than other converters considered environmentally friendly and are comparable to other converters that currently contain halogens.

### Motorola

Motorola has developed a line of products that are bromine-free. The company initially acted to switch flame retardants out of concern about the dioxins and furans released during burning. Motorola then searched for an alternative that was actually superior in several criteria, including: provides better or equal flame retardance; does not diminish the functionality of the product; does not pose a risk to the environment or health; does not reduce any physical property requirements; meets cost requirements; and meets availability requirements. Motorola found several viable alternatives and now offers a line of cell phones that use bromine-free circuit breakers.<sup>109</sup>

In addition, a few manufacturers are actively developing safer alternatives for users of Deca and other brominated flame retardants. RTP Company, based in Winona, Minnesota, has developed a family of chemicals, Non-Halogen Compounds, which it markets as both safe for the environment and uncompromising for industry's needs.<sup>110</sup>

# Why We Face this Crisis: Failure of U.S. and State Toxics Policy

---

Many people think, incorrectly, that the U.S. government would not allow chemicals to enter the market if they were not safe. In truth, the regulatory process has worked the opposite way the public believes it should.

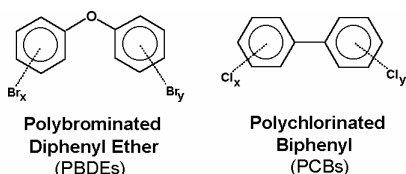
In 1976, Congress passed the primary law regulating toxic chemicals, the Toxic Substances Control Act (TSCA), which grandfathered all existing chemicals on the market into use without

health effects testing or analysis. Most of these chemicals emerged in the 1940s and 1950s when few laws governed chemical safety. Today, the Environmental Protection Agency (EPA) reviews new chemicals that come onto the market but does not require full health effects testing for approval. With 2,000 new chemicals introduced each year, EPA approves an average of seven new chemicals each day.

## Toxic Chemicals Missed By Regulation

---

Stories of the mass production, marketing and release of a dangerous chemical that damages the public's health and the environment are all too familiar. PBDEs are not the first chemical to slip through the cracks of federal regulation. PBDEs are remarkably similar to polychlorinated biphenyls (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.



PCBs were first manufactured in 1927 and marketed for use as insulators for electric products. The first evidence of adverse health impacts were reported in the 1930s when workers at plants producing PCBs became ill. A few early independent scientific studies documented that PCBs were bio-accumulating in the U.S. food

chain. It was later learned that Monsanto Company sponsored studies that falsely concluded that PCBs were not carcinogenic.<sup>111</sup> In 1968, a major human health disaster in Japan with PCB-contaminated cooking oil resulted in the accidental poisoning of 1,800 people. This and a series of other industrial accidents led to the passage of the Toxic Substances Control Act of 1976, effectively banning PCBs. This is the only chemical the U.S. Congress has ever banned, although it has regulated five under its TSCA authority. The good news is that since the 1976 ban, body burden levels of PCBs have declined.

Thirty years after this PCB ban, however, scientists still discover new health effects in people with no occupational or accidental PCB exposures. In 1994, studies found that the remaining levels of PCBs in the general population were high enough to affect thyroid hormone balance in mothers and their nursing infants.<sup>112</sup> Further bad news is that the PCBs may be working together with PBDEs to harm human health, thereby extending the toxic legacy of PCB contamination well into the future.<sup>113</sup>

## Current Law Leaves EPA with Little Power to Protect Public Health

---

The U.S. government's regulation of chemicals is based on the premise that chemicals are presumed innocent until they are proven to harm human health.

Throughout its nearly 30-year history, TSCA has rarely been amended, but clearly fails to effectively regulate toxic chemicals. Since the law's inception, the Environmental Protection Agency has never used its authority to ban a chemical and has only offered regulations on five different chemicals, including PCBs, which Congress ordered regulated. EPA's lax regulation can be attributed to the unreasonably high burden of proof the law places on the agency to show that a chemical poses an unreasonable risk to human health and the environment.

TSCA divides all the chemicals on the market into two categories: existing chemicals and new chemicals. Existing chemicals are chemicals already on the market before 1980. These make up approximately 99 percent by volume of the chemicals on the market today. Existing chemicals are considered safe until EPA can establish that

they pose an unreasonable risk to people's health or the environment, that the benefits of action outweigh the risks of inaction, and that EPA is employing the least burdensome method when taking action.<sup>114</sup>

Companies that wish to introduce new chemicals to the U.S. market must notify EPA at least 90 days before producing or importing a new chemical, and EPA has been able to ensure review of these chemicals. The new chemicals program, however, could be improved by increasing the testing requirements of new chemicals.

EPA should have the authority and means to guarantee chemicals on the market are safe for human health and the environment. In its 1998 review of high production volume chemicals, EPA estimated the cost for a full round of basic screening tests, including tests for reproductive and developmental toxicity, at about \$205,000 per chemical.<sup>115</sup> The chemical industry, with profits of \$17 billion per year, should pay this price to protect both health and the environment.<sup>116</sup>

## Swift Government Action Can Prevent Future Harm

We need not resign ourselves to bloodstreams, infants, or breast milk endlessly contaminated with toxic chemicals. The story of lead in U.S. gasoline provides an apt example of how regulating and finding substitutes for toxic chemicals can reduce their levels in human bodies and protect public health.

### Blood Lead Levels Decrease with Reduced Exposure

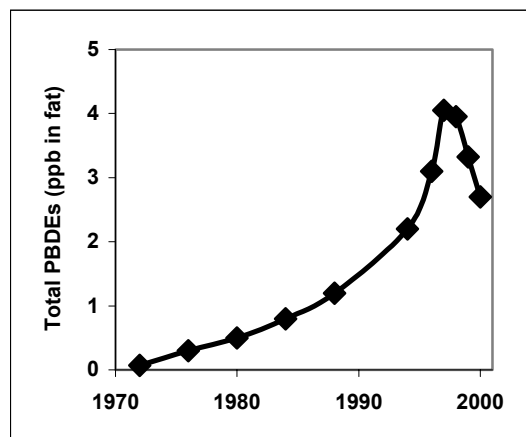
The negative neurological health effects associated with lead exposure have been known for decades and suspected for centuries. In 1976, the federal government instituted new regulations on the use of lead in gasoline. Seven years later, a study in the *New England Journal of Medicine* looked at the change in blood lead levels between 1976 and 1980. The authors of the study found that blood lead levels decreased dramatically (by approximately 37 percent) over the four-year period. They attributed this decrease to the regulations that had reduced the lead content in gasoline during the same time period.<sup>117</sup>

### PBDEs in Sweden

Studies of Swedish women's milk samples showed PBDE levels increasing over the years 1972-1997, doubling every five years. As a result, the Sweden quickly phased out certain PBDEs. Recent studies of milk samples from Swedish women report that PBDE levels have decreased since this action went into effect (Figure F).<sup>118</sup>

The lesson is that regulation works—if a government entity bans or phases out a chemical, the body burdens of that chemical will decline.

**Figure F:** Recent declines in PBDE levels in the breast milk of Swedish women.



*SOURCE:* 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.

## Policy Findings

---

**"For heaven's sake, didn't we learn anything from the issues of DDT and PCBs? It's really time to act." – Aake Bergman, Chair of Stockholm's University's Environmental Chemistry Department**

Individual European countries have long paid attention to the health risks associated with widespread use of toxic flame retardants. Germany originally began the substitution effort in the 1980s with a dioxin directive, which prompted German chemical manufacturers to voluntarily stop manufacturing polybrominated biphenyls and PBDEs in 1989 in order to prevent the continued creation of brominated dioxins.<sup>119</sup> Because Sweden regularly monitors breast milk for the presence of contaminants, scientists were able to detect the problem and prompt Europe to limit exposure by

cutting the use of PBDEs by two-thirds in 1997.

European 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 placed the burden of proof on a company to show that its chemical will not harm the environment or public health.<sup>120</sup>

### Phase Out Toxic Flame Retardants

---

We commend the California legislature for its leadership in protecting children's health by responding to existing evidence and banning the use of Penta and Octa flame retardants. A number of other states across the country have also begun to push for action on toxic flame retardants, including Washington State, Massachusetts, Maryland, Maine, and Michigan. Additionally, the U.S. EPA has taken a positive step toward reducing PBDE production nationwide, by reaching an agreement with Great Lakes Chemical to phase out production of two PBDEs.

However, these steps fall short of what is needed to fully prevent future exposure to these dangerous chemicals. There are still unexplored aspects of the toxicity of Deca flame retardants, and complete study will take many years. However, the evidence indicates that immediate action on this chemical is warranted in California and the rest of the United States. Given the magnitude of the potential threat to public health, the rapidly increasing levels of

exposure, and the availability of alternatives, the United States should phase out the use of Deca as well as other brominated flame retardants. While allowing for time to develop and identify safe and viable alternatives to Deca, the United States should implement this phase-out as soon as possible in order to prevent further exposure of future generations to these toxic chemicals. In the absence of federal action, individual states should lead the way in phasing out these chemicals.

Industry can follow the lead of those companies turning to alternatives. Great Lakes Chemical ceasing production of Penta and Octa three years in advance of California's ban is a first step, but the potential dangers of continued production of Deca need to be addressed. We call on chemical manufacturers to develop safer alternatives, and call on manufacturers of Deca-containing products to aggressively research ways of making products that will meet fire-safety standards without threatening children's health.

## Reform Chemical Policy

---

Chemicals that are untested or known to be hazardous and chemicals that can contaminate the developing fetus should not be on the market or in widespread use and distribution. U.S. and California chemicals policy should 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 United States and California must prevent exposure to toxic chemicals when there is evidence of potential harm.

Currently, manufacturers can put chemicals on the market before detection methods have even been developed to test for the presence of the chemical in air, water, soil, or our bodies. The burden then

falls on federal and state governments to develop these analytical methods—an expensive and time-consuming process. The costs of developing analytical methods and as well as methods to test for a chemical's safety should fall to the manufacturers who stand to profit from the product.

The case of Deca and PBDEs present a suitable case study of the failings of current policy. Groundbreaking measures have been taken in Scandinavia and Europe to protect people's health from the threats of toxic chemicals; California has taken important steps as well, banning two forms of brominated flame retardants. Governments need to take the next step and require safety testing of chemicals before they are introduced to the market.

## **Appendix A: Major Manufacturers of Toxic Flame Retardants in the U.S.**

---

There are 144 facilities in the United States that produce or process Deca, and there are two main U.S. manufacturers: Great Lakes Chemical Company and Albemarle Corporation.

### **Great Lakes Chemical Company**

The leading manufacturer of brominated flame retardants is Great Lakes Chemical Company, headquartered in West Lafayette, Indiana. Great Lakes Chemical is the sole U.S. producer of Penta product and one of a handful of producers of Octa and Deca. It sold approximately \$410 million worth of brominated flame retardants in 2000.<sup>121</sup> In FY2002, Great Lakes Chemical earned more than \$1.4 billion in revenue and reported a gross profit of \$331.3 million.<sup>122</sup> Following the announcement of a new chemical to replace Penta, Great Lakes agreed to a voluntary phase out of the manufacture of both Penta and Octa by the end of 2004.<sup>123</sup> It is inconclusive whether or not this new chemical, known as Firemaster 550, will prove to be safe for human health, although Great Lakes claims the U.S. EPA has provided a “favorable environmental assessment.”<sup>124</sup>

### **Albemarle Corporation**

The second largest producer of brominated flame retardants, Richmond, Virginia-based Albemarle Corporation, is the leading U.S. producer of Deca. In 2000, approximately \$250 million of Albermarle’s

sales were from brominated flame retardants. In 2002, Albemarle had a reported revenue of \$980 million and a gross profit of \$235 million.<sup>125</sup>

In November of 2003, Albemarle also developed an alternative to Penta, even though it does not currently produce Penta. The replacement, SAYTEX RX 8500 is a brominated flame retardant, but is reactive instead of additive, so its molecules are chemically bound to the material to which it is added. This would make it less likely for the chemical to escape into the environment. This new flame retardant can be used in a variety of foams in furniture, bedding, cars, and packaging. However, it has not yet been fully reviewed by EPA for health risks.<sup>126</sup>

### **Others**

In addition to domestic manufacturers, there are at least six other manufacturers of PBDEs globally: Dead Sea Bromines/Eurobrome in the Netherlands, Atofina in France, Tosoh in Japan, Matsinaga in Japan, Nippo in Japan, and Great Lakes Chemical in the United Kingdom.<sup>127</sup> Because there are so many overseas manufacturers, an American phase-out of manufacturing of just a few types of flame retardant will not be sufficient to stop the flow of Deca and other PBDEs into the domestic market.

## Appendix B: Number of Facilities that Produce, Process or Use Deca in the U.S., By State

State	# of Facilities	Activities and Uses (see key)
AL	1	7,8
AR	4	1,4,7,8
CA	5	7,8
CT	4	7,8,9,14
FL	1	7
GA	4	7
IL	2	7,8
IN	5	7,8
KS	1	11
KY	2	7
LA	1	12
MA	12	7,8
MD	1	8
MI	6	1,3,7,8
MN	5	6,7,8,9,12
MO	1	7
MS	1	7
NC	12	7,8
NE	1	8
NH	1	7
NJ	3	7,8
NY	3	6,7,8
OH	9	7,8
OR	1	12
PA	8	7,8
RI	3	7,8,10
SC	9	7,8
TN	6	7
TX	8	1,3,7,8,12
VA	5	7,8
WA	1	8
WI	2	7,8

SOURCE: Toxics Release Inventory, 2002.

### Key to Activities and Uses:

- |                          |                             |
|--------------------------|-----------------------------|
| 1. Produced              | 8. Article Component        |
| 2. Imported              | 9. Repackaging              |
| 3. Used, Processed       | 10. Chemical Processing Aid |
| 4. Sale Distribution     | 11. Manufacture Aid         |
| 5. Byproduct             | 12. Ancillary/Other Uses    |
| 6. Reactant              | 13. Manufacture Impurity    |
| 7. Formulation Component | 14. Process Impurity        |

## End Notes

---

- <sup>1</sup> Limin X. Clegg, Eric J. Feuer, Douglas N. Midthune, Michael P. Fay, Benjamin F. Hankey, "Impact of Reporting Delay and Reporting Error on Cancer Incidence Rates and Trends," *J Natl Cancer Inst*, Vol. 94, No. 20, October 16, 2002.
- <sup>2</sup> Learning disabilities: KA Kavale, SR Forness, and CT Ramey, "Co-variants in learning disability and behavior disorders: An examination of classification and placement issues," *Advances in Learning and Behavioral Disabilities* 12:1-42, 1998; as cited in: Ted Schettler et al., Physicians for Social Responsibility and the Clean Water Fund, *In Harm's Way: Toxic Threats to Child Development*, May 2000; Autism increase: Thomas Maugh, "State Study Finds Sharp Rise in Autism Rate," *Los Angeles Times*, 16 April 1999.
- <sup>3</sup> March of Dimes, "Premature Birth Rate in U.S. Reaches Historic High; Now Up 20 Percent Since 1981," Press Release, downloaded from [http://www.marchofdimes.com/aboutus/10651\\_10763.asp](http://www.marchofdimes.com/aboutus/10651_10763.asp), 3 February 2004.
- <sup>4</sup> U.S. EPA, Chemical Information Collection and Data Development (Testing), *Chemical Hazard Data Availability Study, 2002*.
- <sup>5</sup> National Research Council Commission on Life Sciences, *Scientific Frontiers in Developmental Toxicology and Risk Assessment*, January 2000.
- <sup>6</sup> See, for example, the American Chemistry Council's "2003 Year in Review" Report, available at [www.acnewsmedia.com](http://www.acnewsmedia.com).
- <sup>7</sup> Bromine Science and Environmental Forum (BSEF), "Major Brominated Flame Retardants Volume Estimates," available at [www.bsef-site.com](http://www.bsef-site.com), 21 January 2003.
- <sup>8</sup> Sonia Lunder and Renee Sharp, Environmental Working Group, *Mothers' Milk: Record levels of toxic fire retardants found in American mothers' breast milk*, September 2003.
- <sup>9</sup> P. Eriksson et al., "Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment?" *Environmental Health Perspectives*, 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," *Toxicological Science*, 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," *Toxicological Science*, 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. Ohm, 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.
- <sup>10</sup> A study found that a dose of 0.8 milligrams of PBDEs per kilogram of weight given to infant mice on their tenth day of life produced permanent developmental damage, including abnormal behavior and impaired learning skills. An average mouse is 10% to 20% fat. If the mouse is assumed to absorb 100% of the administered dose, and contains 15% body fat, then levels of PBDEs will be 5,300 parts per billion (ppb) in the fat.
- <sup>11</sup> J. Friedman, Rockefeller University News Release, "Body Weight Regulated by Newly Discovered Hormone," 27 July 1995.
- <sup>12</sup> Anita Mazdai, et al., "Polybrominated Diphenyl Ethers in Maternal and Fetal Blood Samples," *Environmental Health Perspectives*, 111(9), July 2003.
- <sup>13</sup> 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.
- <sup>14</sup> P Eriksson et al, "Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment?," *Environmental Health Perspectives*, 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," *Toxicological Science*, 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," *Toxicological Science*, 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. Ohm, 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.
- <sup>15</sup> 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; 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.
- <sup>16</sup> S.P. Porterfield, C.E. Hendrich, "The role of thyroid hormones in prenatal and neonatal neurological development-current perspectives," *Endocrinology Review*, 14(94-106), 1993.
- <sup>17</sup> 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.
- <sup>18</sup> 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.
- <sup>19</sup> 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.
- <sup>20</sup> 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 [<sup>3</sup>H]arachidonic acid release in rat cerebellar granule neurons," *Toxicological Science*, 68(451-457), 2002.
- <sup>21</sup> Walter Lichtensteiger, et al., "Effect of polybrominated diphenylether and PCB on the development of the brain-gonadal axis and gene expression in rats," *Organohalogen Compounds*, 61(84-87), 2003. Sergio Kuriyama, Ibrahim Chahoud, "Maternal exposure to low dose 2,2', 4,4', 5-pentabromo diphenyl ether (PBDE 99) impairs male reproductive performance in adult rat offspring," *Organohalogen Compounds* 61(92-95), 2003.
- <sup>22</sup> Chris Talsness, et al., "Ultrastructural changes in the ovaries of adult offspring following a single maternal exposure to low-dose 2,2', 4,4', 5-pentabromodiphenyl ether," *Organohalogen Compounds*, 61(88-91), 2003.
- <sup>23</sup> Sergio Kuriyama, Ibrahim Chahoud, "Maternal exposure to low dose 2,2', 4,4', 5-pentabromo diphenyl ether (PBDE 99) impairs male reproductive performance in adult rat offspring," *Organohalogen Compounds*, 61(92-95), 2003.
- <sup>24</sup> National Toxicology Program, "Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies)," 1986. 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.
- <sup>25</sup> 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's lymphoma," *Oncology Research*, 10(429-432), 1998.
- <sup>26</sup> P.O. Darnerud, A. Thuvander, "Studies on Immunological 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).
- <sup>27</sup> Baccarelli, A, P Mocarrelli, DG Patterson Jr., M Bonzini, AC Pesatori, N Caporaso and MT Landi, "Immunologic Effects of Dioxin: New Results from Seveso and Comparison with Other Studies," *Environmental Health Perspectives*, 110(1169-1173), 2002.
- <sup>28</sup> G. Fernlof et al, "Lack of Effects of Some Individual Polybrominated Diphenyl Ether (PBDE) and Polychlorinated Biphenyl Congeners on Human Lymphocyte Functions in Vitro," *Toxicology Letters*, 90(2-3):189-197.
- <sup>29</sup> Kellyn Betts, "Deca PBDE flame retardant gets around," *Environmental Science and Technology*, January 8 2004.
- <sup>30</sup> Dr. Michael Warhurst, Friends of the Earth, "Brominated Flame Retardants," viewed at [website.lineone.net/~mwarhurst/bfr.html](http://website.lineone.net/~mwarhurst/bfr.html), June 21, 2002.
- <sup>31</sup> Environment Daily, "Norway Detects 'Firsts' on Retardant Seepage," 1364, 16 January 2003.
- <sup>32</sup> Robert Hale et al., Virginia Insititue of Marine Science, "Persistent pollutants in land-applied sludges," *Nature*, 412(140-141), 2001.
- <sup>33</sup> Robert Hale et al., Virginia Insititue of Marine Science, "Persistent pollutants in land-applied sludges," *Nature*, 412(140-141), 2001.
- <sup>34</sup> Robert Hale et al., Virginia Insititue of Marine Science, "Persistent pollutants in land-applied sludges," *Nature*, 412(140-141), 2001.
- <sup>35</sup> 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.
- <sup>36</sup> 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., Greenpeace, *The Presence of Brominated Flame Retardants and Organotin Compounds in Dusts Collected from Parliament Buildings from Eight Countries*, 2001.
- <sup>37</sup> David Santillo, et al., Greenpeace, *Consuming Chemicals: Hazardous chemicals in house dust as an indicator of chemical*

---

exposure in the home, January 2003.

<sup>38</sup> Ruthann Rudel, et al., "Phthalates, Alkylphenol, Pesticides, Polybrominated Diphenyl Ethers, and Other Endocrine Disrupting Compounds in Indoor Air and Dust," *Environmental Science and Technology*, 29 December 2003.

<sup>39</sup> Betts, Kellyn, "Deca PBDE flame retardant gets around," *Environmental Science and Technology*, January 8 2004.

<sup>40</sup> A. Sjodin 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.

<sup>41</sup> T. Strandman, J. Koistinen, and T. Variainen, "Polybrominated diphenyl ethers (PBDEs) in placenta and human milk," *Organohalogen Compounds*, 47(61-64), 2000.

<sup>42</sup> A. Lione, "Polychlorinated Biphenyls and Reproduction," *Reproductive Toxicology*, 2(83-89), 1988.

<sup>43</sup> A. Madzai, et al., "Polybrominated Diphenyl Ethers in Maternal and Fetal Blood," *Environmental Health Perspectives*, 2003.

<sup>44</sup> J.L. Jacobson and S.W. Jacobson, "Intellectual Impairment in Children Exposed to Polychlorinated Biphenyls in Utero," *New England Journal of Medicine*, 783-789, 1998.

<sup>45</sup> Jane E. Brody, "Report Links Prenatal PCB Exposure with Child Development," *New York Times*, 12 September 1996.

<sup>46</sup> S. Steingraber, "To Breastfeed or Not to Breastfeed is Not the Question," and "Breastfeeding, Biomonitoring, and the Media," *Mothering Magazine*, 122(63-67), Jan./Feb. 2004.

<sup>47</sup> American Academy of Pediatrics Work Group on Breastfeeding, "Breastfeeding and the Use of Human Milk," *Pediatrics*, 100(6), 1 December 1997.

<sup>48</sup> Solomon, Gina and Pilar Weiss, "Chemical Contaminants in Breast Milk: Time Trends and Regional Variability," *Environmental Health Perspectives*, 110(6), June 2002.

<sup>49</sup> Natural Resources Defense Council, "Healthy Milk, Healthy Baby: Chemical Pollution and Mother's Milk," [www.nrdc.org/breastmilk](http://www.nrdc.org/breastmilk).

<sup>50</sup> Ibid.

<sup>51</sup> American Academy of Pediatrics Work Group on Breastfeeding, "Breastfeeding and the Use of Human Milk," *Pediatrics*, 100(6), 1 December 1997.

<sup>52</sup> 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.

<sup>53</sup> Europe's Environmental News Service, "Flame Retardant Controls Confirmed," *Environment Daily*, 1388, 19 February 2003.

<sup>54</sup> Environmental News Service, "EU Lawmakers Vote Broad Fire Retardant Ban," 6 September 2001.

<sup>55</sup> European Union Environment Agency, *Updated Risk Assessment of Bis(pentabromophenyl) Ether (decabromodiphenyl ether)*, *Environmental Draft*, November 2003.

<sup>56</sup> 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/](http://europa.eu.int/comm/enterprise/chemicals/markrestr/) on 10 Feb 2003.

<sup>57</sup> 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/](http://europa.eu.int/comm/enterprise/chemicals/markrestr/) on 10 Feb 2003.

<sup>58</sup> Environmental Protection Agency press release, "Brominated Flame Retardants to be Voluntarily Phased Out," Nov 3, 2003, available at <http://yosemite.epa.gov/opa/admpress.nsf/b1ab9f485b098972852562e7004dc686/26f9f23c42cd007d85256dd4005525d2?OpenDocument>.

<sup>59</sup> Press release, "Thanks to New Technology, Great Lakes Chemical Corporation Announces that it will Cease Production of Penta-BDE Flame Retardant by End of 2004," 3 November, 2003, available at: [http://biz.yahoo.com/prnews/031103/clm043\\_1.html](http://biz.yahoo.com/prnews/031103/clm043_1.html).

<sup>60</sup> Diskin, Colleen, "Concerns Rise Over Flame Retardant," *Seattle Times*, 26 December, 2003, quoting Dave Deegan of EPA.

<sup>61</sup> M.L. Hardy, "The toxicology of the three commercial polybrominated diphenyl oxide (ether) flame retardants," *Chemosphere* 46, 757-77, 2002.

<sup>62</sup> 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.

<sup>63</sup> H. Viberg, A. Fredriksson, and Per Eriksson, "Neurotoxicity of Different Polybrominated Diphenyl Ethers, Including PBDE 209," *Organohalogen Compounds* 65, 9-11, 2003.

<sup>64</sup> Peter Lindberg, Ulla Sellström, Lisbeth Häggberg, and Cynthia A. de Wit, "Higher Brominated Diphenyl Ethers and Hexabromocyclododecane Found in Eggs of Peregrine Falcons (*Falco peregrinus*) Breeding in Sweden," *Environmental Science and Technology*, 38(1), 93-96, 2004.

- <sup>65</sup> A. Sjodin et al, "Flame Retardant Exposure: Polybrominated Diphenyl Ethers in Blood from Swedish Workers," *Environmental Health Perspectives*, 107(8), 643-648, 1999; A. Schecter, et al., "Congener Specific Measurement of Polybrominated Diphenyl Ethers in 47 Individual Milk Samples From Nursing Mothers in the U.S.A.," *Organohalogen Compounds* 61, 13-16, 2003.
- <sup>66</sup> H. Stapleton, M. Alaei, and J. Baker, "Debromination of Decabromodiphenyl Ether by Juvenile Carp (*Cyprinus Carpio*)," *Organohalogen Compounds* 61, 21-24, 2003; G. Soderstrom, U. Sellstrom, C. de Wit, and M. Tysklind, "Photolytic Debromination of Decabromodiphenyl Ether (BDE 209)," *Environmental Science and Technology*, 38(1), 127-132, 2004; T. Herrmann, B. Schilling, and O. Papke, "Photolysis of PBDEs in Solvents by Exposure to Daylight in a Routine Laboratory," *Organohalogen Compounds* 63, 361-364, 2003; 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.
- <sup>67</sup> Bromine Science and Environmental Forum (BSEF), "Major Brominated Flame Retardants Volume Estimates," available at [www.bsef-site.com](http://www.bsef-site.com), 21 January 2003.
- <sup>68</sup> Sean M. Hays, Colleen A. Cushing, David W. Pyatt, Kelley C. Holicky, and Dennis J. Paustenbach, "Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (DBDPO)," *Organohalogen Compounds* 61, 251-254, 2003. This paper is based on work that Exponent, Inc. presented for the Brominated Flame Retardant Industry Panel as part of the panel's sponsorship of Deca under the U.S. Environmental Protection Agency's Voluntary Children's Chemical Evaluation Program (VCCEP), and was supported by the Bromine Science and Environmental Forum.
- <sup>69</sup> H. Stapleton, M. Alaei, and J. Baker, "Debromination of Decabromodiphenyl Ether by Juvenile Carp (*Cyprinus Carpio*)," *Organohalogen Compounds* 61, 21-24, 2003.
- <sup>70</sup> Betts, Kellyn, "New Research Challenges Assumptions about Popular Flame Retardant," *Environmental Science & Technology*, 6 November 2003.
- <sup>71</sup> A. Kierkegaard, et al, "Dietary Uptake and Biological Effects of Decabromodiphenyl Ether in Rainbow Trout (*Oncorhynchus Mykiss*)," *Environmental Science & Technology*, 33(10), 1612-1617, 1999.
- <sup>72</sup> Betts, Kellyn, "New Research Challenges Assumptions about Popular Flame Retardant," quoting Heather Stapleton, *Environmental Science & Technology*, 6 November 2003; R. Letcher, I. D'Sa, K. Valters, H. Li, E. Bennett, and M. Alaei, "Polybrominated Diphenyl Ethers and Hydroxylated and Methoxylated Analogues in Detroit River Fish," *Organohalogen Compounds* 61, 29-32, 2003.
- <sup>73</sup> M. La Guardia, R. C. Hale, and E. Harvey, "Are Waste Water Treatment Plants Sources for Polybrominated Diphenyl Ethers?," presented at the Annual Meeting of the Society of Environmental Toxicology and Chemistry (SETAC), 2003.
- <sup>74</sup> M. La Guardia, R. C. Hale, and E. Harvey, "Are Waste Water Treatment Plants Sources for Polybrominated Diphenyl Ethers?," presented at the Annual Meeting of the Society of Environmental Toxicology and Chemistry (SETAC), 2003.
- <sup>75</sup> 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," *Organohalogen Compounds* 35 (447-450) 1998; Watamabe 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* 35, 953-959, 1987.
- <sup>76</sup> Kellyn Betts, "Deca PBDE Flame Retardant Gets Around," *Environmental Science and Technology*, 8 January 2004.
- <sup>77</sup> T. Herrmann, B. Schilling, and O. Papke, "Photolysis of PBDEs in Solvents by Exposure to Daylight in a Routine Laboratory," *Organohalogen Compounds* 63, 361-364, 2003.
- <sup>78</sup> T. Herrmann, B. Schilling, and O. Papke, "Photolysis of PBDEs in Solvents by Exposure to Daylight in a Routine Laboratory," *Organohalogen Compounds* 63, 361-364, 2003.
- <sup>79</sup> G. Soderstrom, U. Sellstrom, C. de Wit, and M. Tysklind, "Photolytic Debromination of Decabromodiphenyl Ether (BDE 209)," *Environmental Science and Technology*, 38(1), 127-132, 2004.
- <sup>80</sup> M. Tysklind et al, "Abiotic Transformation of Polybrominated Diphenylethers (PBDEs): Photolytic Debromination of Decabromo Diphenyl Ether," abstract presented at the Second International Workshop on Brominated Flame Retardants, Stockholm, Sweden, 14-16 May 2001.
- <sup>81</sup> 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, 717-728, 2002.
- <sup>82</sup> Peter Lindberg, Ulla Sellström, Lisbeth Häggberg, and Cynthia A. de Wit, "Higher Brominated Diphenyl Ethers and Hexabromocyclododecane Found in Eggs of Peregrine Falcons (*Falco peregrinus*) Breeding in Sweden," *Environmental Science and Technology*, 38(1), 93-96, 2004.
- <sup>83</sup> G. Soderstrom, U. Sellstrom, C. de Wit, and M. Tysklind, "Photolytic Debromination of Decabromodiphenyl Ether (BDE 209)," *Environmental Science and Technology*, 38(1), 127-132, 2004; M. La Guardia, R. C. Hale, and E. Harvey, "Are Waste Water Treatment Plants Sources for Polybrominated Diphenyl Ethers?," presented at the Annual Meeting of the Society of Environmental Toxicology and Chemistry (SETAC), 2003; P. Lepom, T. Karasyova, and G. Sawal, *Organohalogen Compounds*

- 58, 209-212, 2002; A. Kierkegaard, et al, "Dietary Uptake and Biological Effects of Decabromodiphenyl Ether in Rainbow Trout (*Oncorhynchus Mykiss*)," *Environmental Science & Technology*, 33(10),1612-1617, 1999; H H. Stapleton, M. Alaei, and J. Baker, "Debromination of Decabromodiphenyl Ether by Juvenile Carp (*Cyprinus Carpio*)," *Organohalogen Compounds* 61, 21-24, 2003.
- <sup>84</sup> H. Viberg, A. Fredriksson, and Per Eriksson, "Neurotoxicity of Different Polybrominated Diphenyl Ethers, Including PBDE 209," *Organohalogen Compounds* 65, 9-11, 2003.
- <sup>85</sup> Kristina Jakobsson, Kaj Thuresson, Lars Rylander, Andreas Sjödin, Lars Hagmar, Ake Bergman, "Exposure to Polybrominated Diphenyl Ethers and Tetrabromobisphenol A among Computer Technicians," *Chemosphere*, 46(5), 709-716, February 2002.
- <sup>86</sup> K. Rothenbacher, European Brominated Flame Retardant Industry Panel, "Industry Comments on the CSTEE Opinion on the Results of the Risk Assessment of Bis(pentabromophenyl) Ether," October 2002, downloaded from [www.bsrf-site.com](http://www.bsrf-site.com).
- <sup>87</sup> American Chemistry Council's Brominated Flame Retardant Industry Panel for the Voluntary Children's Chemical Evaluation Program (VCCPEP), *Report of the Peer Consultation Meeting On Decabromodiphenyl Ether*, September 2003; Sean M. Hays, Colleen A. Cushing, David W. Pyatt, Kelley C. Holicky, and Dennis J. Paustenbach, "Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (DBDPO)," *Organohalogen Compounds* 61, 251-254, 2003.
- <sup>88</sup> Kristina Jakobsson, Kaj Thuresson, Lars Rylander, Andreas Sjödin, Lars Hagmar, Ake Bergman, "Exposure to Polybrominated Diphenyl Ethers and Tetrabromobisphenol A among Computer Technicians," *Chemosphere*, 46(5), 709-716, February 2002.
- <sup>89</sup> WWF UK, *ContaminATION: National Biomonitoring Survey 2003*, downloaded from [www.wwf.org](http://www.wwf.org), January 2004.
- <sup>90</sup> A. Schecter, et al, "Congener Specific Measurement of Polybrominated Diphenyl Ethers in 47 Individual Milk Samples From Nursing Mothers in the U.S.A.," *Organohalogen Compounds* 61, 13-16, 2003.
- <sup>91</sup> S. Lunder and R. Sharp, Environmental Working Group, *Mothers' Milk: Record Levels of Toxic Fire Retardants Found in American Mothers' Milk*, September 2003.
- <sup>92</sup> G. Koren, *Maternal-Fetal Toxicology (2<sup>nd</sup> Ed.)*, Marcel Dekker, Inc. 1994.
- <sup>93</sup> S.P. Porterfield, C.E. Hendrich, "The role of thyroid hormones in prenatal and neonatal neurological development-current perspective," *Endocrine Reviews* 14, 94-106, 1993.
- <sup>94</sup> H. Viberg, A. Fredriksson, and Per Eriksson, "Neurotoxicity of Different Polybrominated Diphenyl Ethers, Including PBDE 209," *Organohalogen Compounds* 65, 9-11, 2003.
- <sup>95</sup> H. Viberg, A. Fredriksson, and Per Eriksson, "Neurotoxicity of Different Polybrominated Diphenyl Ethers, Including PBDE 209," *Organohalogen Compounds* 65, 9-11, 2003; 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.
- <sup>96</sup> P. Eriksson, C. Fischer, and A. Fredriksson, "Co-exposure to a Polybrominated Diphenyl Ether (PBDE 99) and an Ortho-Substituted PCB (PCB 52) Enhances Developmental Neurotoxic Effects," *Organohalogen Compounds* 61, 2003; 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.
- <sup>97</sup> P. Eriksson et al, "Brominated Flame Retardants: A Novel Class of Developmental Neurotoxicants in Our Environment?," *Environmental Health Perspectives* 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," *Toxicological Science* 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," *Toxicological Science* 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. Ohm, 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.
- <sup>98</sup> Thomas McDonald, Cal/EPA, "A Perspective on the Potential Health Risks of PBDEs," *Chemosphere* 46, 745-755, 2002; R. Letcher, I. D'Sa, K. Valters, H. Li, E. Bennett, and M. Alaei, "Polybrominated Diphenyl Ethers and Hydroxylated and Methoxylated Analogues in Detroit River Fish," *Organohalogen Compounds* 61, 29-32, 2003.
- <sup>99</sup> National Toxicology Program (NTP), *Toxicology and Carcinogenesis Studies of Decabromodiphenyl Oxide (CAS No. 1163-19-5) in F344/N Rats and B6C3F1 Mice (Feed Studies)*. NTP, Research Triangle Park, North Carolina, 1986; Thomas McDonald, CalEPA, "A Perspective on the Potential Health Risks of PBDEs," *Chemosphere* 46, 745-755, 2002.
- <sup>100</sup> Jui-hwa Peng, Chean-yeh Chen, Chin-wang Huang, and Yuan-wu Chen, "Determination of Polybrominated Diphenyl Ethers and Polybrominated Dibenzo-p-dioxins/dibenzofurans in Flue Gas Analysis," *Organohalogen Compounds* 61, 231-234, 2003.
- <sup>101</sup> P. Eriksson, E. Jakobsson, G. Marsh, A. Bergman, "Photo Decomposition of Brominated Diphenyl Ethers in Methanol/Water,"

- 
- presented at the Second International Workshop on Brominated Flame Retardants, 14-16 May 2001, Stockholm, Sweden; H. Olsman et al, "Formation of Dioxin-like Compounds as Photo-products of Decabrominated Diphenyl Ether (DeBDE) During UV-Irradiation," *Organohalogen Compounds* 58, 41-44, 2002.
- <sup>102</sup> 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; Phone conversation with Steve Scheifers, 22 January 2004.
- <sup>103</sup> World Health Organization, International Labour Organization, United Nations Environment Programme, *Environmental Health Criteria For Chlorinated Paraffins*, 1996.
- <sup>104</sup> Travis Madsen, Susan Lee, and Teri Olle, Environment California Research and Policy Center, *Growing Threats: Toxic Flame Retardants and Children's Health*, 2003.
- <sup>105</sup> Toshiba Press Release, "Toshiba Develops Environmentally Sophisticated Package for Transistor ICs," 27 April, 1998, available at [http://www.toshiba.co.jp/about/press/1998\\_04/pr2701.htm](http://www.toshiba.co.jp/about/press/1998_04/pr2701.htm).
- <sup>106</sup> Todd Brady and Greg Clemons, "Going BFR-Free," presented at BFR Conference and Roundtable, September 24-25, 2002.
- <sup>107</sup> "The Ericsson list of banned substances in products," viewed January 22, 2004, at [http://www.ericsson.com/sustainability/supplier\\_guides.shtml](http://www.ericsson.com/sustainability/supplier_guides.shtml).
- <sup>108</sup> Press release, "DC/DC modules are DfE and high performance at standard module prices," viewed January 2004, at [www.ericsson.com](http://www.ericsson.com).
- <sup>109</sup> 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; Phone conversation with Steve Scheifers, 22 January 2004.
- <sup>110</sup> RTP Company, "Innovative Bulletin: Non-Halogen Compounds, Customized Thermoplastics for Flame Retardant Compliance," available at [www.rtpcompany.com](http://www.rtpcompany.com).
- <sup>111</sup> Dan Fagin, Marianne Lavell, Center for Public Integrity, *Toxic Deception*, 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
- <sup>112</sup> 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.
- <sup>113</sup> 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.
- <sup>114</sup> Lowell Center for Sustainable Production, *The Promise and Limits of the United States Toxic Substances Control Act*, October 10, 2003.
- <sup>115</sup> U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics, *Chemical Hazard Data Availability Study*, April 1998.
- <sup>116</sup> Bureau of Economic Analysis documentation of corporate profits by industry, viewed at: <http://www.bea.doc.gov/bea/newsrel/gdp303f.xls>.
- <sup>117</sup> JL Annett, et al., "Chronological trend in blood lead levels between 1976 and 1980," *New England Journal of Medicine*, 308(23), 9 June 1983.
- <sup>118</sup> Mazdai, Anita, et al., "Polybrominated Diphenyl Ethers in Maternal and Fetal Blood Samples," *Environmental Health Perspectives*, 111(9), July 2003.
- <sup>119</sup> Environmental News Service, "EU Lawmakers Vote Broad Fire Retardant Ban," 6 September 2001.
- <sup>120</sup> Greenpeace Press Release, "Ban on New Brominated Flame Retardant in the Netherlands Lets Everyone Breathe Easier," 30 Jan. 2003.
- <sup>121</sup> Ivan Lerner, "Flame Retardant Producers Are Guardedly Optimistic," *Chemical Market Reporter*, May 7, 2001.
- <sup>122</sup> "Great Lakes Chemical Corporation Company Profile," available at: <http://biz.yahoo.com/ic/10/10674.html>.
- <sup>123</sup> Environmental Protection Agency press release, "Brominated Flame Retardants to be Voluntarily Phased Out," November 3, 2003.
- <sup>124</sup> Great Lakes Chemical Corporation press release, "Thanks to New Technology, Great Lakes Chemical Corporation Announces That it will Cease Production of Penta-BDE Flame Retardant by End of 2004," 3 November, 2003, available at: [http://biz.yahoo.com/prnews/031103/clm043\\_1.html](http://biz.yahoo.com/prnews/031103/clm043_1.html).
- <sup>125</sup> "Albemarle Corporation Company Profile," available at: <http://finance.yahoo.com/q/ks?s=ALB>.
- <sup>126</sup> Albemarle Corporation Press Release, "Albemarle Develops Flame Retardant to Replace PentaBDE," 13 November, 2003.
- <sup>127</sup> Sharon Collins, "Do flame-retardants save lives or endanger children?" CNN Headline News, 5 May, 2003, available at: <http://www.cnn.com/2003/TECH/science/05/05/hln.hot.earth.pbde/>; U.S. Department of Health and Human Services, Public Health Service Agency for Toxic Substances and Disease Registry, *Draft Toxicological Profile for Polybrominated Biphenyls and*

