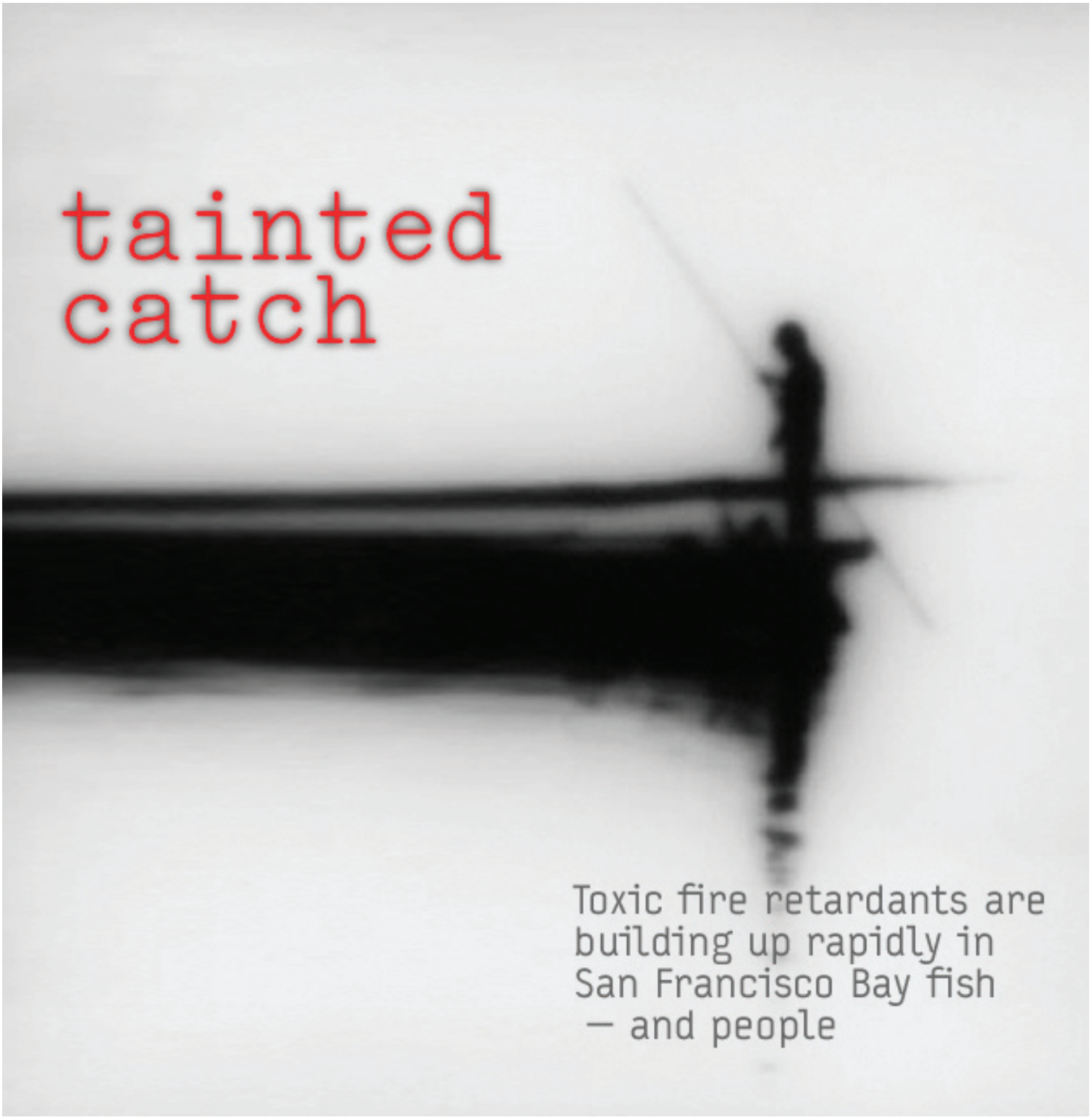

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EWG is a nonprofit research organization with offices in Washington, DC and Oakland, CA. EWG uses the power of information to educate the public and decision-makers about a wide range of environmental issues, especially those affecting public health.

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Executive Summary

Levels of a little-known class of neurotoxic chemicals found in computers, TV sets, cars and furniture are building up rapidly in key indicator species of San Francisco Bay fish, according to tests by the Environmental Working Group (EWG.)

Analysis of six species of Bay fish, conducted for EWG by a California state toxics lab, detected polybrominated diphenyl ethers (PBDEs) in every fish sampled. The tests compared fish caught by local anglers with archived samples caught in 1997, and found that PBDE levels more than doubled in halibut and more than tripled in striped bass. Striped bass and halibut are the two most commonly eaten species of Bay fish, and as large, mobile, carnivorous species, are good indicators of overall toxic contamination in the Bay.

These are the first findings for PBDEs in Bay fish. They add to the evidence that the Bay Area is a hotspot for exposure to bromine-based chemicals, widely used in commercial flame retardants, that many scientists warn are "the next PCBs" — a notorious class of chemicals banned in 1977 after evidence that they cause cancer and build up in people and the environment. The European Union has banned two of the most commonly used PBDEs, effective next year, but in the United States they remain virtually unregulated by either state or federal authorities.

PBDEs and other brominated fire retardants (BFRs) are similar in chemical structure to PCBs, which are still found in the bodies of people and animals more than 20 years after they were removed from commercial products in the United States. Recent research on animals has shown that exposure to low levels of PBDEs can cause permanent neurological and developmental damage including deficits in learning, memory and hearing, changes in behavior, and delays in sensory-motor development. Most at risk are pregnant women, developing fetuses, infants and young children, and to a lesser extent, the 10 million Americans with hypothyroidism.

Every day, a typical American comes in contact with dozens, if not hundreds, of consumer goods that contain PBDEs, including electronics, electrical cables, carpets, furniture, and textiles.

Although the pathway by which PBDEs and other brominated fire retardants get into the environment is largely still a mystery, the chemicals are now found worldwide in house dust, indoor and outdoor air and the water and sediments of rivers, estuaries and oceans. PBDEs have been found in the tissues of whales, seals, birds and bird eggs, moose, reindeer, mussels, eels, and dozens of species of freshwater and marine fish.

Rapid Increases in Humans

PBDEs are also building up rapidly in the bodies of people. Levels in Swedish breast milk samples were 55 times higher in 1997 than in 1972. The few breast milk samples collected from U.S. women indicate even higher levels of PBDEs in the bodies of first-time mothers than found in Europe and Canada. Already, scientists say, most Americans may carry in their bodies levels of PBDEs that have been found to cause serious, permanent neurological damage in laboratory animals.

Though still limited, the data on elevated levels of PBDEs in the Bay Area are disturbing. The levels of PBDEs found in San Francisco Bay fish are much higher than those found in commonly eaten fish species from Europe, Japan, the Pacific Northwest and the Great Lakes. Consumption of contaminated fish is believed to be a major route of PBDE exposure for adults. Earlier studies of PBDEs in the blood and breast tissue of Bay Area women, and of harbor seals from San Francisco Bay, have found levels from three to 60 times higher than levels measured in people and animals in Europe. Ninety-five percent of the type of PBDEs that bioaccumulate most readily is used in North America, and much of that amount goes into polyurethane foam sold in California, but it is unknown exactly why contamination is so high in the Bay Area.

In the fall of 2002, EWG researchers collected 22 fish from six of the most commonly eaten species at 10 locations around San Francisco Bay. Analysis conducted under contract by the state Department of Toxics Substances Control's Hazardous Materials Laboratory in Berkeley found that every sample contained seven different PBDEs, in concentrations ranging from trace amounts to more than 60 parts per billion (ppb) in fish tissue. We also tested for PBDEs in fish samples archived from 1997, and found that in five years, levels of the chemicals had increased in four of six species tested.

The California Legislature is considering a ban on some types of PBDEs in consumer products by 2008. AB 302 by Assemblywoman Wilma Chan of Alameda, which passed the Assembly in May 2003 and is pending a vote in the state Senate, would make California the first state in the nation to regulate PBDEs. The bill is an

important first step, but additional action will be necessary to fully protect public health. Some industries, notably many computer makers, are already moving toward safer alternatives, but the rapid buildup of PBDEs in people, animals and the environment makes it imperative that all brominated flame retardants must be phased out quickly.

The Next PCBs?

As highly flammable synthetic materials have replaced less-combustible natural materials in consumer products, chemical fire retardants have become ubiquitous in consumer products. Of the many different kinds of fire retardants, one of the most common is a class of bromine-based chemicals known as polybrominated diphenyl ethers, or PBDEs. Today PBDEs are in thousands of products, in which they typically comprise 5 to 30 percent of product weight. [1] During manufacturing, PBDEs are simply mixed in to the plastic or foam product, rather than chemically binding to the material as some other retardants do, making PBDEs more likely to leach out.

There are 209 structural variants, or congeners, of PBDEs, classified by the number of bromine atoms in a molecule of the chemical: Penta-BDEs have five bromine atoms, octa-BDEs have eight, deca-BDEs have 10, and so on. The commercial PBDE flame retardants are actually mixtures of several different congeners, with the three major products called Deca, Penta, and Octa. (The common name of the commercial product can be somewhat misleading; the Penta product, for example, is actually a mixture of 40 percent tetra-BDE, 45 percent penta-BDE and 6 percent hexa-BDE congeners.) Worldwide, Deca is the most widely used of the PBDEs with 83 percent of the global market by weight, followed by Penta with 11 percent and Octa with 6 percent. [2]

PBDEs are the chemical cousins of PCBs, another family of persistent and bioaccumulative toxins that came to the attention of regulators only after millions of pounds had been released into the environment. In the 26 years since PCBs were banned, numerous studies have documented permanent, neurological impairment to the developing child from low level PCB exposure. [3-7] Recent evidence suggests PBDEs and PCBs may work together to cause adverse health effects. Not only do PBDEs appear to be acting through the same pathways as PCBs and dioxins, but a 2003 study found that early exposure of lab animals to a combination of PCBs and PBDEs affected motor skills ten times more strongly than exposure to the individual chemicals. [8, 9]

PBDE use has skyrocketed in the last three decades, with Penta production almost doubling between 1992 and 2001. [2, 10] The market took off after the ban of a previously popular class of fire retardants, polybrominated biphenyls or PBBs, following

the catastrophic contamination of cattle feed in Michigan during 1973 and 1974 that exposed nine million people to tainted meat and dairy products. [11] Today, half of the PBDEs used worldwide are used in North America, with 73 million pounds being used in 2001. [2] An unknown amount of PBDEs, probably millions of pounds, is also imported into the country each year in manufactured goods. Chemical industry analysts say the North American market for brominated flame retardants is $1 billion a year and growing by about 3.7 percent annually; the European market is a little more than half that size. [12]

The Bromine Oligopoly

Worldwide, eight companies manufacture PBDEs, with the two largest in the U.S.: Great Lakes Chemical Corp. of West Lafayette, Ind., and Albemarle Corp. of Richmond, Va. In 2002, Great Lakes reported total sales for all products of $1.4 billion, up 4 percent from the previous year. Albermarle reported sales of $980 million, up 7 percent. [13, 14] To Americans familiar with toxics issues, the corporations are notorious as the manufacturers of methyl

Products Often Containing PBDEs

Materials used in	Types of PBDEs used	Examples of consumer products
Plastics	Deca, Octa, Penta	Computers, televisions, hair dryers, curling irons, copy machines, fax machines, printers, coffee makers, plastic automotive parts, lighting panels, PVC wire and cables, electrical connectors, fuses, housings, boxes and switches, lamp sockets, waste-water pipes, underground junction boxes, circuit boards, smoke detectors
Textiles	Deca, Penta	Back coatings and impregnation of home and office furniture, carpets, automotive seating, aircraft and train seating
Polyurethane foam	Penta	Home and office furniture (couches and chairs, carpet padding, mattresses and mattress pads) automobile, bus, plane and train seating, sound insulation panels, imitation wood, packaging materials
Rubber	Deca, Penta	Conveyor belts, foamed pipes for insulation, rubber cables
Paints and laquers	Deca, Penta	Marine and industry protective laquers and paints

Source: WHO 1994 [1], Danish EPA 1999 [103]

bromide, a volatile, acutely toxic, ozone-depleting pesticide gas used to fumigate strawberries, tomatoes and other crops. (Albemarle also has the dubious distinction of being a spin-off of Ethyl Corp., whose leaded gasoline additive was banned in the U.S. in 1972.) The main areas of bromine production in the world are southeastern Arkansas, where Great Lakes and Albemarle pump it from underground pools of brine, and Israel, where a company named Dead Sea Bromine extracts it from the briny inland sea. A chemical industry journal describes the global trade in brominated chemicals as "an oligopoly controlled by Albemarle, Great Lakes and the Dead Sea Bromine Group." [15]

Despite their heavy use, until recently data were scarce on the toxicity or environmental fate of PBDEs. But in the last few years, it has become clear that PBDEs and other brominated flame retardants have joined PCBs, DDT and dioxin on the list of persistent, bioaccumulative chemicals contaminating people, animals and the environment everywhere on Earth. PBDEs are now found in house dust, sewage sludge and the water and sediments of rivers, estuaries and oceans. They've been found in the tissues of whales, seals, birds and bird eggs, moose, reindeer, mussels, eels, and dozens of species of freshwater and marine fish. [16-21] Like scores of other industrial chemicals, they have also been found in human breast milk, fat and blood.

The reach of PBDE pollution is global, found essentially everywhere scientists have looked: Belgium, Canada, Czech Republic, Denmark, England, Finland, Germany, Greenland, Ireland, Israel, Japan, Korea, the Netherlands, Norway, Portugal, Russia, Sweden, Switzerland, Taiwan, and numerous U.S. locations. [16, 17, 19, 22-25] PBDEs can travel great distances. They've been found in birds and marine mammals in remote locations including the North Sea, the Baltic Sea and the Arctic Ocean. [26]

Of greatest concern is the exponential rate of PBDE increase in the environment. PBDEs in California harbor seals increased by a factor of 100 between 1989 and 1998, and in Lake Ontario trout by a factor of more than 300 between 1978 and 1998. [27, 28] Similar dramatic increases have been seen in human blood samples from Norway, ringed seals from the Canadian arctic, and gull eggs from the Great Lakes region. [18, 29, 30] In each of these studies, the time it took for PBDEs to double in concentration was remarkably short — from less than two years to five years.

U.S. Dominates Global Use of PBDEs

The problem is global, but the U.S. is clearly a hotspot. The average PBDE concentration found in the breast tissue of California women was among the highest yet reported — three times higher than Swedish tissue samples, 10 times higher than German blood samples and Canadian milk samples, and 25 times higher than Spanish tissue samples. [27]

It is still unknown why U.S. levels are so much higher than in other industrialized nations, but part of the explanation is the kind of PBDEs favored by American manufacturers. North America uses the lion's share of all the various PBDE products — 44 percent of global Deca production by weight and 40 percent of Octa — but uses an estimated 95 percent of global Penta production. [2] The commercial Penta product is almost exclusively used in flexible polyurethane foam for home and office furniture, carpet padding, and mattresses. But only about 7.5 percent of the more than 2.1 billion pounds of foam produced in the United States each year contains penta-DBE. The majority of the Penta-laden foam is sold in California, where components of upholstered furniture are required to meet stringent fire retardancy standards. [31] Research shows that Penta is by far the most likely of the PBDEs to be absorbed by and build up in living organisms.

A separate but related concern is that PBDEs can form polybrominated dioxins and furans (PBDD/Fs) when heated or burned — in a municipal solid waste incinerator, for example. [32] Low levels of the very similar polychlorinated dioxins and furans are known to cause cancer, birth defects and chloro-acne. PBDD/Fs have recently been measured in human tissue samples and the environment in Japan. [32, 33]

Global Use of PBDEs in 2001
(in thousands of pounds)

Commerical PBDE Product	Americas	Europe	Asia	Other	Total	Percent used in the Americas
Deca	54,010	16,760	50,710	2,315	123,700	44%
Penta	15,650	331	331	221	16,530	95%
Octa	3,307	1,345	3,307	397	8,356	40%

Source: BSEF 2002 [93]

PBDEs in Bay Area Fish and People

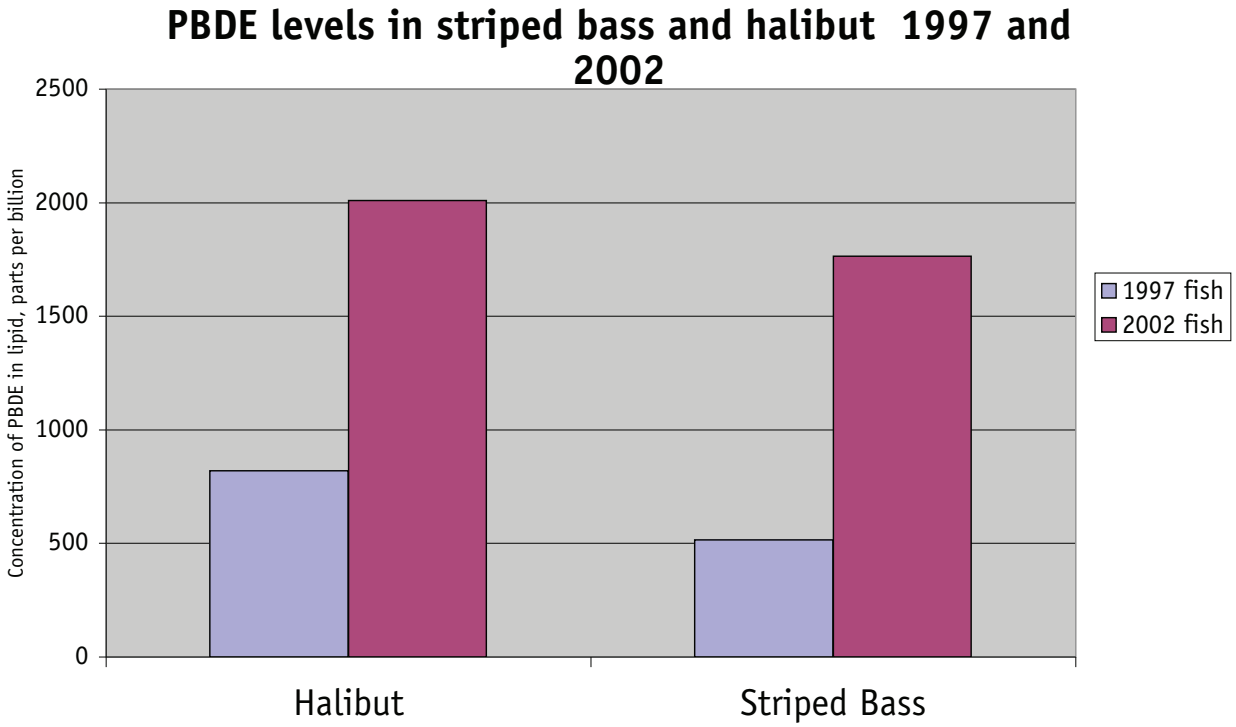
Results of EWG fish sampling

From September to November 2002, EWG researchers visited public piers and other fishing locations around greater San Francisco Bay. We asked anglers to donate fish caught that day they planned to eat. We collected 22 fish from six of the 10 most commonly caught and eaten species in the Bay: halibut, striped bass, white croaker (also called kingfish), walleye surfperch, jacksmelt and leopard shark. Halibut, bass and shark samples were collected from anglers on private or charter boats around South San Francisco or in San Pablo Bay. All other samples were donated by fishermen at public piers in San Francisco, South San Francisco, Alameda, Berkeley, Richmond and Point Pinole. EWG researchers prepared the samples as the species are typically eaten — skinning the shark, halibut and bass but leaving the skin on the croaker, surfperch and smelt. [34]

Samples of the same species caught in 1997 were obtained from the fish tissue archives of the Regional Monitoring Program For Trace Substances (RMP), part of the San Francisco Estuary Institute. The RMP collects and tests Bay fish every three years for PCBs, mercury, pesticides and other contaminants, and in future will include testing for PBDEs. The RMP samples were selected to include fish from the same general areas of the Bay as the fish EWG collected in 2002. Because the RMP did not collect walleye surfperch, we compared shiner surfperch from 1997 to the walleye surfperch we collected in 2002.

Both sets of samples were analyzed under contract by the Public Health Institute in collaboration with California Department of Toxic Substance Control's (DTSC's) Hazardous Materials Lab in Berkeley, where they were tested for 11 different classes of PBDEs by scientists recognized worldwide as pioneers in research on brominated fire retardants. Every sample analyzed by the lab was found to contain the seven most common PBDEs, and four other PBDE congeners were found in some fish. The samples contained levels of PBDEs ranging from 1 to 62 parts per billion (ppb) wet weight.

PBDE levels varied widely between fish species and between individuals of the same species. This variation may stem from difference in the fat content of the particular fish, their diet,



age, metabolism, location in the Bay and/or differences in the way they each species was prepared. While the fish samples compared fish collected from similar geographic areas, there are some differences in the locations represented in the 1997 and 2002 fish. RMP samples were typically collected from boats in the deeper reaches of the Bay, whereas our samples were donated by anglers, in most instances fishing in the shallower, and possibly more polluted, waters from public piers.

In general, white croaker and surfperch had the highest PBDE levels in fish fillets; leopard shark and jacksmelt had the lowest levels. Both the wide range of contaminant levels and the higher concentrations of chemicals in croaker and surfperch have been reported in previous studies of PCBs and persistent pesticides in the same species and locations of the Bay. [34] The single most contaminated fish was a white croaker collected at the Richmond Inner Harbor, a location known for high levels of pollution. This specimen had 62 ppb of PBDEs wet weight.

Comparing EWG's 2002 samples to the RMP's 1997 samples suggests that the concentrations of PBDEs in the fat of San Francisco Bay fish increased rapidly in striped bass and halibut over the past five years. PBDE levels in striped bass fat were

PBDE levels in 2002 San Francisco Bay Fish, ppb wet weight

Fish species	Average Level of PBDEs in fish tissue *	Lowest value	Highest value	Average level of PBDEs in fish fat*	Number of fish tested
Leopard Shark	1	1	NA	474	1
Jacksmelt	6	2	10	282	5
Halibut	13	2	28	2009	4
Striped Bass	17	11	21	1756	4
Walleye Surfperch	22	9	39	672	4
White Croaker	40	17	62	651	4
Overall average	16	1	62	974	22

Sum of the seven most common congeners (PBDE 28, 33, 47, 99, 100, 153, 154)

* NOTE The overall concentration of PBDEs in fish fillets is very different than the PBDE concentration in fish fat. We report the concentrations in fish fillets here because they are more relevant when considering potential human exposure. Concentrations in fish fat are a better way to track changes in contaminant levels over time, because the concentrations of fat in individual fish can vary widely.

3.4 times higher and in halibut fat were 2.4 times higher than samples collected in 1997. According to the California Department of Health Services, bass and halibut are the most commonly eaten Bay fish, and as larger, mobile, carnivorous fish at the top of the food chain, are key indicators of overall Bay contamination. Leopard shark fill a similar ecological niche on the top of the food chain, but our analysis included only one sample from 2002, which makes it less likely to detect a time trend if it were to exist. Less of an increase was observed in white croaker, a smaller, fattier fish that eats lower on the food chain. Surfperch and jacksmelt did not increase.

Based on our analysis, we calculate a doubling rate for PBDE concentrations of 2.8 years for bass and 3.9 years for halibut. The bass and halibut species were roughly the same length and had similar levels of fat when analyzed in 1997, which indicates that the differences we observed are not likely due to different age or fat contents of the fish. The detailed results of our analysis are presented in the Appendix.

PBDEs in Seals, Birds and Water from the Bay

The high level of PBDE contamination and rapid increase in PBDE levels in bass and halibut samples was no surprise. PBDEs have been documented in San Francisco Bay harbor seals, bird eggs, water and sediments. [27, 35, 36] Over the past 20 years, a rising tide of PBDEs has been detected by almost every study that looked at trends over time. In 2002, researchers at DTSC found that PBDE levels in San Francisco Bay Harbor Seals doubled every 1.8 years between 1989 and 1998. [27] Recently published data show a doubling time of 1.6 years in fish collected in the headwaters of the Columbia River in Washington state between 1992 and 2000. [37] PBDE levels in the fat of trout in Lake Ontario increased steadily between 1978 and 1998, by a factor of more than 300. [38] Similar increases in PBDEs over a 15-to-20 year period have been found in Arctic ringed seals and beluga whales. [18, 39]

The levels of PBDEs measured in Bay fish are much higher than those reported for a variety of commonly eaten fish in a variety of locations. These include wild and farmed salmon [40-42], commonly eaten fish in Japan, Sweden, Finland [43-45], similar species in the Great Lakes, Pacific Northwest and Bering Sea, and Washington state trout. [46-49] However, the levels detected in Bay fish are still lower than levels reported in studies of bottom feeding fish like carp collected in Virginia, and fish living downstream from a plastics and textile manufacturer in Sweden. Consumption of fish is thought to be a major route of PBDE exposure for adults. [31]

Comparison of PBDE levels in fish fat 1997 and 2002

Fish Species	Average levels in 1997 fish	Average levels in 2002 fish	Percent increase
Leopard Shark	438	474	8%
Jacksmelt	312	282	no increase
Halibut	821	2009	145%
Striped Bass	516	1764	240%
Surfperch	903	672	no increase
White Croaker	564	652	15%

Concentration of PBDE in lipid, parts per billion

The rapid buildup of PBDEs in the human body was first documented in 1999 by Swedish researchers, who examined archived breast milk samples collected over a 25-year span. They found a 60-fold increase in the concentrations of PBDEs in breast milk between 1972 and 1997 — equivalent to a doubling every 5 years. They noted that the increase was startling, given that levels of many persistent chemicals declined sharply in the same period. [50, 51] Later, Canadian researchers reported a 15-fold increase in PBDE levels in the breast milk of women in Vancouver, B.C. between 1992 and 2002 — a doubling every 2.6 years. [52]

The Swedish findings led to additional studies and the eventual ban of most PBDEs in the European Union, beginning in 2004. However, Europeans' exposure to PBDEs are likely much lower than Americans'. The few breast milk samples collected from U.S. women indicate even higher levels of PBDEs in the bodies of first-time mothers than found in Europe and Canada. [53] There are no historical archives of breast milk samples from the U.S., so it is not possible to track the trend of PBDE buildup in American women as meticulously as it has been documented in Sweden. But the available data are disturbing.

Levels in U.S./Bay Area Women Highest Worldwide

The average PBDE concentrations found in breast tissue, blood, and breast milk samples from studies of U.S. women are the highest yet reported in the world. Two recently published studies

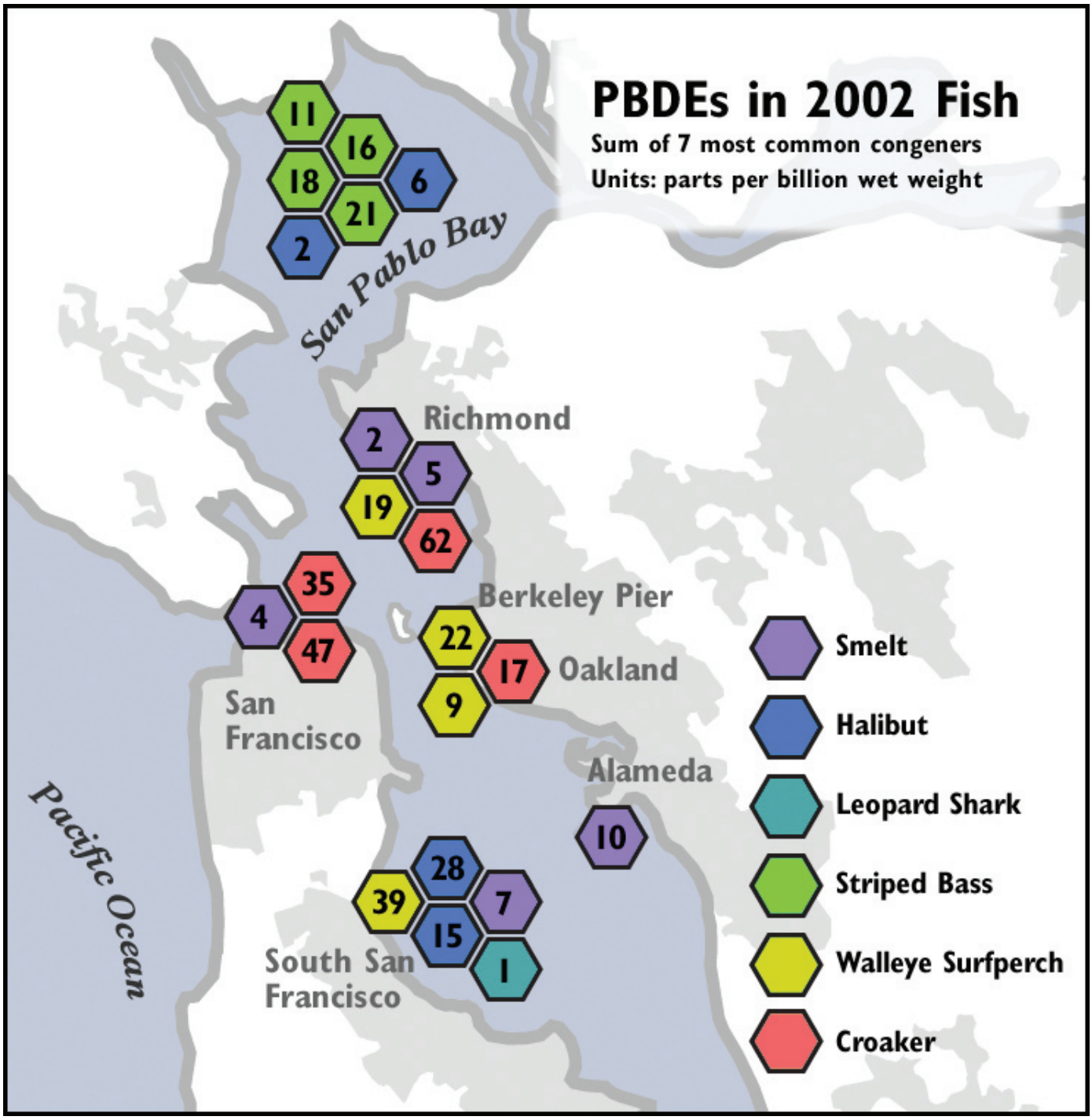
indicate that PBDE levels in Bay Area women have risen by at least a factor of 3 to 5 since PBDEs were first introduced in commercial products about 30 years ago. They also found levels of PBDEs three to five times higher than a study of German blood samples, and 12 to 30 times higher than women's breast milk in Japan, Sweden and Finland. [20, 43, 50, 54] About one in 12 Bay Area women in the two studies had more than 100 ppb of the most common PBDE in her body fat — more than 100 times higher than average levels found in Swedish women during this time period. [55]

A growing body of evidence shows a very low threshold for PBDE to cause permanent impacts to the development of the nervous system. It is hard to say whether PBDE concentrations have reached that point, but scientists are concerned that the margin between known contamination levels and levels that cause health effects in laboratory animals is low — and shrinking rapidly.

Levels of PBDE-47 in California women
parts per billion in lipid

Study	Year of sample collection	Average level of PBDEs	Range	Number of participants
Bay Area women's blood	1960s	Non-detectable	All below 10	42
Bay Area women's breast tissue	late 1990s	29	5 to 196	32
Bay Area women's blood	late 1990s	50	4 to 511	50

Source: Petreas 2003 [55]


PBDEs in 2002 Fish
Sum of 7 most common congeners
Units: parts per billion wet weight
San Pablo Bay
Richmond
Berkeley Pier
Oakland
Alameda
San Francisco
South San Francisco
Pacific Ocean
11
16
18
6
21
2
2
5
19
62
35
4
47
22
17
9
10
28
39
7
15
1
Smelt
Halibut
Leopard Shark
Striped Bass
Walleye Surfperch
Croaker

Sidebar

San Francisco Bay Fish: Who's At Risk?

The PBDEs found in EWG and DTSC's tests join a list of known toxins in San Francisco Bay Fish, including PCBs, mercury and dioxins. As a result, state health officials advise that most adults should eat no more than two meals a month of fish from the Bay. The exceptions, however, are crucial.

The Office of Environmental Health Hazard Assessment (OEHHA) says women who are pregnant, breastfeed or considering pregnancy, and children 5 and under, should eat no more than one meal of Bay fish a month. Additional warnings have been issued against eating certain fatty fish, older fish and fish from more severely polluted parts of the Bay.

To further reduce toxic exposure, the Department of Health Services (DHS) says people who eat Bay fish should:

- Eat younger, smaller and less fatty fish.
- Eat several species of fish from a variety of locations.

Fish Consumption Advisories for the San Francisco Bay

Fish Species/Location	All adults	Women who are pregnant, breastfeeding or considering pregnancy (within one year), and children ages 5 and younger
All species in the San Francisco Bay	No more than 2 meals per month	No more than 1 meal per month
Striped bass	No fish over 35 inches long	No fish over 27 inches long
Leopard shark	No advisory	No fish over 24 inches long
Croaker, surfperch, bullhead, gobies or shellfish	None of these species caught in the Richmond Harbor Channel	

OEHHA http://www.oehha.ca.gov/fish/general/sfbaydelta.html and http://www.oehha.ca.gov/fish/preg/index.html

- Skin and trim the fish to remove fat.
- Eat only the fillets, not organs or eggs.
- Cook the fish thoroughly to kill parasites.
- Bake, broil, grill or steam the fish to drain juices, which can reduce toxic levels by a third or more.

Despite official advisories, there is ample evidence that anglers are still risking their health or others' health by eating Bay fish.

In 1998-99, DHS interviewed 1,300 people fishing at more than 150 sites around the greater Bay. Eighty-five percent of the anglers said they eat their catch; the rest give the fish away away or release them). More than half of those surveyed said they share their catch with a woman of child-bearing age or a young child, who are most at risk from exposure. DHS estimated that the average person fishing in San Francisco Bay eats seven ounces of fish in a meal. Other studies by two nonprofit groups, the Asian Pacific Environmental Network and Save the Bay, say some people eat considerably more than that.

About six in 10 of the anglers surveyed by DHS said they had a general awareness that the state had issued fish advisories for the Bay, but only a third could name even one of the precautions to limit consumption of contaminated fish. Disturbingly, DHS found that the anglers' knowledge of the advisories correlated with their race and income: Poorer anglers and people of color were less likely to know about the government's advice to limit fish consumption.

The survey found a number of other indicators that people of color are more at risk of eating unhealthy levels of contaminated Bay fish. African-Americans and Asian-Americans were more likely to exceed the advisory level of two meals a month. African-Americans and Asian-Americans, plus Latinos, also were the most common consumers of croaker, the species that had the highest level of PBDEs, and which other studies have found to contain the highest levels of PCBs and pesticides.Since people of color in the Bay Area are more likely to live in neighborhoods near toxic pollution, anglers in those communities are also more likely to fish in the more highly contaminated parts of the Bay.

Section Reference:

California Department of Health and Human Services (DHS), Environmental Health Investigations Branch and San Francisco Estuary Institute. 2001. San Francisco Bay Seafood Consumption Study. March, 2001. http://www.sfei.org/rmp/sfcindex.htm

Health Risks of PBDEs

A growing body of research in laboratory animals has linked PBDE exposure to an array of adverse health effects including thyroid hormone disruption, permanent learning and memory impairment, behavioral changes, hearing deficits, delayed puberty onset, fetal malformations and possibly cancer. Research also shows that exposure to brominated flame retardants in utero or infancy leads to much more significant harm than adult exposure, and at much lower levels. Many questions still remain, but almost every month brings new evidence that PBDEs pose a significant health risk to developing animals and are very likely to pose a significant health risk to fetuses, infants and children.

These findings echo what researchers have learned about the structurally similar, but much better known, PCBs. Used primarily as electrical insulators, PCBs were found to be rapidly building up in people and animals before they were banned in 1977. Although levels are now declining, PCBs persist in the environment and cause a number of well-documented health problems. Recent studies have shown that PBDEs can act in concert with PCBs and other chemicals through similar mechanisms to increase their effects. [8, 9, 56]

Most unsettling, comparison of PBDE concentrations in the bodies of American women with the levels shown to harm the health of laboratory animals, the margin of safety is slim or may already be eclipsed. If PBDE levels in people continue to rise at anywhere near current rates, any remaining gap will likely be closed within a few years.

Many of the known health effects of PBDEs are thought to stem from their ability to disrupt the body's thyroid hormone balance, by depressing levels of the T3 and T4 hormones important to metabolism. In adults, hypothyroidism can cause fatigue, depression, anxiety, unexplained weight gain, hair loss and low libido. This can lead to more serious problems if left untreated, but the consequences of depressed thyroid hormone levels on developing fetuses and infants can be devastating. [57] One study, for instance, found that women whose levels of T4 measured in the lowest 10 percent of the population during the first trimester of pregnancy were more than 2.5 times as likely to have a child with an IQ of less than 85 (in the lowest 20

percent of the range of IQs) and five times as likely to have a child with an IQ of less than 70, meeting the diagnosis of "mild retardation." [58]

Even short-term exposures to commercial PBDE mixes or individual congeners can alter thyroid hormone levels in animals, and the effects are more profound in fetuses and offspring than in adults. [59-64] These results aren't surprising, but are ominous as data in humans indicate that pregnancy itself stresses the thyroid, and developing fetuses and infants don't have the thyroid hormone reserves adults do to help buffer insults to the system. [65]

Most studies on thyroid hormone disruption by PBDEs have been very short — with exposures of 14 days or less. The real question is how low doses over the long term affect the body's thyroid hormone balance. The answer is important, because the entire U.S. population is exposed daily to low levels of PBDEs, and studies of other thyroid hormone disrupters have found that long-term exposures can cause more serious harm at lower levels of exposure. [66] Although no direct link could be made, one study found higher rates of hypothyroidism among workers exposed to brominated flame retardants on the job. [67]

Because the developing brain is known to be extremely sensitive to exposure to toxins, researchers have begun to examine whether short-term exposures to PBDEs at critical times could have long-term effects. The results are troubling: Doses administered to fetal or newborn mice and rats caused deficits in learning, memory and hearing, changes in behavior, and delays in sensory-motor development. Many of these effects were found to worsen with age and the effects were seen with the higher-weight PBDEs (the usually less harmful deca-BDE) as well as the more readily absorbed lower-weight congeners.

Just One Dose May Be Harmful

Experiments have shown that just one dose of PBDEs at a critical point in brain development can cause lasting harm. [68-70] In two different studies a small dose — as little as 0.8 milligrams per kilogram of bodyweight per day (mg/kg-day) — given to 10-day-old mice caused "deranged spontaneous behavior," significant deficits in learning and memory and reduced ability to adapt to new environments, with these problems often becoming more pronounced with age. [69, 70] This research also demonstrated the heightened sensitivity of the brain at certain critical phases of development and the importance of timing: While earlier exposures caused "significantly impaired spontaneous motor behavior" and "persistent neurotoxic effects," no effects were seen in mice that were exposed later on during development,

despite having similar levels of PBDEs (or their metabolites) in the brain. [69]

Other animal studies have shown that early exposure to PBDEs, often at relatively low levels, can lead to delays in sensory-motor development, hearing deficits, as well as changes in activity levels and fear responses. [68, 71, 72] At this point, scientists do not understand exactly how PBDEs affect neurological development. But there is evidence that PBDEs and/or their metabolites are in fact acting through several different mechanisms, including mimicking thyroid hormones, increasing their rate of clearance in the body, and interfering with intracellular communication. [73]

In addition to their effects on thyroid hormones and neurological development, PBDEs have been linked to a gamut of other health impacts, from subtle to dramatic. For example, two new studies found that early exposure to PBDEs delayed the onset of puberty in male and female rats and decreased the weight of male reproductive organs. [74, 75] In studies of pregnant animals, PBDE exposure was associated with retarded weight gain, enlarged livers and raised serum cholesterol. [76, 77] In utero exposures have also been associated with serious harm to the fetus, including limb and ureter malformation, enlarged hearts, bent ribs, fused stemebrae, delayed bone hardening, and lower weight gain. [76-79] The malformations of the fetus were consistently seen at levels much lower than doses harmful to the mothers — the lowest being 2 and 5 mg/kg-day, respectively.

The few studies that have looked at changes in organ structure have found that semi-chronic PBDE exposure can cause thyroid hyperplasia and enlarged livers at relatively low doses (10 mg/kg-day) and other adverse effects such as hyaline degeneration, focal necrosis and deformation in the kidney, hyperplastic nodules in the liver, decreased hemoglobin and red blood cell counts at higher doses. [76, 78, 80]

Only one PBDE congener has been tested for causing cancer, in a single study more than 15 years ago. High doses of deca-BDE given to rats and mice caused liver, thyroid and pancreas tumors. [80] Deca-BDE is the least easily absorbed and the most rapidly eliminated of the PBDEs, and recent research indicates that other congeners can cause genetic recombination in cells, a sign of likely carcinogenicity. [81] As a result, scientists believe that the congeners with fewer bromines are likely to be more carcinogenic than deca-BDE and have urged that such tests be conducted. [73]

Unanswered Questions

There are many unanswered questions about the health effects of PBDEs. For example, it is unclear to what extent PBDEs are metabolized by the body and what health effects these breakdown products might have. Because the composition of many commercial mixes hasn't been well characterized, there may be harmful congeners or other chemical contaminants that scientists aren't even looking at. [19] And there remains the question of the health effects of polybrominated dioxins and furans, formed when PBDEs are heated or burned.

One of the major debates centers on the health effects of the various PBDE congeners. Scientists have found that PBDEs with fewer bromines (including penta-BDE) are almost totally absorbed by the body, slowly eliminated, highly bioaccumulative, and cause health effects at relatively low levels. In contrast, PBDEs with more bromines (including octa- and deca-BDE) are less readily absorbed, less bioaccumulative, more quickly eliminated by the body, and generally cause health effects at higher doses. [73] But new research suggests that deca-BDE may be more toxic than previously thought. [82] And maybe even more importantly other recent research shows that when exposed to sunlight, the higher-weight, less harmful congeners can be chemically converted to the more bioaccumulative, better absorbed and more toxic lower-weight congeners. [83-86]

Although there are significant differences between how the environment and organisms deal with the various PBDE congeners, the bottom line is that all have the potential to cause serious environmental and health problems — some alone, some through their breakdown products, others by interacting with other toxic chemicals. The chemical industry, trying to save a highly profitable product, is pushing the notion that certain PBDEs are harmless. The evidence already available argues the opposite: to prevent a bad situation from getting worse, all PBDEs should be banned now.

Scientists have really only begun to examine the potential health effects of PBDEs, but as more studies are conducted, the threshold for health effects continues to be set lower and lower, similar to the regulatory trend with lead, mercury and PCBs. One of the lowest doses of PBDEs found so far to harm lab animals was a 2002 study of newborn mice showing neurodevelopmental damage at concentrations of 4 ppb in brain tissue. [69] Many women in the two recent California studies had PBDE levels above this level in her body fat. Scientists at the California Environmental Protection Agency's Office of Environmental Health Hazard Assessment used the state data, plus studies from Indiana and

Texas, to project PBDE levels in the entire U.S. population. If the data used in the model are representative, as many as 15 million Americans have a PBDE body burden of more than 400 ppb — a hundred times the concentration known to cause permanent effects in laboratory animals. [9]

The highest concentrations of PBDEs measured to date in U.S. breast milk are only slightly lower than level of the PBDE cousin, PCBs, shown to cause significant irreparable harm (lower IQ and memory and attention deficits) to a developing baby. [87] Researchers estimate that if the increase in PBDE levels in our bodies continues at this rate, U.S. women will exceed these levels within 6 to 12 years. The costs of this damage will be an increased need for special education, decreased lifetime earnings for affected children, and treatment for the learning, developmental and behavioral disabilities that already affect nearly 12 million U.S. children. [88]

Regulatory Failure

The evidence against PBDEs was strong enough that bans were proposed in Germany, Sweden and the Netherlands in the mid-1980s and early 1990s. Industrial users of the chemicals agreed to voluntarily phase them out in Germany in 1986, with the manufacturers and users in the other two countries later following suit. In 1993 Germany placed official restrictions on PBDE use because of their tendency to release dioxins when burned under its Dioxin Ordinance. [85] As concern spread to other countries, the European Union launched a scientific review of the safety of PBDEs, originally with respect to electronics waste. In February 2003, the EU announced a ban on two common PBDEs (Penta and Octa) in all products as of August, 2004. [89] The EU is also considering a ban on Deca for use in electronic products by July 2006. Pending the completion of further studies, the EU Chemicals Inspectorate will decide whether to ban on Deca in other non-electronic products as well as of 2006. [90]

Even before the ban takes effect, the early efforts to reduce PBDE use in Europe are paying off. Researchers have found that PBDE levels in Swedish breast milk rose exponentially from 1972 to 1997, but since that year have begun to decline: PBDE levels in Swedish women dropped about 30 percent between 1997 and 2001. [91] These results are encouraging. This shows that if regulations are enacted and PBDE use ceases or declines, the human body burden of PBDEs will also decrease after a lag-time of several years or more.

Despite that fact that PBDE concentrations in Americans and their environment are at least ten times higher than those found in Europe, the U.S. government has so far done nothing to counter this rapidly escalating problem. PBDEs are virtually unregulated for use in commercial products. In 1994, EPA determined that the waste stream from the production of Octa and Deca "should not be listed as hazardous." [92] The only other regulation governing PBDEs is the requirement that companies manufacture or use large amounts of Deca report their chemical emissions under the Toxics Release Inventory.

California Bill a First Step

State legislation was introduced in California this year to ban the use of several types of PBDEs by 2008. While this bill, AB 302 by

Assemblywoman Wilma Chan of Oakland, is a welcome first step, it lacks some key provisions to assure that these chemicals are removed from our homes and our bodies as quickly as possible:

- AB 302 exempts the most widely used PBDE product (Deca), which is common in electronics produced in the United States, but the European Union has been considering banning Deca in electronic products by 2006. This is troublesome, as numerous studies have shown that the types of PBDEs in this commercial product can break down into other congeners that are much more bioaccummulative and bioreactive (and are included in the proposed California legislation).

- As passed by the Assembly, the bill gives PBDE producers and users until 2008 to stop using the chemicals. But if PBDE use continues at its current rate for five years, another 365 million pounds of PBDEs will be put into American couches, easy chairs, cars, planes, buses and other consumer products. [93]

- The bill also doesn't require manufacturers to label PBDE-containing products. As it is now, it would remain impossible to know whether the couch or computer you buy contains PBDEs. Labeling would have allowed consumers to make more informed decisions, providing extra incentive to manufacturers and users to speed their conversion to new fire retardants, materials, or design.

Not surprisingly, the European and California drives to regulate PBDEs have met fierce opposition from manufacturers and users. In 1997 Great Lakes, Albemarle, Dead Sea Bromine and other companies formed the Bromine Science and Environmental Forum (BSEF). Ostensibly dedicated to providing "extensive scientific information on bromine and bromine products" and facilitating "open communication about bromine products across the globe," the Forum is in fact a lobbying front dedicated to casting doubt on the mounting evidence against brominated chemicals. [94]

For example, BSEF denies that the burning of bromine-contaminated waste increases the formation of dioxins and furans, though numerous studies show otherwise. The group also tries to downplay the environmental and public health threats of PBDEs, claiming the chemicals are only used "in controlled applications where emissions to the environment are highly unlikely." [94] Yet

volumes of evidence show that PBDEs are not only escaping into the environment, but that they have become a ubiquitous global pollutant.

Chemical manufacturers been vocal opponents against the California legislation. Great Lakes Chemical, which says it spends $2 million a year to lobby against BFR regulation, pushed for a longer phase-out date and the exemption for Deca. [95]

Affordable Replacements

For most uses of PBDEs there are already chemical replacements on the marketplace at equivalent or slightly higher cost. Aluminum trihydroxide and various phosphorous-based compounds are some of the most common alternatives. But rather than replacing one chemical with one that may turn out to be even more toxic, the answer is to redesign products so that chemical flame retardants are not needed to meet fire safety regulations. The U.S. Consumer Product Safety Commission recently reported: "CPSC laboratory tests have demonstrated that the properties of actual filling materials have little or no effect on the small open flame ignition resistance of full-scale chairs" — in other words, the use of flame retardants in foam does little to improve upon the fire safety of foam furniture. [96] For other products, simply increasing the density of polyurethane foam can eliminate the need for chemical flame retardants. This can also be achieved using manufacturing materials that are naturally less flammable. [97]

Some U.S. companies have begun to phase out PBDEs, even without a regulatory mandate. Computer and electronics companies such as Apple, Ericsson, IBM, Intel, Motorola, Panasonic, Phillips and Sony are already producing some PBDE-free products, and some have committed to completely phasing out PBDEs and other brominated flame retardants. [97] The furniture giant IKEA has phased out BFRs in all its products by changing product design, using naturally less-flammable materials, and employing alternative flame retardants if needed. Hickory Springs of Conover, N.C., a major polyurethane foam producer, is working with Akzo Nobel, a chemical manufacturer, to test a non-halogenated phosphorous-based flame retardant. Hickory Springs says it was motivated by requests from companies such as IKEA, Crate & Barrel and Eddie Bauer to stop using PBDEs. [98]

Unfortunately, data on the toxicity of the alternative fire retardants already in use or under development is scarce. This is largely because of well-documented shortcomings of the nation's toxics laws. The chief regulatory statute for commercial chemicals,

the Toxic Substances Control Act (TSCA), is infamous for the lack of authority it provides the Environmental Protection Agency. [99] The looming PBDE crisis is another disturbing illustration of the failures of a regulatory system that allows persistent, bioaccumulative toxins on the marketplace before they have been adequately tested for safety.

Under the current system, the EPA reviews new chemicals through a process that does not require health and safety test data and that discourages voluntary testing. Companies submit only basic toxicity data for fewer than half of all applications for new chemicals, and the government approves 80 percent of these with no restrictions and no requests for tests. Eight of 10 new chemicals win approval in less than three weeks, at an average rate of seven a day. [99]

No Safety Studies on Many Toxic Chemicals

Worse, when TSCA was enacted in 1976, more than 63,000 chemicals already in use were "grandfathered" — granted blanket approval for continued use in consumer and industrial products. In 1998, the EPA and the nonprofit Environmental Defense Fund reviewed all of the toxicity and environmental fate studies publicly available and found no information — not a single test — for 43 percent of the 2,600 chemicals produced in the highest volumes in the U.S. [99, 100]

The chemical industry has since agreed to do more tests to assess potential toxicity to children for a select number of the most widely use chemicals under the Voluntary Children's Chemical Exposure Program (VCCEP). The three most widely used PBDEs were included in the first group of 23 chemicals to be assessed as part of this program, but the usefulness of the VCCEP program is highly limited. Its purpose is to make "health effects, exposure, and risk information" of these chemicals available and provide "the means to understand the potential health risks to children." [101] But because the program is voluntary, chemical manufacturers are unlikely to hand over any information that might be damning for their chemical products, nor do they have much incentive to fill any significant scientific data gaps that are identified in the process.

There is no question that fire safety is important and that making products fire-resistant can save lives. Chemical flame retardants have become ubiquitous over the last few decades, but a wide variety of fire safety strategies exist. Using less-flammable materials or changing the product design so that it is inherently

more fire resistant are chemical-free solutions. Using less toxic chemicals as flame retardants is another option. We do not have to expose ourselves to toxins to protect ourselves from fire.

EWG recommends:

- The EPA must ban all PBDEs as quickly as possible — no later than 2006.

- In the interim, all products containing PBDEs must be labeled so that consumers have the option of choosing products without them.

- All potential replacement fire retardants must be adequately tested to ensure that they are neither persistent, nor bioaccumulative, nor toxic. Changes in product design that decrease the need for chemical fire retardants should be encouraged over simply switching to a different chemical.

- A nationwide biomonitoring program is needed to identify chemicals that are accumulating in our bodies and in the environment, and determine whether levels are increasing or decreasing.

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Appendix

Appendix: Summary data for 1997 and 2002 fish

Fish samples Fish species	Year	Number in sample	Fish length (cm)	Reported moisture	Percent lipid, wet weight
walleye surfperch	2002	4	19 (16-21)	72 (71-85)	3.2 (2.4-4.0)
shiner surfperch	1997	40	12 (11-15)	76 (71-78)	2.5 (1.7-3.9)
halibut	2002	4	72 (69-76)	76 (75-86)	0.5 (0.2-0.9)
	1997	3	76 (59-92)	75 (73-77)	0.3 (0.2-0.4)
jacksmelt	2002	5	35 (33-37)	72 (67-75)	3.2 (0.5-7.4)
	1997	15	26 (21-29)	74 (71-76)	2.1 (1.4-3.2)
white croaker	2002	4	30 (28-31)	72 (70-74)	6.3 (5.2-7.4)
	1997	10	26 (22-29)	71 (70-73)	6.7 (5.2-7.4)
striped bass	2002	4	59 (55-69)	77 (75-78)	1.1 (0.6-2.1)
	1997	6	58 (50-66)	76 (75-77)	0.9 (0.8-1.0)
leopard shark	2002	1	114	79	0.3
	1997	12	96 (92-102)	76 (75-78)	0.25 (0.18-0.34)

Wet weight values, parts per billion

Fish species	Year	tri-BDE (PBDE 28+33)	tetra-BDE (PBDE-47)	penta-BDE (PBDE-99+100)	hexa-BDE (PBDE-153+154)	sum of 7 congeners
walleye surfperch	2002	0.5	17.2	4.3	0.2	22.2
halibut	2002	0.2	8.4	3.2	0.8	12.6
jacksmelt	2002	0.2	2.3	2.5	0.5	5.5
white croaker	2002	0.9	27.4	10.1	1.7	40.1
striped bass	2002	0.3	10.3	4.5	1.4	16.6
leopard shark	2002	0.1	0.9	0.3	0.1	1.4

Lipid adjusted values, parts per billion

Fish species	Year	tri-BDE (PBDE 28+33)	tetra-BDE (PBDE-47)	penta-BDE (PBDE-99+100)	hexa-BDE (PBDE-153+154)	sum of 7 congeners
walleye surfperch	2002	15	508	133	16	672
shiner surfperch	1997	27	669	185	23	903
halibut	2002	24	1286	548	151	2009
	1997	24	641	154	2	821
jacksmelt	2002	11	122	121	29	282
	1997	15	158	124	15	312
white croaker	2002	13	444	166	28	652
	1997	14	395	131	25	564
striped bass	2002	30	1021	526	179	1756
	1997	24	349	118	26	516
leopard shark	2002	26	310	110	28	474
	1997	70	300	60	8	438