

Brominated Flame Retardants: A Burning Issue



ACSH PRESENTS

Brominated Flame Retardants: A Burning Issue

By William P. Kucewicz

Art Director:
Jennifer Lee

AUGUST 2006



AMERICAN COUNCIL ON SCIENCE AND HEALTH
1995 Broadway, 2nd Floor, New York, NY 10023-5860
Phone: (212) 362-7044 • Fax: (212) 362-4919
URLs: <http://acsh.org> • <http://HealthFactsAndFears.com>
E-mail: acsh@acsh.org

TABLE OF CONTENTS

Executive Summary	01
Introduction	01
How Flame Retardants Work	01
Residential Fires Cost Lives	03
Flame Retardants Save Lives	04
Efforts to Ban PBDEs	05
Warnings from Environmental Groups	08
Human Exposure to PBDEs	09
Studies Find No Health Risk	11
DecaBDE in the Environment	13
Conclusions	13
References	14

ACSH WOULD LIKE TO THANK THE FOLLOWING PEOPLE, WHO REVIEWED THIS PAPER.

Gordon W. Gribble, Ph.D.
The Dartmouth Professor of Chemistry
Dartmouth College

Roy F. Spalding, Ph.D.
Professor - Agronomy & Horticulture
University of Nebraska

Theodore R. Holford, Ph.D.
Head, Division of Biostatistics
Yale University School of Medicine

Mark J. Utell, M.D.
Professor of Medicine and Environmental
Medicine
University of Rochester Medical Center

William O. Robertson, M.D.
Medical Director
Washington Poison Center

James J. Worman, Ph.D.
Department of Chemistry
Rochester Institute of Technology

ACSH accepts unrestricted grants on the condition that it is solely responsible for the conduct of its research and the dissemination of its work to the public. The organization does not perform proprietary research, nor does it accept support from individual corporations for specific research projects. All contributions to ACSH—a publicly funded organization under Section 501(c)(3) of the Internal Revenue Code—are tax deductible.

Copyright © 2006 by American Council on Science and Health, Inc.
This book may not be reproduced in whole or in part, by mimeograph or any other means, without permission.

Executive Summary

A class of brominated flame retardants known as polybrominated diphenylethers (PBDEs) is under assault from environmental activists and regulators both in the United States and overseas. Flame retardants give people more time to escape a fire by delaying flashover, the explosive-like eruption of flames responsible for most of the fatalities and property damage in residential fires. PBDEs are particularly effective flame retardants and have long been widely used in the manufacture of televisions and other electrical equipment, furniture, and mattresses.

Fire retardants truly save lives. Their use in television cabinets alone is estimated to save 190 lives a year in the U.S. In the United Kingdom, where materials used in many home furnishings must be fire-resistant, researchers reckon the regulations have spared about 1,150 lives and prevented almost 13,500 injuries over the course of a decade.

Nevertheless, U.S. and European regulators have effectively banned two of the three most prominent PBDE flame retardants. An assortment of states, environmental groups, and foreign governments, moreover, is seeking to ban the third one (i.e., decaBDE) as well, even though there is no credible evidence that the chemical represents a danger to humans or the environment. Numerous studies, in fact, have concluded that our exposure to the compound is minimal and does not pose an adverse health risk for people at expected exposures.

Current evidence shows that the benefits of PBDE flame retardants, in terms of lives saved and injuries prevented, far outweigh any demonstrated or likely negative health effects from their use.

Introduction

Life-saving flame-retardant chemicals are under assault. Ignoring the vitally important role these compounds play in preventing or slowing fires, environmental activists advocate banning certain flame retardants on the grounds that biomonitoring studies have found trace amounts of the chemicals in humans, including in breast milk. They hope to get various governmental authorities in the U.S. and overseas to impose strict prohibitions on these flame-retardant chemicals.

At issue is a class of brominated flame retardants known as polybrominated diphenylethers (PBDEs). Within this group, three commercial mixtures of PBDEs are: penta-, octa-, and decaBDE. Each product is a mixture of diphenyl ethers with varying degrees of bromination. (ECB 2002b) These particular PBDEs have widely and frequently been used as flame retardants in furniture foam (pentaBDE); plastics for TV cabinets, consumer electronics, wire insulation, and backcoatings for draperies and upholstery (decaBDE); and plastics for personal computers

and small appliances (octaBDE). The chemicals increase valuable escape time in cases of fire by slowing both ignition and the rate of fire growth. (USEPA 2005a)

Efforts to ban these chemicals are deadly serious business. In the U.S., someone dies in a fire every two hours and ten minutes, and the vast majority (85 percent) of these non-firefighter, civilian deaths occur in home fires. The fire death rate is 14.8 persons for every one million Americans. (BFRIP 2002) In view of the public debate and regulatory reviews of PBDEs, a look at some flame-retardancy facts is in order.

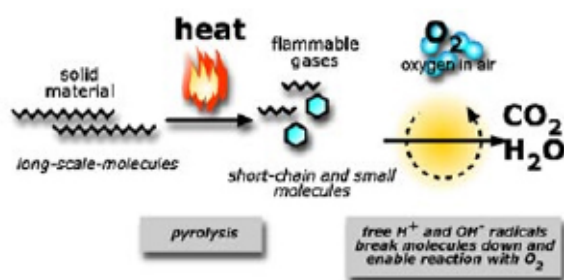
How Flame Retardants Work

Chemical flame retardants have been around since Roman times, when they were used to prevent siege towers from catching fire. The first flame-retardant patent was issued in England in 1735, and the use of these compounds to make plastics flame resistant began in the early 1970s. To understand how flame retardants work, it's

necessary to understand how things burn. (BSEF 2005a)

Solid materials don't burn directly. In a process known as pyrolysis, heat must first decompose the materials, releasing flammable gases. When these gases burn with oxygen in the ambient air, visible flames appear. To be more specific (Figure 1), what are known as high-energy "free radicals" (i.e., H^+ and OH^- in the gas phase) break down molecules, freeing carbon atoms that react with oxygen (O_2) and burn, releasing both carbon dioxide (CO_2) and water (H_2O). (EFRA 2005)

Figure 1. Pyrolysis: How Things Burn



Source: EFRA 2005

Flame retardants, in effect, remove the free radicals, reducing both the generation of heat and the production of flammable gases. This either prevents a fire from erupting or at least slows the combustion process considerably. (EFRA 2005)

Flame retardants thus give people more precious time to escape a fire prior to flashover. (Thomson 2004) Flashover occurs when a relatively small, slowly developing fire, spreading systematically across adjacent fuel surfaces within a room, suddenly transitions to a much larger and more dangerous fire in which all flammable surfaces, including ceilings, walls, and floors, are involved. It is estimated that roughly 20 percent of reported fires in the United States that transition to flashover are responsible for 80 percent of the fire deaths and property damage in buildings. (BFRL 2004) By delaying flashover, flame retardants can mean the difference between living and

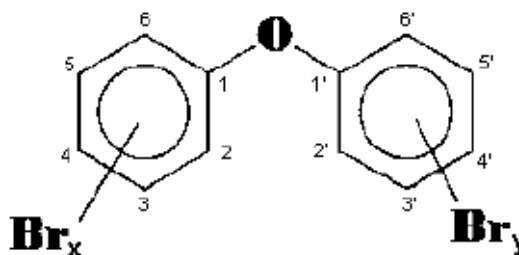
dying. (Thomson 2004) Flame retardants, in fact, can increase the amount of escape time by a factor of 15, meaning that instead of having just 10 seconds, say, to flee with your life, flame retardants may give you as much two and a half minutes to escape. (BSEF 2005a)

Brominated flame retardants (BFRs) are a family of chemicals commonly used in domestic and industrial appliances and equipment such as computers, TV's, mobile phones, furniture, insulation boards, mattresses, and many others. Nine in 10 electrical and electronic appliances contain BFRs. BFRs are also used in textiles for upholstered furniture. (BSEF 2005a)

Among different BFRs, polybrominated diphenyl ethers (PBDEs) are particularly useful for several reasons: 1) they release their flame-retarding bromine (Br) atoms in the same general temperature range at which many household items combust; 2) they combine easily with different plastics and other materials in the manufacturing process, making the resulting products (e.g., TV sets) fire resistant; and 3) they are highly cost-effective. (BFRIP 2002)

DecaBDE derives its effectiveness in retarding flame from the bromine atoms on the diphenyl oxide molecule (Figure 2). Bromine is one of the few elements able to provide flame retardancy in the gas phase of combustion. DecaBDE's high bromine content makes it very effective in retarding flame and also highly cost-efficient. (BFRIP 2002)

Figure 2. Chemical Formula of Brominated Diphenyl Ethers



Source: WHO 1994

About four-fifths of decaBDE production is used in high-impact polystyrene electrical or electronic products, such as television cabinets. (Hardy 2002) These applications are important in improving the safety of consumer products since potentially flammable polymers are in close contact with possible ignition sources, notably electricity. (Hays 2003) PBDEs typically comprise 5 to 30 percent of a product's overall weight. (CSOR 2004)

DecaBDE also is used to flame-retard upholstery fabric and textiles found in homes, offices, and motor vehicles. The chemical is encapsulated in latex and applied as a fabric back-coat, which significantly limits potential releases and exposures via direct contact with the flame-retarded surface. DecaBDE, however, is an additive flame retardant, which means it is physically combined with the material instead of being chemically combined. This creates the possibility that decaBDE may diffuse out of the treated material. (Hays 2003, BFRIP 2002, ECB 2002)

Residential Fires Cost Lives

Fires in the U.S between 1995 and 2004 cost 4,023 lives and injured another 21,704 on average per year (excluding the events of Sept. 11, 2001), according to the U.S. Fire Administration. In 2004 alone, an estimated 1,550,500 fires nationwide killed 3,900 persons and injured an additional 17,785; direct financial losses approached \$9.8 billion. (USFA, 2005a) Residential fires represented about 27 percent of all fires in 2004 and 78 percent of structure fires. Of all civilian fire fatalities (i.e., non-fighter fatalities), almost 82 percent occurred in the

home, and about 84 percent of these fires were in single-family homes or duplexes. (NFPA, 2004)

Young children and the elderly are often the victims of residential fires, accounting for 22 percent of all fire-related deaths and injuries in 2002. Residential fires injured or killed an estimated 2,490 children age 14 or younger, with children under the age of 5 representing 56 percent of child fire casualty deaths, according to the U.S. Fire Administration. Fires and burns were the third leading cause of unintentional fatal injuries to children age 14 or younger in 2002; they were also the third leading cause of unintentional injuries to infants, and the 12th overall cause of unintentional injuries to children age 14 or younger. (USFA 2005b) An estimated 2,320 older adults were injured or killed in residential fires in 2002. Smoking was the leading cause of these fires (25 percent), which typically ignited upholstered furniture and bedding. (USFA 2005c)

Internationally, the total cost of fires to society is estimated at about 1 percent of the world's gross domestic product (GDP), with property losses alone amounting to 0.2 percent of global GDP. Not surprisingly, a great deal of research has gone into preventing the spread of flame using barriers and flame retardants. The use of flame retardants, coupled with governmentally mandated fire-safety regulations and strict building codes, are reflected in the varying percentages of deaths per fire from continent to continent (Table 1). The lowest fire fatality rates are in North America and Australia, followed by South America and Africa. European fire fatalities are slightly above the worldwide average, but the rate of fire-related deaths in Asia is triple the global mean. (Harding, Crompton 2000)

Continent	Population (millions)	Fires per Year (millions)	Fire Deaths per Year (thousands)	Deaths per Fire* (percent)
Europe	720	2.2	25.0	0.011
Asia	3,660	1.0	30.0	0.030
North America	470	2.3	6.5	0.003
South America	340	0.5	2.5	0.005
Africa	780	0.8	5.0	0.006
Australia	30	0.1	0.3	0.003
Total	6,000	6.9	69.3	0.010
*Calculations added.				

Table 1. Global Fires and Fire Deaths by Continent

Source: Harding and Crompton 2004

Flame Retardants Save Lives

A study of the safety benefits of decaBDE and other brominated flame retardants (BFRs) in the U.S. found that an estimated 190 lives are saved annually because of the use of these flame retardants in television cabinets alone. Their use in electrical wire/cable insulation is estimated to save an additional 80 lives per year, and their application to draperies likely saves 10 more lives a year. All told, decaBDE and other flame inhibitors save an estimated 280 lives in the U.S. each year. (BFRIP 2002)

In studying the relationship between incidence of fire-related deaths and injuries and the use of flame retardants, the United Kingdom is perhaps the world's best venue, principally because in November 1988 it began instituting rules requiring all fabric and polyurethane foam used in the construction of furniture and mattresses be fire resistant. The rules were later expanded to other filling materials and also applied to used furnishings for resale. The UK may thus be considered a touchstone against which to assess fire-safety regulations promulgated in other countries. (Surrey 2000, 2002, 2005)

Surrey University's Polymer Research Center, which has published a series of studies relating to Britain's flame-retardants regulations, finds substantial benefits in reduced numbers of serious fires, reduced fatal and non-fatal injuries, and reduced financial losses since the introduction of the "Furniture Fire Safety Regulations 1988." The risk of death in residential fires in the UK, for example, dropped from 17 per million people per annum (pmp pa) in 1988 to 10 pmp pa in 2005, a 41 percent reduction. It is estimated that about half of this reduction (or around 20 percent) relates to the increased use of effective residential smoke

alarms and around half (or about 21 percent) can be ascribed to the 1988 fire-safety regulations. The effect of reduced tobacco smoking in the UK over the period was considered negligible. (Surrey 2005)

Cumulatively, from 1988 to 2002, it is estimated the 1988 UK furniture regulations alone saved 1,150 lives and prevented 13,442 injuries (Figure 3). Smoke alarms in Britain and the upholstered furniture regulations combined prevented an estimated 44,314 residential fires, saved 4,287 lives, and prevented 39,257 non-fatal injuries. As for property damage, the savings from 1988 to 2000 is calculated at £182 million per year (or around \$300 million) or a 12-year total of about £2.2 billion (or about \$3.6 billion). (Surrey 2005)

If current trends continue, the rate of UK residential fire deaths is likely to decline to between 5 and 6 pmp pa by 2010 to 2015, which would place Britain in the "best of class" group of countries with the best fire-safety records. (Surrey 2005) (California has already achieved this distinction, having implemented statewide fire-safety standards for residential upholstered furniture in 1976. California is the only state in the nation with such home-furnishings requirements, although federal fire-safety standards do apply to commercial furniture. [Hays 2003, CPSC 2001])

In Britain, fire-safe furniture and smoke alarms

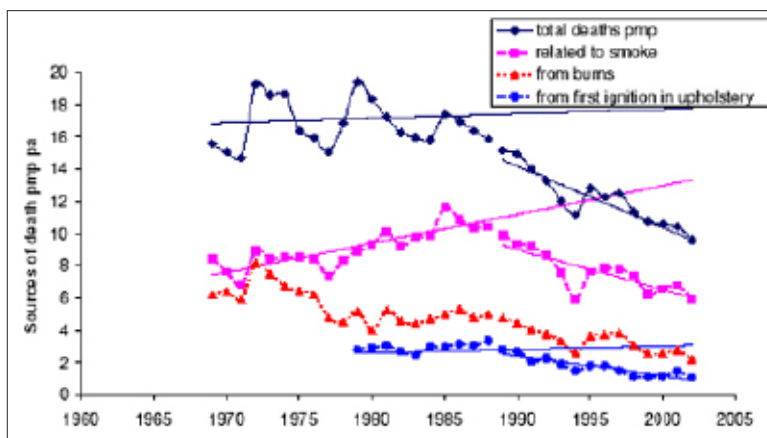


Figure 3. Pre- and Post-1988 Trends in UK Fatal Fire Injuries in Dwellings
Source: Surrey 2005

are expected to bring residential fire fatalities down to between 300 and 360 per year over the next decade from an actual 898 fire deaths in 1988 and 561 in 2002. By comparison, in France, which has no residential furniture fire-safety regulations and where the penetration of smoke alarms in residences is near zero, residential fire deaths for an equivalent population are calculated at 680. (Surrey 2005, Deheuvelds 2004)

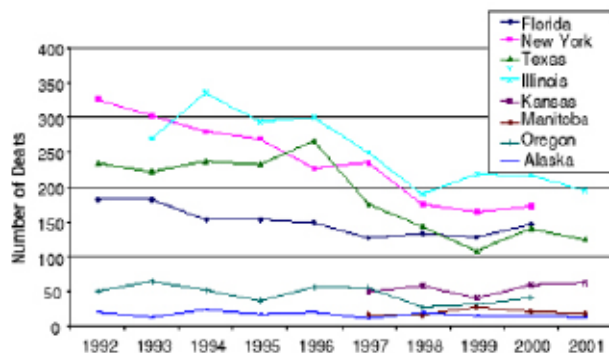
The University of Surrey researchers, reviewing U.S. fire data, reached a startling conclusion:

The progressive decline in the risks of death in fire in the USA appears to correlate well with the steady increase in the number (and probably the effectiveness) of smoke alarms in US residences....

However, despite the progressive decline in national US residential fire death rates, the US sustains a much higher risk of residential fire death in comparison to the UK. Some care is required when making international comparisons because it also is clear that there can be very wide variations of fire risk within countries and within regions. In the USA, state-to-state comparisons [Figure 4] indicate that the risk of death in fire can vary from below 5 pmp pa to over 30 pmp pa. This same wide range of risk exists in Europe and in the rest of the world. It is clear that climate, lifestyle, and cultural differences may account for some of this variation but it also true that sufficient exceptions exist to indicate that the effectiveness of good fire protection regulation and the presence of good fire countermeasures are also important factors.

By adopting furniture fire safety legislation at the federal level, combined with ongoing promotion of fire alarm installation and maintenance, it might be possible to eventually reduce the national average death rate due to fire in the US to 6 pmp pa. If this was achieved the number of additional lives saved in 2000 would be around 7.6 pmp pa. or 2,139 people per year. (Surrey2005)

Figure 4. Trends in State Fire Deaths in the U.S. from 1992 to 2001



Source: Surrey 2005

Efforts to Ban PBDEs

Both European and U.S. regulators have taken steps to ban pentaBDE and octaBDE, while permitting continued use of the third prominent PBDE flame retardant, decaBDE.

Following an exhaustive 10-year risk assessment that included the evaluation of 588 studies, the European Union (EU) decided on Oct. 13, 2005 to exempt decaBDE from its "Directive on the Restriction of Hazardous Substances," meaning the compound may continue to be used as a flame retardant in its 25 member countries.* The EU risk assessment had concluded that the use of decaBDE does not pose risks to humans or the environment. (BSEF 2005c) Since the risk assessment of decaBDE has concluded that there is at present no need for measures to reduce the risks for consumers beyond those which are being applied already, the EU found that decaBDE can be exempted from any restrictions on its use until further notice. Should new evidence lead to a different conclusion of the risk assessment, the decision would be re-examined and amended, if appropriate. (EC 2005) The EU left standing an earlier directive requiring the separation of decaBDE, as well as other BFRs, from electrical and electronics (E&E) equipment waste prior to recovery and recycling. (BSEF 2005c)

* EU member countries: Austria, Belgium, Cyprus, Czech Republic, Denmark, Estonia, Finland, France, Germany, Greece, Hungary, Ireland, Italy, Latvia, Lithuania, Luxembourg, Malta, Poland, Portugal, Slovakia, Slovenia, Spain, Sweden, the Netherlands, and United Kingdom.

The EU banned the marketing of two other BFRs – namely, pentaBDE and octaBDE – as well as articles containing more than 0.1 percent of either chemical, effective Aug. 15, 2004. (EU 2003) Citing a risk to breast-feeding infants, a series of EU Risk Assessment Reports expressed concern that the bioaccumulative properties (i.e., the tendency of a substance, such as a toxic chemical, to accumulate in various tissues of a living organism) of pentaBDE and octaBDE could cause concentrations in breast milk to rise. It further said risks to infants from exposure to pentaBDE and octaBDE via cows' milk is likely to be similar to or greater than that from human breast milk. (ECB 2000, ECB 2002a, ECB 2002b, ECB 2003a, ECB 2003b)

The U.S. Environmental Protection Agency (EPA) cited the EU risk assessments in deciding to issue a *de facto* ban on the production or purchase of penta- and octaBDE in the United States. Using a regulatory device known as a “Significant New Use Rule,” the EPA since Jan. 1, 2005 has required prior notification of any plans to manufacture or import pentaBDE and octaBDE. (USEPA 2004)

In particular, the agency called for specific measures to limit risks from pentaBDE because of “concerns for effects on the local aquatic (sediment) and terrestrial environment as a consequence of exposure arising from polyurethane foam production [and] concerns for secondary poisoning to the environmental spheres both locally and regionally as a consequence of exposure arising from production and/or use of polyurethane foams.” (ECB 2000, USEPA 2004) For octaBDE, the EPA cited the EU's concern about the risk of “secondary poisoning via the earthworm route for the hexabromodiphenyl ether component in the commercial octabromodiphenyl ether product from the use in polymer applications.” (ECB 2003a, USEPA 2004)

The EPA states the case against PBDEs thusly:

Although use of flame retardants saves lives

and property, there have been unintended consequences. There is growing evidence that PBDEs persist in the environment and accumulate in living organisms, as well as toxicological testing that indicates these chemicals may cause liver toxicity, thyroid toxicity, and neurodevelopmental toxicity. Environmental monitoring programs in Europe, Asia, North America, and the Arctic have found traces of several PBDEs in human breast milk, fish, aquatic birds, and elsewhere in the environment. Particular congeners [i.e., related chemical substances], tetra- to hexabrominated diphenyl ethers, are the forms most frequently detected in wildlife and humans. The mechanisms or pathways through which PBDEs get into the environment and humans are not known yet, but could include releases from manufacturing or processing of the chemicals into products like plastics or textiles, aging and wear of the end consumer products, and direct exposure during use (e.g., from furniture). (USEPA 2005a)

The agency in promulgating its “Significant New Use Rule” explained that the lower brominated PBDEs (tetraBDE, pentaBDE, and hexaBDE) found in the commercial pentaBDE and octaBDE products are “the congeners most often detected in the environment and for which human health and environmental concerns are greater. These factors, taken together, raise concerns for potential adverse effects in people and wildlife over time if these substances should continue to be produced, released, and built up in the environment.” (USEPA 2004, USEPA 2006)

The EPA action followed a voluntary phase-out by Great Lakes Chemical Corp. (GLC), the only U.S. manufacturer of pentaBDE and octaBDE, which ceased production of both chemicals by the end of 2004. In announcing its decision to act preemptively, GLC (now part of Chemtura, Inc.) explained that the EPA has concluded that PBDEs do not constitute a risk to the public, while conceding that there is growing public concern about the persistence and bioaccumulation of penta

products. Accordingly, GLC decided to transition away from pentaBDE in “an orderly manner, years ahead of any legislatively-mandated deadlines.” (The EPA considers a chemical with a half-life of more than 120 days to be “persistent” and less than 60 days to be not a concern. It considers a chemical with a bioaccumulation factor of more than 5,000 to be “bioaccumulative” and less than 1,000 to be not a concern.) (GLC 2004, USEPA 2004)

Separately, the EPA’s Integrated Risk Information System (IRIS), a database of human health effects from exposure to various substances, says penta- and octaBDE are not classifiable as to human carcinogenicity because no human data or animal data are available. (IRIS 2006a, IRIS 2006b) DecaBDE, however, is considered a “possible human carcinogen,” based on no human data but instead limited evidence of carcinogenicity in animals – specifically, significantly increased incidences of neoplastic liver nodules in male and female rats and increased incidences of hepatocellular adenomas or carcinomas (combined) in male mice. (IRIS 2006c)

In assessing risk for health effects other than cancer and gene mutations from chronic chemical exposure, an EPA working group coined new terminology (e.g., reference dose, or RfD). NOEL (also referred to as NOAEL) is the highest experimentally determined dose without a statistically or biologically significant adverse effect. In cases in which a NOEL has not been demonstrated experimentally, the term lowest-observed-adverse-effect level (LOAEL) is used. The RfD is a benchmark dose operationally derived from the NOAEL by consistent application of order-of-magnitude uncertainty factors (UFs). The modifying factor (MF) is an additional uncertainty factor whose magnitude depends on the professional assessment of the completeness of the overall data base and the number of species tested. Ranging from zero to 10, the default value for the MF is 1. (USEPA 1993)

Beyond the federal manufacture and import restrictions, a number of states have either enact-

ed legislation or are considering proposals banning the use of pentaBDE and octaBDE. Notably, however, a California State Senate research report concluded, like the EU’s multiyear risk assessment, that decaBDE does not pose a risk to public health and thus should not be proscribed. (CSOR 2004)

The EPA, meanwhile, is evaluating potential PBDE substitutes through its New Chemicals Program and its Design for the Environment Program’s Furniture Flame Retardancy Partnership with private industry. In the case of pentaBDE, the working group says no single alternative is likely but it has identified 14 chemical formulations that are potentially viable substitutes in large-scale production of low-density flexible polyurethane foam. EPA assessed the hazards, potential exposures, and tendency to bioaccumulate and persist in the environment for the chemicals. The industry panel continues to look for other flame-retardant compounds. (USEPA 2005b)

Additionally, the EPA is engaged in the Voluntary Children’s Chemical Evaluation Program (VCCEP), working with chemical manufacturers to provide data to enable the public to understand the potential health risks to children associated with certain chemical exposures. Through VCCEP, industry-sponsored risk assessments for pentaBDE, octaBDE, and decaBDE were developed to evaluate the potential risks to children and prospective parents from potential exposure scenarios. In September 2005, EPA issued its Data Needs Decision on PBDEs, asking manufacturers to provide the needed data by volunteering to conduct a mix of tests. (USEPA 2005a)

Substitutes for decaBDE also are being studied for use to electronic enclosures, such as TV set backs, and textiles, as well as replacements for pentaBDE in flame retardant furniture foam. The most cost-effective non-halogenated substitutes for decaBDE high-impact polystyrene (e.g., TV backs) involve changing the resin system and the use of phosphorous-based flame retardants. The

most cost-effective non-halogenated substitutes include: blends of polycarbonate and acrylonitrile-butadiene-styrene, using the flame retardant bis-phenol A diphosphate; polycarbonate, utilizing phosphate esters; and blends of high-impact polystyrene and polyphenylene oxide, which includes the flame retardant resorcinol bis diphenyl phosphate. Other substitutes such as metal, wood, or enclosures based on polylactide are possible but are not widely employed because of cost and performance issues. (Lowell 2005)

In regard to textiles, the most common chemically applied decaBDE substitutes available for natural cellulosic fibers such as cotton, wool, rayon, and linen are dimethylphosphono (N-methylol) propionamide (phosphonic acid) and tetrakis (hydroxymethyl) phosphonium salt (or chloride) compound with urea. For synthetic fabrics such as acrylic, acetate, nylon, and polypropylene, some decaBDE substitutes are available, but they often have limited durability because of their water solubility and tendency to wash out during laundering. (Lowell 2005)

The Swedish government has said it intends to propose a restriction on the use of decaBDE in textiles, furniture, and cable applications. DecaBDE's use in automobiles and in electrical and electronic equipment would not be affected. The government further said it is launching a study of two other BFRs – hexabromocyclododecane (HBCD), used mainly in textile coatings and thermal insulation foams, and tetrabromobisphenol A (TBBPA), primarily used in laminates (e.g., epoxy resins) for printed wiring boards – with an intent to ban the chemicals. It was taking the action, it said, because it felt the EU was moving too slowly in regard to the two compounds. (EBFRIP 2005)

Australia decided in June 2005 to commence a process to assess the potential effects on public health and the environment of certain BFRs, including decaBDE. China is preparing legislation similar to that approved by the EU concerning the handling of E&E waste containing BFRs. And Norway, which isn't an EU member, is considering legislation to partially restrict the use of

decaBDE. Some foreign governments have objected to the Norwegian proposals, however, and the final outcome remains undecided. (EBFRIP 2005)

Warnings from Environmental Groups

Environmentalists have made PBDE flame retardants a *cause célèbre*. Greenpeace, for instance, has held demonstrations to get electronics makers to stop using the chemicals. (Sandoval 2005) The Swedish Society for Nature Conservation has expressed opposition to the use of brominated flame retardants (BFRs), claiming they are absorbed and stored in the body and pose “serious health consequences that are a threat to the development and well-being of future generations.” (SSNC 2005) A pair of U.S.-based environmental groups have found BFRs in dust samples swiped from computers (Hines 2004), and Los Angeles-based Environment California has criticized the EU for failing to act on decaBDE, citing concerns that the compound breaks down into “its more toxic cousins octa and penta.” (Environment California 2003)

A report by Northwest Environment Watch in Seattle found decaBDE in 24 of the 40 women tested, with 10 of the women carrying deca levels above 1 part per billion. The highest level detected was 4.3 ppb. It said even the deca congener alone exceeds the levels of total PBDEs typically found in European and Japanese populations. Levels of deca in the most exposed residents of the Pacific Northwest are comparable to those of Swedish electronics dismantlers, who are occupationally exposed to decaBDE. (NEW 2004)

Washington Physicians for Social Responsibility maintain that PBDEs, which share chemical properties with banned PCBs, are ubiquitous in our environment. This group states that studies have linked these chemicals to serious health effects, including memory impairment, and learning and behavioral problems in laboratory animals, and that they have also been associated with disruption of thyroid hormone balance, non-

Hodgkin's lymphoma in humans, and a variety of cancers in rodents. (WPSR 2004)

People for Puget Sound cite studies that have found PBDEs in Puget Sound orcas, salmon, ospreys, Columbia River fish, and Northwest human breast milk. The studies they cite refer to levels found in orca whales 2-10 times higher than those found in other whales around the world, and the levels found in breast milk were 20 to 40 times higher than levels found in Europe and Japan. (PPS 2004)

Canadian and Norwegian scientists recently reported finding PBDEs in the fat tissue of adult and subadult female polar bears sampled between 1999 and 2002 from sub-populations in Arctic Canada, eastern Greenland, and Svalbard, and in males and females collected from 1994 to 2002 in northwestern Alaska. (Muir 2005)

The Environment California Research & Policy Center, a San Francisco public interest research group (PIRG), maintains decaBDE "poses a threat to human health" because it decomposes into forms that are "more toxic and more easily absorbed by the body" and also because it "may be neurotoxic," affecting the nervous system and impairing motor skills. "Deca," the PIRG report added, "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." (Kucher 2004)

Human Exposure to PBDEs

Studies have found decaBDE in human tissue, but the route of exposure is unknown. (CSOR 2004) Recent data suggests that PBDE body burdens continue to rise in North American wildlife and humans, and that PBDEs levels among individuals within a population vary widely, by approximately 50-fold. The reasons for this variability are not well understood, but likely relate to differences in exposure and host differences that affect uptake and elimination. (CSOR 2004)

Levels of PBDEs among residents of North America are approximately 40 to 70 times higher than those of individuals in Europe or Japan. This disparity is likely a result of the fact that more than 95 percent of the world's use of the penta-BDE technical mixture (whose congeners are highly bioaccumulative) is in the Americas. (CSOR 2004)

Current total PBDE levels in U.S. blood samples are the highest reported worldwide to date, according to a University of Texas study, with 2 pooled samples measuring 61.7 and 79.7 parts per billion (ppb) lipid. In a series of 39 individual analyses, the range was 4.6 to 365.5 ppb, with a median of 29 ppb and a mean of 52.6 ppb. (Schechter 2005)

A study comparing potential PBDEs inhalation exposures of three groups of workers (i.e., clerks working in front of computer screens full-time, workers at an electronics dismantling plant, and a control group of hospital cleaning workers) found decaBDE in the blood of individuals from all three groups. Plasma levels of decaBDE were significantly higher in the electronics dismantling workers than in the other two groups, apparently resulting from inhalation of particle-bound decaBDE in the air of the dismantling plant, as high ambient levels of particle-bound decaBDE were recorded at the plant. Electronics dismantling involved grinding plastic goods in a shredder. DecaBDE in the blood of cleaning workers and clerks was presumed to be due to ingestion of food contaminated with decaBDE. The total median PBDE concentrations in the serum from

workers at the electronics-dismantling plant, clerks, and cleaners were 37, 7.3, and 5.4 pmol/g lipid weight, respectively. The results show that decaBDE is bioavailable (i.e., the ability of a drug or other substance to be absorbed and used by the body) and that occupational exposure to PBDEs occurs at the electronics-dismantling plant. (Sjödin 1999)

It has been suggested that the main sources of exposure of the general population to decaBDE are likely to be inhaled air and contact with dust at home or in the office. The efficiency of absorption of decaBDE following inhalation has not been investigated, but a large proportion of inhaled particles containing decaBDE are likely to be ultimately swallowed and only a small proportion of the decaBDE in these particles is likely to be absorbed. (Searl 2003)

In experimental animals fed standard laboratory diets, pentaBDE mixtures can decrease circulating thyroid hormone and liver vitamin A concentrations. A substantial number of pregnant women and their children have marginal vitamin A status, potentially increasing their risk of adverse effects to pentaBDE exposure. The results support the concept that marginal vitamin A status in pregnant women may increase the risk for PBDE-induced disruptions in vitamin A and thyroid hormone homeostasis. (Ellis-Hutchings, 2006)

To assess blood concentrations of PBDEs and polychlorinated biphenyls (PCBs) and their polychlorobiphenylol (OH-PCB) metabolites in humans with a high seafood intake, researchers obtained samples from pregnant women in the Faroe Islands in 1994-1995 and from their children at 7 years of age to examine maternal transfer of the compounds to their child, age-dependent metabolism, and temporal changes. Children at 7 years of age, it found, are exposed to PCBs at levels only slightly below those of their mothers, and the increased 4-OH-CB107 concentrations in children could be due to age-related differences in PCB metabolism. The PBDE concentrations were similar in both mothers and their children.

The main persistent organic pollutant concentrations in the children are most probably due to other environmental exposure than maternal transfer. (Fangstrom 2005)

The EPA has launched a project plan, including a dedicated webpage (www.epa.gov/oppt/pbde), to further assess and evaluate PBDEs and human health and to weigh possible substitutes. In unveiling the project, the federal agency noted that PBDEs level in humans and the environment are generally higher in North America than in other parts of the world and cited potential concerns about liver toxicity, thyroid toxicity, developmental toxicity, and developmental neurotoxicity. (USEPA 2006b)

The U.S. Department of Agriculture, meanwhile, is conducting research on the absorption, disposition, metabolism, and excretion (ADME) of PBDEs. Studies of ADME in rats have been conducted on commercial pentaBDE, octaBDE, and decaBDE mixtures. Rodent ADME studies have also been conducted on BDE congeners 47, 99, 100, 154, and 209. Studies in rats are planned for BDEs 153 and 183, along with further study of BDE-209. (USEPA 2006b)

Studies Find No Health Risk

At least four leading institutions – two international, and two U.S.-based – have reviewed decaBDE for potential health risks to humans, and all four evaluations concluded that exposures to decaBDE were minimal and not likely to pose an adverse health risk. (Hays 2003) (CSOR 2004)

Specifically, the World Health Organization (WHO) evaluated the manufacture and formulation of decaBDE into polymers and concluded that exposure of the general population to decaBDE is insignificant. (WHO 1994) The National Academy of Sciences (NAS) and the Consumer Product Safety Commission (CPSC) assessed the use of decaBDE in textiles (National Academy of Sciences 2000; Babich and Thomas 2001), and the European Chemicals Bureau examined exposures to decaBDE via the general environment. (ECB 2002) These four evaluations concluded that exposures via the respective pathways did not pose any adverse health risks to the general population. (Hays 2003)

Additionally, the California Senate Office of Research (CSOR) reviewed the potential hazards from decaBDE and reached the following conclusion:

Based on an analysis of the likely potential harm to humans posed by decaBDE and the known human exposures to this chemical, it does not appear that human exposure to decaBDE is occurring at a level that is likely to be unsafe for human health or development given the current level of scientific knowledge. At this time, it would be premature to add decaBDE to the list of banned PBDEs contained in AB 302 [a California law banning the sale of products containing two classes of PBDE – pentaBDE and octaBDE – as of 2008]. (CSOR 2004)

As for the exposure of infants and children to decaBDE, current levels of decaBDE in the U.S. are not likely to represent an adverse health risk for children (Figure 5). The study looked at six different pathways by which children might become exposed to decaBDE; it then combined

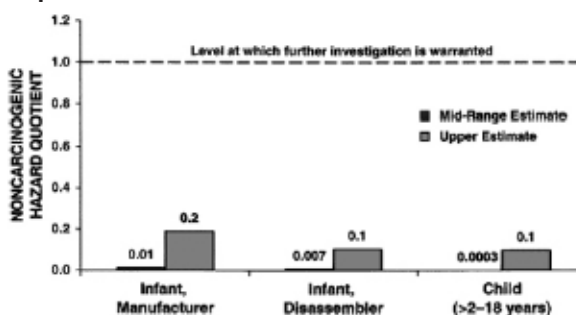
these into three exposure scenarios, which aggregated all exposures for a given population: (Hays 2003)

1. Child (0–2 years) ingesting breast milk from a mother who manufactures decaBDE, mouthing decaBDE-containing plastic electronic products, mouthing decaBDE-containing fabric, and being exposed via the general environment,
2. Child (0–2 years) ingesting breast milk from a mother who disassembles electronics, mouthing decaBDE-containing plastic electronic products, mouthing decaBDE-containing fabric, and being exposed via the general environment,
3. Child (>2–18 years) being exposed via the general environment. (Hays 2003)

Like the findings of the WHO, CPSC, the EU, and the NAS, each of which concluded that decaBDE does not pose a health risk to humans, this assessment of the exposure of infants and children in the U.S. to decaBDE was consistent with their conclusions that current levels of exposure do not endanger the health of infants or children, including those ingesting breast milk from mothers occupationally exposed to decaBDE. (Hays 2003)

German researchers furthermore found the reported levels of decaBDE detected in breast milk by Veith et al. (2005) would not be expected to be hazardous to infant health. There is a substantial margin of safety in the predicted intake of decaBDE by breastfeeding babies and EU standards. There is also a substantial margin of safe-

Figure 5. Hazard Quotients for Children's Exposure to DecaBDE



Source: Hays 2003

ty in comparison to the lowest effects level reported by Viberg et al. (2003) for developmental toxicity in mice. (BfR 2005)

There are no reports of any adverse clinical health effects in humans associated with touching, inhaling or ingesting decaBDE. In an NAS review, no data were identified on immunological, neurological, reproductive, developmental, or carcinogenic effects of decaBDE following dermal exposure, inhalation exposure, or oral exposure. In animal studies, no clinical signs of toxicity or death occurred in rats that received single gavage doses as high as 5,000 mg/kg (NAS 2000), which is equal to 5,000,000 ppb.

Similarly, a U.S. Consumer Product Safety Commission (CPSC) risk assessment for residential upholstered furniture concluded decaBDE was not likely to present a hazard to consumers. In both adults and children, dermal exposure was the primary route of exposure to decaBDE; the contribution from inhalation of particles was negligible. In all cases evaluated, the Hazard Index (HI) was less than 1 – that is, the potential exposures were below those of concern. (CPSC 2001)

The California Senate Office of Research also found insufficient evidence to warrant a ban of decaBDE in California. It found that human exposure to decaBDE is not occurring at a level that is likely to be unsafe for human health or development given the current level of scientific knowledge. They concluded that at this time, it would be premature to ban PBDEs. (CSOR 2004)

DecaBDE has not been shown to be a hazard to human health in anything close to the dosages that have been observed or that are likely to occur, according to a submission to Washington State authorities by the Boeing Company. No study, it noted, has found the compound to be toxic, although vague concerns have been raised about neurological damage in high dosages in experimental animals. DecaBDE's half-life of 12 days in humans cannot be considered persistent. (Thomson 2004)

The results of animal experiments further suggest that most ingested decaBDE is rapidly excreted

through the gastrointestinal tract. Both animal and human data indicate decaBDE is largely removed within a few days of exposure and that there is little long-term retention of decaBDE in tissue. Evidence further suggests the commercial form of decaBDE is not absorbed by the gut and passes out of the body quickly through feces. (Searl 2003) (Raupe 2003)

Neither is decaBDE a cancer risk. According to the U.S Public Health Service's Subcommittee on Flame Retardants, there is "no evidence that [decaBDE] posed either a non-cancer or cancer hazard regardless of exposure route." (USPHS 2001)

Another risk assessment of exposure to decaBDE and the potential human health effects reached the following conclusions:

- Levels of exposure of the general population to decaBDE are much too low to give rise to an adverse effect on health.
- Levels of exposure of workers recycling electrical goods appear to be much too low to give rise to an adverse effect on health.
- Levels of exposure of computer technicians and office workers using electrical equipment are unlikely to be substantially greater than for other members of the general population and are much too low to give rise to an adverse effect on health.
- Historical levels of exposure during the production of decaBDE may have been higher; however, there is little evidence to suggest that exposure to decaBDE during production has been an important cause of ill health in exposed workers.
- The presence of decaBDE in the blood of recycling workers confirms that they are exposed to decaBDE and is suggestive of slightly higher levels of exposure to decaBDE than typical for the general population.
- The presence of decaBDE in the blood of recycling workers does not indicate that they are being exposed to quantities of decaBDE that are likely to be hazardous to health. (Searl 2003)

DecaBDE in the Environment

DecaBDE, largely because of its physical properties, does not pose a significant risk to the environment. Sunlight does not degrade decaBDE. (Jafvert and Hua 2001) DecaBDE is poorly soluble, and its presence in natural waters is mainly associated with suspended particulate matter, which is an important carrier for all PBDE species. (de Boer 2003) DecaBDE has a strong affinity to bind to organic carbon in the water column and sediment. (Hardy, 2002a) Most studies of the concentrations of PBDE in the water column have failed to find detectable levels of these compounds. DecaBDE does not accumulate in wildlife or food and unlike other persistent organic chemicals has not been reported to occur in wildlife or sediments. (Searl 2003)

Concerns have been raised over the potential for decaBDE to act as a source for tetra- and pentaBDE. Researchers at the University of Victoria, Canada conclude that the distribution of “penta species” found in the environment near urban and industrial regions arise from a weighted mix of commercial penta- and octaBDE mixtures in present use rather than from environmental debromination of decaBDE. (Rayne and Ikononou 2002)

Dutch researchers report similar results. In a study

of the occurrence of PBDEs in the aquatic environment of the Netherlands, UK, Ireland, and the southern North Sea, sediment core analyses confirm a decaBDE increase. However, a parallel increase of penta-mix-related congeners was not found, except in one Norwegian core. Because indications of increasing nona- and octaBDEs were not found, it is unlikely that penta- and hexaBDEs are being formed from decaBDE, unless at a very slow rate. The results support the view that decaBDE in the environment is not responsible for the presence of penta-product components in biota (fish, breast milk, etc.). (de Boer 2001)

Most important, PBDEs occur naturally. Several dozen are known to be marine natural products – mainly sponge metabolites – and at least two such natural PBDEs are identical to synthetic PBDEs. These two natural PBDEs were recently isolated from whale blubber (from a True’s beaked whale, *Mesoplodon mirus*) and shown by C-14 isotopic analysis to be naturally produced and not synthetic. The PBDEs having both natural and anthropogenic origins are methoxylated PBDEs MeO-BDE-17 and MeO-BDE-68. (Reddy 2004, Teuten 2005) The significance of the findings are this: when PBDEs are found in fish and marine mammals, it is entirely possible, even likely, that they are of natural origin. DecaBDE itself, however, has yet to be found to be a marine (sponge or bacterial) natural product.

Conclusions

There is no credible medical or scientific basis upon which to support a decaBDE flame-retardants ban. Studies have failed to demonstrate any health risks to the general population. Banning flame retardants, though, has the dire potential to increase the risk of death from fire and raise the number of associated injuries.

DecaBDE’s flame-retardant and life-saving properties are undeniable. By reducing the incidence of fires and, perhaps more important, by giving occupants valuable extra time to flee a fire, PBDEs are a clear benefit to public health in reducing fire injuries and fatalities in the United States and worldwide. Finding trace amounts in the human body or in environmental samples is not an indication of harm. Even studies of human breast milk failed to show adverse effects on infants or children.

Furthermore, when interpreting biomonitoring data as a measure of the background level of chemical exposures from the environment, Dennis Paustenbach and

David Galbraith offer an important caveat in *Biomonitoring: Measuring Levels of Chemicals in People – and What the Results Mean*. to wit, since in most cases the source of the chemical being measured in the biological samples will not be known, there is also a large risk of misinterpreting the data, and perhaps the most common misperception is that the mere detection of a chemical in our bodies suggests a health hazard rather than simply providing a measure of exposure. (Paustenbach 2005)

The efforts to ban decaBDEs, in sum, are misguided. Were such bans adopted, they would have the potential to engender very real and potentially tragic threats to public health from fires that would otherwise have been prevented or delayed with the use of products incorporating brominated flame retardants. By any measure, the benefits to public health and safety of decaBDEs outweigh the risks related to their production and use.

References

- Babich MA and Thomas TA, CPSC staff exposure and risk assessment of flame retardant chemicals in residential upholstered furniture. Consumer Product Safety Commission, Bethesda, MD, 2001.
- Brominated Flame Retardant Industry Panel (BFRIP), American Chemistry Council, "Voluntary Children's Chemical Evaluation Program (VCCRP): Data Summary," Dec. 17, 2002.
- Bromine Science and Environmental Forum (BSEF 2005a), "What Are Brominated Flame Retardants? What Are They Used For?" 2005.
- Bromine Science and Environmental Forum (BSEF 2005b), "BFR-Related Legislation in the United States," November 2005.
- Bromine Science and Environmental Forum (BSEF 2005c), "Legislation – Regulatory Overview in the EU," November 2005.
- Building and Fire Research Laboratory (BFRL), Technology Administration, U.S. Commerce Department, "Reduced Risk of Flashover," Dec. 12, 2004.
- California Senate Office of Research (CSOR), "Polybrominated Diphenyl Ethers (Pbdes): Potential Hazards from DecaBDE, and Unresolved Issues from AB 302, prepared by Kip Wiley and Brendan McCarthy, June 2004.
- de Boer J (de Boer 2001), Aldridge J, Allchin C, Bennett M, Boon JP, Brandsma S, van Hesseligen J, Law R, Lewis W, Morris S, Tjoen-A-Choy MR, and Zegers B, "Polybrominated Diphenylethers in the Aquatic Environment," Netherlands Institute for Fisheries Research (RIVO) report (Draft 110501), Number: C023/01, June 29, 2001.
- de Boer J (de Boer 2003), Wester PG, van-der Horst A, Leonards PE, "Polybrominated diphenyl ethers in influents, suspended particulate matter, sediments, sewage treatment plant and effluents and biota from the Netherlands." *Environmental Pollution* 122, 2003, 63-74.
- Dehevels P, Pierre-Lotu-Viaud D (Dehevels 2004), "Report on the recent evolution of fire accidents in the United Kingdom," Laboratoire de Statistique Théorique et Appliquée – Université Pierre et Marie Curie, Paris, and Frontier Economics for the French Commission de la Sécurité des Consommateurs and ACFSE, 2004.
- Ellis-Hutchings RG, Cherr GN, Hanna LA, Keen CL, "Polybrominated diphenyl ether (PBDE)-induced alterations in vitamin A and thyroid hormone concentrations in the rat during lactation and early postnatal development," *Toxicol Appl Pharmacol*, March 29, 2006.
- Environment California, "EU Shows Reluctance to Act on Dangerous Flame Retardant, Despite Evidence of Harm," press release, Dec. 5, 2003.
- European Brominated Flame Retardant Industry Panel (EBFRIP), "Sweden Draft Ban Under EU Scrutiny as Unjustified Barrier to Trade," press release, Nov. 14, 2005.
- European Chemicals Bureau (ECB 2000), European Union risk assessment report: Diphenyl Ether, Pentabromo Derivative (pentabromophenyl ether), CAS No: 32524-81-0, EINECS No: 251-084-2, Institute for Health and Consumer Protection. Volume 5, Final Report, August 2000.
- European Chemicals Bureau (ECB 2002a), Diphenyl Ether, Octabromo Derivative, CAS No: 32536-52-0, EINECS No: 251-087-9, Final Report, European Commission, Joint Research Centre, 2002.
- European Chemicals Bureau (ECB 2002b), European Union risk assessment report: Bis(pentabromophenyl) ether. CAS No: 1163-19-5, EINECS No: 214-604-9, Institute for Health and Consumer Protection. Vol. 17, 2002.
- European Chemicals Bureau (ECB 2003a), Diphenyl Ether, Octabromo Derivative, CAS No: 32536-52-0, EINECS No: 251-087-9, Summary Risk Assessment Report, European Commission, Joint Research Centre, 2003.
- European Chemicals Bureau (ECB 2003b), Institute for Health and Consumer Protection, European Commission, Joint Research Centre, Bis(Pentabromophenyl) Ether, CAS No: 1163-19-5, EINECS No: 214-604-9, Summary Risk Assessment Report, 2003.
- European Commission, Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE 2002a), "Opinion on the results of the Risk Assessment of: Bis(pentabromophenyl)ether," Human Health Part, CAS No.: 1163-19-5, EINECS No.: 214-604-9, Feb. 22, 2002.
- European Commission, Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE 2002b), "Opinion on the results of the Risk Assessment of: Bis(pentabromophenyl)ether," Environmental and Human Health Part, CAS No.: 1163-19-5, EINECS No.: 214-604-9, 2002.
- European Commission (EC 2005), "Commission Decision of 13 October 2005," amending Annex to Directive 2002/95/EC of the European Parliament and of the Council on the restriction of the use of certain hazardous substances in electrical and electronic equipment, *Official Journal of the European Union* (En) L 271/48, Oct. 15, 2005.
- European Flame Retardant Association (EFRA), "How Flame Retardants Work," The European Chemical Industry Council, 2005.
- European Union (EU 2002a) Directive 2002/95/EC of The European Parliament and of the Council of 27 January 2003, on restricted use of certain hazardous substances in electrical and electronic equipment, *Official Journal of the European Union*, February 13, 2003.
- European Union (EU 2002b) Directive 2002/96/EC of The European Parliament and of the Council of 27 January 2003, on waste electrical and electronic equipment, *Official Journal of the European Union*, February 13, 2003.

- European Union (EU 2003) Directive 2003/11/Ec of the European Parliament and of the Council, re pentabromodiphenyl ether, octabromodiphenyl ether, *Official Journal of the European Union*, Feb. 6, 2003.
- Fangstrom B. (Fangstrom 2005), Hovander L, Bignert A, Athanassiadis I, Linderholm L, Grandjean P, Weihe P, Bergman A., "Concentrations of polybrominated diphenyl ethers, polychlorinated biphenyls, and polychlorobiphenyls in serum from pregnant Faroese women and their children 7 years later," *Environ Sci Technol.* 2005 Dec 15;39(24):9457-63.
- Federal Institute for Risk Assessment [of Germany] (BfR 2005), "Residues of retardants in breast milk in Germany with special consideration of polybrominated diphenyl ethers (PBDEs)," Federal Environmental Agency (UBA), Action Programme Environment and Health (APUG), 2005.
- Great Lakes Chemical Corp. (GLC 2004), "Thanks to New Product Technology, Great Lakes Chemical Corporation announces that it will cease production of Penta-PBDE Flame Retardant by end of 2004," press release, 2004.
- Great Lakes Chemical Corp. (GLC 2005), "Crompton Corporation and Great Lakes Chemical Corporation Announce Merger to Create Major New Specialty Chemicals Company," press release, March 9, 2005.
- Harding P and Crompton G, "Melting in the Heat," *Asia Pacific Coatings Journal*, Vol. 13, No. 4, p. 16, August 2000, as cited in Thomson, 2004.
- Hardy ML (Hardy 2002), "A comparison of the properties of the major commercial PBDPO/PBDE product to those of major PBB and PCB products." *Chemosphere* 46, 2002,717-28.
- Hays SM, Cushing CA, Leung H, Pyatt DW, Holicky KC, and Paustenbach DJ, "Exposure of Infants and Children in the U.S. to the Flame Retardant Decabromodiphenyl Oxide (decaBDE)," *Journal of Children's Health*: Vol. 1, No. 4, pp. 449-475 (2003).
- Hines M, "Is the Dust on Your Computer Toxic?" CNETNews.com, June 3, 2004.
- Integrated Risk Information System (IRIS 2006a), "Pentabromodiphenyl ether; CASRN 32534-81-9," U.S. Environmental Protection Agency, Jan. 3, 2006.
- Integrated Risk Information System (IRIS 2006b), "Octabromodiphenyl ether; CASRN 32536-52-0," U.S. Environmental Protection Agency, Jan. 3, 2006.
- Integrated Risk Information System (IRIS 2006c), "Decabromodiphenyl ether (DBDPE); CASRN 1163-19-5," U.S. Environmental Protection Agency, Jan. 3, 2006.
- Jafvert C and Hua I, 2001. "Final Report. Photochemical reactions of decabromodiphenyl oxide and 2,2',4,4'-tetrabromodiphenyl oxide," submitted to American Chemistry Council, Brominated Flame Retardant Industry Panel.
- Kociba RJ, Frauson LO, Huniston CG, et al. 1975. "Results of a two-year dietary feeding study with decabromodiphenyl oxide (DBDPO) in rats." *J. Combust. Toxicol.* 2(4): 267-285.
- Kucher Y and Purvis M (Kucher 2004), "Body of Evidence: New Science in the Debate over Toxic Flame Retardants and Our Health," U.S. PIRG Education Fund, Environment California Research & Policy Center, February 2004.
- Lowell Center for Sustainable Production, The (Lowell 2005), "Decabromodiphenylether: An Investigation of Non-Halogen Substitutes in Electronic Enclosure and Textile Applications," University of Massachusetts Lowell, prepared by Pure Strategies, Inc., April 2005.
- Muir DCG (Muir 2005), Backus S, Derocher AE, Dietz R, Evans TJ, Gabrielsen GW, Nagy J, Norstrom RJ, Sonne C, Stirling I, Taylor MK, and Letcher RJ, "Brominated Flame Retardants in Polar Bears (*Ursus maritimus*) from Alaska, the Canadian Arctic, East Greenland, and Svalbard," *Environ. Sci. Technol.*, American Chemical Society, Dec. 10, 2005.
- NAS (National Academy of Sciences), Toxicological risks of selected flame-retardant chemicals. NAS, National Research Council, Commission on Life Sciences, Board on Environmental Studies and Technology, Committee on Toxicology, Subcommittee on Flame-Retardant Chemicals. National Academy Press, Washington, DC, 2000.
- National Fire Protection Association (NFPA), *Fire Loss in the U.S. During 2004: Abridged Report*.
- Norris JM, Ehrmantraut JW, Gibbons CL, et al. 1973. "Toxicological and environmental factors involved in the selection of decabromodiphenyl oxide as a fire retardant chemical." *App. Polym. Symp.* 22: 195-219.
- Norris JM, Kociba RJ, Schwetz BA, et al. 1975. "Toxicology of octabromodiphenyl and decabromodiphenyl oxide." *Environ. Health Perspect.* 11: 153-161.
- Northwest Environment Watch (NEW 2004), Comments on the Washington State PBDE Chemical Action Plan, Nov. 9, 2004.
- Paustenbach D, Galbraith D, *Biomonitoring: Measuring Levels of Chemicals in People – and What the Results Mean*, American Council on Science and Health, August 2005.
- People for Puget Sound (PPS 2004), Comments on the Draft Washington State PBDE Chemical Action Plan, Nov. 11, 2004.
- Raupe, Bebe, "Scientists Find No Significant Health Risk From Flame Retardant Decabromodiphenyl," *Chemical Regulation Reporter*, April 7, 2003.
- Rayne S and Ikononou MG (Rayne and Ikononou 2002), "Reconstructing Source Polybrominated Diphenyl Ether Congener Patterns From Semipermeable Membrane Devices in the Fraser River, British Columbia, Canada: Comparison to Commercial Mixtures," *Environmental Toxicology and Chemistry*, Vol. 21, No. 11, pp. 2292-2300, 2002.

- Reddy CM (Reddy 2004) et al., "Radiocarbon Evidence for a Naturally Produced, Bioaccumulating Halogenated Organic Compound," *Environmental Science & Technology*, 38 (7), 1992-1997, 2004.
- Sandoval G, "Greenpeace: HP stands for 'Harmful Products,'" CNET News.com, Dec. 6, 2005.
- Schecter A (Schecter 2005), Päpke O, Tung KC, Joseph J, Harris TR, Dahlgren J, Polybrominated Diphenyl Ether Flame Retardants in the U.S. Population: Current Levels, Temporal Trends, and Comparison With Dioxins, Dibenzofurans, and Polychlorinated Biphenyls, *Journal of Occupational & Environmental Medicine*. 47(3), 199-211, March 2005.
- Searl A, "Review of the Potential Human Exposure to decaDBE and the Associated Risks to Health," Institute of Occupational Medicine, Nov. 17, 2003.
- Sjödin A (Sjödin 1999), Hagmar L, Klasson-Wehler E, Kronholm-Diab K, Jakobson E, and Bergman Å, "Flame Retardant Exposure: Polybrominated Diphenyl Ethers in Blood from Swedish Workers," *Environmental Health Perspectives*, Volume 107, Number 8, August 1999.
- Swedish Society for Nature Conservation (SSNC), "The SSNC Believes That:" "The Effects of Various Brominated Flame Retardants," and "What Are Brominated Flame Retardants?" 2005.
- Teuten EL, Xu L, and Reddy CM (Teuten 2005), "Two Abundant Bioaccumulated Halogenated Compounds Are Natural Products," *Science*, Vol. 307, pp. 917-920, Feb. 11, 2005.
- Thomson K, Environmental Affairs, Boeing Company, comments submitted to Department of Ecology, State of Washington, regarding Draft PBDE Chemical Action Plan, Nov. 14, 2004.
- University of Surrey (Surrey 2002), "International Fire Statistics and the Potential Benefits of Fire Counter-Measures," Emsley AM, Lim L, and Stevens GC, Polymer Research Centre, "FR2002" Conference, London, February, 2002.
- University of Surrey (Surrey 2005), "International Fire Statistics and the Potential Benefits of Fire Counter-Measures," Emsley AM, Lim L, and Stevens GC, Polymer Research Centre, and Peter Williams, Department of Mathematics and Statistics, May 2005.
- U.S. Consumer Product Safety Commission (CPSC), "Briefing Package on Upholstered Furniture Flammability: Regulatory Options," October 2001.
- U.S. Environmental Protection Agency's (USEPA 1993), "Reference Dose (RfD): Description and Use in Health Risk Assessments," Background Document 1A, March 15, 1993.
- U.S. Environmental Protection Agency (USEPA 2004), 40 CFR Part 721, "Certain Polybrominated Diphenylethers: Proposed Significant New Use Rule," *Federal Register*, Vol. 69, No. 233, p. 70404, Dec. 6, 2004.
- U.S. Environmental Protection Agency (USEPA 2005a), Office of Pollution Prevention & Toxics, "Polybrominated diphenylethers," Dec. 30, 2005.
- U.S. Environmental Protection Agency (USEPA 2005b), Design for the Environment, Furniture Flame Retardancy Partnership, "Environmental Profiles of Chemical Flame-Retardant Alternatives for Low-Density Polyurethane Foam," September 2005.
- U.S. Environmental Protection Agency (USEPA 2006a), Office of Pollution Prevention & Toxics, "Polybrominated diphenylethers (PBDEs) Significant New Use Rule (SNUR) Questions and Answers," Jan. 12, 2006.
- U.S. Environmental Protection Agency (USEPA 2006b), "Polybrominated Diphenyl Ethers (PBDEs) Project Plan," March 2006.
- U.S. Fire Administration (USFA 2005a), U.S. Department of Homeland Security, "National Fire Statistics," Sept. 22, 2005.
- U.S. Fire Administration (USFA 2005b), National Fire Data Center, U.S. Department of Homeland Security, *Residential Fires and Child Casualties: Topical Fire Research Series*, Volume 5, Issue 2, April 2005.
- U.S. Fire Administration (USFA 2005c), National Fire Data Center, U.S. Department of Homeland Security, *Residential Fires and Older Adult Casualties: Topical Fire Research Series*, Volume 5, Issue 3, June 2005.
- U.S. Public Health Service, National Institute of Occupational Safety and Health, letter to U.S. Consumer Product Safety Commission, Feb. 15, 2001.
- Vieth B, Rudiger T, Ostermann B, Mielke H (2005) Rückstände von Flammenschutzmitteln in Frauenmilch aus Deutschland unter besonderer Berücksichtigung von polybromierten Diphenylethern (PBDE). Report of the Bundesinstitute für Risikobewertung. Forderkennzeichen (UFOPLAN) 202 61 218/03
- Vieth B, Hermann T, Mielke H, Ostermann B, Papke O, Rudiger T (2004) PBDE levels in human milk: the situation in Germany and potential influencing factors – a controlled study. *Organohalogen Compounds* 66, 2643-2648, 2004.
- Voluntary Children's Chemical, Evaluation Program (VCCEP), "Report of the Peer Consultation Meeting On, Decabromodiphenyl Ether," April 2 and 3, 2003, American Chemistry Council's Brominated, Flame Retardant Industry Panel, Sep. 30, 2003.
- Washington Physicians for Social Responsibility (WPSR 2004), Comments to the Draft PBDE Chemical Action Plan, Nov. 9, 2004.
- WHO (World Health Organization), Environmental health criteria 162: Brominated diphenyl ethers. International Programme on Chemical Safety, Geneva, Switzerland, 1994.
- WHO (World Health Organization), Environmental health criteria 192: Flame Retardants: A General Introduction, International Programme on Chemical Safety, Geneva, Switzerland, 1997.

ACSH BOARD OF TRUSTEES

Frederick Anderson, Esq. <i>McKenna Long & Aldridge</i>	James E. Enstrom, Ph.D., M.P.H. <i>University of California, Los Angeles</i>	Thomas Campbell Jackson, M.P.H. <i>Pamela B. Jackson and Thomas C. Jackson Charitable Fund</i>	Kenneth M. Prager, M.D. <i>Columbia University Medical Center</i>
Nigel Bark, M.D. <i>Albert Einstein College of Medicine</i>	Jack Fisher, M.D. <i>University of California, San Diego</i>	Elizabeth McCaughey, Ph.D. <i>Committee to Reduce Infection Deaths</i>	Katherine L. Rhyne, Esq. <i>King & Spalding LLP</i>
Elissa P. Benedek, M.D. <i>University of Michigan Medical School</i>	Hon. Bruce S. Gelb <i>New York, NY</i>	Henry I. Miller, M.D. <i>The Hoover Institution</i>	Lee M. Silver, Ph.D. <i>Princeton University</i>
Norman E. Borlaug, Ph.D. <i>Texas A&M University</i>	Donald A. Henderson, M.D., M.P.H. <i>University of Pittsburgh Medical Center</i>	Rodney W. Nichols <i>Indo-US Science & Technology Forum</i>	Thomas P. Stossel, M.D. <i>Harvard Medical School</i>
Michael B. Bracken, Ph.D., M.P.H. <i>Yale University School of Medicine</i>			Elizabeth M. Whelan, Sc.D., M.P.H. <i>American Council on Science and Health</i>

ACSH FOUNDERS CIRCLE

Christine M. Bruhn, Ph.D. <i>University of California, Davis</i>	A. Alan Moghissi, Ph.D. <i>Institute for Regulatory Science</i>	Stephen S. Sternberg, M.D. <i>Memorial Sloan-Kettering Cancer Center</i>	Robert J. White, M.D., Ph.D. <i>Case Western Reserve University</i>
Taiwo K. Danmola, C.P.A. <i>Ernst & Young</i>	John Moore, Ph.D., M.B.A <i>Grove City College, President Emeritus</i>	Lorraine Thelian <i>Ketchum</i>	
Thomas R. DeGregori, Ph.D. <i>University of Houston</i>	Albert G. Nickel <i>Lyons Lavey Nickel Swift, Inc.</i>	Kimberly M. Thompson, Sc.D. <i>Massachusetts Institute of Technology</i>	

ACSH EXECUTIVE STAFF

Elizabeth M. Whelan, Sc.D., M.P.H., President

ACSH BOARD OF SCIENTIFIC AND POLICY ADVISORS

Ernest L. Abel, Ph.D. <i>C.S. Mott Center</i>	Robert L. Brent, M.D., Ph.D. <i>Thomas Jefferson University / A. I. duPont Hospital for Children</i>	Michael D. Corbett, Ph.D. <i>Omaha, NE</i>	J. Gordon Edwards, Ph.D. <i>San José State University</i>
Gary R. Acuff, Ph.D. <i>Texas A&M University</i>	Allan Brett, M.D. <i>University of South Carolina</i>	Morton Corn, Ph.D. <i>John Hopkins University</i>	George E. Ehrlich, M.D., M.B. <i>Philadelphia, PA</i>
Julie A. Albrecht, Ph.D. <i>University of Nebraska, Lincoln</i>	Kenneth G. Brown, Ph.D. <i>KBinc</i>	Nancy Cotugna, Dr.Ph., R.D., C.D.N. <i>University of Delaware</i>	Michael P. Elston, M.D., M.S. <i>Western Health</i>
James E. Alcock, Ph.D. <i>Glendon College, York University</i>	Gale A. Buchanan, Ph.D. <i>Adel, GA</i>	H. Russell Cross, Ph.D. <i>National Beef</i>	William N. Elwood, Ph.D. <i>Key West, FL</i>
Thomas S. Allems, M.D., M.P.H. <i>San Francisco, CA</i>	George M. Burditt, J.D. <i>Bell, Boyd & Lloyd LLC</i>	James W. Curran, M.D., M.P.H. <i>Rollins School of Public Health, Emory University</i>	Stephen K. Epstein, M.D., M.P.P., FACEP <i>Beth Israel Deaconess Medical Center</i>
Richard G. Allison, Ph.D. <i>American Society for Nutritional Sciences</i>	Edward E. Burns, Ph.D. <i>Texas A&M University</i>	Charles R. Curtis, Ph.D. <i>Ohio State University</i>	Myron E. Essex, D.V.M., Ph.D. <i>Harvard School of Public Health</i>
John B. Allred, Ph.D. <i>Ohio State University</i>	Francis F. Busta, Ph.D. <i>University of Minnesota</i>	Ilene R. Danse, M.D. <i>Bolinas, CA</i>	Terry D. Etherton, Ph.D. <i>Pennsylvania State University</i>
Philip R. Alper, M.D. <i>University of California, San Francisco</i>	Elwood F. Caldwell, Ph.D., M.B.A. <i>University of Minnesota</i>	Robert M. Devlin, Ph.D. <i>University of Massachusetts</i>	R. Gregory Evans, Ph.D., M.P.H. <i>St. Louis University Center for the Study of Bioterrorism and Emerging Infections</i>
Karl E. Anderson, M.D. <i>University of Texas Medical Branch, Galveston</i>	Zerle L. Carpenter, Ph.D. <i>Texas A&M University</i>	Seymour Diamond, M.D. <i>Diamond Headache Clinic</i>	William Evans, Ph.D. <i>University of Alabama</i>
Dennis T. Avery <i>Hudson Institute</i>	Robert G. Cassens, Ph.D. <i>University of Wisconsin, Madison</i>	Donald C. Dickson, M.S.E.E. <i>Gilbert, AZ</i>	Daniel F. Farkas, Ph.D., M.S., P.E. <i>Oregon State University</i>
Ronald P. Bachman, M.D. <i>Kaiser-Permanente Medical Center</i>	Ercole L. Cavalieri, D.Sc. <i>University of Nebraska Medical Center</i>	Ralph Dittman, M.D., M.P.H. <i>Houston, TX</i>	Richard S. Fawcett, Ph.D. <i>Huxley, IA</i>
Robert S. Baratz, D.D.S., Ph.D., M.D. <i>International Medical Consultation Services</i>	Russell N. A. Cecil, M.D., Ph.D. <i>Albany Medical College</i>	John E. Dodes, D.D.S. <i>National Council Against Health Fraud</i>	Owen R. Fennema, Ph.D. <i>University of Wisconsin, Madison</i>
Stephen Barrett, M.D. <i>Allentown, PA</i>	Rino Cerio, M.D. <i>Barts and The London Hospital Institute of Pathology</i>	Theron W. Downes, Ph.D. <i>Michigan State University</i>	Frederick L. Ferris, III, M.D. <i>National Eye Institute</i>
Thomas G. Baumgartner, Pharm.D., M.Ed. <i>University of Florida</i>	Morris E. Chafetz, M.D. <i>Health Education Foundation</i>	Michael P. Doyle, Ph.D. <i>University of Georgia</i>	David N. Ferro, Ph.D. <i>University of Massachusetts</i>
W. Lawrence Beeson, Dr.P.H. <i>Loma Linda University School of Public Health</i>	Bruce M. Chassy, Ph.D. <i>University of Illinois, Urbana-Champaign</i>	Adam Drewnowski, Ph.D. <i>University of Washington</i>	Madelon L. Finkel, Ph.D. <i>Weill Medical College of Cornell University</i>
Sir Colin Berry, D.Sc., Ph.D., M.D. <i>Institute of Pathology, Royal London Hospital</i>	Martha A. Churchill, Esq. <i>Milan, MI</i>	Michael A. Dubick, Ph.D. <i>U.S. Army Institute of Surgical Research</i>	Kenneth D. Fisher, Ph.D. <i>Office of Disease Prevention and Health</i>
Barry L. Beyerstein, Ph.D. <i>Simon Fraser University</i>	Emil William Chynn, M.D., FACS., M.B.A. <i>New York Eye & Ear Infirmary</i>	Greg Dubord, M.D., M.P.H. <i>Toronto Center for Cognitive Therapy</i>	Leonard T. Flynn, Ph.D., M.B.A. <i>Morganville, NJ</i>
Steven Black, M.D. <i>Kaiser-Permanente Vaccine Study Center</i>	Dean O. Cliver, Ph.D. <i>University of California, Davis</i>	Edward R. Duffie, Jr., M.D. <i>Savannah, GA</i>	William H. Foege, M.D., M.P.H. <i>Emory University</i>
Blaine L. Blad, Ph.D. <i>Kanosh, UT</i>	F. M. Clydesdale, Ph.D. <i>University of Massachusetts</i>	Leonard J. Duhl, M.D. <i>University of California, Berkeley</i>	Ralph W. Fogleman, D.V.M. <i>Doylestown, PA</i>
Hinrich L. Bohn, Ph.D. <i>University of Arizona</i>	Donald G. Cochran, Ph.D. <i>Virginia Polytechnic Institute and State University</i>	David F. Duncan, Dr.P.H. <i>Duncan & Associates</i>	Christopher H. Foreman, Jr., Ph.D. <i>University of Maryland</i>
Ben W. Bolch, Ph.D. <i>Rhodes College</i>	W. Ronnie Coffman, Ph.D. <i>Cornell University</i>	James R. Dunn, Ph.D. <i>Averill Park, NY</i>	F. J. Francis, Ph.D. <i>University of Massachusetts</i>
Joseph F. Borzelleca, Ph.D. <i>Medical College of Virginia</i>	Bernard L. Cohen, D.Sc. <i>University of Pittsburgh</i>	Robert L. DuPont, M.D. <i>Institute for Behavior and Health</i>	Glenn W. Froning, Ph.D. <i>University of Nebraska, Lincoln</i>
Michael K. Botts, Esq. <i>Ankeny, IA</i>	John J. Cohrsens, Esq. <i>Public Health Policy Advisory Board</i>	Henry A. Dymsha, Ph.D. <i>University of Rhode Island</i>	Vincent A. Fulginiti, M.D. <i>Tucson, AZ</i>
George A. Bray, M.D. <i>Pennington Biomedical Research Center</i>	Gerald F. Combs, Jr., Ph.D. <i>USDA Grand Forks Human Nutrition Center</i>	Michael W. Easley, D.D.S., M.P.H. <i>International Health Management & Research Associates</i>	Robert S. Gable, Ed.D., Ph.D., J.D. <i>Claremont Graduate University</i>
Ronald W. Brecher, Ph.D., C.Chem., DABT <i>GlobalTox International Consultants, Inc.</i>			Shayne C. Gad, Ph.D., D.A.B.T., A.T.S. <i>Gad Consulting Services</i>

William G. Gaines, Jr., M.D., M.P.H.
Scott & White Clinic

Charles O. Gallina, Ph.D.
Professional Nuclear Associates

Raymond Gambino, M.D.
Quest Diagnostics Incorporated

Randy R. Gaugler, Ph.D.
Rutgers University

J. Bernard L. Gee, M.D.
Yale University School of Medicine

K. H. Ginzel, M.D.
University of Arkansas for Medical Science

William Paul Glezen, M.D.
Baylor College of Medicine

Jay A. Gold, M.D., J.D., M.P.H.
Medical College of Wisconsin

Roger E. Gold, Ph.D.
Texas A&M University

Reneé M. Goodrich, Ph.D.
University of Florida

Frederick K. Goodwin, M.D.
The George Washington University Medical Center

Timothy N. Gorski, M.D., F.A.C.O.G.
University of North Texas

Ronald E. Gots, M.D., Ph.D.
International Center for Toxicology and Medicine

Henry G. Grabowski, Ph.D.
Duke University

James Ian Gray, Ph.D.
Michigan State University

William W. Greaves, M.D., M.S.P.H.
Medical College of Wisconsin

Kenneth Green, D.Env.
American Interprise Institute

Laura C. Green, Ph.D., D.A.B.T.
Cambridge Environmental, Inc.

Saul Green, Ph.D.
Zol Consultants

Richard A. Greenberg, Ph.D.
Hinsdale, IL

Sander Greenland, Dr.P.H., M.S., M.A.
UCLA School of Public Health

Gordon W. Gribble, Ph.D.
Dartmouth College

William Grierson, Ph.D.
University of Florida

Lester Grinspoon, M.D.
Harvard Medical School

F. Peter Guengerich, Ph.D.
Vanderbilt University School of Medicine

Caryl J. Guth, M.D.
Advance, NC

Philip S. Guzelian, M.D.
University of Colorado

Terry J. Hartman, Ph.D., M.P.H., R.D.
The Pennsylvania State University

Clare M. Hasler, Ph.D.
*The Robert Mondavi Institute of Wine and Food Science,
University of California, Davis*

Robert D. Havener, M.P.A.
Sacramento, CA

Virgil W. Hays, Ph.D.
University of Kentucky

Cheryl G. Healtson, Dr.PH.
*J.L. Mailman School of Public Health of Columbia
University*

Clark W. Heath, Jr., M.D.
American Cancer Society

Dwight B. Heath, Ph.D.
Brown University

Robert Heimer, Ph.D.
Yale School of Public Health

Robert B. Helms, Ph.D.
American Enterprise Institute

Zane R. Helsel, Ph.D.
Rutgers University, Cook College

James D. Herbert, Ph.D.
Drexel University

Gene M. Heyman, Ph.D.
McLean Hospital/Harvard Medical School

Richard M. Hoor, Ph.D.
Savannah, GA

Theodore R. Holford, Ph.D.
Yale University School of Medicine

Robert M. Hollingworth, Ph.D.
Michigan State University

Edward S. Horton, M.D.
Joslin Diabetes Center/Harvard Medical School

Joseph H. Hotchkiss, Ph.D.
Cornell University

Steve E. Hruddy, Ph.D.
University of Alberta

Susanne L. Huttner, Ph.D.
University of California, Berkeley

Robert H. Imrie, D.V.M.
Seattle, WA

Lucien R. Jacobs, M.D.
University of California, Los Angeles

Alejandro R. Jadad, M.D., D.Phil., F.R.C.P.C.
University of Toronto

Rudolph J. Jaeger, Ph.D.
Environmental Medicine, Inc.

William T. Jarvis, Ph.D.
Loma Linda University

Michael Kamrin, Ph.D.
Michigan State University

John B. Kaneene, D.V.M., M.P.H., Ph.D.
Michigan State University

P. Andrew Karam, Ph.D., CHP
MJW Corporation

Philip G. Keeney, Ph.D.
Pennsylvania State University

John G. Keller, Ph.D.
Olney, MD

Kathryn E. Kelly, Dr.P.H.
Delta Toxicology

George R. Kerr, M.D.
University of Texas, Houston

George A. Keyworth II, Ph.D.
Progress and Freedom Foundation

Michael Kirsch, M.D.
Highland Heights, OH

John C. Kirschman, Ph.D.
Emmaus, PA

Ronald E. Kleinman, M.D.
Massachusetts General Hospital/ Harvard Medical School

Leslie M. Klevay, M.D., S.D. in Hyg.
*University of North Dakota School of Medicine and Health
Sciences*

David M. Klurfeld, Ph.D.
U.S. Department of Agriculture

Kathryn M. Kolasa, Ph.D., R.D.
East Carolina University

James S. Koopman, M.D, M.P.H.
University of Michigan School of Public Health

Alan R. Kristal, Dr.P.H.
Fred Hutchinson Cancer Research Center

David Kritchevsky, Ph.D.
The Wistar Institute

Stephen B. Kritchevsky, Ph.D.
Wake Forest University Baptist Medical Center

Mitzi R. Krockover, M.D.
SSB Solutions

Manfred Kroger, Ph.D.
Pennsylvania State University

Laurence J. Kulp, Ph.D.
University of Washington

Sandford F. Kuvin, M.D.
*University of Miami School of Medicine/ Hebrew
University of Jerusalem*

Carolyn J. Lackey, Ph.D., R.D.
North Carolina State University

J. Clayburn LaForce, Ph.D.
University of California, Los Angeles

Pagona Lagioui, M.D., Ph.D.
University of Athens Medical School

James C. Lamb, IV, Ph.D., J.D., D.A.B.T.
The Weinberg Group

Lawrence E. Lamb, M.D.
San Antonio, TX

William E. M. Lands, Ph.D.
College Park, MD

Lillian Langseth, Dr.P.H.
Lyda Associates, Inc.

Brian A. Larkins, Ph.D.
University of Arizona

Larry Laudon, Ph.D.
National Autonomous University of Mexico

Tom B. Leamon, Ph.D.
Liberty Mutual Insurance Company

Jay H. Lehr, Ph.D.
Environmental Education Enterprises, Inc.

Brian C. Lentle, M.D., FRCPC, DMRD
University of British Columbia

Floy Lilley, J.D.
Fernandina Beach, FL

Paul J. Lioy, Ph.D.
UMDNJ-Robert Wood Johnson Medical School

William M. London, Ed.D., M.P.H.
Charles R. Drew University of Medicine and Science

Frank C. Lu, M.D., BCFE
Miami, FL

William M. Lunch, Ph.D.
Oregon State University

Daryl Lund, Ph.D.
University of Wisconsin

George D. Lundberg, M.D.
Medscape General Medicine

Howard D. Maccabee, Ph.D., M.D.
Alamo, CA

Janet E. Macheledt, M.D., M.S., M.P.H.
Houston, TX

Roger P. Maickel, Ph.D.
Purdue University

Henry G. Manne, J.S.D.
George Mason University Law School

Karl Maramorosch, Ph.D.
Rutgers University, Cook College

Judith A. Marlett, Ph.D., R.D.
University of Wisconsin, Madison

James R. Marshall, Ph.D.
Roswell Park Cancer Institute

Mary H. McGrath, M.D., M.P.H.
University of California, San Francisco

Alan G. McHughen, D.Phil.
University of California, Riverside

James D. McKean, D.V.M., J.D.
Iowa State University

Patrick J. Michaels, Ph.D.
University of Virginia

Thomas H. Milby, M.D., M.P.H.
Walnut Creek, CA

Joseph M. Miller, M.D., M.P.H.
Durham, NH

William J. Miller, Ph.D.
University of Georgia

Dade W. Moeller, Ph.D.
Harvard University

Grace P. Monaco, J.D.
Medical Care Management Corp.

Brian E. Mondell, M.D.
Baltimore Headache Institute

John W. Morgan, Dr.P.H.
California Cancer Registry

Stephen J. Moss, D.D.S., M.S.
*New York University College of Dentistry/ Health
Education Enterprises, Inc.*

Brooke T. Mossman, Ph.D.
University of Vermont College of Medicine

Allison A. Muller, Pharm.D
The Children's Hospital of Philadelphia

Ian C. Munro, F.A.T.S., Ph.D., FRCPath
Cantox Health Sciences International

Harris M. Nagler, M.D.
*Beth Israel Medical Center/ Albert Einstein College of
Medicine*

Daniel J. Ncayiyana, M.D.
Durban Institute of Technology

Philip E. Nelson, Ph.D.
Purdue University

Joyce A. Nettleton, D.Sc., R.D.
Denver, CO

John S. Neuberger, Dr.P.H.
University of Kansas School of Medicine

Gordon W. Newell, Ph.D., M.S., F-A.T.S.
Cupertino, CA

Thomas J. Nicholson, Ph.D., M.P.H.
Western Kentucky University

Steven P. Novella, M.D.
Yale University School of Medicine

James L. Oblinger, Ph.D.
North Carolina State University

Deborah L. O'Connor, Ph.D.
University of Toronto/ The Hospital for Sick Children

John Patrick O'Grady, M.D.
Tufts University School of Medicine

James E. Oldfield, Ph.D.
Oregon State University

Stanley T. Omaye, Ph.D., F-A.T.S., FACN, C.N.S.
University of Nevada, Reno

Michael T. Osterholm, Ph.D., M.P.H.
University of Minnesota

Michael W. Pariza, Ph.D.
University of Wisconsin, Madison

Stuart Patton, Ph.D.
Pennsylvania State University

James Marc Perrin, M.D.
Mass General Hospital for Children

Timothy Dukes Phillips, Ph.D.
Texas A&M University

Mary Frances Picciano, Ph.D.
National Institutes of Health

David R. Pike, Ph.D.
University of Illinois, Urbana-Champaign

Thomas T. Poleman, Ph.D.
Cornell University

Gary P. Posner, M.D.
Tampa, FL

John J. Powers, Ph.D.
University of Georgia

William D. Powrie, Ph.D.
University of British Columbia

C.S. Prakash, Ph.D.
Tuskegee University

Marvin P. Pritts, Ph.D.
Cornell University

Daniel J. Raiten, Ph.D.
National Institute of Health

David W. Ramey, D.V.M.
Ramey Equine Group

R.T. Ravenholt, M.D., M.P.H.
Population Health Imperatives

Russel J. Reiter, Ph.D.
University of Texas, San Antonio

William O. Robertson, M.D.
University of Washington School of Medicine

J. D. Robinson, M.D.
Georgetown University School of Medicine

Bill D. Roebuck, Ph.D., D.A.B.T.
Dartmouth Medical School

David B. Roll, Ph.D.
The United States Pharmacopeia

Dale R. Romsos, Ph.D. <i>Michigan State University</i>	Michael B. Shermer, Ph.D. <i>Skeptical Magazine</i>	Ronald D. Stewart, O.C., M.D., FRCPC <i>Dalhousie University</i>	John Weisburger, M.D., Ph.D. <i>Institute for Cancer Prevention/ New York Medical College</i>
Joseph D. Rosen, Ph.D. <i>Cook College, Rutgers University</i>	Sidney Shindell, M.D., LL.B. <i>Medical College of Wisconsin</i>	Martha Barnes Stone, Ph.D. <i>Colorado State University</i>	Janet S. Weiss, M.D. <i>The ToxDoc</i>
Steven T. Rosen, M.D. <i>Northwestern University Medical School</i>	Sarah Short, Ph.D., Ed.D., R.D. <i>Syracuse University</i>	Jon A. Story, Ph.D. <i>Purdue University</i>	Simon Wessley, M.D., FRCP <i>King's College London and Institute of Psychiatry</i>
Kenneth J. Rothman, Dr.P.H. <i>Boston University School of Public Health</i>	A. J. Siedler, Ph.D. <i>University of Illinois, Urbana-Champaign</i>	Michael M. Sveda, Ph.D. <i>Gaithersburg, MD</i>	Steven D. Wexner, M.D. <i>Cleveland Clinic Florida</i>
Stanley Rothman, Ph.D. <i>Smith College</i>	Marc K. Siegel, M.D. <i>New York University School of Medicine</i>	Glenn Swogger, Jr., M.D. <i>Topeka, KS</i>	Joel Elliot White, M.D., F.A.C.R. <i>Danville, CA</i>
Stephen H. Safe, D.Phil. <i>Texas A&M University</i>	Michael S. Simon, M.D., M.P.H. <i>Wayne State University</i>	Sita R. Totini, Ph.D. <i>University of Minnesota</i>	Carol Whitlock, Ph.D., R.D. <i>Rochester Institute of Technology</i>
Wallace I. Sampson, M.D. <i>Stanford University School of Medicine</i>	S. Fred Singer, Ph.D. <i>Science & Environmental Policy Project</i>	Steve L. Taylor, Ph.D. <i>University of Nebraska, Lincoln</i>	Christopher F. Wilkinson, Ph.D. <i>Wilmington, NC</i>
Harold H. Sandstead, M.D. <i>University of Texas Medical Branch</i>	Robert B. Sklaroff, M.D. <i>Elkins Park, PA</i>	James W. Tillotson, Ph.D., M.B.A. <i>Tufts University</i>	Mark L. Willenbring, M.D., Ph.D. <i>National Institute on Alcohol Abuse and Alcoholism</i>
Charles R. Santerre, Ph.D. <i>Purdue University</i>	Anne M. Smith, Ph.D., R.D., L.D. <i>Ohio State University</i>	Dimitrios Trichopoulos, M.D. <i>Harvard School of Public Health</i>	Carl K. Winter, Ph.D. <i>University of California, Davis</i>
Sally L. Satel, M.D. <i>American Enterprise Institute</i>	Gary C. Smith, Ph.D. <i>Colorado State University</i>	Murray M. Tuckerman, Ph.D. <i>Winchendon, MA</i>	James J. Worman, Ph.D. <i>Rochester Institute of Technology</i>
Lowell D. Satterlee, Ph.D. <i>Vergas, MN</i>	John N. Sofos, Ph.D. <i>Colorado State University</i>	Robert P. Upchurch, Ph.D. <i>University of Arizona</i>	Russell S. Worrall, O.D. <i>University of California, Berkeley</i>
Jeffrey W. Savell <i>Texas A&M University</i>	Roy F. Spalding, Ph.D. <i>University of Nebraska, Lincoln</i>	Mark J. Utell, M.D. <i>University of Rochester Medical Center</i>	Steven H. Zeisel, M.D., Ph.D. <i>University of North Carolina</i>
Marvin J. Schissel, D.D.S. <i>Roslyn Heights, NY</i>	Leonard T. Sperry, M.D., Ph.D. <i>Barry University</i>	Shashi B. Verma, Ph.D. <i>University of Nebraska, Lincoln</i>	Michael B. Zemel, Ph.D. <i>Nutrition Institute, University of Tennessee</i>
Edgar J. Schoen, M.D. <i>Kaiser Permanente Medical Center</i>	Robert A. Squire, D.V.M., Ph.D. <i>Johns Hopkins University</i>	Willard J. Visek, M.D., Ph.D. <i>University of Illinois College of Medicine</i>	Eckhard E. Ziegler, M.D. <i>University of Iowa</i>
David Schottenfeld, M.D., M.Sc. <i>University of Michigan</i>	Ronald T. Stanko, M.D. <i>University of Pittsburgh Medical Center</i>	Lynn Waishwell, Ph.D., C.H.E.S. <i>University of Medicine and Dentistry of New Jersey, School of Public Health</i>	
Joel M. Schwartz, M.S. <i>American Enterprise Institute</i>	James H. Steele, D.V.M., M.P.H. <i>University of Texas, Houston</i>	Donald M. Watkin, M.D., M.P.H., F.A.C.P. <i>George Washington University</i>	
David E. Seidemann, Ph.D. <i>Brooklyn College</i>	Robert D. Steele, Ph.D. <i>Pennsylvania State University</i>	Miles Weinberger, M.D. <i>University of Iowa Hospitals and Clinics</i>	
Patrick J. Shea, Ph.D. <i>University of Nebraska, Lincoln</i>	Judith S. Stern, Sc.D., R.D. <i>University of California, Davis</i>		

The opinions expressed in ACSH publications do not necessarily represent the views of all members of the ACSH Board of Trustees, Founders Circle and Board of Scientific and Policy Advisors, who all serve without compensation.

ACSH STAFF

Julianne M. Chickering <i>Research Associate</i>	Patricia A. Keenan <i>Executive Assistant to the President</i>	Cheryl E. Martin <i>Associate Director</i>	Todd Seavey <i>Director of Publications</i>
Judith A. D'Agostino <i>Administrative Assistant</i>	A. Marcial C. Lapeña <i>Accountant</i>	Gilbert L. Ross, M.D. <i>Executive and Medical Director</i>	Jeff Stier, Esq. <i>Associate Director</i>
Jaclyn Eisenberg <i>Research Assistant</i>	Jennifer Lee <i>Art Director</i>	Tara McTeague <i>Development Assistant</i>	
Ruth Kava, Ph.D., R.D. <i>Director of Nutrition</i>	Molly Lee <i>Research Assistant</i>		

PUBLICATIONS ORDER FORM

TITLE

QUANTITY

QUANTITY

BOOKS

America's War on "Carcinogens"—\$15.95 each
Are Children More Vulnerable to Environmental Chemicals?—\$19.95 each
Cigarettes: What the Warning Label Doesn't Tell You—\$19.95 each
Qty X \$19.95 = \$
A Citizen's Guide to Terrorism Preparedness and Response—\$11.95 each
New Yorker's Guide to Terrorism Preparedness and Response—\$11.95 each
Qty X \$11.95 = \$

BOOKLETS AND SPECIAL REPORTS (\$5.00 each)
A Comparison of the Health Effects of Alcohol Consumption and Tobacco Use in America
A Summary of America's War on "Carcinogens"
AIDS in New York City: Update 2001
Alzheimer's Disease: A Status Report For 2002
Anthrax: What You Need to Know
Avian Influenza, or "Bird Flu": What You Need to Know
Biomonitoring: Measuring Levels of Chemicals in People and What the Results Mean
Biotech Pharmaceuticals and Biotherapy
Biotechnology and Food
California's Proposition 65 and Its Impact on Public Health
Cancer Clusters: Findings Vs. Feelings
Chemoprevention of Breast Cancer
Chemoprevention of Coronary Heart Disease
Does Nature Know Best? Natural Carcinogens
Eating Safely: Avoiding Foodborne Illness
The Efficacy, Safety and Benefits of bST and pST
Endocrine Disrupters: A Scientific Perspective
Environmental Tobacco Smoke
The Facts About "Dirty Bombs"
Facts About Fats
Facts About "Functional Foods"
Facts Versus Fears (fourth edition)
Fat Replacers
Feeding Baby Safely
Good Stories, Bad Science: A Guide for Journalists to the Health Claims of "Consumer Activist" Groups
Health and Safety Tips for Your Summer Vacation
Holiday Dinner Menu
The Irreversible Health Effects of Cigarette Smoking
Kicking Butts in the Twenty-First Century: What Modern Science Has Learned about Smoking Cessation
Making Sense of Over-the-Counter Pain Relievers
Moderate Alcohol Consumption and Health
Much Ado About Milk
Nutrition Accuracy in Popular Magazines 1997-1999
Nutrition Accuracy in Popular Magazines 1995-1996
Of Mice and Mandates: Animal Experiments, Human Cancer Risk and Regulatory Policies
Postmenopausal Hormone Replacement Therapy

Regulating Mercury Emissions From Power Plants: Will It Protect Our Health?
Risk Factors for Prostate Cancer: Facts, Speculation and Myths
Risk Factors of Breast Cancer
The Role of Beef in the American Diet
The Role of Eggs in the Diet
School Buses and Diesel Fuel
Silicone-Gel Breast Implants: Health and Regulatory Update 2000
Smoking and Women's Magazines
Sugar Substitutes and Health
Teflon and Human Health: Do the Charges Stick?
Assessing the Safety of the Chemical PFOA
Traces of Environmental Chemicals in the Human Body: Are They a Risk to Health?
Vegetarianism
Vitamins and Minerals
Weighing Benefits and Risks in Pharmaceutical Use: a Consumer's Guide
Writing about Health Risks: Challenges and Strategies
Qty X \$5.00 = \$
SPECIAL RELEASES (\$3.00 each)
A Critical Assessment of "Lies, Damned Lies, & 400,000 Smoking-Related Deaths"
Analysis of Alleged Health Risk from DBCP in Drinking Water
Estrogen and Health: How Popular Magazines Have Dealt with Hormone Replacement Therapy
Rumble in the Bronx: Mass Hysteria and the "Chemicalization" of Demonology
Safe, Long-lasting Pressure-Treated Wood
Should Long-Chain Polyunsaturated Fatty Acids Be Added to Infant Formula?
Three Mile Island: A 20th Anniversary Remembrance
Qty X \$3.00 = \$
BROCHURES (\$1.00 each)
What's the Story: The Scientific Facts About...
Drug-Supplement Interaction
The Dry-Cleaning Chemical Perc
Eggs
Health Claims Against Cosmetics: How Do They Look in the Light?
The Role of Milk in Your Diet
Olestra
MMT's
Pressure-Treated Wood
Weighing the Benefits and Risks of Your Medications
Qty X \$1.00 = \$

MEMBERSHIP / PAYMENT

A MEMBERSHIP APPLICATION

A financial contribution to ACSH entitles you to receive all new ACSH publications as they are released, and a 25% discount on all additional ACSH publication purchases. All contributions are tax-deductible as permitted by law.

Yes, I want to join ACSH. I wish to donate

- ☐ Member \$50-249
- ☐ Contributor \$1,000-4,999
- ☐ Friend \$250-999
- ☐ Supporter \$5,000-9,999

B PUBLICATION SUMMARY

Books @ \$19.95 \$

Books @ \$11.95 \$

Booklets and Special Reports \$

Special Releases \$

Brochures \$

Subtotal \$

ACSH Member Discount (deduct 25%) —

Publication Total \$

PUBLICATION BULK RATES
Call ACSH for special discounts on Book orders of 50 copies or more.

- BOOKLETS AND SPECIAL REPORTS
- ☐ Non-Members
1-499 copies \$5.00 each
500-999 copies \$3.00 each
1000+ copies \$2.50 each
- ☐ ACSH Members
1-499 copies \$3.75 per copy
500 or more copies see regular bulk rates

C INTERNATIONAL POSTAGE AND HANDLING CHARGES ARE AS FOLLOWS:

- (Overseas orders must be prepaid in U.S. currency, or charged to Visa or Mastercard)
- 1-2 copies Add \$3.00 to total
- 3-6 copies Add \$7.00 to total
- 7-9 copies Add \$12.00 to total
- 10 or more copies Please contact ACSH

INTERNATIONAL POSTAGE FOR BOOKS ONLY:

- 1-2 copies Add \$8.00 to total
- 3-6 copies Add \$16.00 to total
- 7-9 copies Add \$26.00 to total
- 10 or more copies Please contact ACSH

ORDER SUMMARY

A Membership Total \$

B Publications Total \$

C International Postage (if applicable) \$

GRAND TOTAL \$

MAIL TO:

AMERICAN COUNCIL ON SCIENCE AND HEALTH
1995 Broadway • 2nd Floor • New York • NY 10023
acsh.org; HealthFactsAndFears.com

Or call: (212) 362-7044 • Or fax: (212) 362-4919 • Or e-mail: orders@acsh.org