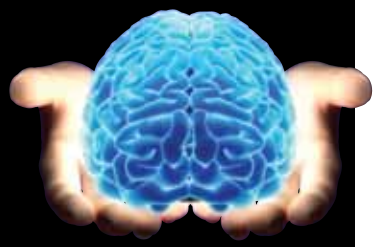
MIND, DISRUPTED

How toxic chemicals change how we think and who we are



Polybrominated Diphenyl Ethers (PBDEs)

What Are PBDEs?

Polybrominated diphenyl ethers, or PBDEs, are a class of flame retardant chemicals added to many consumer products found in the home, office, automobiles, and airplanes.[1] Three mixtures used widely—penta-BDE, octa-BDE, and deca-BDE—made up 14%, 6%, and 80% of the 1999 worldwide production of PBDEs, respectively.[2] Usually found in electronics, such as TVs, and used in some furniture foams, fabrics, and kitchen appliances, the industry voluntarily ended production in the United States of the formulations of penta and octa in 2004 after high levels were found in breast milk. However, North America made up a large percentage of global demand for these PBDEs, 97.5 % for penta and 35.9% for octa, and there is still a large reservoir of products containing these PBDEs in the US.[3] The deca formulation is currently produced and used primarily in plastic electronics such as televisions and computer casings. It is also used in the upholstery covers of items such as furniture and car seats.[4,5]

PBDEs are part of a larger chemical class called polyhalogenated aromatic hydrocarbons (PHAHs) which include other highly toxic chemicals such as polychlorinated biphenyls (PCBs) and dioxins.[2] PHAHs are intrinsically hazardous because of their chemical make-up: (1) they are stable, meaning they persist in the environment and do not break down easily; (2) they are lipophilic, meaning they build up in fatty tissues of living organisms; and (3) they have toxic properties, including the potential to act as endocrine disruptors.[6] Their persistence and fat solubility allow them to both biomagnify and bioaccumulate, meaning they build up in the bodies of animals and humans as they move through the food chain. While not all PBDEs meet these criteria, a significant portion have been found to exhibit these characteristics. Although the main component in deca-BDE, BDE-209, has a relatively short half-life in people,[1] animal studies show that the liver breaks down BDE-209 into the more persistent and bioaccumulative forms of hepta-BDE, octa-BDE, and nona-BDE.[7,8] There are relatively few data available concerning the extent to which this occurs in humans.

How Are We Exposed?

There are many ways that humans are exposed to PBDEs, including ingestion of contaminated foods and incidental intake of PBDE-contaminated dust.[8] PBDEs are not permanently bound to the products in which they are used. Some PBDEs are semi-volatile and may be released from PBDE containing products. Other PBDEs are released from furniture, electronics and other products as they physically degrade. It is not entirely clear how deca is released from products, though some think it might be released from physical abrasion or deterioration of the product. Deca-BDE breaks down into more toxic by-products and may volatize when exposed to ultraviolet (UV) light.[9] Although penta-BDE and octa-BDE are no longer produced in the United States, exposure continues from old computers, furniture, fabrics, and other consumer sources that were made before the discontinuation.[10]

Occupational exposure may also occur for workers in electronics recycling, computer repair, and rubber manufacturing facilities.[11] PBDEs are found in sewage sludge used as agricultural fertilizers and may be a source of exposure for workers applying contaminated fertilizers.[11]

Ingestion of PBDEs in fatty foods such as meat and dairy products is thought to be a major route of exposure,[12] as PBDEs are fat-seeking and build up in the food web. Indoor exposures to dust and contaminated air are also thought to be major routes, as well as direct absorption through the skin from dust or products.[13] Relatively little is known about the routes of deca-BDE exposure. It is estimated that the highest intake of deca-BDE results from household dust.[13,14,15]

People may also be exposed through consumption of fish and other marine animals, because these organisms have been found to contain high levels of deca-BDE.[16] The high levels in marine environments may be due to atmospheric deposition (when contaminants from the air come down to the earth's surface through rain, snow, falling particles, and the absorption of gas) of deca-BDE as well as sewage discharge into the oceans.[16]

For infants, breast milk is a major source of PBDEs. The PBDEs that have accumulated in women may be passed to children through breastfeeding.[17] It is important to note that breastfeeding is still considered best for a baby's health.

PBDEs in Our Bodies

Due to their widespread use, persistence and bioaccumulative properties, PBDEs have been found in humans at high levels. A recent study by scientists from the U.S. Centers for Disease Control and Prevention (CDC) found PBDEs in nearly all 2,040 participants in a sample representative of the 2003–2004 U.S. population.[18] In this study, 3 congeners were found in over 90% of the participants: 97% had levels of BDE-47 (2,2′,4,4′-hexabromodiphenyl ether), 93% had levels of BDE-100 (2,2′,4,4′,6-pentabromodiphenyl ether), and 93% had levels of BDE-153 (2,2′,4,4′,5,5′-hexabromodiphenyl ether) above the limit of detection.[18] Another study showed that 5 percent of American women have levels of PBDEs that are close to the levels linked to reproductive problems in animals.[1] PBDEs have been found in mothers' breast milk and in the blood of mothers and their babies.[19,20] People of the Arctic may experience an even higher risk of exposure due to their traditional subsistence diet rich in fat from marine mammals.[21,] Concentrations of PBDEs have grown over the years in marine mammals due to atmospheric transport of chemicals into the north (transported long distances from areas of production and use via air and ocean currents) and bioaccumulation.[2]

What Does Exposure to PBDEs Mean for Our Health?

The presence of environmental chemicals in the human body does not necessarily imply that they are causing adverse health effects; however, environmental chemical exposures can and do affect human health. It is important to note that both the dosage and the timing of exposure have significant effects on any potential health outcome.

The following information is intended to inform the reader about the current state of knowledge on the health effects of PBDEs, including both human and animals studies.

Relatively little is known about the health effects of PBDEs. Overall there are few studies on health effects in the general population; however, health effects have been detected in rigorous animal based studies. Nevertheless, research has found associations between PBDEs and many adverse health effects, including:

Neurodevelopmental Effects

There are few published human studies on the health effects of environmental exposure to PDBEs. One study on Dutch children found that prenatal PBDE exposure affected childhood development. Exposed children had worse fine motor skills and

Reducing Your Exposure

You can minimize your exposure to PBDEs by taking the following steps:

PBDE-free Furniture

- Before purchasing furniture, find out which companies offer PBDE-free products. The following websites can help:
  - Clean Production Action: *www.cleanproduction.org/Flame.Alternatives.php*
  - Pollution in People: *www.pollutioninpeople.org/safer/products*
- Contact the company directly if you cannot figure out if the manufacturer uses PBDEs.
- Choose furniture made with less flammable fabrics like leather, wool and cotton.

PBDEs in Foam Padding

- Foam items purchased before 2005 can potentially contain PBDEs. Completely covering these items in fabric can limit potential exposure.
- Avoid reupholstering foam furniture.
- Be very careful when removing old carpet. Try to keep your work area separated from the rest of the house and thoroughly clean up the area.

Reduce Your Dust Exposure

- Wash your hands often to remove dust particles that your hands pick up throughout the day on everything you touch.
- Use a wet rag or cloth while dusting to avoid kicking up the dust in the air.
- If possible, use a vacuum fitted with a HEPA filter. These vacuums can trap smaller particles of dust and will be more likely to remove contaminants from your home.
- Vehicles have been exempt from recent PBDE laws and high levels have been found inside cars.[5] Removing dust with a wet cloth and keeping car seat cushions in good repair will help to reduce your exposure.

PBDE-free Electronics

- Many companies are beginning to make electronics with alternatives to PBDEs. Certain PBDE-free products are available from Canon, Dell, HP, Intel, Erickson, Apple, Acer, Nokia, Motorola, LG Electronics, and Sony.

Consider Eating Less Fat:

- Consider choosing leaner meat and poultry cuts.
- Consider removing fat that you see on meat and fish whenever possible.
- Choose cooking methods that remove excess fat such as broiling, grilling, and roasting.

attention, but better coordination, visual perception and behavior. The mothers in this study had serum PBDE concentrations in the low ppb range.[22] Another study of children prenatally exposed to PDBEs showed decreases in mental and physical development. PBDEs 47, 99 and 100 were correlated with lower developmental scores, while PBDE 153 was not. The most highly exposed children had significantly lower developmental scores than the less exposed children.[23]

A recent peer-reviewed scientific consensus statement by the Collaborative on Health and the Environment's Learning and Developmental Disabilities Initiative states, "Recent studies have left little doubt that PBDEs are developmental neurotoxicants in animals and lead to changes in motor activity and reduced performance on learning and memory tests."[24,25,26] Numerous studies on rodents suggest that neonatal exposure to PBDEs permanently affects learning and memory functions, impairs motor activity, and is linked to aberrations in spontaneous motor behavior and hyperactivity.[27,28,29,30,31,32,33]

Researchers have also found that some of the developmental effects of exposure become worse over time. In a study of PBDE exposure in mice, spontaneous behavior and habituation capability (both indicators of the ability of individuals and the species to survive in the wild) were permanently damaged by neonatal exposure to PBDEs and worsened with age.[27] Exposure to hexa-BDE has been linked to other developmental neurotoxic effects in mice, including changes in spontaneous behavior and impairments in learning and memory, which also appear to worsen with age.[34] A recent study in adult mice found consistent results which show that deca-BDE can be just as toxic to neurodevelopment as other PBDEs. In this study, exposure to deca-BDE was linked to hyperactivity and reduced or lack of habituation, also worsening with age.[35]

Thyroid Problems

Some PBDE congeners are structurally similar to thyroid hormone and have been shown to disrupt (by decreasing, increasing, or mimicking) the biological action of thyroid hormone.[36] In a study of newborn babies, high PBDE levels in cord blood were associated with decreased levels of thyroid hormones. PBDE concentrations ranged from below 10 ppb to several hundred ppb in the blood.[37] In a recent study of adult men who consume sport fish, researchers found that exposure to PBDEs at levels similar to those found in the general US population were associated with increased thyroid hormone T_4 (thyroxine).[38] Men in this study with the highest body burdens of PBDEs also showed increased thyroid antibodies, a risk factor for thyroid diseases such as chronic autoimmune thyroiditis and Graves' disease.[38] PBDE exposure has also been linked to hypothyroidism in adults. In an occupational exposure study at a deca-BDE and decabromobiphenyl manufacturing plant, 4 out of 35 exposed workers showed clinical hypothyroidism, while none of the 89 unexposed workers exhibited thyroid dysfunction.[39]

Corresponding animal studies have also shown that PBDE exposure is linked to decreased circulating concentrations of thyroid hormone[14,40] and decreased thyroid weight in adult rodent offspring.[12] Exposure to deca-BDE has been linked to thyroid hyperplasia in rodent studies. In a 2-year study by the National Toxicology Program, deca-BDE was found to increase the incidence of follicular cell hyperplasia (proliferation of follicular cells, which are responsible for making thyroid hormones) in the thyroid gland in male and female mice.[41]

Reproductive Effects

PBDEs can be both mildly estrogenic[42] and anti-androgenic compounds.[43,44] They have been correlated to cryptorchidism (undescended testes), in newborn boys exposed through breast milk with a median concentration of 4.16 ppb(lw) vs. 3.16 ppb(lw) for controls.[45] PBDEs have been shown to permanently impair sperm development in rodent studies.[14] Exposure to BDE-153, has been associated with a decrease in testicle size and the sperm concentration in humans.[46] Exposure to PBDEs in household dust at levels commonly encountered in the US has been linked with lower levels of androgens (male hormones) in adult men.[47] PBDEs have also been associated with delay of puberty in both male and female rodents and changes in sexual development and gender-specific sexual behavior.[48] Exposure to PBDEs has been linked to low birth weight, birth defects, reduced weight gain during pregnancy, changes in ovary cells and reduced sperm count.[49,50] The breakdown products of PBDEs may inhibit human aromatase, an enzyme important in the formation of androgens and estrogens (male and female hormones), and in skeletal development. However, these results were found at concentrations above current human exposure levels. [51]

Cancer

One study suggests that *in utero* exposure to PBDE concentrations is associated with an increased risk of testicular cancer in men, although the study has limitations and further studies would help to shed light on this association.[52] The Agency for Toxic Substances and Disease Registry (ATSDR) lists deca-BDE as a possible human carcinogen based on the development of liver tumors in rats.[8]

Regulation of PBDEs

There have been many steps forward in the regulation of PBDEs in recent years, but much more is needed to ensure the safety of public health. Internationally, Sweden was the first to initiate a phase-out of PBDEs in the late 1990s, followed by the European Union—first phasing out penta-BDE and octa-BDE in 2003 and then deca-BDE in 2006.[53] In 2008, Norway banned the use of deca-BDE in new consumer products.[54] Although no production occurred there, Canada prohibited the future manufacture of all PBDEs and placed resrictions on the import of certain PBDEs.[55]

The Stockholm Convention on Persistent Organic Pollutants, a United Nations treaty on persistent bioaccumulative substances, placed tetra-, penta-, hexa- and hepta-PBDEs under provisions

of the Convention for global phase-out after determining they posed a risk to environmental and human health.

In the United States, industry voluntarily ended production of penta-BDE and octa-BDE in 2004, but deca-BDE is still permitted for use in consumer products.[20] In December, 2009 two U.S. producers of deca-BDE and the largest U.S. importer announced commitments to end production, importation, and sales of deca-BDE for most uses by December, 2012.[56] Washington, Maine, Oregon, and Vermont are currently discontinuing the use and manufacture of deca-BDE,[57,58] and 12 states have banned penta- and octa-BDE, with other states across the nation aiming for similar legislation.

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Fact sheets on toxic chemicals for the *Mind, Disrupted* Biomonitoring Project provided by the Alaska Community Action on Toxics (www.akaction.net) and Commonweal (www.commonweal.org). For more information, please visit the *Mind, Disrupted* website at www.minddisrupted.org, or contact Pam Miller at pkmiller@akaction.net or Sharyle Patton at spatton@igc.org.