



# **PUBLIC HEALTH STATEMENT**

## **Polybrominated Diphenyl Ethers (PBDEs)**

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**Division of Toxicology and Human Health Sciences**

**March 2017**

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This Public Health Statement summarizes the Division of Toxicology and Human Health Science's findings on PBDEs, tells you about them, the effects of exposure, and describes what you can do to limit that exposure.

The U.S. Environmental Protection Agency (EPA) identifies the most serious hazardous waste sites in the nation. These sites make up the National Priorities List (NPL) and are sites targeted for long-term federal clean-up activities. U.S. EPA has not found PBDEs in any of the 1,832 current or former NPL sites. The total number of NPL sites evaluated for PBDEs is not known. But the possibility remains that as more sites are evaluated, sites with PBDEs may be identified. This information is important because these future sites may be sources of exposure, and exposure to PBDEs may be harmful.

If you are exposed to PBDEs, many factors determine whether you'll be harmed. These include how much you are exposed to (dose), how long you are exposed (duration), and how you are exposed (route of exposure). You must also consider the other chemicals you are exposed to and your age, sex, diet, family traits, lifestyle, and state of health.

### **WHAT ARE PBDEs?**

PBDEs are flame-retardant chemicals that were added to a variety of consumer products to make them difficult to burn. These substances are not single chemical compounds, but rather mixtures of several brominated substances. The entire family of PBDEs consists of 209 possible substances that are referred to as congeners.

There were three important commercial PBDE mixtures (i.e., penta-, octa-, and deca- bromodiphenyl ethers [BDEs]). DecaBDE's main use was for electronic enclosures, such as television cabinets. OctaBDE was largely used in plastics for business equipment. PentaBDE was principally used in foam for cushioning in upholstery. PentaBDE and octaBDE mixtures were voluntarily withdrawn from the U.S. marketplace by their manufacturers at the end of 2004 and decaBDE was not to be manufactured or imported into the United States after December 31, 2013. In 2003, the European Union (EU) passed a

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Directive to ban the marketing and use of penta- and octaBDE that took effect in 2004. In 2008, the use of decaBDE was restricted by a EU's Restriction of Hazardous Substances (RoHS) Directive.

#### **WHAT HAPPENS TO PBDEs WHEN THEY ENTER THE ENVIRONMENT?**

PBDEs can be released into the air, water, and soil at places where they are produced or used. Despite the phase out of penta-, octa-, and decaBDE, vast amounts of consumer products still contain PBDEs, and these products are intended to be used for several more years. Some of these products include older televisions, computers, and furniture containing polyurethane foam.

PBDEs have very low water solubility, and when these substances are released to water, they typically bind to sediment. PBDEs in consumer items put in landfills may leach through the soil into groundwater. This is not likely to be a problem, however, because these substances generally bind strongly to soil particles, and therefore, do not move easily through soil layers.

Soils and sediments are major sinks for PBDEs. Various food items, including fish, meat, and dairy products, have been shown to contain low concentrations of PBDEs.

#### **HOW MIGHT I BE EXPOSED TO PBDEs?**

Humans can be exposed to PBDEs in a wide variety of ways, including eating contaminated foods or contaminated dusts/soils, breathing in contaminated air, or having skin contact with contaminated soil/dust/commercial products.

The primary route of exposure to PBDEs for the general population of the United States is from ingestion of contaminated dust in indoor environments, including both personal residences and work-place environments. PBDEs have been detected in residential house dust, which you can breathe in or swallow in low concentrations. This can occur because PBDEs are physically mixed into consumer products from which they have the potential to escape into the environment when conditions are ideal. Ingestion of house dust (and to a lesser degree skin exposure to house dust) accounts for between 80 and 90% of total

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PBDE exposures of the general population. The remaining exposure to PBDEs in the United States is from food ingestion. You may be exposed to PBDEs through ingestion of contaminated foods, particularly those with high fat content, such as fatty fish. In breastfeeding infants, breast milk may be a major source of PBDE exposure because PBDEs can accumulate in breast milk. Due to the chemical nature of PBDEs, they have not been detected in water to any significant extent; therefore, drinking water is not expected to be a major route of exposure to PBDEs. While exposure to dust appears to be the major exposure pathway for the general population of North American residents, PBDE exposure through dietary routes appears to be more important for European communities.

PBDEs have been detected in air samples, indicating that people can also be exposed by inhalation. Consumer products such as computer and electronic equipment (e.g., televisions) treated with PBDEs can continue to release these substances to air over time.

PBDEs can enter soil from discarded products (e.g., in landfills). Biosolids may also contain PBDEs; therefore, they may be inadvertently released to soils from the use of biosolids that are applied to add nutrients to farmlands. If you touch soil containing PBDEs, a small amount of PBDEs may pass through your skin into the bloodstream; ingestion of soil can lead to higher PBDE exposure. This route may be especially important for children who display a lot of hand to mouth activity.

#### **HOW CAN PBDEs ENTER AND LEAVE MY BODY?**

PBDEs can enter your body from food, air, water, or soil. The ways that PBDEs might enter and leave your body depend on the chemical structures of the congener components. The higher-brominated PBDEs, particularly decaBDE, act somewhat differently in the body than do lower-brominated PBDEs. If you breathe air that contains PBDEs, or swallow food, water, soil, or dust contaminated with PBDEs, the lower-brominated congeners are more likely than decaBDE to enter your body through your lungs and stomach and pass into the bloodstream.

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Once PBDEs are in your body, the congeners might partially change into breakdown products called metabolites.

PBDEs and their metabolites can leave your body, mainly in the feces and a very small amount in urine. DecaBDE, with an apparent half-time of 15 days, tends to be eliminated from your body faster than lower-brominated PBDEs, with apparent half-times as high as 94 days. Lower brominated PBDEs can stay in your body for many years, stored mainly in body fat. DecaBDE also accumulates in body fat, but to a lesser degree. Both lower-brominated PBDEs and decaBDE can concentrate in breast milk fat and can enter the bodies of children through breastfeeding. Lower-brominated PBDEs and decaBDE also can enter the bodies of unborn babies through the placenta.

#### **HOW CAN PBDEs AFFECT MY HEALTH?**

Nothing definite is known about the health effects of PBDEs in people. The majority of information regarding toxicity of PBDEs and their breakdown products (metabolites) is from animal studies; however, several recent studies have evaluated associations between PBDE concentrations in human tissues (e.g., blood, breast milk) and various health effects. Due to differences in how decaBDE is absorbed and stored in your body, decaBDE is expected to be less toxic than lower-brominated PBDEs.

Rats and mice that ingested small amounts of lower-brominated PBDEs during early development had neurobehavioral changes and damage to their reproductive systems as adults. Altered neurobehavior was also observed in rats and mice that ingested decaBDE during early development, but at doses higher than observed for lower-brominated PBDEs. Adult rats and mice that ingested moderate amounts of lower-brominated PBDEs for short periods of time had mainly thyroid and liver effects. Additional findings from short-term animal studies suggest that some PBDEs might impair the immune system. Animals exposed to PBDEs by skin contact showed signs of skin irritation only if they had been scratched.

As with short-term exposure, rats and mice that ingested PBDEs for longer periods during early development also showed neurobehavioral changes; again, effects occurred at higher doses with

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decaBDE. Evidence from human studies is also suggestive of an association between PBDE exposure and altered neurodevelopment. Adult rats and mice that ingested small amounts of lower-brominated PBDEs over several weeks or months developed effects in the male reproductive system, thyroid, and liver. Adult animals that ingested small amounts of decaBDE over several weeks or months developed effects in the pancreas (diabetes), nervous system, immune system, and reproductive system. Evidence for PBDE-mediated effects from human studies in systems other than the developing nervous system is inconclusive or non-existent.

We don't know if PBDEs can cause cancer in people, although liver tumors developed in rats and mice that ate extremely large amounts of decaBDE throughout their lifetime. Lower-brominated PBDEs have not yet been tested for cancer in animals.

The International Agency for Research on Cancer (IARC) has classified PBDE as a Group 3 carcinogen (*not classifiable as to its carcinogenicity to humans*) based on inadequate evidence of carcinogenicity in humans and inadequate or limited evidence in experimental animals. The EPA assigns the cancer category Group D (*not classifiable as to human carcinogenicity*) to mono-, di-, tri-, tetra-, penta-, hexa-, octa-, and nonaBDEs and reports "*inadequate information*" to classify the specific congeners 2,2',4,4'-tetraBDE, 2,2',4,4',5-pentaBDE, and 2,2',4,4',5,5'-hexaBDE. However, EPA assigns a classification of "*suggestive evidence of carcinogenic potential*" for decaBDE. The Department of Health and Human Services has not classified PBDEs as carcinogens. The American Conference of Governmental Industrial Hygienists (ACGIH) has no data regarding cancer classifications for PBDEs.

### HOW CAN PBDEs AFFECT CHILDREN?

This section discusses potential health effects of PBDE exposure in humans from when they're first conceived to 18 years of age.

Studies indicate that infants and toddlers have higher exposures to PBDEs compared to older children or adults. The most likely way that infants might be exposed to PBDEs is from breast milk containing

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PBDEs, although fetuses in the womb could also be exposed. Toddlers and older children are exposed to PBDEs in generally the same way as are adults, mainly by ingesting contaminated household dust and food. However, soil/dust ingestion in small children (age 1–5 years) is much higher than in older children and adults. Because of their smaller weight, children’s intake of PBDEs per kilogram (or pound) of body weight may be greater than that of adults. Children who live near hazardous waste sites might accidentally eat some PBDEs by putting dirty hands or other soil/dirt covered objects in their mouths, or through eating without washing their hands. Some children also eat dirt on purpose. It is also possible that children could be exposed to PBDEs following transport of the chemical on clothing from the parent’s workplace to the home.

As indicated above, young children can be exposed to PBDEs both before birth and from breast milk. Both lower-brominated PBDEs and decaBDE have been found in breast milk, and they can be transferred to babies and young children. In general, however, any risks from exposures in mother’s milk are outweighed by the benefits of breastfeeding. You should consult your health care provider if you have any concerns about PBDEs and breastfeeding. Since the fetus and child are still developing, effects of PBDEs might be more significant if exposure occurs during the periods before and soon after birth.

Evidence suggests that fetuses and young children are more susceptible to PBDEs than adults. Subtle behavioral changes have been observed in animals exposed to PBDEs within the first 2 weeks of life, and results from human studies are suggestive of an effect of PBDEs on neurodevelopment in children, including impaired cognitive development (comprehension, memory), impaired motor skills, increased impulsivity, and decreased attention. One study reported that early PBDE exposure was a risk factor for the development of Attention Deficit Hyperactivity Disorder (ADHD); however, another study did not find a link between PBDE exposure and ADHD. One study investigating potential associations between early PBDE exposure and autism did not find a link between maternal PBDE serum levels and autistic behaviors in 4–5-year old children. One possible explanation for the observed behavioral effects might be related to changes in the thyroid, because development of the nervous system is dependent on thyroid hormones. Damage to developing reproductive organs and immune suppression have also been observed in animals exposed to PBDEs during development. It is unknown if these effects occur in human children.

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PBDEs have not caused birth defects in animals or impaired the ability for rats or mice to become pregnant or stay pregnant.

#### **HOW CAN FAMILIES REDUCE THE RISK OF EXPOSURE TO PBDEs?**

If your doctor finds that you have been exposed to significant amounts of PBDEs, ask whether your children might also be exposed. Your doctor might need to ask your state health department to investigate.

Ingestion and dermal contact with indoor dust containing PBDEs is the major exposure pathway to residents of the United States. Dust containing PBDEs can collect on your hands and be ingested through hand-to-mouth activities; regular hand washing may decrease PBDE exposure from this route.

Additionally, PBDE exposure may be decreased by regular vacuuming and cleaning of air ducts and filters to reduce indoor dust levels.

Since many older consumer products such as televisions, computers, and furniture containing polyurethane foam contain PBDEs, replacing older products with newer ones that do not contain these substances may decrease residential PBDE exposure.

#### **ARE THERE MEDICAL TESTS TO DETERMINE WHETHER I HAVE BEEN EXPOSED TO PBDEs?**

PBDEs and their breakdown products (metabolites) can be measured in human blood, hair, and breast milk. However, the detection of PBDEs or their metabolites cannot predict the kind of health effects that might develop from that exposure. Because PBDEs and their metabolites either leave the body or are distributed to body fat fairly rapidly, the tests need to be conducted within days if an acute, high-level exposure is suspected.

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#### **WHAT RECOMMENDATIONS HAS THE FEDERAL GOVERNMENT MADE TO PROTECT HUMAN HEALTH?**

The federal government develops regulations and recommendations to protect public health. Regulations can be enforced by law. Federal agencies that develop regulations for toxic substances include the Environmental Protection Agency (EPA), the Occupational Safety and Health Administration (OSHA), and the Food and Drug Administration (FDA). Recommendations provide valuable guidelines to protect public health but cannot be enforced by law. Federal organizations that develop recommendations for toxic substances include the Agency for Toxic Substances and Disease Registry (ATSDR) and the National Institute for Occupational Safety and Health (NIOSH).

Regulations and recommendations can be expressed as “not-to-exceed” levels; that is, levels of a toxic substance in air, water, soil, or food that do not exceed a critical value usually based on levels that affect animals; levels are then adjusted to help protect humans. Sometimes these not-to-exceed levels differ among federal organizations. Different organizations use different exposure times (an 8-hour workday or a 24-hour day), different animal studies, or emphasize some factors over others, depending on their mission.

Recommendations and regulations are also updated periodically as more information becomes available. For the most current information, check with the federal agency or organization that issued the regulation or recommendation.

EPA requires that companies that transport, store, or dispose of monobrominated diphenyl ether (monoBDE) (or diphenyl ether with one bromine attached to the structure, represented by Chemical Abstracts Service [CAS] Registry Number 101-55-3; Resource Conservation and Recovery Act [RCRA] waste number U030) follow the rules and regulations of the federal hazardous waste management program because it has been listed (U-list) as a hazardous waste due to toxicity concerns. EPA also limits the amount of monoBDE put into publicly owned waste water treatment plants. To minimize exposure of

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people to monoBDE, EPA requires that industry tell the National Response Center each time 100 pounds or more of monoBDE have been released to the environment.

OSHA has not set permissible exposure limits (PELs) to protect workers against adverse health effects resulting from exposure to PBDEs. NIOSH has not recommended guidelines for worker exposure limits.

#### **WHERE CAN I GET MORE INFORMATION?**

If you have any questions or concerns, please contact your community or state health or environmental quality department, or contact ATSDR at the address and phone number below. ATSDR can also provide publicly available information regarding medical specialists with expertise and experience recognizing, evaluating, treating, and managing patients exposed to hazardous substances.

- Call the toll-free information and technical assistance number at 1-800-CDCINFO (1-800-232-4636) or
- Write to:  
Agency for Toxic Substances and Disease Registry  
Division of Toxicology and Human Health Sciences  
1600 Clifton Road NE  
Mailstop F-57  
Atlanta, GA 30329-4027

Toxicological profiles and other information are available on ATSDR's web site:  
<http://www.atsdr.cdc.gov>.

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