Polybrominated Diphenyl Ethers (PBDEs)

Polybrominated diphenyl ethers (PBDEs) are a group of brominated flame retardant chemicals that have been incorporated into a variety of manufactured products, including foam cushioning used in furniture and plastics used in televisions and computers. Flame retardants are intended to slow the rate of ignition and fire growth, allowing more time for people to escape from a fire or extinguish it.

All PBDEs have a common structure of a diphenyl ether molecule, which may have from 1–10 bromine atoms attached; each particular PBDE variant is referred to as a congener. In theory, there could be as many as 209 PBDE congeners, but a much smaller number of congeners are commonly found in the commercial PBDE mixtures and in measurements of PBDEs in humans and the environment.

Three commercial PBDE mixtures have been used in manufactured products since the 1960s and 1970s, when these chemicals came into use,[1] with each mixture made up of congeners with varying degrees of bromination. The commercial pentabromodiphenyl ether (pentaBDE) and octabromodiphenyl ether (octaBDE) mixtures have not been manufactured or imported in the United States since 2004. The pentaBDE mixture, made up primarily of four- and five-bromine congeners, was used almost entirely in flexible polyurethane foam in furniture, mattresses, carpet padding, and automobile seats; and the octaBDE mixture, made up primarily of seven- and eight-bromine congeners, was used in acrylonitrile-butadiene-styrene (ABS) plastic for certain electric and electronic devices.

A third product, the commercial decabromodiphenyl ether (decaBDE) mixture, is still manufactured and used in the United States. The decaBDE mixture, made up almost entirely of the 10-bromine congener, has been used primarily in high-impact polystyrene (HIPS) plastic that was frequently used to make the back part of television sets, and in other electronic devices. However, there are indications that the use of decaBDE in electronic devices has declined in recent years, particularly since restrictions on the use of decaBDE in electronics were implemented in Europe beginning in 2008. DecaBDE is also used as a flame retardant on certain types of textiles; in electrical products, including uses in vehicles and airplanes; and in certain building materials. The major U.S. importers and manufacturers of decaBDE have announced that this mixture will be phased out by the end of 2013.[2] As use of PBDEs is reduced, they are being replaced by other flame-retardant chemicals or by materials that are inherently resistant to fire. EPA has conducted an assessment of alternatives to commercial pentaBDE,[3] and is conducting a similar assessment of alternatives to commercial decaBDE.[4]

PBDEs can be released into the environment at various points in their lifecycle, from their production and application to consumer products to their release from discarded products in landfills. Since PBDEs are not chemically bound to the products in which they are used, they can easily migrate into the surrounding air, dust, soil, and water. Although production and use of the commercial PBDE mixtures has been phased out (pentaBDE and octaBDE) or will soon be

phased out (decaBDE), it is likely that PBDE congeners will continue to be present in the environment for many years. This is because products previously manufactured with PBDEs (e.g., sofas) will stay in use for many years. PBDEs will continue to be released from these products while they are in use, and these releases may continue when the products are disposed of or recycled. PBDEs are persistent in the environment, so even if there were no further releases they would continue to be detected for many years.

Exposure studies, focusing on selected PBDE congeners that were most predominant in the commercial mixtures or that are frequently measured in environmental samples, have concluded that the presence of PBDEs in house dust and in foods are both important contributors to PBDE exposures for people of all ages, and that exposures from house dust are generally greater than those from food.[1,5-11]

Studies conducted in multiple locations have consistently found PBDEs in U.S. house dust at levels greater than those found in other countries.[12-14] This is likely due to greater use of PBDE-containing products in homes in the United States than in other countries. Within the United States, the highest levels of three frequently measured PBDE congeners in dust have been found in California. The three congeners were all components of commercial pentaBDE mixtures, and the authors of these studies observed that the elevated levels may be due to California requirements for flame resistance in residential furniture that are not applicable in other states.[13,15] A study conducted in adults found a stronger association between direct contact with PBDE-containing materials and PBDE blood levels than between PBDE-contaminated house dust and PBDE blood levels.[16]

A second pathway of exposure to PBDEs is through diet. PBDEs are generally persistent chemicals that accumulate in fat tissue, so they are commonly found in foods derived from animals.[17-19] Information about how PBDEs enter the food web is limited, but release from manufacture of the PBDEs or of PBDE-containing products; release of PBDEs from products while they are in use; and release from products when disposed of or recycled are all likely contributors to PBDEs in the environment. PBDEs have been measured in a variety of supermarket foods, with the highest levels found in fish and other foods of animal origin.[18,20] A California study found associations between pork and poultry consumption and the levels of PBDEs measured in blood of children ages 2 to 5 years.[13]

Levels of PBDEs measured in blood are substantially greater in North America than in Europe and Asia, a difference that appears to be due to the higher levels of PBDEs in house dust in North America.[7,12,21,22] Studies comparing archived and current samples of blood and pooled serum from various locations in the United States have shown marked increases in PBDE levels since the late 1970s.[23,24]

Early-life exposures to PBDEs may be elevated in a number of ways. A child's exposure to PBDEs begins in the prenatal period, as PBDEs have been measured in cord blood, fetal blood, and placental tissue[25-27] and continues in early infancy due to the presence of PBDEs in breast milk.[11,22,28-31] Levels of PBDEs in breast milk are higher in North America than elsewhere,[12] and

estimated intakes of PBDEs are substantially greater for a breastfeeding infant than exposures that occur during other life stages.[6,22]

Exposures are also elevated for young children up to age 7 years. While few studies have measured concentrations of PBDEs in young children, one large study conducted in Australia found that levels of PBDEs in blood are greatest for children ages 2 to 5 years, compared with older children and adults.[32] A study of 20 young children (ages 1.5 to 4 years) in various locations throughout the United States found that their PBDE blood levels were consistently higher than those of their mothers.[33] A study of California children ages 2 to 5 years found PBDE blood levels generally greater than those measured in California residents ages 12 to 60 years.[13,15] A study of 7-year-old Mexican-American children living in California reported PBDE blood levels that were three times the levels found in their mothers during pregnancy.[34]

The elevated exposures observed for young children are likely due to increased exposure to house dust, based on several studies that have estimated exposures based on measured levels of PBDEs in house dust, air, and food.[6,7,9,35] Infants and small children may have the highest exposure to PBDEs in house dust due to their frequent and extensive contact with floors, carpets, and other surfaces where dust gathers, as well as their frequent hand-to-mouth activity.[36] However, children of all ages (as well as adults) are likely to be exposed to dust contaminants through hand-to-mouth activity and other ingestion pathways, such as the settling of dust onto food and food preparation surfaces in the kitchen, as well as inhalation and absorption of PBDEs through the skin.[1,9,22]

Concerns about the health effects of PBDEs are based largely on laboratory animal studies, along with findings of the limited number of human epidemiological studies that have been conducted to date. A primary concern from the animal studies is for effects on the developing brain and nervous system, including effects on learning, memory, and behavior.[37-39] A study of children in New York City found significant associations between children's prenatal exposure to PBDEs and performance on IQ tests at ages up through 6 years.[40] A second epidemiological study conducted in the Netherlands found that prenatal exposure to PBDEs was associated with reduced scores on some tests of neurological development and improved scores on other tests at ages 5 to 6 years.[41]

PBDEs are suspected endocrine disruptors.[37] Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.[42,43] Given the importance of hormones in human physiology, there is concern in the scientific community over the potential for endocrine disruptors to adversely affect children's health, particularly in reproduction, early and adolescent development, and behavior.

Animal and human studies indicate that PBDEs may alter circulating levels of thyroid hormones.[37,44-47] Moderate deficits in maternal thyroid hormone levels during early pregnancy have been linked to reduced childhood IQ scores and other neurodevelopmental effects, as well as unsuccessful or complicated pregnancies.[48] Animal studies have found that PBDE exposure at critical stages of fetal development reduced levels of male hormones or caused other changes

relevant to male reproductive development.[37,47,49-51] An epidemiological study of boys born in Denmark and Finland found that increased levels of PBDEs in breast milk were associated with an increased risk of cryptorchidism (undescended testes),[52] an effect that may be related to hormone disruption during critical stages of development.[53,54] Also, a study of Mexican immigrant women in California found effects on fertility (increased time to pregnancy) with increasing PBDE levels; this finding may be related to hormonal activity of PBDEs.[55]

The following indicator presents the best nationally representative data on PBDE levels in women of child-bearing age. Indicator B8 presents median blood serum levels of PBDEs for women ages 16 to 49 years. Although data are available only for a single two-year period at this time, the data provide a baseline that will be updated with PBDE measurements over time from subsequent survey cycles.

Indicator B8: PBDEs in women ages 16 to 49 years: Median concentrations in blood serum, by race/ethnicity and family income, 2003–2004

About the Indicator: Indicator B8 presents concentrations of PBDEs in blood serum of U.S. women ages 16 to 49 years. The data are from a national survey that collects blood specimens from a representative sample of the population every two years, and then measures the concentration of PBDEs in the blood serum. The indicator presents comparisons of PBDEs in blood serum for women of different race/ethnicities, and for women of different income levels. The focus on women of child-bearing age is based on concern for potential effects in children born to women who have been exposed to PBDEs.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for PBDEs. NHANES is designed to assess the health and nutritional status of the civilian noninstitutionalized U.S. population and is conducted by the National Center for Health Statistics, part of the Centers for Disease Control and Prevention (CDC). Interviews and physical examinations are conducted with approximately 10,000 people in each two-year survey cycle. CDC's National Center for Environmental Health measures concentrations of environmental chemicals in blood and urine samples collected from NHANES participants. Summaries of the measured values for more than 200 chemicals are provided in the *Fourth National Report on Human Exposure to Environmental Chemicals*.[56]

PBDE Congeners

Indicator B8 presents blood serum levels of PBDEs in women of child-bearing age. There are 209 possible PBDEs, referred to as "congeners," which are defined by the number of bromine atoms (from 1 to 10) and their position in the chemical structure. Each congener is assigned a specific brominated diphenyl ether (BDE) number, such as BDE-47 (a tetrabromodiphenyl ether congener – four bromine atoms). Most of these congeners have not been detected in the manufactured PBDE mixtures and have not been measured in environmental or human samples.

PBDE concentrations are measured in blood serum. PBDEs are lipophilic, meaning that they tend to accumulate in fat. Serum PBDEs concentrations are measured and expressed on a lipid-adjusted basis, as these values better represent the amount of PBDEs stored in the body compared with unadjusted values.[56] The indicator uses lipid-adjusted concentrations, meaning that the concentration of PBDEs in serum is divided by the concentration of lipid in serum. The resulting units are nanograms of PBDE per gram of lipid (ng/g lipid) in serum.[i]

[i] Serum levels of PBDEs can also be reported without lipid adjustment. Both the lipid-adjusted values and the unadjusted "whole weight" values are reported in CDC's *Fourth National Report on Human Exposure to Environmental Chemicals*.

Concentrations of PBDEs in blood serum have been measured in a representative subset of NHANES participants ages 12 years and older in the 2003–2004 survey cycle. NHANES sampled for 10 PBDE congeners in 2003–2004, including those most frequently measured in environmental and human samples. These include BDEs 17, 28, 47, 66, 85, 99, 100, 153, 154, and 183. Most of these 10 congeners were components of the pentaBDE mixture that was used in polyurethane foam for furniture, mattresses, and automotive seating. Some of the congeners measured in NHANES were components of the octaBDE mixture, used in plastics for some household electric devices. The primary congener comprising the decaBDE formulation, BDE-209, was not measured in NHANES in 2003–2004.

Indicator B8 was calculated by summing together the measured values of the 10 congeners for each woman 16 to 49 years sampled in NHANES; this approach is commonly used in studies assessing levels of PBDEs in human blood samples and environmental samples.[1] Data are insufficient at this time to assess and quantify differences in toxicity of the measured PBDE congeners, or to inform approaches other than an unweighted summation of the 10 congeners.

If a congener was not detected in a particular blood sample, a default value below the detection limit was assigned for purposes of calculating the summed total for the sampled individual.[ii] This assumption has a small impact on the reported blood levels of PBDEs, because almost all women sampled had values well above the detection limit for at least some congeners. BDEs 47, 100 and 153 were each detected in more than 90% of women ages 16 to 49 years.

In 2003–2004, a sum of the 10 measured PBDEs is available from NHANES for 2,040 individuals ages 12 years and older, including 540 women ages 16 to 49 years. One or more PBDE congeners were detected in 99% of the individuals sampled in NHANES 2003–2004, and in 99% of women ages 16 to 49 years.[iii] The median sum of the ten PBDE congeners among NHANES participants in 2003–2004 was 38 ng/g lipid and the 95th percentile sum was 307 ng/g lipid.

Birth Rate Adjustment

Indicator B8 uses measurements of PBDEs in blood of women ages 16 to 49 years to represent the distribution of PBDE exposures to women who are pregnant or may become pregnant. However, women of different ages have a different likelihood of giving birth. For example, in 2003–2004, women aged 27 years had a 12% annual probability of giving birth, and women aged 37 years had a 4% annual probability of giving birth.[57] A birth rate-adjusted distribution of women's PBDE levels is used in calculating this indicator,[iv] meaning that the data are weighted using the age-specific probability of a woman giving birth.[58]

[ii] The default value used for non-detect samples is equal to the limit of detection divided by the square root of 2.
[iii] The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.
[iv] There may be multiple ways to implement an adjustment to the data that accounts for birth rates by age. The National Center for Health Statistics has not fully evaluated the method used in ACE, or any other method intended to accomplish the same purpose, and has not used any such method in its publications. NCHS and EPA are working together to further evaluate the birth rate adjustment method used in ACE and alternative methods.

Data Presented in the Indicator

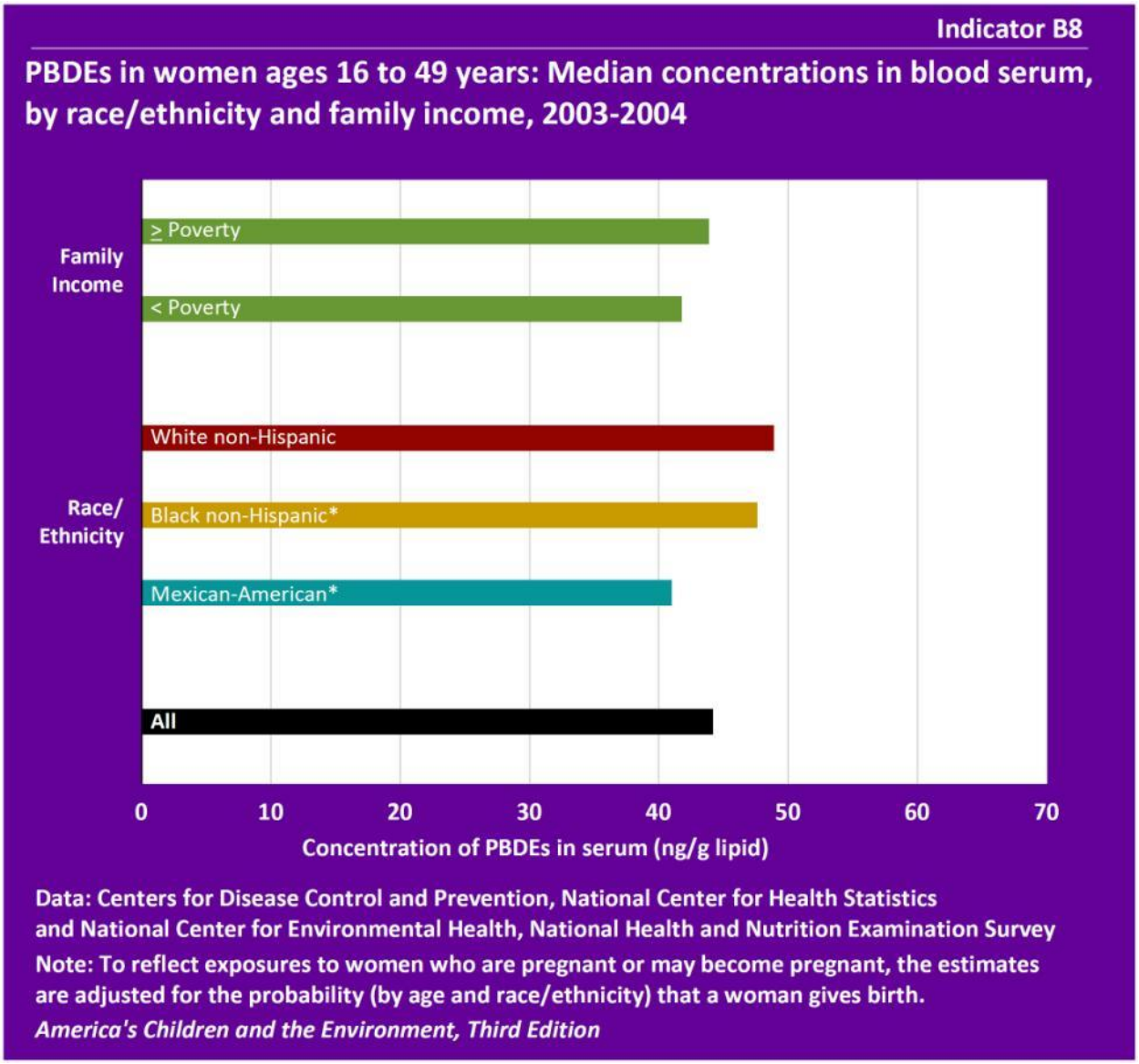
Indicator B8 presents median concentrations of PBDEs in blood serum for women ages 16 to 49 years of different races/ethnicities and levels of family income, using NHANES data from 2003–2004.[v]

Three race/ethnicity groups are presented in Indicator B8: White non-Hispanic, Black non-Hispanic, and Mexican-American. The data are also tabulated across three income categories: all incomes, below the poverty level, and greater than or equal to the poverty level.

Additional information on how median blood serum levels of PBDEs vary by race/ethnicity and family income for children ages 12 to 17 years is presented in a supplemental data table for this indicator.

Please see the Introduction to the Biomonitoring section for an explanation of the term "median" and information on the statistical significance testing applied to this indicator.

[v] Unlike other biomonitoring indicators in this report, 95th percentile PBDE levels are not provided in a supplementary table. This is because most 95th percentile PBDE values do not meet ACE statistical reliability criteria. There is more uncertainty in 95th percentile estimates for PBDEs than for other chemicals because data are only available for two years (2003–2004) at this time. Similarly, separate values are not provided considering both race/ethnicity and income simultaneously, nor are values provided for the "All Other Races/Ethnicities" category, because (with data from only one NHANES cycle available at this time) such estimates lack statistical reliability.



*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

Data characterization

- Data for this indicator are obtained from an ongoing continuous survey conducted by the National Center for Health Statistics.
- Survey data are representative of the U.S. civilian noninstitutionalized population.
- PBDEs are measured in blood samples obtained from individual survey participants.

- The median concentration of PBDEs in blood serum of women ages 16 to 49 years was 44 ng/g lipid in 2003–2004.

- White non-Hispanic women and Black non-Hispanic women had the highest median PBDE levels at 49 and 48 ng/g lipid, respectively.
  - The differences by race/ethnicity were generally not statistically significant without accounting for differences by age and income across race/ethnicity groups. After accounting for differences by age and income across race/ethnicity groups, PBDE levels in White non-Hispanic women were statistically significantly greater than levels in Black non-Hispanic women. Also after adjustment, PBDE levels in Black non-Hispanic women were statistically significantly greater than levels in Mexican-American women.
- Among women of child-bearing age, there was little difference in median PBDE concentrations in blood serum between income groups.
- The median concentration of PBDEs in children ages 12 to 17 years overall was 53 ng/g lipid. The median concentration of PBDEs for children with family incomes below the poverty level was 63 ng/g lipid, and 50 ng/g lipid for children at or above poverty level. (See Table B8a.)
  - The difference in median PBDE concentration between the income groups was not statistically significant.
- Among children ages 12 to 17, White non-Hispanic children and Black non-Hispanic children had the lowest median PBDE levels at 48 and 50 ng/g lipid. Mexican-American children had median PBDE levels of 63 ng/g lipid. (See Table B8a.)
  - These differences by race/ethnicity were not statistically significant.

Polybrominated Diphenyl Ethers (PBDEs)

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Table B8. PBDEs in women ages 16 to 49 years: Median concentrations in blood serum, by race/ethnicity and family income, 2003-2004

Race / Ethnicity	Median concentration of PBDEs in serum (ng/g lipid)
All Races/Ethnicities *(n=540)*	44.2
White non-Hispanic *(n=233)*	48.9
Black non-Hispanic *(n=132)*	47.6*
Mexican-American *(n=131)*	41.0*
All Other Races/Ethnicities† *(n=44)*	NA**
Income	
All Incomes‡ *(n=540)*	44.2
< Poverty Level *(n=156)*	41.8
≥ Poverty Level *(n=352)*	43.9

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTES:

- Values below the limit of detection are assumed equal to the limit of detection divided by the square root of 2.
- To reflect exposures to women who are pregnant or may become pregnant, the estimates are adjusted for the probability (by age and race/ethnicity) that a woman gives birth. The intent of this adjustment is to approximate the distribution of exposure to pregnant women. Results will therefore differ from a characterization of exposure to adult women without consideration of birth rates.

† The "All Other Races/Ethnicities" category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.

Table B8a. PBDEs in children ages 12 to 17 years: Median concentrations in blood serum, by race/ethnicity and family income, 2003-2004

Race / Ethnicity	Median concentration of PBDEs in serum (ng/g lipid)
All Races/Ethnicities *(n=464)*	52.9
White non-Hispanic *(n=114)*	47.5*
Black non-Hispanic *(n=176)*	50.4*
Mexican-American *(n=145)*	62.9*
All Other Races/Ethnicities† *(n=29)*	NA**
Income	
All Incomes‡ *(n=464)*	52.9
< Poverty Level *(n=147)*	62.6
≥ Poverty Level *(n=304)*	49.8

DATA: Centers for Disease Control and Prevention, National Center for Health Statistics and National Center for Environmental Health, National Health and Nutrition Examination Survey

NOTE: Values below the limit of detection are assumed equal to the limit of detection divided by the square root of 2.

† The "All Other Races/Ethnicities" category includes all other races or ethnicities not specified, together with those individuals who report more than one race.

‡ Includes sampled individuals for whom income information is missing.

*The estimate should be interpreted with caution because the standard error of the estimate is relatively large: the relative standard error, RSE, is at least 30% but is less than 40% (RSE = standard error divided by the estimate), or the RSE may be underestimated.

** Not available. The estimate is not reported because it has large uncertainty: the relative standard error, RSE, is 40% or greater (RSE = standard error divided by the estimate), or the RSE cannot be reliably estimated.