Phthalates

Phthalates are a class of manufactured chemicals commonly used to increase the flexibility of plastics in a wide array of consumer products. More than 470 million pounds of phthalates are produced or imported in the United States each year.¹

By far the most common use of phthalates is in the production of polyvinyl chloride (PVC) products.² PVC is the second most commonly used plastic in the world, and is present in pipes and tubing, construction materials, packaging, electrical wiring, and thousands of consumer goods.³,⁴ Phthalates are or have been used in wall coverings, tablecloths, floor tiles, furniture upholstery, carpet backings, shower curtains, garden hoses, rainwear, pesticides, some toys, shoes, automobile upholstery, food packaging, medical tubing, and blood storage bags.⁵⁻⁸ Phthalates are not strongly bound in these products and can therefore leach out.⁴⁻¹⁰ Some phthalates are also present in cosmetics, nail polish, hair products, skin care products, and some medications.⁴,⁶,⁷,¹¹,¹²

The Consumer Product Safety Improvement Act of 2008 (CPSIA) banned the use of three phthalates in toys and child care articles at concentrations greater than 0.1 percent: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), and butyl benzyl phthalate (BBzP). CPSIA also restricts the use of di-isononyl phthalate (DINP), di-isodecyl phthalate (DIDP), and di- n-octyl phthalate (DnOP) in toys that can be mouthed and child care articles. The Consumer Product Safety Commission has also appointed a Chronic Hazard Advisory Panel to examine the cumulative health risks of phthalates and phthalate substitutes, and to recommend whether to continue the ban of DINP, DIDP, and DnOP and whether any other phthalates or phthalate substitutes should be banned.¹ As use of phthalates is reduced, they are being replaced by other chemicals, such as di-isononylcyclohexane-1,2-dicarboxylate (DINCH) and di(2-ethylhexyl) terephthalate (DEHT), that also increase the flexibility of plastics.¹³⁻¹⁵ EPA is planning to conduct an assessment of alternatives to several phthalates.¹

For most phthalates, the major route of exposure is food ingestion.⁴,¹⁶⁻¹⁹ However, personal care product use and inhalation are major routes of exposure for certain phthalates.⁴⁻⁸,¹¹,²⁰ Some phthalates have been found at higher levels in fatty foods such as dairy products, fish, seafood, and oils.⁸ Phthalates in a mother’s body can enter her breast milk. Ingestion of that breast milk and infant formula containing phthalates may also contribute to infant phthalate exposure.¹¹ The phthalates that may be present in dust can be ingested by infants and children through hand-to-mouth activities.¹⁰,²² Finally, infants and small children can be exposed to phthalates by sucking on toys and objects made with phthalate-containing plastics.¹⁰

Other minor routes of phthalate exposure include inhalation, drinking contaminated water, and absorption through the skin.¹⁶,¹⁷ Phthalates can be released in small amounts to the air people breathe inside homes or schools from the many consumer products that contain them.⁷,²⁰ People living near phthalate-producing factories or hazardous waste sites may be exposed to phthalates released into the air or ground water where they live.⁵,⁷,⁸ Individuals may be exposed
to phthalates during the use of many personal care products containing phthalates, such as hair products, cosmetics, and lotions.\textsuperscript{11,23,24} Phthalates in these products may be absorbed through contact with the skin or may be inhaled if some of the product is present in the air.\textsuperscript{5} In addition, certain medical devices, such as intravenous tubing or flexible bags containing blood, medications, or nutritional products, contain phthalates. These can be a source of phthalate exposure to children and women of child-bearing age when the tubing or bags are used to administer medications, nutritional products, or blood to the individual. This can be a very significant route of exposure, especially for premature infants in intensive care units.\textsuperscript{25-27}

Phthalate exposures, assessed from urinary concentrations of phthalate metabolites (i.e., breakdown products), appear to be higher for children compared with adolescents and adults. Studies of phthalate metabolites in children’s urine are limited, but the few that have been published have found children’s urinary phthalate metabolite levels to be higher than levels in adults and to decrease with age (i.e., younger children had more phthalate metabolites in their urine than older children did).\textsuperscript{28-30} The exception is monoethyl phthalate (MEP), a metabolite of diethyl phthalate, which has been found to be present in higher levels in adult urine compared with children’s urine.\textsuperscript{28} Levels of MEP are most likely associated with use of consumer products that contain diethyl phthalate, such as detergents, soaps, cosmetics, shampoos, and perfumes.\textsuperscript{5,28}

Some phthalates are suspected endocrine disruptors.\textsuperscript{31-35} Endocrine disruptors act by interfering with the biosynthesis, secretion, action, or metabolism of naturally occurring hormones.\textsuperscript{32,36} 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, development, and behavior. Male laboratory animals exposed to high doses of some phthalates have been known to display elements of “phthalate syndrome,” which includes infertility, decreased sperm count, cryptorchidism (undescended testes), hypospadias (malformation of the penis in which the urethra does not open at the tip of the organ), and other reproductive tract malformations.\textsuperscript{4} A number of animal studies have reported associations between exposure to certain phthalates and changes in male hormone production, altered sexual differentiation, and changes to reproductive organs, including hypospadias.\textsuperscript{37-45} These findings in animal studies, although typically occurring at exposure levels much higher than what the general population may be exposed to, suggest a potential concern for health effects in children as well. The National Research Council has concluded that prenatal exposure to certain phthalates produces reproductive tract abnormalities in male rats, and also concluded that the same effects could plausibly occur in humans.\textsuperscript{4}

There are only a limited number of human studies looking at the relationship between phthalate exposure and hormonal and reproductive health changes. In one study, prenatal exposure to some phthalates at typical U.S. population levels was associated with changes in physical measures of the distance between the anus and the genitals (anogenital distance) in male infants.\textsuperscript{46,47} A shorter anogenital distance has been associated with decreased fertility in animal experiments\textsuperscript{48,49} and a recent human study reported that a shorter anogenital distance in men was associated with decreased semen quality and low sperm count.\textsuperscript{50} Another study reported an association between increased concentrations of phthalate metabolites in breast
milk and altered reproductive hormone levels in newborn boys. The same study did not find an association between breast milk phthalate metabolite concentrations and cryptorchidism.\textsuperscript{51}

Exposure to some phthalates has been associated with neurodevelopmental problems in children in some studies. Two studies of a group of New York City children ages 4 to 9 years reported associations between prenatal exposure to certain phthalates and behavioral deficits, including effects on attention, conduct, and social behaviors.\textsuperscript{52,53} Studies conducted in South Korea of children ages 8 to 11 years reported that children with higher levels of certain phthalate metabolites in their urine were more inattentive and hyperactive, displayed more symptoms of attention-deficit/hyperactivity disorder (ADHD), and had lower IQ compared with those who had lower levels.\textsuperscript{54,55} The exposure levels in these studies are comparable to typical exposures in the U.S. population.

A handful of studies have reported associations between prenatal exposure to some phthalates and preterm birth, shorter gestational length, and low birth weight;\textsuperscript{56-59} however, one study reported phthalate exposure to be associated with longer gestational length and increased risk of delivery by Cesarean section.\textsuperscript{60}

Finally, some researchers have hypothesized that phthalate exposure in homes may contribute to asthma and allergies in children. Two research groups have conducted studies, primarily in Europe, and reported associations between surrogates for potential phthalate exposure in the home and risk of asthma and allergies in children.\textsuperscript{61} Examples of the exposure indicators and outcomes considered in these studies include an association between some phthalates in surface dust and increased risk of runny nose, eczema, and asthma,\textsuperscript{62} and increased risk of bronchial obstruction associated with the presence of PVC in the home.\textsuperscript{63}

The two indicators that follow use the best nationally representative data currently available on urinary phthalate metabolite levels over time for women of child-bearing age and children. The indicators focus on three important phthalates: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (DBP), and butyl benzyl phthalate (BBzP). These three phthalates were chosen because their metabolites are commonly detected in humans and their potential connection to adverse children’s health outcomes is supported by the scientific literature summarized in the following paragraphs.

DEHP is currently the only phthalate plasticizer used in PVC medical devices such as blood bags and plastic tubing. DEHP is also currently used in flooring, wallpaper, and raincoats and has been used in toys, auto upholstery, and food packaging.\textsuperscript{64} DBP is used primarily in latex adhesives, cellulose plastics, dyes, and cosmetics and other personal care products.\textsuperscript{65} The largest use of BBzP is in the production of PVC flooring materials, but it is also used in the manufacture of automotive materials, artificial leather, and food conveyor belts.\textsuperscript{66,67}

In 2006, the National Toxicology Program (NTP) concluded that there is “concern” for effects on reproductive tract development in male infants less than one year old exposed to DEHP. In addition, the NTP also concluded that there is “some concern” (the midpoint on a five-level
scale ranging from “negligible” to “serious” concern)\textsuperscript{1} for effects on reproductive tract development in male children older than one year old exposed to DEHP, and also that there is “some concern” for effects of prenatal DEHP exposure on reproductive tract development in males. Concern was greater for males exposed to high levels of DEHP in the womb or early in life. These conclusions were based primarily on findings from animal studies, as human data are limited and were determined to be insufficient for evaluating the reproductive effects of DEHP.\textsuperscript{64} Some studies have also reported associations of DEHP exposure with increased risk of asthma and bronchial obstruction, increased risk of ADHD symptoms, and altered pregnancy durations.\textsuperscript{55,56,58,60,62,63} Human health studies have reported associations between exposures to DBP and altered reproductive hormone levels in newborn boys, and shifts in thyroid hormone levels in pregnant women.\textsuperscript{51,68} Signs of feminization in young boys (as measured by reduced anogenital distance), altered hormone levels in newborn boys, and increased risk of rhinitis and eczema are health effects that have been associated with BBzP exposure in some studies.\textsuperscript{46,47,51,62} The exposure levels in these studies are comparable to typical exposures in the U.S. population. It is important to note that while the following indicators present data on individual phthalate metabolites, evidence suggests that exposures to multiple phthalates may contribute to common adverse outcomes. The National Research Council has concluded that multiple phthalates may act cumulatively to adversely impact male reproductive development.\textsuperscript{4}

Indicator B9 presents median concentrations of metabolites of DEHP, DBP, and BBzP in urine for women ages 16 to 49 years. Indicator B10 presents median metabolite levels of the same phthalates (DEHP, DBP, and BBzP) in urine for children ages 6 to 17 years. Both indicators have been updated since the publication of America’s Children and the Environment, Third Edition (January 2013) to include data from 2009–2014.

\textsuperscript{1} More information on NTP concern levels is available at http://www.niehs.nih.gov/news/media/questions/sya-bpa.cfm.
Biomonitoring | Phthalates

Indicator B9: Phthalate metabolites in women ages 16 to 49 years: Median concentrations in urine, 1999–2014

Indicator B10: Phthalate metabolites in children ages 6 to 17 years: Median concentrations in urine, 1999–2014

About the Indicators: Indicators B9 and B10 present concentrations of phthalate metabolites in urine of U.S. women ages 16 to 49 years and children ages 6 to 17 years. The data are from a national survey that collects urine specimens from a representative sample of the population every two years, and then measures the concentration of phthalate metabolites in the urine. Indicator B9 presents concentrations of phthalate metabolites in women’s urine over time and Indicator B10 presents concentrations of phthalate metabolites in children’s urine over time. The focus on both women of child-bearing age and children is based on concern for potential adverse effects in children born to women who have been exposed to phthalates and in children exposed to phthalates.

NHANES

The National Health and Nutrition Examination Survey (NHANES) provides nationally representative biomonitoring data for several phthalates. 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 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.69

Phthalate Metabolites

Indicators B9 and B10 present urinary metabolite levels of three important phthalates: di-2-ethylhexyl phthalate (DEHP), dibutyl phthalate (di-n-butyl phthalate and di-isobutyl phthalate) (DBP), and butyl benzyl phthalate (BBzP).

In NHANES and many research studies, biomonitoring of phthalates is conducted by measuring phthalate metabolites in urine rather than the phthalates themselves. This is because phthalates may be present in the sampling and laboratory equipment used to study human exposure levels, and contamination of samples could occur. Also phthalate metabolism is so rapid that the parent phthalate may not appear in urine.5–8,16,70,71 Furthermore, the phthalate metabolites, and not the parent phthalates, are generally considered to be the biologically active molecules.5–8,16,72 Unlike other contaminants that have a tendency to accumulate in the human body, phthalates are metabolized and excreted quickly, with elimination half-lives on the order of hours.5–8,71 Therefore, phthalate metabolites measured in humans are indicative of
recent exposures. All values are reported as micrograms of phthalate metabolites per liter of urine (µg/L).

Concentrations of phthalate metabolites, including those for DEHP, DBP, and BBzP, have been measured in urine from a representative subset of NHANES participants ages 6 and older beginning with the 1999–2000 survey cycle. For DEHP, three metabolites are included: mono-2-ethylhexyl phthalate (MEHP), mono-(2-ethyl-5-oxohexyl) phthalate (MEOHP), and mono-(2-ethyl-5-hydroxyhexyl) phthalate (MEHHP). The urinary levels of MEHP, MEOHP, and MEHHP are summed together, as is common in phthalates research, to provide a more complete picture of an individual’s total DEHP exposure than is given by any individual metabolite.\textsuperscript{57,73-75} The primary urinary metabolites of DBP are mono-n-butyl phthalate (MnBP) and mono-isobutyl phthalate (MiBP). The urinary levels of MnBP and MiBP were measured together for the NHANES 1999–2000 survey cycle, but for the following years were measured separately. Indicators B9 and B10 present the combined urinary levels of MnBP and MiBP for each survey cycle. The primary urinary metabolite of BBzP is mono-benzyl phthalate (MBzP).

Calculation of the DEHP metabolite and DBP metabolite indicator values involves summing together separate measured values (3 metabolites of DEHP, and 2 metabolites of DBP in the survey cycles following 1999–2000). If a metabolite included in the sum was not detected in a sample, a default value below the detection limit was assigned for purposes of calculating the summed total.\textsuperscript{iii}

In 2013–2014, NHANES collected phthalates biomonitoring data for 2,685 individuals ages 6 years and older, including 599 women ages 16 to 49 years and 750 children ages 6 to 17 years. DEHP metabolites were detected in about 58% of all individuals sampled. The frequency of DEHP metabolites detection was 62% in women ages 16 to 49 years,\textsuperscript{iv} and 64% in children ages 6 to 17 years. DBP metabolites and BBzP metabolite were detected in 96% and 98% of all individuals sampled, respectively. The frequency of DBP metabolites detection was 94% in women ages 16 to 49 years, and 98% in children ages 6 to 17 years. The frequency of BBzP metabolite detection was 98% in women ages 16 to 49 years, and 99% in children ages 6 to 17 years. The median and 95\textsuperscript{th} percentile of phthalate levels in urine for all NHANES participants in 2013–2014 were 13 µg/L and 60 µg/L, respectively, for DEHP; 19 µg/L and 84 µg/L, respectively, for DBP; and 5 µg/L and 35 µg/L, respectively, for BBzP. The widespread detection of phthalate metabolites, combined with the fact that phthalates have short half-lives, indicates that phthalate exposure is widespread and relatively continuous.

\textsuperscript{ii} A fourth DEHP metabolite, mono(2-ethyl-5-carboxypentyl) phthalate (MECPP), is now measured in NHANES but was not measured prior to 2003–2004. At least one other DEHP metabolite has been measured in laboratory studies but is not measured in NHANES.

\textsuperscript{iii} The default value used for non-detect samples is equal to the limit of detection divided by the square root of 2.

\textsuperscript{iv} The percentage for women ages 16 to 49 years is calculated with the birth rate adjustment described below.
Individual Variability in Urinary Measurements

NHANES data for phthalates are based on measurements made using a single urine sample for each person surveyed. Due to normal changes in an individual’s urinary output throughout the day, this variability in urinary volume, among other factors related to the measurement of chemicals that do not accumulate in the body, may mask differences between individuals in levels of phthalates. Since phthalates do not appear to accumulate in bodily tissues, the distribution of NHANES urinary phthalate levels may overestimate high-end exposures (e.g., at the 95th percentile) as a result of collecting one-time urine samples. Many studies account for differences in hydration levels by reporting the chemical concentration per gram of creatinine. Creatinine is a byproduct of muscle metabolism that is excreted in urine at a relatively constant rate, independent of the volume of urine, and can in some circumstances partially account for the measurement variability due to changes in urinary output. However, urinary creatinine concentrations differ significantly among different demographic groups, and are strongly associated with an individual’s muscle mass, age, sex, diet, health status (specifically renal function), body mass index, and pregnancy status. Thus, these indicators present the unadjusted phthalate concentrations so that any observed differences in concentrations between demographic groups are not due to differences in creatinine excretion rates. These unadjusted urinary levels from a single sample may either over- or underestimate urinary levels for a sampled individual. However, for a representative group, it can be expected that a median value based on single samples taken throughout the day will provide a good approximation of the median for that group. Furthermore, due to the large number of subjects surveyed, we expect that differences in the concentrations of phthalates that might be attributed to the volume of the urine sample would average out within and across the various comparison groups.

Birth Rate Adjustment

Indicator B9 uses measurements of phthalate metabolites in urine of women ages 16 to 49 years to represent the distribution of phthalate 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. A birth rate-adjusted distribution of women’s phthalate metabolite levels is used in calculating this indicator, meaning that the data are weighted using the age-specific probability of a woman giving birth.

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 Indicators

Indicator B9 presents median concentrations of DEHP, DBP, and BBzP metabolites in urine over time for women ages 16 to 49 years, using NHANES data from 1999–2014.

Indicator B10 presents median concentrations of DEHP, DBP, and BBzP metabolites in urine over time for children ages 6 to 17 years, using NHANES data from 1999–2014.

Additional information on the 95th percentile levels of urinary phthalates and how median levels of phthalate metabolites vary by race/ethnicity and family income for women ages 16 to 49 years is presented in the supplemental data tables for this indicator. Data tables also display the 95th percentile levels of phthalate metabolites and how median levels of phthalate metabolites vary by race/ethnicity, family income and age for children ages 6 to 17 years.

NHANES only provides phthalate metabolite data for children ages 6 years and older, which means that the indicator is not able to capture the exposure of premature infants, some of whom may have high levels of phthalate exposure due to the use of medical equipment containing phthalates; or young children, whose play and mouthing behaviors may increase their exposure to phthalates in toys and house dust.

Please see the Introduction to the Biomonitoring section for an explanation of the terms “median” and “95th percentile,” a description of the race/ethnicity and income groups used in the ACE3 biomonitoring indicators, and information on the statistical significance testing applied to these indicators.
The estimate is not reported because the metabolites MEOHP and MEHHP were not measured in 1999–2000.

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.
- Phthalate metabolites are measured in urine samples obtained from individual survey participants.

From 2001–2002 to 2007–2008, the median level of DEHP metabolites in urine of women ages 16 to 49 years varied between 41 μg/L and 51 μg/L, and was 51 μg/L in 2007–2008. The median level of DEHP metabolites decreased to 14 μg/L in 2013–2014.
- There was a statistically significant trend in median DEHP metabolites over 2001–2002 to 2013–2014.
From 1999–2000 to 2007–2008, the median level of DBP metabolites in urine of women ages 16 to 49 years varied between 27 μg/L and 36 μg/L, and was 36 μg/L in 2007–2008. The median level of DBP metabolites decreased to 20 μg/L in 2013–2014.

- There was a statistically significant trend in median DBP metabolites over 2007–2008 to 2013–2014. However, the trend was not statistically significant after adjusting for differences in age, race/ethnicity, and income.

From 1999–2000 to 2007–2008, the median level of BBzP metabolite in urine of women ages 16 to 49 years varied between 10 μg/L and 14 μg/L, and was 12 μg/L in 2007–2008. The median level of BBzP metabolite decreased to 6 μg/L in 2013–2014.

- There was a statistically significant trend in median BBzP metabolite over 2007–2008 to 2013–2014. However, the trend was not statistically significant after adjusting for differences in age, race/ethnicity, and income.

From 2001–2002 to 2007–2008, the concentration of DEHP metabolites in the 95th percentile varied between 462 μg/L and 578 μg/L, and was 567 μg/L in 2007–2008. There was an increasing trend in the 95th percentile concentration of DBP metabolites in women of child-bearing age, from 128 μg/L in 1999–2000 to 160 μg/L in 2007–2008. From 1999–2000 and 2007–2008, the concentration of BBzP metabolite varied between 68 μg/L and 100 μg/L, and was 70 μg/L in 2007–2008. (See Table B9a.)

- The increasing trend for DBP metabolites at the 95th percentile from 1999–2000 to 2007–2008 was statistically significant after accounting for differences by age, race/ethnicity, and income.

From 2007–2008 to 2011–2014, the 95th percentile concentrations of DEHP metabolites decreased from 567 μg/L to 57 μg/L, the 95th percentile concentrations of DBP metabolites decreased from 160 μg/L to 100 μg/L, and the 95th percentile concentration of BBzp metabolite decreased from 70 μg/L to 43 μg/L. (See Table B9a.)

- These decreasing trends were all statistically significant, except for the BBzP trend, before adjusting for differences in age, race/ethnicity, and income.

The concentrations of DEHP metabolites in the 95th percentile ranged from 4 to 14 times higher than the median levels presented in this graph. The concentrations of DBP metabolites and BBzP metabolite in urine at the 95th percentile ranged from 4 to 9 times higher than the median levels presented in this graph. (See Table B9 and B9a.)

For the years 2011–2014, Black non-Hispanic women of child-bearing age had higher median concentrations of all the phthalate metabolites shown here compared with White non-Hispanic women, Mexican-American women, and women of “All Other Races/Ethnicities,” although these differences were frequently not statistically significant. (See Table B9b.)

Median levels of urinary phthalate metabolites varied by family income. For the years 2011–2014, women living below the poverty level had higher concentrations of phthalate metabolites in their urine compared with women living at or above the poverty level. (See Table B9b.)

- The difference between income groups was statistically significant for the DBP metabolites and the BBzP metabolite. The difference between income groups was not statistically significant for the DEHP metabolites.
The estimate is not reported because the metabolites MEOHP and MEHHP were not measured in 1999–2000.

**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.
- Phthalate metabolites are measured in urine samples obtained from individual survey participants.

From 2001–2002 to 2007–2008, the median level of DEHP metabolites in urine of children ages 6 to 17 years varied between 45 μg/L and 62 μg/L, and was 45 μg/L in 2007–2008. From 2007–2008 to 2013–2014, the median level of DEHP metabolites in urine of children ages 6 to 17 years decreased to 16 μg/L.
There was a statistically significant trend in median DEHP metabolites over 2007–2008 to 2013–2014.

From 1999–2000 to 2007–2008, the median level of DBP metabolites in urine of children ages 6 to 17 years varied between 36 μg/L and 42 μg/L, and was 41 μg/L in 2007–2008. From 2007–2008 to 2013–2014, the median level of DBP metabolites in urine of children ages 6 to 17 years decreased to 25 μg/L.

There was a statistically significant trend in median DBP metabolites over 2007–2008 to 2013–2014, before accounting for differences in age, sex, race/ethnicity, and income.

The median level of BBzP metabolite in urine of children ages 6 to 17 years decreased from 25 μg/L in 1999–2000 to 8 μg/L in 2013–2014. This decreasing trend was statistically significant.

At the 95th percentile, there was an increasing trend in the concentration of DEHP metabolites in children, from 387 μg/L in 2001–2002 to 564 μg/L in 2007–2008. From 1999–2000 to 2007–2008, the concentration of DBP metabolites varied between 166 μg/L and 191 μg/L, and was 191 μg/L in 2007–2008. The concentration of BBzP metabolites varied between 104 μg/L and 151 μg/L, and was 107 μg/L in 2007–2008. (See Table B10a.)

The increasing trend for DEHP metabolites from 2001–2002 to 2007–2008 was statistically significant.

From 2007–2008 to 2013–2014, the 95th percentile concentration of DEHP metabolites in children decreased from 564 μg/L to 69 μg/L, the 95th percentile concentration of DBP metabolites decreased from 191 μg/L to 103 μg/L, and the 95th percentile concentration of BBzP metabolite decreased from 107 μg/L to 56 μg/L. (See Table B10a.)

These decreasing trends were all statistically significant.

Among children ages 6 to 17 years, the concentration of DEHP metabolites in urine at the 95th percentile ranged from 4 to 12 times higher than the median levels presented in this graph. The concentrations of metabolites of DBP and BBzP in the 95th percentile ranged from 4 to 7 times higher than the median levels. (See Table B10 and B10a.)

For the years 2011–2014, children living below poverty level had higher median concentrations of all the phthalate metabolites detected in their urine compared with children living at or above poverty level. (See Table B10b.)

The difference between income groups for DEHP metabolites was statistically significant after accounting for differences in age, sex, and race/ethnicity. The difference between income groups for BBzP metabolite was statistically significant before accounting for differences in age, sex, and race/ethnicity.

For the years 2011–2014, Black non-Hispanic children had higher median concentrations of all the phthalate metabolites shown here compared with all the other race/ethnicity groups. (See Table B10b.)

The differences were all statistically significant.

For the years 2011–2014, children ages 6 to 10 years had higher median levels of phthalate metabolites in their urine compared to adolescents ages 16-17 years. These differences were relatively small for DEHP metabolites and DBP metabolites but greater for BBzP metabolite. (See Table B10c.)
The age group differences for BBzP were statistically significant after accounting for differences in sex, race/ethnicity, and income.
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