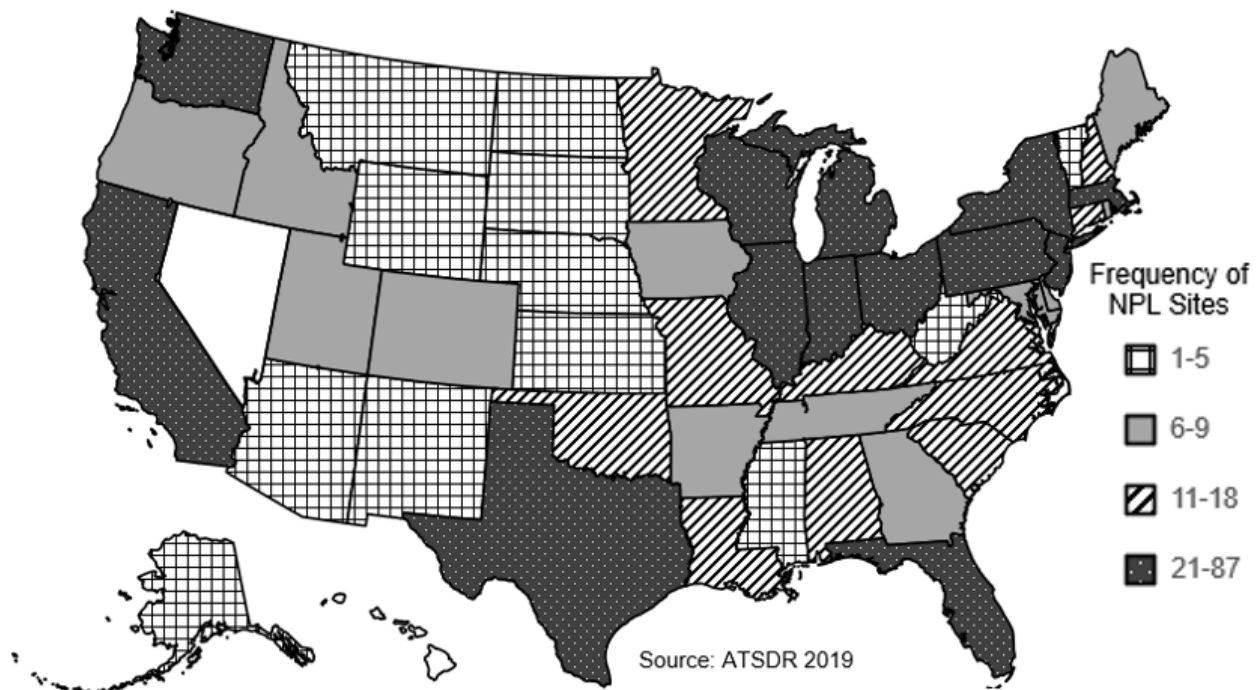


CHAPTER 5. POTENTIAL FOR HUMAN EXPOSURE

5.1 OVERVIEW

DEHP has been identified in at least 757 of the 1,867 hazardous waste sites that have been proposed for inclusion on the EPA National Priorities List (NPL) (ATSDR 2019). However, the number of sites in which DEHP has been evaluated is not known. The number of sites in each state is shown in Figure 5-1. Of these sites, 750 are located within the United States, 1 is located in the Virgin Islands, 1 is located in Guam, and 6 are located in Puerto Rico (not shown).

Figure 5-1. Number of NPL Sites with DEHP Contamination



- The most likely route of exposure for the general public to DEHP is through ingestion of food, inhalation or ingestion of house dust, and dermal contact with consumer products containing DEHP. Occupational exposures may be significant in some settings. However, the highest DEHP exposures result from medical procedures.
- DEHP is ubiquitous in the environment, although usually at low levels. The majority of DEHP in the environment sticks to soils and sediment.
- DEHP tends to sorb strongly to soils and sediments and to bioconcentrate in aquatic organisms. Biodegradation is expected to occur under aerobic conditions. The dominant fate pathway is determined by local environmental conditions.

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DEHP is a widely used chemical that enters the environment both through disposal of industrial and municipal wastes in landfills and by leaching into consumer products stored in plastics. It tends to sorb strongly to soils and sediments and to bioconcentrate in aquatic organisms; however, biomagnification of DEHP in the food chain is not expected to occur due to metabolism. Biodegradation is expected to occur under aerobic conditions. Sorption, bioconcentration, and biodegradation are likely to be competing processes, with the dominant fate being determined by local environmental conditions, such as pH, soil texture, and oxygen levels.

The principal route of human exposure to DEHP is oral. Much of the monitoring database is old and might not represent current exposures, especially since the uses of DEHP in certain applications has been changing (CPSIA 2008; Wilkinson and Lamb 1999). The U.S. Department of Health and Human Services estimates that the average U.S. adult exposure to DEHP is on the order of 3–30 $\mu\text{g}/\text{kg}/\text{day}$ (NTP 2006). Populations residing near hazardous waste disposal sites or municipal landfills might be subject to higher than average levels of DEHP in ambient air and drinking water. Even so, the concentrations of DEHP in these media will be greatly limited by the low volatility and low water solubility of DEHP.

Occupational exposures might be significant, but the highest exposures to DEHP result from medical procedures such as blood transfusions (e.g., estimated upper bound limit of 8.5 $\text{mg}/\text{kg}/\text{day}$) or hemodialysis (e.g., estimated upper bound limit of 0.36 $\text{mg}/\text{kg}/\text{day}$), during which DEHP might leach from plastic equipment into biological fluids (FDA 2001). Exposures of neonates to DEHP can be especially high as a result of some medical procedures; TPN administration (e.g., estimated upper bound limit of 2.5 $\text{mg}/\text{kg}/\text{day}$), and extracorporeal membrane oxygenation (ECMO) (e.g., estimated upper bound limit of 14 $\text{mg}/\text{kg}/\text{day}$) (FDA 2001). A report published by the European Union Scientific Committee on Emerging and Newly-Identified Health Risks estimated that the highest acute/short-term exposures to DEHP were from the plastics (intravenous bags and lines) used during blood transfusions or ECMO (SCENIHR 2016). Maximum exposures to DEHP during these procedures were estimated at 8–10 $\text{mg}/\text{kg}/\text{day}$. The highest risk from chronic treatment comes from patients undergoing hemodialysis, with a maximum reported exposure of 2.2 $\text{mg}/\text{kg}/\text{day}$ (SCENIHR 2016).

When DEHP is present in the environment, it is usually at very low levels. DEHP was a ubiquitous laboratory contaminant, which made it difficult to determine low levels accurately due to the potential for false identification of elevated phthalate concentrations from sample contamination. In recent years, DEHP-free laboratory equipment has been made available, reducing the potential for contaminating a sample.

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5.2 PRODUCTION, IMPORT/EXPORT, USE, AND DISPOSAL**5.2.1 Production**

DEHP is a member of a group of compounds commonly referred to as the phthalate esters, which are predominantly used as plasticizers in flexible products made from PVC (CPSC 2010a). DEHP is produced by the esterification of phthalic anhydride with 2-ethylhexyl alcohol in the presence of an acid catalyst (CPSC 2010a). Phthalate plasticizers can be produced using this reaction in batch methods or in highly automated continuous operations (TURI 2006). DEHP can also be manufactured by the dimerization of butyraldehyde (Cadogan and Howick 2001). The production volume of DEHP in the United States was 120,000 metric tons (265 million pounds) in 2002 (TURI 2006). Production and/or use in the United States in 2006 was reported as 45,000–230,000 tons (90–460 million pounds) (Zolfaghari et al. 2014). Worldwide production was estimated to be 2 million metric tons (4.4 billion pounds) in 2004 (Erythropel et al. 2014). Worldwide production of DEHP is decreasing, mainly related to the regulations being enforced against certain uses of DEHP (Zolfaghari et al. 2014).

The Chemical Data Reporting (CDR) rule, which was enacted through the Toxic Substances Control Act (TSCA), requires manufacturers including importers of chemicals to provide EPA with information on the production and use of these chemicals in commerce. The Chemical Data Reporter indicated that there were 37 U.S.-based companies that either manufactured or imported DEHP to the United States in 2016 (CDR 2016). Most companies reported the production or import volume as confidential business information (CBI); however, in 2015 it was reported that at least 10,196,363 pounds were produced or imported from 19 companies.

Table 5-1 summarizes the number and location of U.S. facilities that reported the use and production of DEHP in 2018 (TRI18 2020). The Toxics Release Inventory (TRI) data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list.

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Table 5-1. Facilities that Produce, Process, or Use DEHP

State ^a	Number of facilities	Minimum amount on site in pounds ^b	Maximum amount on site in pounds ^b	Activities and uses ^c
AL	1	1,000	9,999	8
AR	4	1,000	999,999	7, 8, 11
CA	8	100	9,999,999	7, 8, 14
CT	1	100	999	7
GA	3	100,000	999,999	8, 9
IL	2	1,000	99,999	7, 8
IN	5	100	9,999	7, 8, 12
LA	1	10,000	99,999	1, 5, 12
MA	4	10,000	999,999	8, 10, 11
MI	4	0	99	7
MO	6	100	999,999	7, 8
MS	4	1,000	99,999	7, 8
NC	10	0	999,999	1, 2, 3, 5, 7, 8, 13, 14
NJ	5	10,000	499,999,999	2, 3, 4, 7, 8, 9
NY	3	10,000	999,999	7, 8
OH	14	100	999,999	7, 8, 9, 12
OR	3	10,000	99,999	7, 8, 12
PA	3	10,000	999,999	2, 3, 7, 8
PR	3	1,000	999,999	8
RI	1	100,000	999,999	7, 14
SC	5	1,000	999,999	7, 8, 12, 14
TN	8	1,000	9,999,999	1, 2, 3, 4, 5, 7, 8, 9, 10, 12, 13, 14
TX	7	1,000	999,999	7, 8, 10
VA	2	100,000	999,999	7, 8
WA	3	1,000	999,999	7, 8, 9
WI	6	100	999,999	7, 8, 9
WV	1	10,000	99,999	7, 8

^aPost office state abbreviations used.

^bAmounts on site reported by facilities in each state.

^cActivities/Uses:

- | | | |
|----------------------|-----------------------------|--------------------------|
| 1. Produce | 6. Reactant | 11. Manufacture Aid |
| 2. Import | 7. Formulation Component | 12. Ancillary |
| 3. Used Processing | 8. Article Component | 13. Manufacture Impurity |
| 4. Sale/Distribution | 9. Repackaging | 14. Process Impurity |
| 5. Byproduct | 10. Chemical Processing Aid | |

Source: TRI18 2020 (Data are from 2018)

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Decreasing demand for DEHP due to continued concern over health effects will impact future production volumes (Zolfaghari et al. 2014).

5.2.2 Import/Export

Estimated annual imports and exports from the United States in 2006 were reported to be approximately 69 and 13 million pounds, respectively (CPSC 2010a). The Chemical Data Reporter (CDR 2016) indicated a downward trend in annual imports, with estimated production or import of at least 28 million pounds in 2011 and 10 million pounds in 2015.

5.2.3 Use

DEHP was principally used as a plasticizer in the production of flexible PVC products, with about 97% of DEHP produced being used for this purpose (CPSC 2010a). DEHP is generally used to dissolve monomers to facilitate their crosslinking into polymers (e.g., the conversion of vinyl chloride into PVC) (Chaudhary et al. 2016). The polymerization process retains a portion of the plasticizer, and the retention of DEHP in polymers such as PVC increases flexibility, reduces hardness, and decreases tensile strength of the plastic (Chaudhary et al. 2016). Plastics made with DEHP can be found in many common items such as wall coverings, tablecloths, floor tiles, furniture upholstery, shower curtains, garden hoses, swimming pool liners, rainwear, baby pants, dolls, toys, shoes, automobile upholstery and tops, packaging film and sheet, sheathing for wire and cable, medical tubing, and blood storage bags. PVC is also used to produce disposable medical examination and surgical gloves, flexible tubing used to administer parenteral solutions, tubing used in hemodialysis treatment, syringes, and blood, dialysis, and storage bags (CPSC 2010a; NTP 1989). Current restrictions on the use of DEHP in PVC materials has led manufacturers to find alternatives to DEHP. In an effort to reduce use of DEHP, current or proposed replacement plasticizers that may reduce toxicity include citrate-based plasticizers, such as acetyl tri-*n*-butyl citrate (ATBC), as well as 1,2-cyclohexanedicarboxylic acid, diisononyl ester (DINCH), di(2-ethylhexyl) adipate (DEHA), trioctyltrimellitate (TOTM), and di(2-ethylhexyl) terephthalate (DEHT or DOTP) (CPSC 2010b; EPA 2012; Messerlian et al. 2017b; Tickner et al. 2001).

DEHP is also used as a plasticizer in products such as polyvinyl acetate, polyvinyl butyral, natural and synthetic rubber, chlorinated rubber, ethyl cellulose, nitrocellulose, and polyurethane resins (CPSC 2010a). DEHP plasticizer use in medical devices and industrial/commercial products accounts for 25 and 45% of the overall consumption of DEHP, respectively (CPSC 2010a). In 2017, the European Union

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passed the new Medical Device Regulation, which restricted the use of DEHP and other substances of very high concern by 2020 and encourages the use of alternatives (Hansen 2019).

Numerous nonplasticizer uses of DEHP have been reported and account for <3–5% of the national use of DEHP (CPSC 2010a). These uses include as a solvent in erasable ink and ultrasound gel, as a carrier for pesticides, in ceramics, in cosmetics, in vacuum pump oil, as a component of dielectric fluids in electrical capacitors, to detect leaks in respirators, in paints, lacquers, and adhesives, and in testing the efficiency of air filtration systems (CPSC 2010a; Mannsville Chemical Products Corporation 1990; Messerlian et al. 2017b; NTP 1989).

Because of concerns regarding health effects from exposure to DEHP, many toy manufacturers have discontinued use of all phthalates in their products (Wilkinson and Lamb 1999). The use of DEHP in domestically produced teethingers and rattles has also been discontinued (CPSC 1999). In 2008, Congress permanently banned DEHP in any amount >0.1% in children's toys and certain childcare articles, such as those to help sleeping, feeding, sucking, or teething of children ≤ 3 years old (CPSIA 2008). Risk assessments have supported this permanent ban (CPSC 2014; Liroy et al. 2015).

DEHP has been removed from or replaced as a plasticizer in most food packaging products (CDC 2016); however, the FDA still approves its use as an indirect additive in food contact substances as a component of or surface lubricant for adhesives, coatings, paper and paperboard, acrylic polymers, cellophane, and metallic foil (FDA 1999a, 1999b, 1999c, 1999d, 1999e, 1999f, 1999g). Finally, in the future, polyolefin metallocene elastomers might replace flexible applications for PVC and other plastics altogether because they provide flexibility without the need for plasticizers. DEHP has also been replaced with DINCH in some ultrasound gels (Messerlian et al. 2017b).

5.2.4 Disposal

When DEHP (as a commercial chemical product or chemical intermediate) becomes a waste, its disposal is regulated by law, as shown in Chapter 7. DEHP disposal is regulated under the Resource Conservation and Recovery Act (RCRA). Regulations promulgated under this Act control the treatment, storage, and disposal of waste DEHP. Land disposal restrictions are the responsibility of the EPA Office of Solid Waste. In 2018, it was estimated that about 643,000 pounds of waste DEHP were transported from production facilities or points of usage for disposal, including publicly owned treatment works (TRI18

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2020). No data were located regarding the quantity of waste DEHP that was disposed of by any specific means. No data were located regarding trends in DEHP disposal.

Bioremediation of DEHP-contaminated soils has been studied through bench experiments. It has been reported that 89% removal of DEHP, with an initial concentration of 5.51 mg/g dry soil, was achieved in 76 days through the addition of nutrients and inoculum to the soil (Carrara et al. 2011). However, these bench studies cannot be inferred directly to field use, as parameters such as DEHP adsorption to organic matter in soil will vary; therefore, *in situ* and intrinsic bioremediation studies in various soil conditions are needed. Carrara et al. (2011) performed pilot *ex situ* bioremediation tests on tropical soils using a slurry-phase reactor and were able to achieve 99% removal of DEHP in 49 days.

5.3 RELEASES TO THE ENVIRONMENT

The Toxics Release Inventory (TRI) data should be used with caution because only certain types of facilities are required to report (EPA 2005). This is not an exhaustive list. Manufacturing and processing facilities are required to report information to the TRI only if they employ ≥ 10 full-time employees; if their facility is included in Standard Industrial Classification (SIC) Codes 10 (except 1011, 1081, and 1094), 12 (except 1241), 20–39, 4911 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4931 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4939 (limited to facilities that combust coal and/or oil for the purpose of generating electricity for distribution in commerce), 4953 (limited to facilities regulated under RCRA Subtitle C, 42 U.S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited S.C. section 6921 et seq.), 5169, 5171, and 7389 (limited to facilities primarily engaged in solvents recovery services on a contract or fee basis); and if their facility produces, imports, or processes $\geq 25,000$ pounds of any TRI chemical or otherwise uses $>10,000$ pounds of a TRI chemical in a calendar year (EPA 2005).

Industrial manufacturers, processors, and users of DEHP are required to report the quantities of this substance released to environmental media annually (EPA 2005). The data compiled in the TRI (TRI18 2020) are for releases in 2018 to air, water, soil, and transfers for offsite disposal. These data are summarized in Table 5-2. Total releases of DEHP to the environment in 2018 were approximately 711,000 pounds (approximately 322 metric tons) (TRI18 2020).

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Table 5-2. Releases to the Environment from Facilities that Produce, Process, or Use DEHP^a

State ^c	RF ^d	Air ^e	Reported amounts released in pounds per year ^b						
			Water ^f	UI ^g	Land ^h	Other ⁱ	Total release		
							On-site ^j	Off-site ^k	On- and off-site
AL	1	26,280	0	0	0	0	26,280	0	26,280
AR	4	1,738	7	0	2,354	0	2,494	1,604	4,098
CA	8	1,389	4	0	7,190	0	1,393	7,190	8,583
CT	1	0	0	0	0	311	0	311	311
GA	3	295	0	0	2,943	0	295	2,943	3,238
IL	2	29	0	0	1,900	0	29	1,900	1,929
IN	5	132	0	0	4,453	0	132	4,453	4,585
LA	1	0	0	0	0	0	0	0	0
MA	4	191	0	0	0	0	191	0	191
MI	4	250	33	0	0	0	250	33	283
MO	6	1,544	5	0	43,438	880	1,544	44,323	45,867
MS	4	42	0	0	98,260	0	42	98,260	98,301
NC	10	2,011	6	0	33,509	0	2,011	33,515	35,526
NJ	5	26	0	0	0	1,040	26	1,040	1,066
NY	3	2,143	0	0	0	0	2,143	0	2,143
OH	14	641	5	0	11,903	17,491	641	29,399	30,039
OR	3	60	0	0	19,688	0	19,748	0	19,748
PA	3	2	0	0	3,900	0	2	3,900	3,902
PR	3	24	0	0	0	0	24	0	24
RI	1	1,717	0	0	594	0	1,717	594	2,311
SC	5	201	1,564	5	142	250	201	1,961	2,162
TN	8	5,564	15	0	11,524	62	6,029	11,136	17,165
TX	7	6	0	395,800	0	0	6	395,800	395,806
VA	2	261	0	0	0	0	261	0	261
WA	3	1,603	0	0	0	0	1,603	0	1,603
WI	6	271	0	0	4,235	0	271	4,235	4,506
WV	1	255	10	0	500	0	510	255	765

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Table 5-2. Releases to the Environment from Facilities that Produce, Process, or Use DEHP^a

State ^c	RF ^d	Air ^e	Reported amounts released in pounds per year ^b						
			Water ^f	UI ^g	Land ^h	Other ⁱ	Total release		
							On-site ^j	Off-site ^k	On- and off-site
Total	117	46,674	1,649	395,805	246,532	20,034	67,843	642,851	710,694

^aThe TRI data should be used with caution since only certain types of facilities are required to report. This is not an exhaustive list. Data are rounded to nearest whole number.

^bData in TRI are maximum amounts released by each facility.

^cPost office state abbreviations are used.

^dNumber of reporting facilities.

^eThe sum of fugitive and point source releases are included in releases to air by a given facility.

^fSurface water discharges, wastewater treatment-(metals only), and publicly owned treatment works (POTWs) (metal and metal compounds).

^gClass I wells, Class II-V wells, and underground injection.

^hResource Conservation and Recovery Act (RCRA) subtitle C landfills; other onsite landfills, land treatment, surface impoundments, other land disposal, other landfills.

ⁱStorage only, solidification/stabilization (metals only), other off-site management, transfers to waste broker for disposal, unknown

^jThe sum of all releases of the chemical to air, land, water, and underground injection wells.

^kTotal amount of chemical transferred off-site, including to POTWs.

RF = reporting facilities; UI = underground injection

Source: TRI18 2020 (Data are from 2018)

Industrial releases are only a fraction of the total environmental releases of DEHP. Release of DEHP into the environment is thought to originate from diffuse sources, mainly from end-uses of DEHP (e.g., as an additive to plastics) by leaching or evaporating (Clara et al. 2010). Disposal of plastic products containing DEHP (Section 5.2.4) is also a possible source of environmental release (Bauer and Herrmann 1997; EPA 1981). Quantitative information on releases of DEHP to specific environmental media is discussed below.

5.3.1 Air

Estimated releases of 46,674 pounds (~21 metric tons) of DEHP to the atmosphere from 117 domestic manufacturing and processing facilities in 2018, accounted for about 7% of the estimated total environmental releases from facilities required to report to the TRI (TRI18 2020). These releases are summarized in Table 5-2.

As presented in Chapter 4, DEHP has a relatively low vapor pressure and Henry's law constant, as well as a relatively high octanol/water partition coefficient and soil sorption coefficient. This combination of

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properties is consistent with a chemical that is found to only a limited extent in air (Staples et al. 1997). Nonetheless, DEHP appears to be a common air contaminant that is present globally in low ng/m^3 concentrations (Section 5.5.1), although specific information that quantifies emissions of DEHP to air appears to be insufficient to account for this apparent widespread presence. For example, while monitoring data show that elevated fallout concentrations of DEHP are associated with industrial activity (Thurén and Larsson 1990), elevated fallout concentrations were only seen near a stack, and no elevated concentrations could be seen 2 km away from the stack. In addition, these authors could not correlate DEHP fallout rates with specific sources or transport routes on a nationwide basis in Sweden. They found no “distributional patterns or gradient,” which possibly suggests that any local patterns were obscured by DEHP contribution from other sources or that emission sources of roughly equal magnitude are diffuse. By contrast, a pattern associating distance from sources and concentration was seen with DEHP by Ritsema et al. (1989) in Lake Yssel in the Netherlands, while for other lower-molecular-weight phthalate esters, no pattern was evident. The authors suggested that an upstream source was the dominant mechanism by which DEHP enters the lake, not atmospheric deposition.

Emissions of DEHP to air can occur due to volatilization from sludge used in wastewater treatment plants. Lee et al. (2019c) collected sludge samples from 40 wastewater treatment facilities in South Korea and investigated the occurrence and emissions of phthalates from this source. DEHP was the dominant phthalate found in the sludge samples with levels ranging from 1,400 to 1,000,000 ng/g (71,000 ng/g mean). Using these data, an average emission of 1,310 kg/day was estimated from wastewater treatment plants in Korea.

The possibility of many diffuse sources of DEHP is potentially supported by some of the uses. For example, some of the products that use DEHP include thin sheets and coatings, such as floor tiles, shower curtains, tablecloths, and furniture upholstery. These products characteristically have large surface area-to-volume ratios, which might allow DEHP to volatilize more readily relative to other products with smaller surface area-to-volume ratios. Liang et al. (2019) developed a multi-media indoor fate model to estimate levels of phthalates such as DEHP in indoor air environments. The model accounted for emissions from common housing materials and sorption and resuspension from surfaces such as flooring, ceilings, furniture, and carpet. Steady-state DEHP levels in air from a typical residential home were estimated as 0.14 $\mu\text{g}/\text{m}^3$ and 80–46 000 $\mu\text{g}/\text{g}$ in settled dust on various surfaces. Shinohara and Uchino (2020) measured the emissions of DEHP to indoor air and dust from a PVC sheet over a 2-week period using a passive sampler. DEHP levels in the surface air on the PVC sheet were in the range of 2.6–3.3 $\mu\text{g}/\text{m}^3$. In a similar study, Shinohara et al. (2019) measured flux rates of DEHP from building

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materials, such as vinyl floorings and wallpaper, using a passive flux sampler. They found that the rates were relatively constant over time, with fluxes in the range of 4.5–6.1 $\mu\text{g}/\text{m}^2\text{-hour}$. Cadogan et al. (1994) and Cadogan and Howick (2001) reported that an indoor flux of 2.3×10^{-4} $\text{mg}/\text{m}^2\text{-second}$ (828 $\mu\text{g}/\text{m}^2\text{-hour}$) at 25 °C has been calculated for all phthalate plasticizers in products such as wall coverings, flooring, upholstery, and wire insulation. These authors used this emission estimate to calculate overall releases of phthalate esters to air. Cadogan and Howick (2001) also noted that approximately 47% of the phthalate ester used is DEHP. Applying this DEHP use percentage to their emission estimates, the total end-use emission of DEHP to the air from indoor household uses in Western Europe in 1990 was approximately 300 tons per year. Emissions from exterior end uses were estimated to be 5,600 tons per year for DEHP (the authors noted that this estimate was not well defined). These estimates support the conclusion that the major sources of DEHP are from end-uses and that these represent a geographically diffuse source. Finally, Jones et al. (1996) estimated that between 0.001 and 3.6 metric tons of DEHP are emitted per year (depending on assumptions about vapor equilibria and mass transfer used in model calculations) from sewer manholes in a large U.S. city having an average DEHP sewage concentration of 26 $\mu\text{g}/\text{L}$.

It has been estimated that <3% of the total U.S. domestic supply of DEHP is released to air (EPA 1981). Based on a reported U.S. production amount in 2002 of about 265 million pounds, discussed in Section 5.2.1, the estimated annual atmospheric emission of DEHP from all sources in the United States was about 8.0 million pounds in 2002.

DEHP may also be released into the air from burning domestic materials that still contain this compound from legacy use as a fire retardant, such as clothing and furnishing (Alexander and Baxter 2016; Lacey et al. 2014). DEHP detected on firefighter protective clothing has been attributed to release of semi-volatile toxic combustion products during structural fires (Alexander and Baxter 2016; Lacey et al. 2014).

5.3.2 Water

Estimated releases of 1,649 pounds (~0.75 metric tons) of DEHP to surface water from 117 domestic manufacturing and processing facilities in 2018, accounted for about 0.23% of the estimated total environmental releases from facilities required to report to the TRI (TRI18 2020). These releases are summarized in Table 5-2.

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As a result of secondary treatment processes in publicly owned treatment works (POTWs), only a small percentage (<3%) of DEHP that enters POTWs is subsequently released to surface water (Yu and Chu 2009; Zolfaghari et al. 2014).

DEHP was detected in 13% of 86 samples of urban storm water runoff evaluated for the National Urban Runoff Program, at concentrations ranging from 7 to 39 ppb (Cole et al. 1984). In some locations, storm and sanitary sewers are separated so that storm water runoff in these locations directly enters surface water. Even in locations with combined storm and sanitary sewers, DEHP is still expected to enter the environment, but probably to a lesser extent. For example, Stubin et al. (1996) reported that DEHP was present in 48% of the influent and 12% of the effluent samples taken from New York City sewage treatment plants during 1989–1993. Thus, storm water runoff, even when it goes through a sewage treatment plant, might enter the environment. In addition, DEHP also appears to be present in the treatment plant influent whether or not it receives storm water. It was reported that raw sewage samples had DEHP concentrations ranging from 3.4 to 34 µg/L and wastewater treatment plant effluent samples had concentrations of 0.083–6.6 µg/L (Clara et al. 2010). Influent at two wastewater treatment plants in eastern Tennessee contained total DEHP levels of 8,572 and 12,160 ng/L, while only one plant had detectable DEHP in its effluent discharge at 300 ng/L (Yu and Chu 2009). DEHP has also been reported in wastewater from a petrochemical plant (Castillo et al. 1998), leachate from industrial and municipal landfills (Brown and Donnelly 1988; Castillo et al. 1998; Ghassemi et al. 1984; Roy 1994), and sewage sludge (O'Connor 1996). It is anticipated that water from all of these sources enters the environment and might contain DEHP. Stubin et al. (1996) noted that DEHP was commonly present (48% of the samples) in municipal sewage treatment plant influent, suggesting that DEHP is present in domestic wastewater. DEHP in domestic wastewater can come from either the source tap water or from activities within the household such as washing floors that contain DEHP, showering using a shower curtain containing DEHP, or washing other DEHP-containing materials.

5.3.3 Soil

Estimated releases of 246,532 pounds (~112 metric tons) of DEHP to soils from 117 domestic manufacturing and processing facilities in 2018, accounted for about 35% of the estimated total environmental releases from facilities required to report to the TRI (TRI18 2020). An additional 395,805 pounds (~180 metric tons), accounted for about 56% of the total environmental emissions, were released via underground injection (TRI18 2020). These releases are summarized in Table 5-2.

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The principal source of DEHP release to land is likely the disposal of industrial and municipal waste to landfills (EPA 1981). Municipal wastes probably contain substantial quantities of DEHP-containing plastics, which might significantly increase the total quantity of DEHP released to land. Based on an estimate that 92% of U.S. domestic supplies of DEHP are released to landfills (EPA 1981) and a reported U.S. domestic production in 2002 of approximately 265 million pounds (Section 5.2.1), it was estimated that about 244 million pounds of DEHP are deposited in landfills annually. Bauer and Herrmann (1997) reported the concentration of DEHP in various fractions of household wastes from the regions of Bayreuth and Straubling in Germany. The wastes included food waste, paper for recycling, unusable paper, cardboard, plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound packing waste, compound materials, and disposable diapers. DEHP was found in all of the fractions. It is anticipated that household waste from continental Europe is similar to the United States, so that the same profile would be expected in both places. Further information on this study is presented in Section 5.5.4 and Table 5-8.

Land application of sewage sludge might also release DEHP to soil. The 1989 National Sewage Sludge Survey estimated that mean DEHP concentrations in sludge range from 55 to 300 ppm, with a national mean of 75 ppm (EPA 1990). It is also estimated that about 42% of sewage sludge generated in the United States annually, or 5.1 billion pounds, is applied to land as biosolids. Another 20% (2.4 billion pounds) is deposited in landfills, and 14% (1.7 billion pounds) is incinerated (EPA 1990). Using the national mean concentration and a total of 7.5 billion pounds of sludge deposited in soils, sludge accounts for approximately 7,500 pounds of DEHP released to soils annually. In the 2009 National Sewage Sludge Survey, DEHP was detected in 84 samples collected from 74 treatment plants in 35 states, at concentrations ranging from 657 to 310,000 $\mu\text{g}/\text{kg}$ (0.657–310 ppm) (EPA 2009a).

DEHP has also been reported in ocean sediments at levels up to 25 ppm at points of urban sewage outfall (Swartz et al. 1985), and in 100% of the sediments in rivers near combined sewer overflows in New Jersey (Iannuzzi et al. 1997). Concentrations of phthalates, including DEHP, are approximately 10 times higher in stream sediments that are influenced by urban activity than in areas under other land-use activities (Lopes and Furlong 2001).

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5.4 ENVIRONMENTAL FATE**5.4.1 Transport and Partitioning**

Air. DEHP is ubiquitous in air at low concentrations (e.g., 0.06–5.0 ng/m³) (Eisenreich et al. 1981; Ligocki et al. 1985a; Lunderberg et al. 2019), in both the vapor phase and associated with particulates, and is subject to both wet (rain and snow) and dry (wind and settling) deposition on the Earth's surfaces (Atlas and Giam 1981; Eisenreich et al. 1981; Ligocki et al. 1985a, 1985b). Eisenreich et al. (1981) calculated that wet and dry deposition of DEHP into the five Great Lakes amounted to approximately 47.7 metric tons per year, which corresponds to an average fallout rate of 16.2 µg/m² per month. A similar average fallout rate of 23.8 µg/m² per month (the range was 5.96–195.5 µg/m² per month) was reported by Thurén and Larsson (1990) for DEHP in Sweden. The authors noted that the deposition rate for DEHP decreased with increasing distance from a smokestack at a phthalate-consuming factory. DEHP has been found in Antarctic surface and sub-surface snow (up to 3 m deep), and in pack ice (Desideri et al. 1994, 1998), as well as in the atmosphere over the Gulf of Mexico (Giam et al. 1980), suggesting that DEHP can be transported for long distances. Thus, the DEHP measured in one part of the world might have originated elsewhere. This transport is likely particle-sorbed DEHP (Atlas and Giam 1981) because vapor-phase DEHP reacts rapidly with hydroxyl radicals in the atmosphere (Section 5.4.2), while particle-sorbed DEHP does not react rapidly with hydroxyl radicals. Nearly half of the DEHP detected in the atmosphere over the Gulf of Mexico was in the particulate phase (Giam et al. 1980). Atmospheric fallout is negatively correlated with temperature; less DEHP is subject to fallout in the summer than in the winter (Staples et al. 1997; Thurén and Larsson 1990). This is in keeping with a higher proportion of the atmospheric DEHP in the vapor state in the warm summer and less in the cold winter, and further indicates that the partitioning between particles and vapor is controlled by vapor pressure.

Water. In water, DEHP is predominantly sorbed to suspended particulates and sediments, but some remains dissolved in the aqueous phase. The vapor pressure of DEHP is extremely low compared to water, indicating that volatilization is not a dominant transport process. Volatilization from water and soil may be expected, based on the Henry's law constant (estimated value 1.71×10^{-5} atm·m³/mol; Staples et al. 1997); however, adsorption to soil and suspended particulate matter in the water column will attenuate the rate of volatilization. It has been estimated that the evaporative half-life of DEHP from water would be about 15 years (EPA 1979), and that only about 2% of DEHP loading of lakes and ponds would be volatilized (Wolfe et al. 1980).

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Sediment and Soil. Adsorption onto soils and sediments is a significant sink for DEHP. DEHP released to water adsorbs strongly to suspended particulates and sediments (Al-Omran and Preston 1987; Staples et al. 1997; Sullivan et al. 1982; Wolfe et al. 1980). Distribution of DEHP between the water column and the sediments was modeled for several types of freshwater aquatic environments (Wolfe et al. 1980). Between 69 and 99% of DEHP was estimated to partition to the sediments. Adsorption of DEHP to marine sediments might be greater than adsorption to freshwater sediments, due to reduced solubility of DEHP in saltwater and a salting-out effect (Al-Omran and Preston 1987; Sullivan et al. 1982; Yuwatini et al. 2013; Zhou and Liu 2000). Levels of DEHP in a marine environment ranged from 0.1 to 0.7 ppb in the water and from 280 to 640 ppb in the suspended particulates (Preston and Al-Omran 1989). DEHP shows greater adsorption to the smaller size particle fractions of suspended particulates or colloids (Al-Omran and Preston 1987; Zhou and Liu 2000). Complexation of DEHP with fulvic acid, a compound associated with humic substances in water and soil, might increase solubilization and thus increase the mobility of DEHP in aquatic systems (Johnson et al. 1977). Ritsema et al. (1989) noted that DEHP in the River Rhine was mainly associated with suspended particulates, but on some sampling days, dissolved DEHP was at a higher concentration than the sorbed material. By contrast, in Lake Yssel, DEHP concentrations in the suspended material were approximately 100 times higher than the dissolved material. In addition, the authors reported that a distinct concentration gradient was noted across the lake, suggesting that DEHP entered the lake from the River Yssel rather than nonpoint sources as was the case with some other phthalates.

Other Media. Percolation of DEHP through the soil to groundwater might occur during times of rapid infiltration. DEHP concentrations were generally reduced by infiltration through a soil column, but all column effluents contained measurable levels (Hutchins et al. 1983). In hazardous waste sites, the presence of common organic solvents such as alcohols and ketones might increase the solubility of relatively insoluble compounds such as DEHP, thereby increasing the amounts that might leach from the waste site into subsoil and groundwater (Nyssen et al. 1987). This is consistent with the measurement of DEHP in leachate of some landfills at levels in excess of its usual water solubility (Section 5.3.2).

Bioconcentration of DEHP has been observed in invertebrates, fish, and terrestrial organisms. Mean bioconcentration factors (BCFs) have been reported for algae ($3,173 \pm 3,149$, two species), mollusks ($1,469 \pm 949$, five species), crustacea ($1,164 \pm 1,182$, four species), insects ($1,058 \pm 772$, three species), polychaetes (422, one species), fish (280 ± 230 , five species), and amphibians (605, one species) have been compiled by Staples et al. (1997). Residues of DEHP have been found in the organs of terrestrial animals such as rats, rabbits, dogs, cows, and humans (EPA 1979). However, accumulation of DEHP will be

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minimized by metabolism, and biomagnification of DEHP in the food chain is not expected to occur (EPA 1979; Johnson et al. 1977; Mackintosh et al. 2004; Staples et al. 1997; Wofford et al. 1981). Several metabolites of DEHP might be detected in animal tissues (Johnson et al. 1977). Uptake of DEHP from soil by plants has also been reported (EPA 1986; O'Connor 1996).

5.4.2 Transformation and Degradation

Air. Reaction of DEHP vapor with hydroxyl radicals in the atmosphere has been predicted, with an estimated half-life of about 6 hours using the Atmospheric Oxidation Program (Meylan and Howard 1993). The atmospheric half-life, however, is expected to be longer for DEHP adsorbed to atmospheric particulates. Based on the estimated half-life alone, extensive transport of DEHP would not be expected and concentrations in Antarctic snow would not be predicted. Nonetheless, DEHP appears to be present in urban and rural atmospheres (Section 5.5.1), and its transport might be mainly in the sorbed state. Data confirming this degradation pathway have not been located. Direct photolysis and photooxidation are not likely to be important (Wams 1987).

Water. Biodegradation might be an important fate process for DEHP in water under aerobic, but not anaerobic, conditions (O'Connor et al. 1989; O'Grady et al. 1985; Sugatt et al. 1984; Tabak et al. 1981; Thomas et al. 1986). DEHP was significantly biodegraded (>95%) after gradual acclimation of the microbial population over a period of about 3 weeks under conditions of the static-flask and shake-flask screening tests (Sugatt et al. 1984; Tabak et al. 1981). In the shake flask study using an acclimated inoculum, initial biodegradation was low on days 2 and 3, but increased 5–10-fold by days 6 and 7; degradation to carbon dioxide was 87% at 28 days (Sugatt et al. 1984). The reported half-life of DEHP due to microbial activity in river water is about 1 month (Wams 1987). In freshwater, degradation has been reported to range from 0 to >99% and is dependent on many variables including temperature (Staples et al. 1997). Reported removal of DEHP from aqueous systems by activated sludge biodegradation under aerobic conditions ranged from 70 to >99%, and from 0 to 90% in wastewater depending on the microbial strains present and other variables (Kurane 1986; Nasu et al. 2001; O'Grady et al. 1985; Pradeep et al. 2015; Staples et al. 1997). In spite of the many reported rapid degradation rates, DEHP has been found in sewage sludge (O'Connor 1996) and in sewage treatment plant effluents (Stubin et al. 1996), indicating that under actual sewage treatment plant conditions (which are more rigorous than environmental waters), DEHP is not always completely degraded, but rather becomes sorbed to sludge solids. Nonetheless, DEHP does not appear to be accumulating in the environment so that biodegradation

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is removing the apparent constant influx of DEHP. Under anaerobic conditions, biodegradation of DEHP is slower (O'Connor et al. 1989; Staples et al. 1997; Wams 1987).

Chemical hydrolysis of DEHP occurs too slowly to be important (Howard 1989; Staples et al. 1997). The estimated half-life for DEHP hydrolysis in water is 100 years (Wams 1987). DEHP can undergo indirect photolysis in sunlit surface waters. Yu et al. (2019) demonstrated that nitrate or ferric ions facilitated the photodegradation of DEHP via oxidation with photochemically generated hydroxyl radicals and naturally occurring fulvic acids in water also promoted the photolysis of DEHP in surface waters via indirect photolysis whereby the fulvic acids absorb photons in the environmental ultraviolet (UV) spectrum and transfer energy from their excited state directly to DEHP resulting in the photodegradation of DEHP.

Sediment and Soil. Biodegradation of DEHP also occurs in soil, but at a slower rate than in water, since adsorption onto the soil organic matter reduces the availability of DEHP for degradation (Carrara et al. 2011; Cartwright et al. 2000; Cheng and Lin 2000; Wams 1987). According to Cartwright et al. (2000), DEHP is reported to be recalcitrant in soil and, as such, is predicted to account for the majority of phthalate contamination in the environment. Many other environmental factors, in addition to soil organic content, influence the rate of DEHP biodegradation (Cartwright et al. 2000; Gejlsbjerg et al. 2001). The half-life of DEHP in a silt loam (38.6% sand, 45.0% silt, 16.4% clay, 3.8% organic carbon and pH = 6.0) ranged from 24.2 to 29.6 days (He et al. 2018). The half-life in a soil with low organic matter (38.7% sand, 44.4% silt, 16.9% clay, 0.6% organic carbon and pH = 5.8) was shown to be considerably longer, 94.1±4.3 days; however, the half-life tended to decrease as amendments such as compost and biochar were added, which increased the amount of organic matter in the soil.

In sediments, optimum degradation of DEHP occurred at high concentrations, warm temperatures, and in a nutrient-rich system (Johnson et al. 1984). Biodegradation rates in sediments, like soil, can also decrease with increasing sorption, showing that DEHP has the inherent capacity to be quickly degraded by microbes; sorption will cause longer half-lives in natural sediments (Kickham et al. 2012). Anaerobic biodegradation of DEHP in sediments was reported to occur, but more slowly than under aerobic conditions (Chang et al. 2005; Johnson et al. 1984).

5.5 LEVELS IN THE ENVIRONMENT

Reliable evaluation of the potential for human exposure to DEHP depends, in part, on the reliability of supporting analytical data from environmental samples and biological specimens. Concentrations of

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DEHP in unpolluted atmospheres and in pristine surface waters are often so low as to be near the limits of current analytical methods. In reviewing data on DEHP levels monitored or estimated in the environment, it should also be noted that the amount of chemical identified analytically is not necessarily equivalent to the amount that is bioavailable.

One problem that is encountered when reviewing the concentrations of DEHP in environmental water samples is evaluating the accuracy of the reported values of DEHP dissolved in water. Many of the concentrations of DEHP that have been reported for environmental water samples often exceed the solubility of DEHP in distilled or deionized water (Staples et al. 1997). Evaluating the values is complicated by the fact that a true solubility of DEHP in water has been difficult to determine experimentally, with values ranging between 0.0006 and 0.40 mg/L depending on the method of analysis (Staples et al. 1997). In addition, the solubility of DEHP in aqueous environmental media can be greatly affected by the types and concentration of dissolved organics in the sampled water; for example, humic substrates in landfill leachates (Staples et al. 1997). Another complication to determining the concentration of DEHP in environmental water samples is the possible introduction of DEHP from other sources (Howard et al. 1985). For example, the measurement of DEHP in water can be confounded by a number of sampling problems. Samples can be contaminated by additional amounts of DEHP contained in sampling devices and laboratory containers. Since DEHP is a common laboratory contaminant, laboratory and field blanks often show concentrations similar to those in the media under study. Sampling of water through the air-water interface can be contaminated by DEHP that is contained in surface films, due to the limited solubility of DEHP in water and a density that is slightly lower than water. Consequently, the reliability of the values that have been reported to represent the concentration of DEHP dissolved in water will have to be judged upon the quality of the sampling and analytical techniques used to measure DEHP in aqueous environmental media.

Table 5-3 shows the lowest limits of detection that are achieved by analytical analysis in environmental media. An overview summary of the range of concentrations detected in environmental media is presented in Table 5-4.

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Table 5-3. Lowest Limit of Detection Based on Standards^a

Media	Detection limit	Reference
Air	16 µg/m ³ (workplace) <1 ng/m ³ (outdoor) ^b	OSHA 1994 Thurén and Larsson 1990
Drinking water	0.46 µg/L	EPA 1995
Surface water and groundwater	0.27 µg/L	EPA 1996
Soil	27.9 µg/kg	USGS 2006
Sediment	23 µg/kg	Fernández-González et al. 2017
Whole blood	20 ng/mL	Kambia et al. 2001

^aDetection list based on using appropriate preparation and analytics. These limits may not be possible in all situations.

^bFor a sampling volume of 300–400 m³ collected at a flow rate of 4.5 m³/day.

Table 5-4. Summary of Environmental Levels of DEHP

Media	Low	High	Mean
Outdoor air (ng/m ³)	<0.4	65	5.0
Indoor air (ng/m ³)	20	240	109
Dust (g/kg)	2.38	4.10	3.24
Surface water (µg/L)	<0.002	137	0.21
Groundwater (µg/L)	Not detected	470	15.7
Drinking water (µg/L)	0.16	170	0.55
Rainwater (µg/L)	0.004	0.68	0.17
Wastewater (µg/L)	0.01	4,400	27
Sediments (µg/kg)	0.00027	218	1.4
Soil (µg/kg)	0.03	1,280	0.03
Sludge (g/kg)	0.000420	58.3	0.301

Source: NTP 2006

Detections of DEHP in air, water, and soil at NPL sites are summarized in Table 5-5.

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Table 5-5. DEHP Levels in Water, Soil, and Air of National Priorities List (NPL) Sites

Medium	Median ^a	Geometric mean ^a	Geometric standard deviation ^a	Number of quantitative measurements	NPL sites
Water (ppb)	30	45.5	12.5	319	197
Soil (ppb)	9,300	14,600	20.3	305	190
Air ($\mu\text{g}/\text{m}^3$)	0.03	0.020	2.9	4	4

^aConcentrations found in ATSDR site documents from 1981 to 2019 for 1,867 NPL sites (ATSDR 2019). Maximum concentrations were abstracted for types of environmental media for which exposure is likely. Pathways do not necessarily involve exposure or levels of concern.

5.5.1 Air

As presented in Chapter 4, DEHP has a relatively low vapor pressure, as well as a relatively high octanol/water partition coefficient and soil sorption coefficient. This combination of properties is consistent with a chemical that is found to only a limited extent in air. Nonetheless, DEHP appears to be ubiquitous in air, with urban air having somewhat higher concentrations than air in rural or uninhabited areas. Its presence in atmospheric samples removed from point source indicates that DEHP is subject to long-range transport. The monitoring studies reported below appear to have taken reasonable efforts to eliminate contamination from their analyses.

Average atmospheric concentrations reported in the literature appear to be within a relatively narrow range regardless of sampling location. DEHP has been reported over the Pacific and Atlantic Oceans at mean levels of approximately $1.4 \text{ ng}/\text{m}^3$ with a range of $0.32\text{--}2.68 \text{ ng}/\text{m}^3$ (Atlas and Giam 1981; Giam et al. 1980). Within the continental United States, DEHP levels over the Great Lakes have been reported at a mean concentration of $2.0 \text{ ng}/\text{m}^3$ with a range of $0.50\text{--}5.0 \text{ ng}/\text{m}^3$ (Eisenreich et al. 1981). However, DEHP was not among the four phthalate esters detected in industrialized areas along the Niagara River (Hoff and Chan 1987). DEHP levels near Lake Chaohu, China were reported to range from 1.229 to $14.418 \text{ ng}/\text{m}^3$ (He et al. 2019).

DEHP has also been noted in outdoor air over Portland, Oregon at a mean level of $0.39 \text{ ng}/\text{m}^3$ with a range of $0.06\text{--}0.94 \text{ ng}/\text{m}^3$ (Ligocki et al. 1985a). The mean (\pm standard deviation) concentration of DEHP in outdoor air near a residence in Contra Costa County, California, was $3.4\pm 0.4 \text{ ng}/\text{m}^3$ (Lunderberg et al. 2019). DEHP was detected in ambient air during a 7-month sampling period from a highly populated area of Mexico City, Mexico (Quintana-Belmares et al. 2018). DEHP levels ranged from 32.8 to

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175.8 $\mu\text{g/g}$ in PM_{10} particulates and from 21.5 to 229.7 $\mu\text{g/g}$ in $\text{PM}_{2.5}$ particulates. DEHP has been noted in outdoor air in Sweden at a median concentration of 2.0 ng/m^3 with a range of 0.28–77.0 ng/m^3 (Thurén and Larsson 1990). In Germany above a forest, DEHP was detected but not quantified (Helmig et al. 1990).

DEHP levels in indoor air might be higher due to slow volatilization from plastic products (Bornehag et al. 2005; EPA 1981; Wams 1987). As noted in Section 5.3.1, Cadogan et al. (1994) reported that an indoor overall emission rate of 2.3×10^{-4} mg/second-m^2 at 25 °C has been calculated for all phthalate plasticizers in products such as wall coverings, flooring, upholstery, and wire insulation.

In an effort to quantify typical indoor chemical exposures, Rudel et al. (2001) measured DEHP (and other compounds) in air samples in various occupational and residential structures. A total of seven air samples were collected from a mobile trailer (two simultaneous samples), two office buildings (two samples), a residential home (one sample), a workplace where plastics were melted (one sample), and a personal air sample collected during an 11-hour period of shopping and errands (one sample). DEHP was detected in four out of seven air samples with the highest measured level (11.5 $\mu\text{g/m}^3$; 11,500 ng/m^3) observed in the workplace where plastics were melted. Szewczyńska et al. (2020) measured levels of DEHP in the workplace air of four companies in Poland involved in the manufacture of plastics and rubber products that could emit DEHP to the air. The maximum concentrations of DEHP in the respirable (aerodynamic diameter particles of $<4 \mu\text{m}$) and inhalable fractions were 1.25 and 6.47 $\mu\text{g/m}^3$, respectively.

Additional studies have quantified residential indoor DEHP air concentrations. In the spring of 2000, DEHP concentrations in five homes located in Tokyo, Japan ranged from 0.04 to 0.06 $\mu\text{g/m}^3$, with a concentration of 0.23 $\mu\text{g/m}^3$ reported in a sixth home (Otake et al. 2001). Thirty-two homes in New York City had a mean DEHP concentration of 0.09 $\mu\text{g/m}^3$ (90 ng/m^3), measured during a 2-week period (Adibi et al. 2008). In another study, Lunderberg et al. (2019) measured DEHP levels in indoor air in a single-family residential home in Contra Costa County, California, from December 7, 2017 to February 4, 2018. The mean (\pm standard deviation) DEHP level was $0.009 \pm 0.016 \mu\text{g/m}^3$ when residents were home and $0.0014 \pm 0.0016 \mu\text{g/m}^3$ when the home was vacant, suggesting that human activities can increase the levels of airborne DEHP.

Emission of DEHP from PVC wall coverings (containing 30% phthalic esters) was measured in a test chamber at room temperature; a maximum concentration of 0.94 $\mu\text{g/m}^3$ was noted for DEHP in air over 14-day test period (Uhde et al. 2001). Increases in DEHP emissions with increasing ambient temperature

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are especially important within car interiors, where DEHP concentrations in air have been shown to range from 1 $\mu\text{g}/\text{m}^3$ at room temperature to 34 $\mu\text{g}/\text{m}^3$ at 65 °C (Uhde et al. 2001).

5.5.2 Water

DEHP has been detected infrequently (11%) in surface water, rainwater, and groundwater in the United States at concentrations generally in the low ppb ($\mu\text{g}/\text{L}$) range. DEHP was detected in drinking water concentrates from several U.S. cities (EPA 1984). Canter and Sabatini (1994) reported that the Biscayne aquifer in Florida had a maximum DEHP concentration of 8,600 $\mu\text{g}/\text{L}$, but no DEHP was detected in the municipal well fields that draw water from that aquifer. Eckel et al. (1993) also reported the presence of DEHP in the groundwater in Florida. DEHP was detected in samples from Long Island public water supply wells and groundwater collected between 1997 and 2011 at concentrations of 2.0–39 and 2.0–4.6 $\mu\text{g}/\text{L}$, respectively (NYDEC 2014). In water samples collected from private wells in close proximity to gas drilling in Pavillion, Wyoming, DEHP was detected in 15 of 41 wells at concentrations ranging from 0.15 to 9.80 $\mu\text{g}/\text{L}$ (ATSDR 2010). In an analysis of occurrence data from public water systems from the Six-Year Review of National Primary Drinking Water Regulations conducted by the EPA (2009a), DEHP was detected in 3,098 of 27,667 systems (11%) in 42 states, which collectively serve more than 45 million people at concentrations ranging from 0.05 to 250 $\mu\text{g}/\text{L}$. DEHP was detected in 460 systems at concentrations above the maximum contaminant level (MCL) of 6 $\mu\text{g}/\text{L}$, which serve a population >11 million (EPA 2009b).

DEHP was detected in 24% of 901 surface water samples recorded in the STORET database at a median concentration of 10 ppb ($\mu\text{g}/\text{L}$) (Staples et al. 1985). DEHP was also found in water samples from several U.S. rivers (DeLeon et al. 1986; Hites 1973; Sheldon and Hites 1979). Reported concentrations ranged from 0.5 to 1 ppb ($\mu\text{g}/\text{L}$). DEHP was detected at concentrations of <2,000 ng/L in surface water collected from the Fremont Creek and Sulphur Creek in Capitol Reef National Park and the Grotto and North Creek in Zion National Park in 2015 (NPS 2016). Average concentrations of DEHP in seawater ranging from 0.005 to 0.7 ppb ($\mu\text{g}/\text{L}$) have also been reported (Giam et al. 1978; McFall et al. 1985a).

DEHP was detected in petrochemical plant wastewaters and industrial landfill leachate at <0.1–30 $\mu\text{g}/\text{L}$ (Castillo et al. 1998) and in New York City municipal treatment plant effluents up to 50 $\mu\text{g}/\text{L}$ (Stubin et al. 1996). Roy (1994) reported a range of 34–7,900 $\mu\text{g}/\text{L}$ in U.S. landfill leachate.

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Bauer and Herrmann (1997) reported that DEHP was present in the leachate from various fractions of household wastes from the regions of Bayreuth and Straubling in Germany. The wastes included food waste, paper for recycling, unusable paper, cardboard, plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound packing waste, compound materials, and disposable diapers. Approximately 50 kg of these wastes were cut into 5–10 cm pieces, placed in laboratory fermenters, and then flooded with water. Stable methanogenic conditions were obtained in 3 months. Leachate from a mixture of all waste categories except food waste contained a maximum of 147 µg/L of DEHP, while leachate from a mixture of waste categories limited to plastic films, other plastics, textiles, 8–40 mm screened fraction, <8 mm screened fraction, compound materials contained a maximum of 56 µg/kg DEHP. The authors were careful to exclude inadvertent sources of phthalate esters. This report demonstrates that DEHP is present in European household waste and that it leaches from that waste to percolating water. DEHP was detected in untreated and treated wastewater and surface runoff from traffic roads in Europe (Clara et al. 2010).

5.5.3 Sediment and Soil

DEHP was detected in both marine and freshwater sediments at average levels ranging from 6.6 to 1,500 ppb. Maximum values were usually observed near industrial effluent discharge points (Fallon and Horvath 1985; Murray et al. 1981; Ray et al. 1983; Velinsky et al. 2011). One study, measuring historical contamination of sediment in the tidal Anacostia River in Washington, DC, found that DEHP concentrations were the highest in the upper 200–300 cm with a subsurface maximum of up to 7,500 ng/g dry weight, showing only a slight decrease in concentration towards the sediment-water interface (Velinsky et al. 2011). In the New York Bight (a sector of the Middle Atlantic Ridge adjoining the New York and New Jersey shorelines), which is an area containing disposal sites for dredging mud, sewage sludge, and industrial acid waste, DEHP has been measured in sediments at concentrations ranging from 0.1 to 10.1 ppm (Friedman et al. 2000). Iannuzzi et al. (1997) reported that DEHP was present in every sediment sample taken adjacent to combined sewer overflows to the Passaic River in New Jersey at concentrations between 960 and 27,000 µg/kg (a total of 40 samples). Of the 431 stream bed sediments collected from throughout the United States, 39.2% showed DEHP concentrations, with a median concentration of 180 µg/kg (the high concentration was 17,000 µg/kg) (Lopes et al. 1997). DEHP was reported in 40% of 367 sediment samples recorded in the STORET database at a median concentration of 1,000 ppb (Staples et al. 1985) and in sediments near a hazardous waste site (Hauser and Bromberg 1982).

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Current monitoring data for DEHP in soil were not located. One study measuring phthalate esters in five soils and leachate-sprayed soils from Pennsylvania and New York in the Susquehanna River basin in 1979 reported DEHP concentrations of 0.001–1.2 mg/kg (Russell and McDuffie 1983).

5.5.4 Other Media

DEHP has been found in several kinds of food. Fish and other seafood have been reported to be contaminated with concentrations ranging from 2 to 32,000 ppb (DeVault 1985; Giam and Wong 1987; Giam et al. 1975; McFall et al. 1985b; Ray et al. 1983; Stalling et al. 1973; Williams 1973). DEHP was detected in 33% of 139 biota samples (not necessarily edible) recorded in the STORET database at a median concentration of 3,000 ppb (Staples et al. 1985). DEHP has also been reported in processed canned and frozen fish in Canada at concentrations up to 160 ppb (Williams 1973).

DEHP can become an indirect additive in packaged foods due to its use in plastic wraps, heat seal coatings for metal foils, closure seals for containers, paper packaging with a plastic film, and printing inks for food wrappers and containers (Cao 2010; Gao et al. 2014). Table 5-6 summarizes the detections of DEHP in various foods and beverages. As discussed in Section 5.6, food is the primary source of DEHP exposure in the general population.

Table 5-6. Concentration of DEHP in Food

Food	Concentration of DEHP (µg/g)		
	Minimum	Maximum	Median
Beverages	0.006	1.7	0.043
Cereal	0.02	1.7	0.05
Dairy (excluding milk)	0.059	16.8	0.96
Eggs	<0.01	0.6	0.12
Fats and oils	0.7	11.9	2.4
Fish	0.00005	32.0 ^a	0.001
Fruits	<0.02	0.11	0.02
Grains	<0.1	1.5	0.14
Meat, not processed	<0.01	0.8	0.05
Milk	<0.005	1.4	0.035
Nuts and beans	<0.08	0.8	0.045
Poultry	0.05	2.6	0.9
Processed meat	<0.1	4.32	0.45
Vegetables	0.0098	2.2	0.048
Infant formula, powdered	<0.012	0.98	0.12

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Table 5-6. Concentration of DEHP in Food

Food	Concentration of DEHP ($\mu\text{g/g}$)		
	Minimum	Maximum	Median
Infant formula, liquid	<0.005	0.15	0.006
Baby food	0.01	0.6	0.12
Other food	<0.01	25	0.05

^aFrom DeVault 1985.

DEHP = di(2-ethylhexyl)phthalate

Source: NTP 2006

Serrano et al. (2014) reviewed 17 studies measuring phthalate concentrations in United States and international foods and found DEHP levels in poultry, cooking oils, and cream-based dairy products often exceeded $\geq 300 \mu\text{g/kg}$ ($0.300 \mu\text{g/g}$). DEHP was detected in 74% of 72 individual food samples purchased in Albany, New York (Schechter et al. 2013). The mean and median values of DEHP in these food items are provided in Table 5-7.

Table 5-7. Mean and Median Values of DEHP in Food

Food item	Mean ^a ($\mu\text{g/g}$)	Median ($\mu\text{g/g}$)
Beverages	0.00385	0.00189
Milk	0.0486	0.0486
Other dairy	0.144	0.0928
Fish	0.0317	0.0396
Fruit/vegetables	0.00625	0.00185
Grain	0.0616	0.0506
Beef	0.00185	0.00185
Pork	0.300	0.0206
Poultry	0.0186	0.0148
Meat and meat products	0.101	0.007
Vegetable oils	0.117	0.0489
Condiments	0.0304	0.0206
Infant food	0.0751	0.0294

^aMean values were calculated substituting one-half the limit of detection for each non-detect.

Source: Schechter et al. 2013

In addition to fish (discussed above), DEHP has been detected in such foods as milk, cheese, meat, margarine, eggs, cereal products, baby food, and infant formula (Cerbulis and Byler 1986; EPA 1981;

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Petersen and Breindahl 2000). Most samples contained <1 ppm DEHP, but fatty foods had higher levels. Combined data from Europe, North America, and Asia show that the foods with the highest DEHP concentrations were animal fats, spices, and nut/nut spreads (Wormuth et al. 2006). Although one study found that levels of DEHP in fatty foods such as milk, cheese, and meat did not differ significantly from background levels (CMA 1986), high levels of DEHP in "blank" samples and other analytical problems indicate that laboratory contamination might have confounded the results. Chocolate bars contained DEHP at levels up to 2.4 ppm (Castle et al. 1989).

DEHP has also been detected in beverages. DEHP was detected in soft drinks at concentrations ranging from 0.03 to 3.50 ng/L and in different types of milk powder at levels up to 25.1 µg/kg (Khedr 2013). DEHP has been detected in 61.7% of bottled water tested from 21 countries across the world. The mean concentration worldwide was 3.42 ± 8.94 µg/L, with a maximum concentration of 94.1 µg/L (Luo et al. 2018). Military packaged water, filled in polyethylene terephthalate bottles in Afghanistan, contained a maximum DEHP concentration of 0.6 µg/L (Greifenstein et al. 2013). The maximum allowable limit for DEHP in bottled water in the United States is 6 µg/L (FDA 2016). Based on the survey by Luo et al. (2018), 14.2% of the 379 brands of bottled water tested worldwide contained DEHP at levels above the U.S. maximum allowable limit. Countries with the highest average levels were Thailand (61.1 µg/L), Croatia (8.8 µg/L), Czech Republic (6.3 µg/L), Saudi Arabia (6.2 µg/L), and China (6.1 µg/L).

DEHP has been detected in indoor dust samples. In an effort to quantify typical indoor chemical exposures, Rudel et al. (2001) measured DEHP (and other compounds) in dust air samples in various occupational and residential structures. A total of six dust samples were collected from an office building (one sample) and three residential homes (five samples). DEHP was detected in all dust samples, with concentrations ranging from 69.4 to 524 µg/g dust and a mean concentration of 315 µg/g dust. Øie et al. (1997) reported that sedimented dust samples from 38 dwellings in Oslo, Norway contained an average of 640 µg/mg sedimented dust (100–1,610 µg/g), while suspended particulate matter from six dwellings contained an average of 600 µg/g (240–940 µg/g). In a study of 390 homes in Sweden, DEHP was found in nearly all dust samples collected (99.1%) from 346 children's bedrooms at mean and median concentrations of 1.31 and 0.77 mg/g dust, respectively (Bornehag et al. 2005). DEHP was detected in 99% of house dust samples collected from 167 homes in California between 2010 and 2011 at a median concentration of 187 µg/g dust (Philippat et al. 2015).

Blood products available for transfusions might be contaminated with DEHP due to leaching from the plastic equipment used to collect and store the blood. The concentration of DEHP increases with storage

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time (Inoue et al. 2005). Reported concentrations of DEHP in blood products stored in PVC bags are: whole blood (2–620 ppm); platelet concentrates (23.4–267 ppm); red cell concentrates (4.3–152 ppm); and plasma (4.3–1,230 ppm) (Ching et al. 1981; Cole et al. 1981; Contreras et al. 1974; Dine et al. 1991; FDA 2001; Inoue et al. 2005; Jaeger and Rubin 1972; Loff et al. 2000; NTP 2000; Rock et al. 1978; Shintani 2000; Sjöberg et al. 1985c; Vessman and Rietz 1974). DEHP was also detected in intravenous fluids, such as saline and glucose, used for parenteral therapy of hospitalized patients, at levels ranging from 9 to 13 ppb (Ching et al. 1981). Karle et al. (1997) reported that DEHP concentrations at the end of the blood prime in ECMO circuits in an *in vitro* study had mean values of 18.3, 21.8, and 19.3 µg/mL for different circuits and was dependent on the surface area of each circuit. After 3 days, DEHP concentrations in infants averaged 4.9±4.0 µg/mL. Shneider et al. (1991) reported that serum DEHP concentrations varied depending on the nature of the treatment. They reported serum DEHP concentrations ranges of 1.1–5.1 µg/mL for infant cardiopulmonary bypass, 0.4–4.2 µg/mL for pediatric hemodialysis, 5.4–21.5 µg/mL for exchange transfusion, and 18–98 µg/mL for ECMO. Newer circuits using other plasticizers such as trioctyltrimellitate (TOTM) have been shown to reduce exposure to DEHP; however, PVC priming bags that use DEHP as a plasticizer may still result in exposure (Fernandez-Canal et al. 2018).

DEHP was the most common plasticizer in soft PVC products intended for children until the early 1980s and these products may have contained low levels of DEHP. For example, DEHP was detected in four commercial pacifiers at concentrations of 31–42% by weight (Lay and Miller 1987). However, the use of DEHP in domestically produced pacifiers, teething rings, and rattles has been discontinued (CPSC 1999). Yet, some PVC toys manufactured in a small number of foreign countries have been reported to contain up to 11–19% DEHP (Stringer et al. 2000). In 2008, the Consumer Products Safety Commission (CPSC) tested 63 children's plastic toys purchased in the United States, 38 of which were composed of PVC (Babich et al. 2020). DEHP was detected in only 1 out these 38 toys. DEHP was detected above 0.1% in 11 out of 118 samples obtained from PVC composed children's toys in Switzerland (McCombie et al. 2017).

As presented in Section 5.5.2 above, Bauer and Herrmann (1997) reported that mixed household waste contained DEHP. Table 5-8 summarizes the concentration of DEHP detected in various categories of waste. The authors also calculated that 177.5–1,469.5 mg/kg DEHP was present in the waste on a dry-weight basis and constituted the most commonly found phthalate ester, constituting 91.9–93.3% of the total phthalates found in the waste.

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Table 5-8. Concentration DEHP in Categories of Household Waste

Waste fraction	Concentration of DEHP (mg/kg) ^a		
	Mean	Minimum	Maximum
Food waste	64.3	4.8	334.7
8–40 mm Fraction	1,259.1	584.9	2,253.5
<8 mm Fraction	95.5	76.1	132.5
Paper for recycling	29.7	10.0	60.3
Unusable paper	71.1	41.4	106.4
Cardboard	47.4	10.1	70.5
Plastic films	444.9	169.0	907.9
Other plastics	1,027.6	373.8	2,035.3
Textiles	205.7	14.9	686.1
Compound packing waste	151.9	57.7	393.7
Compound materials	16,820.6	7,862.4	26,352.0
Disposable diapers ^b	74.1	14.2	322.2

^aResults are from six extractions except “compound material” for which the results are for nine extractions.

^bDescribed as “nappies” in the original paper.

Source: Bauer and Herrmann 1997

5.6 GENERAL POPULATION EXPOSURE

The general population is exposed to DEHP via oral, dermal, and inhalation routes of exposure. DEHP is present in environmental media and in numerous consumer articles that are used world-wide (Section 5.2.3). Biomonitoring data suggest that 95% of the U.S. population is exposed to DEHP based on detectible levels of DEHP metabolites in urine (Kato et al. 2004). Estimates of the average total daily individual ambient exposure to DEHP in the United States have ranged from 0.21 to 2.1 mg/day (Doull et al. 1999; Huber et al. 1996; Tickner et al. 2001; Zolfaghari et al. 2014). These estimates do not include workplace air exposures or exposures to DEHP off gassing from building materials. In the United States, estimated DEHP exposures for different age groups, reported in $\mu\text{g}/\text{kg}$ body weight/day, were 5.0–7.3 (0–0.5 year), 25.8 (0.6–4 years), 18.9 (5–11 years), 10 (12–19 years), and 8.2 (20–70 years) (Clark et al. 2011). Some of the information presented might not represent current exposures, since there have been recent changes in the use patterns for DEHP; specific examples are discussed in Section 5.2.3.

The National Health and Nutrition Examination Survey (NHANES) periodically uses biomonitoring to provide estimates of exposure to the civilian U.S. population. Chemicals and their metabolites are measured in subsets of participants aged 6–59 years old, meant to be a representative sample of the population. Urine measurements are reported as both the concentration in urine and the concentration

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corrected for urine-creatinine level, which adjusts for urine dilution. Urinary levels of DEHP metabolites, including MEHP, MEHHP, MEOHP, and MECPP, were measured in several NHANES programs assessing exposure to subsets of the general population in the United States from 1999 to 2012 (CDC 2015). MEHP, the primary metabolite of DEHP, formed by hydrolysis, represents only approximately 6% of the total amount of DEHP metabolites excreted through urine. MEHHP, MEOHP, and MECPP, the secondary metabolites of DEHP formed from the metabolism of MEHP, represent approximately 70% of DEHP metabolites in urine, and can be present in amounts roughly 3–5 times higher than MEHP (CDC 2015; TURI 2006). The NHANES results for 1999–2014 are summarized in Tables 5-9, 5-10, 5-11, 5-12, 5-13, 5-14, 5-15, and 5-16 (CDC 2018). Urinary levels were generally higher in women than men and in children than adults. However, urinary levels for all metabolites have shown an overall decrease by approximately 2-fold or greater between 1999 and 2014 for age, gender, and ethnicity that represent a broad mix of the general public, indicating that regulations to reduce general population exposure to DEHP (CDC 2018; CPSIA 2008) may be effective. Still, these findings indicate widespread exposure among the general U.S. population; however, no correlation of these measurements with actual DEHP intake has yet been determined.

Hines et al. (2009a) explored the relationship of phthalate metabolites, including those of DEHP, in urine, serum, saliva, and breast milk and potential routes of exposure using samples collected from 33 lactating mothers in North Carolina; however, phthalates were detected in <50% of the samples collected across matrices, so a correlation could not be made. Only 2% of saliva samples contained detectable levels of DEHP metabolite MECPP (2.3 µg/L). Serum and urine samples contained detectable levels of DEHP metabolites (only MECPP for serum) at >50% of samples. Median concentrations for collective DEHP metabolites in urine samples ranged from 3.6 to 36.8 µg/g creatinine and mean concentrations of MECPP detected in plasma were 2.0–2.3 µg/L. Using an exposure questionnaire, the authors found an inverse correlation with the age of the primary car driven by participants and the urinary concentration of metabolites. This study is limited by the small sample size and low detection rate.

The predominant source of DEHP exposure to the general population by the oral route is through the diet (Doull et al. 1999; Gong et al. 2014; Huber et al. 1996; NTP 2000; Wormuth et al. 2006). Clark et al. (2011) reported that ingestion of food accounts for approximately 95% of total exposure for the toddler through adult age range. Similarly, up to 90% of the daily intake of DEHP in European children and adults is attributed to food consumption (Wormuth et al. 2006). Dietary contribution to the total daily DEHP intake is less in infants and toddlers, approximately 50%, due to differences in dietary patterns (Wormuth et al. 2006). A study in Germany (Koch et al. 2013) found that urinary DEHP metabolites in

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Table 5-9. Uncorrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Total	1999–2000	3.43 (3.19–3.69)	3.20 (3.00–3.60)	7.60 (6.80–8.40)	14.9 (13.5–17.4)	23.8 (19.2–28.6)	2,541
	2001–2002	4.27 (3.80–4.79)	4.20 (3.70–4.90)	9.80 (8.40–11.6)	23.0 (19.1–27.9)	39.2 (31.8–50.0)	2,782
	2003–2004	2.34 (2.10–2.62)	1.90 (1.70–2.40)	5.30 (4.50–6.60)	15.1 (11.4–20.6)	31.0 (21.4–42.0)	2,605
	2005–2006	3.04 (2.78–3.32)	2.50 (2.10–2.80)	6.30 (5.70–7.10)	17.7 (14.0–22.5)	39.7 (28.6–52.1)	2,548
	2007–2008	2.64 (2.29–3.05)	2.20 (1.80–2.50)	5.40 (4.30–6.90)	14.1 (11.2–20.2)	27.6 (19.8–39.8)	2,604
	2009–2010	1.59 (1.41–1.79)	1.51 (1.33–1.71)	3.52 (2.99–4.00)	7.54 (5.96–9.54)	14.1 (9.91–21.1)	2,749
	2011–2012	1.36 (1.25–1.49)	1.40 (1.20–1.50)	3.00 (2.70–3.30)	6.00 (5.30–6.40)	8.70 (7.60–9.70)	2,489
	2013–2014	Not calculated ^b	1.10 (0.900–1.20)	2.30 (2.10–2.60)	4.40 (4.00–5.00)	6.30 (5.60–7.10)	2,685
Age group							
6–11 years	1999–2000	5.12 (4.42–5.92)	4.90 (3.70–6.40)	11.1 (8.30–13.6)	19.0 (13.8–36.1)	35.3 (15.6–130)	328
	2001–2002	4.41 (3.90–5.00)	4.40 (4.10–5.30)	9.30 (7.90–11.7)	19.7 (14.6–25.9)	31.4 (21.8–47.9)	393
	2003–2004	2.84 (2.10–3.84)	2.70 (1.80–4.10)	6.40 (4.40–9.60)	13.9 (7.80–27.6)	27.6 (11.3–64.7)	342
	2005–2006	3.10 (2.78–3.47)	3.00 (2.60–3.30)	6.20 (5.10–7.10)	14.1 (9.40–19.3)	19.7 (14.7–36.6)	356
	2007–2008	2.39 (2.05–2.80)	2.20 (1.70–2.90)	4.50 (3.70–6.20)	8.70 (6.40–13.9)	15.1 (10.6–24.1)	389
	2009–2010	1.64 (1.45–1.85)	1.71 (1.26–2.02)	3.50 (3.09–3.94)	5.95 (4.56–7.56)	8.92 (6.94–12.9)	415
	2011–2012	1.41 (1.23–1.61)	1.50 (1.20–1.80)	2.90 (2.50–3.40)	5.30 (4.10–7.10)	7.60 (6.30–8.80)	396
	2013–2014	1.44 (1.24–1.66)	1.20 (1.00–1.50)	2.70 (2.20–3.20)	5.20 (3.70–8.50)	8.70 (5.20–11.8)	409
12–19 years	1999–2000	3.75 (3.24–4.35)	3.70 (2.90–4.60)	8.10 (6.40–9.40)	15.3 (11.4–20.5)	22.8 (19.1–29.2)	752
	2001–2002	4.57 (3.96–5.27)	4.50 (3.70–5.10)	11.0 (9.50–14.4)	23.0 (17.7–32.7)	42.5 (25.9–57.5)	742
	2003–2004	2.77 (2.25–3.41)	2.50 (2.00–3.00)	6.40 (4.50–8.60)	18.6 (10.2–35.6)	40.6 (20.7–58.4)	729
	2005–2006	3.72 (3.04–4.56)	3.20 (2.40–4.10)	8.80 (6.20–13.3)	22.6 (13.8–43.4)	48.7 (23.1–62.9)	702
	2007–2008	2.99 (2.39–3.75)	2.30 (1.80–2.70)	6.00 (4.40–9.90)	21.1 (11.8–32.8)	37.6 (24.8–74.1)	401
	2009–2010	1.82 (1.52–2.16)	1.66 (1.42–1.94)	3.98 (3.35–4.73)	9.53 (6.27–14.0)	17.6 (9.54–27.4)	420
	2011–2012	1.58 (1.33–1.87)	1.50 (1.00–2.30)	3.90 (3.10–4.40)	6.80 (5.20–10.3)	12.5 (8.90–14.3)	388
	2013–2014	1.43 (1.26–1.62)	1.20 (1.00–1.40)	2.60 (2.00–3.50)	4.90 (4.20–5.80)	8.30 (5.50–10.7)	462
≥20 years	1999–2000	3.21 (2.94–3.51)	3.00 (2.70–3.40)	7.30 (6.40–8.00)	14.5 (12.1–17.0)	22.7 (17.5–27.0)	1,461
	2001–2002	4.20 (3.63–4.86)	4.10 (3.50–5.00)	9.50 (8.10–11.9)	23.5 (18.0–29.8)	39.5 (30.3–57.1)	1,647
	2003–2004	2.23 (2.03–2.44)	1.70 (1.50–2.00)	5.10 (4.50–6.00)	15.1 (10.9–19.7)	29.5 (20.4–40.0)	1,534
	2005–2006	2.94 (2.68–3.21)	2.30 (1.90–2.70)	6.20 (5.60–6.70)	17.7 (13.6–24.5)	41.5 (28.6–54.1)	1,490
	2007–2008	2.62 (2.27–3.02)	2.10 (1.80–2.50)	5.40 (4.20–7.10)	14.4 (11.2–20.2)	27.3 (19.2–40.6)	1,814
	2009–2010	1.55 (1.36–1.78)	1.44 (1.25–1.68)	3.40 (2.88–4.03)	7.39 (5.94–9.58)	14.6 (9.91–21.1)	1,914
	2011–2012	1.33 (1.19–1.48)	1.30 (1.10–1.50)	2.90 (2.50–3.20)	5.90 (5.10–6.40)	8.30 (7.10–9.60)	1,705
	2013–2014	Not calculated ^b	1.00 (0.900–1.20)	2.30 (2.00–2.50)	4.30 (3.80–4.90)	6.10 (5.40–6.80)	1,814

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Table 5-9. Uncorrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	1999–2000	3.68 (3.31–4.10)	3.40 (2.90–3.90)	8.00 (7.40–8.80)	16.0 (14.0–19.0)	25.3 (19.5–36.7)	1,215
	2001–2002	4.31 (3.84–4.83)	4.30 (3.70–5.10)	9.70 (8.30–11.2)	23.0 (16.9–29.8)	37.9 (29.9–48.4)	1,371
	2003–2004	2.56 (2.26–2.90)	2.20 (1.70–2.60)	6.00 (4.60–7.70)	17.2 (11.3–26.3)	33.3 (24.9–55.5)	1,250
	2005–2006	3.40 (3.01–3.85)	2.80 (2.40–3.30)	7.00 (5.70–9.00)	22.6 (15.0–35.8)	49.8 (35.2–67.4)	1,270
	2007–2008	2.77 (2.35–3.27)	2.30 (1.90–2.80)	5.50 (4.20–7.60)	14.4 (11.2–20.5)	28.9 (19.2–40.0)	1,294
	2009–2010	1.83 (1.63–2.05)	1.77 (1.63–1.91)	3.97 (3.49–4.51)	8.63 (6.94–11.9)	18.0 (12.6–29.0)	1,399
	2011–2012	1.51 (1.33–1.70)	1.50 (1.20–1.80)	3.10 (2.60–3.90)	6.10 (5.50–7.00)	9.20 (7.70–11.3)	1,259
	2013–2014	1.29 (1.17–1.41)	1.10 (1.00–1.30)	2.30 (2.00–2.70)	4.20 (3.50–5.00)	5.70 (5.10–6.60)	1,285
Females	1999–2000	3.21 (2.91–3.54)	3.10 (2.80–3.50)	7.10 (5.90–8.50)	13.6 (12.1–17.2)	21.9 (15.6–28.5)	1,326
	2001–2002	4.23 (3.67–4.86)	4.10 (3.50–5.00)	9.80 (8.40–12.2)	23.0 (19.5–28.4)	43.5 (31.4–53.7)	1,411
	2003–2004	2.15 (1.92–2.42)	1.80 (1.50–2.10)	4.90 (4.10–5.70)	13.2 (10.0–18.1)	27.8 (17.5–40.7)	1,355
	2005–2006	2.72 (2.49–2.98)	2.10 (1.90–2.40)	6.00 (5.20–6.80)	13.9 (11.7–17.5)	30.8 (21.9–36.2)	1,278
	2007–2008	2.52 (2.18–2.92)	2.00 (1.70–2.40)	5.10 (4.20–6.30)	14.1 (9.50–21.4)	26.4 (19.2–42.1)	1,310
	2009–2010	1.39 (1.21–1.60)	1.30 (1.12–1.53)	3.00 (2.51–3.62)	6.48 (5.45–8.09)	10.3 (8.33–14.9)	1,350
	2011–2012	1.24 (1.14–1.34)	1.20 (1.00–1.40)	2.90 (2.50–3.10)	5.60 (4.90–6.30)	8.10 (6.80–9.20)	1,230
	2013–2014	Not calculated ^b	1.00 (0.900–1.10)	2.40 (2.10–2.60)	4.80 (4.20–5.30)	7.00 (5.70–8.70)	1,400
Race/ethnicity							
Mexican Americans	1999–2000	3.49 (3.16–3.85)	3.50 (3.10–3.90)	7.00 (5.70–8.60)	13.3 (10.7–18.7)	23.9 (17.4–27.3)	814
	2001–2002	4.32 (3.75–4.98)	4.70 (3.80–5.70)	10.1 (8.50–11.4)	19.6 (16.6–23.0)	28.5 (24.2–39.9)	677
	2003–2004	2.35 (1.87–2.96)	2.20 (1.50–3.00)	5.10 (4.30–6.60)	11.2 (7.50–16.5)	18.5 (11.6–38.2)	652
	2005–2006	2.99 (2.50–3.57)	2.30 (1.70–3.30)	5.70 (4.70–7.60)	18.4 (12.1–30.6)	36.4 (26.2–63.8)	637
	2007–2008	2.89 (2.38–3.50)	2.60 (2.00–3.10)	5.30 (4.40–8.20)	16.9 (10.3–27.3)	30.2 (22.9–34.3)	531
	2009–2010	2.08 (1.84–2.36)	2.10 (1.86–2.40)	4.44 (3.73–5.63)	9.67 (6.89–16.0)	17.9 (13.1–24.3)	566
	2011–2012	1.49 (1.16–1.91)	1.50 (1.00–1.90)	3.40 (2.50–4.30)	6.60 (5.10–8.90)	9.60 (6.60–12.9)	316
	2013–2014	1.55 (1.35–1.78)	1.50 (1.20–1.70)	2.90 (2.20–3.80)	5.60 (4.20–7.00)	7.40 (6.30–9.30)	438
Non- Hispanic blacks	1999–2000	4.82 (3.92–5.93)	5.20 (4.10–5.80)	9.50 (7.60–11.4)	19.5 (12.9–26.5)	29.5 (18.6–60.3)	603
	2001–2002	6.60 (5.57–7.82)	6.70 (5.40–8.10)	15.4 (13.0–18.7)	32.9 (26.5–41.4)	52.6 (41.0–84.0)	703
	2003–2004	3.61 (3.07–4.23)	3.50 (3.00–4.00)	8.50 (7.10–11.4)	22.9 (16.5–28.6)	35.2 (29.3–49.1)	699
	2005–2006	4.09 (3.51–4.75)	3.70 (3.10–4.10)	9.10 (6.90–11.9)	22.5 (17.4–40.7)	59.3 (34.5–86.7)	678
	2007–2008	3.30 (2.98–3.64)	3.20 (2.70–3.60)	7.20 (6.30–8.90)	15.3 (13.3–20.2)	25.5 (19.2–38.1)	597
	2009–2010	2.08 (1.79–2.41)	2.05 (1.70–2.42)	4.79 (3.80–5.95)	10.1 (7.50–14.4)	16.4 (9.60–38.4)	516
	2011–2012	1.89 (1.67–2.13)	2.00 (1.60–2.40)	4.00 (3.40–4.90)	8.00 (6.20–9.70)	11.3 (9.60–14.9)	665
	2013–2014	1.63 (1.39–1.92)	1.50 (1.10–2.00)	3.30 (2.50–4.00)	5.80 (5.10–6.70)	8.10 (6.50–10.5)	609

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Table 5-9. Uncorrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Non-Hispanic whites	1999–2000	3.16 (2.89–3.46)	2.80 (2.50–3.10)	7.40 (6.30–8.40)	14.5 (12.2–17.4)	22.4 (16.9–28.5)	912
	2001–2002	3.85 (3.37–4.40)	3.70 (3.10–4.40)	8.70 (7.80–9.90)	20.9 (17.3–25.9)	37.9 (29.9–49.5)	1,216
	2003–2004	2.14 (1.92–2.39)	1.70 (1.40–1.90)	4.80 (4.00–5.80)	13.6 (9.50–20.0)	31.0 (18.1–48.9)	1,088
	2005–2006	2.83 (2.59–3.10)	2.20 (1.80–2.60)	5.90 (5.30–6.90)	17.0 (13.3–21.6)	36.3 (26.6–51.0)	1,038
	2007–2008	2.44 (2.07–2.88)	2.00 (1.70–2.30)	4.90 (3.70–6.20)	13.1 (8.70–20.1)	25.0 (15.8–42.1)	1,077
	2009–2010	1.41 (1.22–1.62)	1.30 (1.12–1.57)	3.04 (2.53–3.60)	6.12 (5.23–7.65)	11.8 (7.63–21.1)	1,206
	2011–2012	1.21 (1.07–1.37)	1.10 (.900–1.50)	2.70 (2.20–3.10)	4.90 (4.20–6.10)	6.70 (6.00–8.30)	813
	2013–2014	Not calculated ^b	0.900 (0.800–1.00)	2.00 (1.80–2.30)	3.90 (3.20–4.30)	5.50 (4.80–6.30)	987
All Hispanics	2011–2012	1.61 (1.40–1.83)	1.70 (1.30–2.00)	3.90 (3.10–4.70)	7.30 (6.30–8.70)	11.6 (9.20–12.9)	571
	2013–2014	1.55 (1.40–1.73)	1.50 (1.20–1.70)	2.90 (2.50–3.40)	5.20 (4.40–6.30)	7.20 (6.20–9.20)	690
Asians	2011–2012	1.69 (1.44–1.98)	1.70 (1.30–1.90)	3.60 (2.80–4.40)	7.80 (6.60–11.4)	13.8 (10.0–17.8)	352
	2013–2014	Not calculated ^b	1.00 (<LOD–1.20)	2.10 (1.80–2.80)	4.30 (3.50–6.10)	7.90 (4.90–10.5)	289

^aThe limit of detection for survey years 1999–2000, 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.2, 1.0, 0.9, 1.2, 1.1, 0.5, 0.5, and 0.8 µg/L, respectively.

^bNot calculated: the proportion of results below limit of detection was too high to provide a valid result.

CI = confidence interval; MEHP = mono-(2-ethylhexyl)phthalate; LOD = limit of detection; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-10. Creatinine-Corrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Total	1999–2000	3.12 (2.95–3.31)	3.08 (2.82–3.27)	5.88 (5.38–6.25)	10.8 (9.62–12.5)	18.9 (15.0–21.8)	2,541
	2001–2002	4.00 (3.58–4.48)	3.90 (3.44–4.47)	7.94 (7.22–9.02)	18.0 (15.3–21.5)	32.8 (25.2–42.9)	2,782
	2003–2004	2.20 (2.01–2.41)	1.89 (1.68–2.19)	4.31 (3.84–4.74)	10.8 (8.72–13.8)	25.4 (16.7–34.7)	2,605
	2005–2006	2.96 (2.72–3.23)	2.61 (2.37–2.86)	5.69 (5.00–6.47)	13.7 (11.4–17.8)	30.1 (21.2–35.2)	2,548
	2007–2008	2.66 (2.37–2.99)	2.36 (2.11–2.67)	5.15 (4.35–6.00)	11.8 (8.89–15.6)	21.9 (14.6–33.4)	2,604
	2009–2010	1.66 (1.45–1.90)	1.52 (1.36–1.77)	3.09 (2.69–3.60)	6.28 (4.98–8.48)	11.2 (8.33–19.1)	2,749
	2011–2012	1.55 (1.43–1.68)	1.46 (1.33–1.67)	2.73 (2.50–2.92)	4.91 (4.55–5.52)	8.47 (6.72–9.86)	2,487
	2013–2014	Not calculated ^b	1.21 (1.11–1.33)	2.19 (2.05–2.38)	3.82 (3.46–4.15)	5.18 (4.66–5.74)	2,684
Age group							
6–11 years	1999–2000	5.19 (4.55–5.93)	5.37 (4.52–5.95)	9.11 (8.06–11.4)	21.6 (11.5–41.9)	41.9 (13.5–86.2)	328
	2001–2002	5.03 (4.47–5.65)	5.38 (4.51–6.21)	9.90 (7.87–11.5)	21.1 (13.8–28.8)	31.4 (24.3–40.7)	393
	2003–2004	3.00 (2.30–3.93)	2.80 (1.93–4.09)	5.86 (4.69–7.70)	14.3 (8.54–24.4)	28.7 (14.1–45.3)	342
	2005–2006	3.42 (3.08–3.79)	3.26 (2.63–3.92)	6.18 (5.40–6.85)	11.3 (8.96–17.4)	20.7 (11.3–31.8)	356
	2007–2008	2.95 (2.49–3.49)	2.80 (2.17–3.33)	5.42 (3.95–6.51)	10.6 (7.47–14.0)	15.6 (10.6–23.7)	389
	2009–2010	2.13 (1.90–2.40)	2.08 (1.88–2.33)	3.69 (3.13–4.20)	5.83 (4.80–7.95)	8.89 (5.88–20.1)	415
	2011–2012	2.02 (1.81–2.25)	2.07 (1.75–2.44)	3.45 (2.82–4.06)	5.26 (4.60–5.89)	7.15 (5.89–8.17)	395
	2013–2014	1.81 (1.57–2.09)	1.76 (1.53–2.01)	3.13 (2.59–3.86)	6.33 (4.02–8.32)	8.32 (5.53–14.1)	409
12–19 years	1999–2000	2.53 (2.14–2.99)	2.35 (2.05–2.76)	5.83 (4.38–6.29)	9.66 (7.41–11.5)	12.1 (10.5–17.3)	752
	2001–2002	3.53 (3.09–4.03)	3.67 (2.89–4.48)	7.47 (6.51–8.67)	15.2 (11.7–21.9)	25.2 (17.7–32.8)	742
	2003–2004	2.07 (1.74–2.48)	1.88 (1.60–2.23)	4.25 (3.19–5.62)	11.6 (6.83–23.2)	24.8 (11.6–37.9)	729
	2005–2006	2.77 (2.27–3.38)	2.43 (2.03–2.87)	5.24 (4.06–7.75)	15.2 (9.86–23.2)	27.1 (16.0–43.7)	702
	2007–2008	2.33 (1.90–2.86)	2.00 (1.67–2.57)	4.33 (3.55–6.23)	12.6 (8.31–16.3)	21.9 (12.0–45.7)	401
	2009–2010	1.46 (1.20–1.77)	1.33 (1.09–1.61)	2.85 (2.24–3.63)	7.47 (4.31–11.6)	13.2 (8.11–20.9)	420
	2011–2012	1.53 (1.33–1.78)	1.40 (1.12–1.88)	2.79 (2.20–3.88)	5.00 (4.11–6.94)	9.86 (5.00–11.1)	388
	2013–2014	1.16 (1.03–1.31)	1.15 (1.00–1.27)	1.88 (1.67–2.10)	3.35 (2.38–4.07)	4.79 (3.61–6.76)	462
≥20 years	1999–2000	3.03 (2.83–3.25)	2.98 (2.73–3.23)	5.55 (4.90–6.06)	10.0 (8.60–12.9)	17.5 (13.8–22.1)	1,461
	2001–2002	3.97 (3.49–4.52)	3.82 (3.26–4.38)	7.79 (7.00–9.00)	18.3 (15.3–21.8)	34.5 (23.1–47.9)	1,647
	2003–2004	2.14 (1.98–2.31)	1.84 (1.63–2.08)	4.14 (3.78–4.40)	10.5 (8.38–12.9)	25.6 (15.9–36.3)	1,534
	2005–2006	2.94 (2.69–3.23)	2.60 (2.36–2.83)	5.67 (4.77–6.52)	13.8 (11.3–18.1)	33.1 (21.9–47.4)	1,490
	2007–2008	2.69 (2.39–3.02)	2.36 (2.14–2.66)	5.20 (4.38–6.04)	11.8 (8.94–16.6)	22.1 (13.5–37.1)	1,814
	2009–2010	1.65 (1.43–1.90)	1.51 (1.34–1.75)	3.04 (2.63–3.60)	6.24 (4.98–8.61)	11.1 (8.03–19.4)	1,914
	2011–2012	1.51 (1.39–1.64)	1.46 (1.28–1.58)	2.59 (2.38–2.84)	4.81 (4.35–5.51)	8.49 (6.18–11.2)	1,704
	2013–2014	Not calculated ^b	1.19 (1.08–1.31)	2.19 (1.98–2.41)	3.70 (3.25–4.09)	5.00 (4.34–5.40)	1,813

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Table 5-10. Creatinine-Corrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	1999–2000	2.89 (2.60–3.22)	2.76 (2.52–2.96)	5.58 (4.71–6.08)	10.3 (9.35–12.4)	21.6 (14.1–27.7)	1,215
	2001–2002	3.50 (3.08–3.99)	3.33 (2.83–3.90)	7.00 (6.49–7.77)	16.2 (12.8–20.9)	31.6 (20.5–49.4)	1,371
	2003–2004	2.01 (1.82–2.21)	1.71 (1.46–1.89)	4.14 (3.49–4.81)	10.4 (7.68–16.2)	23.3 (15.1–41.1)	1,250
	2005–2006	2.73 (2.43–3.07)	2.30 (2.12–2.61)	5.08 (4.29–6.14)	14.3 (11.2–20.5)	31.0 (21.5–50.9)	1,270
	2007–2008	2.33 (2.03–2.68)	2.00 (1.71–2.34)	4.36 (3.71–5.41)	11.6 (8.33–14.2)	20.2 (14.6–26.3)	1,294
	2009–2010	1.64 (1.45–1.85)	1.52 (1.39–1.74)	3.06 (2.70–3.60)	6.53 (5.18–9.49)	13.0 (8.65–22.2)	1,399
	2011–2012	1.41 (1.28–1.56)	1.36 (1.19–1.58)	2.52 (2.21–2.83)	4.58 (3.77–5.46)	7.19 (6.16–8.79)	1,258
	2013–2014	1.08 (0.984–1.19)	1.03 (0.950–1.12)	1.78 (1.58–2.00)	3.17 (2.73–3.60)	4.30 (3.94–5.06)	1,284
Females	1999–2000	3.36 (3.11–3.63)	3.33 (2.91–3.80)	6.15 (5.55–6.77)	11.1 (9.11–14.0)	17.3 (12.4–24.6)	1,326
	2001–2002	4.54 (4.02–5.13)	4.47 (3.85–5.14)	9.28 (7.94–10.3)	20.3 (16.6–24.4)	34.7 (27.1–42.0)	1,411
	2003–2004	2.40 (2.15–2.69)	2.16 (1.84–2.40)	4.40 (3.97–4.89)	10.9 (8.27–16.0)	27.0 (17.5–34.6)	1,355
	2005–2006	3.20 (2.89–3.55)	2.89 (2.58–3.17)	6.07 (5.00–7.00)	13.3 (10.4–16.3)	28.2 (18.2–37.4)	1,278
	2007–2008	3.02 (2.70–3.38)	2.76 (2.36–3.02)	5.57 (4.90–6.50)	12.1 (8.64–18.2)	24.7 (14.8–44.4)	1,310
	2009–2010	1.68 (1.44–1.96)	1.53 (1.35–1.89)	3.10 (2.68–3.69)	5.75 (4.58–8.03)	10.5 (7.36–17.5)	1,350
	2011–2012	1.70 (1.56–1.85)	1.58 (1.43–1.75)	2.86 (2.63–3.18)	5.36 (4.64–5.89)	8.89 (6.67–13.2)	1,229
	2013–2014	Not calculated ^b	1.48 (1.33–1.64)	2.59 (2.36–2.96)	4.29 (3.70–5.00)	5.74 (5.12–7.70)	1,400
Race/ethnicity							
Mexican Americans	1999–2000	3.16 (2.72–3.68)	3.15 (2.52–3.81)	5.88 (4.86–7.24)	11.6 (9.63–13.1)	15.7 (12.6–23.1)	814
	2001–2002	4.07 (3.60–4.61)	4.18 (3.82–4.90)	7.80 (6.64–9.49)	16.4 (13.6–18.9)	24.9 (19.8–28.7)	677
	2003–2004	2.12 (1.74–2.59)	1.94 (1.50–2.42)	4.06 (3.29–4.93)	9.38 (5.72–15.4)	16.8 (9.86–38.6)	652
	2005–2006	2.69 (2.36–3.07)	2.41 (2.04–2.73)	4.82 (4.09–6.05)	14.3 (10.0–16.9)	27.2 (16.3–40.1)	637
	2007–2008	2.82 (2.38–3.34)	2.45 (2.08–2.89)	5.00 (4.14–6.86)	12.3 (9.58–20.7)	29.0 (17.0–50.3)	531
	2009–2010	2.07 (1.83–2.34)	1.89 (1.69–2.31)	4.06 (3.50–4.98)	8.38 (6.08–11.3)	13.9 (9.70–20.0)	566
	2011–2012	1.68 (1.36–2.06)	1.67 (1.21–2.06)	2.91 (2.46–3.50)	6.23 (4.32–9.05)	11.3 (5.67–18.3)	316
	2013–2014	1.58 (1.35–1.85)	1.48 (1.24–1.83)	2.85 (2.46–3.39)	4.62 (4.07–5.56)	7.13 (5.53–8.04)	438
Non- Hispanic blacks	1999–2000	3.11 (2.59–3.73)	3.13 (2.50–3.61)	5.84 (4.43–7.32)	10.2 (8.05–15.6)	18.4 (11.6–35.2)	603
	2001–2002	4.63 (3.96–5.42)	4.59 (3.97–5.02)	9.93 (7.95–12.4)	21.2 (16.0–33.2)	39.9 (27.7–48.1)	703
	2003–2004	2.56 (2.24–2.92)	2.28 (2.02–2.78)	5.17 (4.48–6.83)	13.2 (10.5–16.2)	27.5 (18.4–36.0)	699
	2005–2006	2.87 (2.45–3.38)	2.41 (2.09–2.78)	5.72 (4.40–7.29)	15.0 (12.0–20.6)	54.4 (18.4–84.0)	678
	2007–2008	2.56 (2.26–2.90)	2.40 (2.17–2.73)	4.77 (4.07–5.76)	11.4 (8.75–15.4)	18.0 (16.1–26.3)	597
	2009–2010	1.51 (1.22–1.86)	1.48 (1.14–1.93)	2.87 (2.26–3.69)	5.73 (3.69–10.7)	10.7 (5.61–19.1)	516
	2011–2012	1.47 (1.29–1.67)	1.44 (1.26–1.75)	2.75 (2.37–3.14)	5.16 (4.17–7.05)	8.69 (6.07–12.1)	665
	2013–2014	1.20 (1.07–1.36)	1.13 (0.957–1.39)	2.11 (1.85–2.44)	3.46 (2.96–4.07)	5.05 (3.74–6.31)	609

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Table 5-10. Creatinine-Corrected Urinary MEHP Concentrations for the U.S. Population from NHANES 1999–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Non- Hispanic whites	1999–2000	3.09 (2.84–3.36)	3.08 (2.73–3.47)	5.87 (5.11–6.67)	10.6 (8.95–13.5)	20.0 (14.0–24.6)	912
	2001–2002	3.81 (3.34–4.35)	3.67 (3.11–4.33)	7.78 (6.74–9.35)	17.0 (14.1–21.8)	32.8 (21.5–46.9)	1,216
	2003–2004	2.12 (1.91–2.35)	1.82 (1.60–2.13)	4.11 (3.49–4.42)	10.7 (7.42–15.1)	27.0 (15.1–37.4)	1,088
	2005–2006	2.98 (2.77–3.21)	2.66 (2.43–2.93)	5.73 (5.00–6.47)	13.4 (11.3–17.8)	27.7 (19.5–37.4)	1,038
	2007–2008	2.55 (2.20–2.94)	2.26 (1.97–2.67)	4.90 (3.99–5.92)	11.0 (7.80–14.8)	20.5 (11.9–30.2)	1,077
	2009–2010	1.58 (1.36–1.84)	1.49 (1.27–1.76)	2.94 (2.50–3.49)	5.64 (4.53–7.96)	10.3 (6.85–18.3)	1,206
	2011–2012	1.47 (1.29–1.68)	1.45 (1.25–1.65)	2.51 (2.17–2.84)	4.43 (3.77–5.00)	6.77 (5.00–9.54)	811
2013–2014	Not calculated ^b	1.16 (1.02–1.31)	2.11 (1.84–2.33)	3.56 (3.00–4.10)	4.68 (4.19–5.62)	987	
All Hispanics	2011–2012	1.80 (1.63–1.98)	1.79 (1.59–2.00)	3.32 (2.86–3.76)	6.37 (5.67–8.07)	11.3 (7.79–14.4)	571
	2013–2014	1.54 (1.38–1.73)	1.43 (1.22–1.67)	2.80 (2.48–3.10)	4.39 (3.98–5.09)	6.61 (5.53–7.64)	690
Asians	2011–2012	2.26 (1.98–2.58)	1.98 (1.71–2.33)	4.18 (3.26–5.00)	8.89 (6.40–12.6)	14.0 (10.0–20.1)	352
	2013–2014	Not calculated ^b	1.43 (<LOD–1.70)	2.76 (2.31–3.13)	5.00 (3.96–5.87)	6.85 (5.17–9.73)	288

^aThe limit of detection (not corrected for creatinine) for survey years 1999–2000, 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.2, 1.0, 0.9, 1.2, 1.1, 0.5, 0.5, and 0.8 µg/L, respectively.

^bNot calculated: proportion of results below limit of detection was too high to provide a valid result.

CI = confidence interval; MEHP = mono-(2-ethylhexyl)phthalate; LOD = limit of detection; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

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Table 5-11. Uncorrected Urinary MEHHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Total	2001–2002	20.0 (17.8–22.5)	20.1 (17.8–22.4)	43.6 (38.0–49.7)	92.3 (77.0–108)	192 (131–256)	2,782
	2003–2004	21.7 (19.3–24.4)	21.2 (18.7–24.1)	49.1 (40.5–56.9)	121 (91.3–164)	266 (165–383)	2,605
	2005–2006	25.5 (23.0–28.2)	23.8 (21.5–26.8)	55.1 (50.2–61.0)	153 (132–180)	306 (240–421)	2,548
	2007–2008	22.1 (18.7–26.0)	20.7 (17.6–23.7)	48.2 (39.8–61.8)	123 (83.7–181)	238 (171–336)	2,604
	2009–2010	12.9 (11.3–14.7)	12.9 (11.4–14.8)	25.8 (22.1–30.3)	53.2 (41.7–75.2)	103 (74.2–149)	2,749
	2011–2012	7.91 (7.47–8.36)	8.30 (7.50–9.00)	16.0 (14.4–17.4)	29.0 (25.8–32.5)	43.1 (40.6–47.2)	2,489
	2013–2014	6.47 (5.98–7.00)	6.90 (6.30–7.70)	13.2 (11.9–14.7)	23.7 (20.7–26.6)	33.7 (28.2–37.6)	2,685
Age group							
6–11 years	2001–2002	33.6 (29.7–37.9)	32.9 (26.9–39.1)	66.9 (49.7–74.0)	127 (103–148)	216 (137–280)	393
	2003–2004	36.9 (28.4–47.9)	36.5 (26.5–47.0)	77.4 (49.1–103)	164 (79.9–350)	318 (164–400)	342
	2005–2006	34.9 (30.8–39.6)	35.7 (31.3–40.7)	68.9 (56.5–76.0)	140 (101–169)	206 (133–401)	356
	2007–2008	28.6 (23.4–34.8)	27.0 (20.1–36.4)	56.1 (45.4–67.8)	113 (76.5–218)	242 (109–351)	389
	2009–2010	15.0 (13.2–17.1)	17.0 (14.1–19.8)	28.9 (24.4–37.4)	49.3 (41.2–70.2)	75.1 (55.2–117)	415
	2011–2012	10.5 (8.82–12.4)	11.8 (10.4–14.2)	23.3 (19.9–26.1)	39.5 (30.6–50.3)	55.4 (41.8–68.5)	396
	2013–2014	9.54 (7.94–11.5)	9.40 (7.50–12.8)	18.7 (16.2–23.0)	36.4 (26.5–44.8)	50.8 (37.3–76.5)	409
12–19 years	2001–2002	24.9 (21.3–29.1)	25.3 (22.9–31.3)	50.6 (40.7–64.5)	107 (78.5–148)	216 (117–330)	742
	2003–2004	28.3 (23.0–34.8)	29.8 (25.9–33.9)	56.9 (45.4–73.7)	157 (84.1–299)	317 (176–553)	729
	2005–2006	34.8 (28.0–43.3)	32.5 (27.1–42.2)	79.5 (66.9–103)	213 (131–384)	424 (232–836)	702
	2007–2008	29.8 (22.5–39.3)	26.6 (20.0–35.4)	66.7 (43.7–96.6)	224 (101–417)	417 (209–615)	401
	2009–2010	15.3 (12.4–18.8)	14.9 (12.0–18.0)	28.8 (22.7–41.0)	70.2 (41.0–110)	117 (61.0–215)	420
	2011–2012	8.55 (6.93–10.6)	8.80 (6.80–10.6)	18.3 (14.3–23.2)	36.0 (27.4–46.6)	56.7 (38.1–99.8)	388
	2013–2014	7.62 (6.43–9.04)	7.70 (6.20–10.3)	15.8 (12.3–19.3)	26.8 (21.9–32.5)	35.3 (27.6–59.5)	462
≥20 years	2001–2002	18.1 (15.7–20.9)	17.8 (14.7–20.7)	39.8 (32.7–48.0)	86.2 (65.7–107)	175 (110–279)	1,647
	2003–2004	19.5 (17.7–21.5)	18.4 (16.6–21.0)	41.9 (36.9–51.2)	107 (88.2–136)	225 (148–384)	1,534
	2005–2006	23.4 (21.1–25.9)	21.4 (19.5–23.7)	48.6 (43.7–55.1)	148 (121–172)	306 (238–421)	1,490
	2007–2008	20.5 (17.4–24.1)	19.6 (17.1–22.2)	46.3 (37.2–59.9)	110 (75.0–169)	214 (157–303)	1,814
	2009–2010	12.4 (10.7–14.3)	12.5 (10.8–14.3)	24.5 (21.1–29.3)	52.5 (40.1–72.6)	104 (72.6–151)	1,914
	2011–2012	7.58 (7.03–8.16)	8.00 (7.20–8.90)	15.0 (13.5–16.9)	26.7 (23.8–30.6)	41.5 (34.7–46.2)	1,705
	2013–2014	6.06 (5.63–6.51)	6.70 (5.70–7.30)	12.3 (11.3–13.4)	22.3 (19.6–24.0)	30.3 (26.5–35.9)	1,814

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-11. Uncorrected Urinary MEHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	2001–2002	22.0 (19.5–24.7)	21.2 (19.4–24.2)	48.0 (41.4–54.4)	94.2 (80.8–110)	212 (130–256)	1,371
	2003–2004	24.1 (20.9–27.9)	22.9 (19.2–27.9)	51.0 (40.5–59.8)	133 (94.8–220)	317 (162–470)	1,250
	2005–2006	29.6 (26.0–33.8)	27.2 (22.9–31.0)	63.1 (54.3–71.9)	180 (143–263)	494 (285–626)	1,270
	2007–2008	23.2 (19.4–27.8)	20.8 (18.1–24.5)	46.0 (39.2–57.3)	132 (85.4–190)	277 (162–349)	1,294
	2009–2010	15.2 (13.1–17.6)	14.6 (12.9–16.4)	29.5 (24.6–36.2)	71.0 (50.3–93.5)	128 (90.6–181)	1,399
	2011–2012	8.71 (7.98–9.51)	9.40 (7.80–10.6)	16.4 (14.2–19.5)	31.1 (26.7–35.8)	46.2 (41.5–55.4)	1,259
	2013–2014	6.98 (6.32–7.71)	7.40 (6.70–8.40)	13.1 (11.7–14.8)	22.7 (20.1–24.9)	32.9 (27.3–36.0)	1,285
Females	2001–2002	18.3 (15.7–21.4)	18.2 (14.9–22.1)	39.8 (34.3–46.0)	86.0 (69.4–115)	170 (119–273)	1,411
	2003–2004	19.7 (17.4–22.2)	19.4 (16.7–22.8)	46.4 (37.5–54.4)	103 (84.1–148)	214 (140–318)	1,355
	2005–2006	22.0 (19.2–25.2)	21.4 (19.5–23.4)	47.1 (42.6–54.3)	135 (113–156)	232 (186–300)	1,278
	2007–2008	21.0 (17.8–24.8)	19.9 (16.8–23.3)	51.0 (39.9–64.6)	121 (76.9–183)	223 (171–336)	1,310
	2009–2010	11.0 (9.58–12.8)	11.6 (9.73–13.5)	22.5 (19.2–27.2)	42.8 (33.2–59.1)	82.2 (53.1–116)	1,350
	2011–2012	7.20 (6.77–7.66)	7.60 (6.90–8.20)	15.5 (14.4–17.1)	27.6 (25.0–30.5)	40.7 (35.1–46.4)	1,230
	2013–2014	6.02 (5.40–6.72)	6.30 (5.30–7.20)	13.4 (11.5–15.3)	24.8 (20.7–27.8)	35.3 (27.4–40.8)	1,400
Race/ethnicity							
Mexican Americans	2001–2002	18.5 (16.2–21.1)	19.1 (16.3–21.6)	36.3 (31.6–44.0)	79.9 (66.4–93.9)	123 (100–161)	677
	2003–2004	18.9 (15.4–23.4)	19.8 (17.6–22.3)	37.5 (30.0–45.6)	72.2 (52.4–115)	116 (71.6–327)	652
	2005–2006	23.0 (18.0–29.3)	19.9 (15.7–23.9)	47.8 (34.8–65.3)	136 (84.6–223)	244 (157–520)	637
	2007–2008	22.7 (18.5–27.7)	19.8 (17.5–23.8)	43.6 (33.5–66.6)	104 (82.8–157)	238 (158–282)	531
	2009–2010	15.3 (12.9–18.2)	15.8 (13.3–18.3)	31.5 (26.6–39.2)	64.5 (46.4–94.9)	108 (70.7–153)	566
	2011–2012	9.13 (7.22–11.5)	9.60 (7.30–11.7)	17.7 (12.7–24.7)	32.1 (24.8–37.7)	50.2 (36.4–56.3)	316
	2013–2014	7.71 (6.53–9.10)	8.00 (7.40–8.80)	14.4 (12.5–16.2)	28.1 (18.9–42.0)	43.7 (31.8–50.3)	438
Non- Hispanic blacks	2001–2002	29.8 (26.1–34.1)	30.9 (27.2–34.3)	61.9 (52.6–69.4)	126 (108–157)	276 (157–339)	703
	2003–2004	30.8 (26.8–35.5)	29.1 (25.3–32.3)	65.6 (53.7–76.3)	154 (113–178)	275 (174–401)	699
	2005–2006	34.8 (30.3–39.9)	30.2 (27.6–33.2)	73.8 (61.0–96.7)	206 (156–275)	395 (274–547)	678
	2007–2008	25.7 (23.1–28.6)	25.8 (22.4–28.9)	55.9 (47.1–64.7)	121 (99.1–150)	184 (137–255)	597
	2009–2010	15.5 (12.8–18.8)	15.7 (13.4–17.7)	30.4 (24.4–35.7)	54.5 (37.7–84.1)	94.6 (51.5–229)	516
	2011–2012	11.3 (10.1–12.7)	11.2 (10.2–12.7)	22.1 (17.4–26.8)	38.8 (34.9–47.9)	56.2 (46.8–65.1)	665
	2013–2014	8.06 (6.79–9.56)	8.80 (7.30–10.4)	16.5 (14.0–18.6)	27.4 (22.8–32.7)	38.7 (32.7–48.1)	609

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-11. Uncorrected Urinary MEHHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Non-	2001–2002	19.1 (16.7–21.9)	19.2 (16.9–21.4)	41.7 (35.3–50.7)	91.1 (75.6–110)	212 (130–275)	1,216
Hispanic	2003–2004	20.8 (18.6–23.3)	19.7 (17.2–22.5)	47.5 (39.4–56.1)	120 (91.3–165)	270 (155–403)	1,088
whites	2005–2006	24.3 (21.9–26.9)	23.0 (21.1–26.0)	54.6 (48.4–61.0)	148 (121–172)	302 (221–421)	1,038
	2007–2008	21.3 (17.6–25.8)	20.2 (16.5–24.1)	46.7 (36.1–65.2)	123 (75.1–203)	277 (161–373)	1,077
	2009–2010	12.2 (10.5–14.1)	12.3 (10.5–14.2)	24.4 (20.7–29.1)	51.7 (37.7–77.1)	104 (72.6–151)	1,206
	2011–2012	7.20 (6.73–7.70)	7.40 (6.80–8.30)	14.6 (12.9–17.2)	25.0 (23.4–27.9)	36.6 (30.6–42.9)	813
	2013–2014	6.05 (5.54–6.61)	6.70 (5.60–7.20)	12.6 (10.8–14.2)	22.4 (19.1–24.6)	30.1 (26.3–35.3)	987
All Hispanics	2011–2012	9.28 (8.07–10.7)	9.40 (7.70–10.8)	18.8 (15.3–22.9)	37.5 (32.1–43.9)	53.8 (46.3–70.6)	571
	2013–2014	7.56 (6.92–8.26)	8.00 (7.40–8.60)	14.4 (13.1–15.5)	27.2 (21.3–37.3)	39.3 (35.1–46.4)	690
Asians	2011–2012	6.85 (5.53–8.48)	6.70 (5.40–7.90)	15.1 (11.5–18.6)	31.8 (22.5–41.9)	64.2 (35.7–82.8)	352
	2013–2014	4.78 (4.07–5.61)	4.50 (3.70–5.60)	10.2 (8.30–12.4)	17.1 (14.6–21.8)	26.7 (17.9–47.0)	289

^aThe limit of detection for survey years 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.0, 0.3, 0.7, 0.7, 0.2, 0.2, and 0.4 µg/L, respectively.

CI = confidence interval; MEHHP = mono-2-ethyl-5-hydroxyhexyl phthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-12. Creatinine-Corrected Urinary MEHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Total	2001–2002	18.8 (17.0–20.7)	16.6 (14.9–18.5)	32.2 (27.8–37.1)	71.1 (58.7–88.3)	143 (101–200)	2,782
	2003–2004	20.4 (18.7–22.3)	17.7 (16.3–19.6)	35.8 (30.5–43.3)	93.5 (74.0–128)	182 (134–262)	2,605
	2005–2006	24.8 (22.4–27.5)	21.4 (19.1–23.4)	46.1 (40.0–52.1)	117 (97.8–148)	235 (197–272)	2,548
	2007–2008	22.2 (19.4–25.5)	19.3 (17.4–22.1)	40.5 (34.1–50.3)	99.3 (69.7–134)	179 (135–252)	2,604
	2009–2010	13.5 (11.6–15.6)	11.9 (10.5–13.9)	22.3 (18.6–26.4)	44.0 (35.3–61.0)	86.3 (59.2–131)	2,749
	2011–2012	8.99 (8.55–9.46)	8.46 (7.90–9.27)	14.1 (13.5–14.9)	25.3 (23.1–27.8)	37.7 (32.9–45.3)	2,487
	2013–2014	6.49 (5.97–7.05)	6.15 (5.67–6.86)	10.5 (9.47–11.7)	18.1 (15.4–20.4)	25.8 (22.9–29.3)	2,684
Age group							
6–11 years	2001–2002	38.2 (34.3–42.6)	34.3 (29.9–38.9)	60.6 (51.9–76.4)	107 (96.3–147)	211 (122–313)	393
	2003–2004	39.0 (31.1–48.9)	36.6 (25.3–49.3)	65.6 (49.8–91.3)	129 (77.1–253)	211 (123–708)	342
	2005–2006	38.5 (34.2–43.3)	37.0 (32.9–40.3)	65.5 (55.4–71.9)	115 (85.1–165)	213 (119–333)	356
	2007–2008	35.2 (29.1–42.5)	31.4 (25.6–37.8)	53.5 (44.8–72.3)	139 (79.9–230)	258 (141–303)	389
	2009–2010	19.6 (17.3–22.2)	18.6 (16.5–21.5)	34.2 (27.3–40.4)	55.4 (44.0–64.8)	72.1 (56.3–140)	415
	2011–2012	14.9 (12.9–17.2)	14.8 (13.0–18.2)	26.5 (22.9–28.2)	42.6 (30.9–46.5)	58.1 (43.2–75.1)	395
	2013–2014	12.0 (10.4–14.0)	11.6 (9.32–14.4)	19.3 (17.2–24.2)	36.9 (24.6–61.7)	61.7 (34.3–130)	409
12–19 years	2001–2002	19.2 (17.0–21.8)	17.8 (15.6–20.0)	34.9 (29.2–42.7)	73.4 (58.4–80.7)	102 (86.6–160)	742
	2003–2004	21.2 (18.1–24.7)	18.6 (16.9–21.7)	38.7 (29.7–53.4)	103 (62.7–209)	212 (100–358)	729
	2005–2006	26.0 (21.2–31.7)	23.7 (18.8–28.4)	49.4 (37.4–74.0)	131 (79.0–228)	278 (132–375)	702
	2007–2008	23.2 (18.1–29.6)	20.0 (14.6–23.9)	46.1 (31.5–66.3)	148 (66.6–234)	234 (146–373)	401
	2009–2010	12.3 (9.73–15.5)	10.5 (8.95–13.3)	20.9 (14.2–31.0)	45.3 (28.9–91.9)	110 (44.6–200)	420
	2011–2012	8.33 (7.38–9.41)	7.60 (6.41–8.29)	12.6 (10.4–15.5)	28.7 (20.1–37.2)	53.9 (31.7–68.8)	388
	2013–2014	6.19 (5.36–7.14)	5.94 (5.00–7.14)	9.82 (8.59–11.5)	16.9 (13.0–21.1)	24.8 (19.9–31.1)	462
≥20 years	2001–2002	17.1 (15.2–19.3)	15.0 (13.3–16.7)	27.7 (23.2–34.0)	63.7 (48.3–86.9)	137 (84.4–203)	1,647
	2003–2004	18.8 (17.5–20.2)	16.3 (15.4–17.5)	31.6 (28.1–35.3)	83.8 (67.2–106)	171 (129–246)	1,534
	2005–2006	23.4 (21.1–26.0)	19.4 (17.6–21.8)	42.5 (37.6–49.2)	115 (93.1–153)	235 (184–298)	1,490
	2007–2008	21.0 (18.5–24.0)	18.5 (16.3–20.2)	38.1 (30.9–49.2)	94.3 (62.6–126)	164 (120–235)	1,814
	2009–2010	13.1 (11.3–15.2)	11.5 (10.1–13.4)	20.9 (17.7–25.1)	40.6 (32.4–64.9)	86.3 (59.2–131)	1,914
	2011–2012	8.61 (8.17–9.07)	8.20 (7.63–8.94)	13.5 (12.6–14.1)	22.5 (20.1–25.3)	32.0 (28.1–39.3)	1,704
	2013–2014	6.10 (5.62–6.63)	5.93 (5.40–6.41)	9.81 (8.89–10.8)	15.4 (14.1–18.3)	22.2 (19.2–26.0)	1,813

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-12. Creatinine-Corrected Urinary MEHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	2001–2002	17.9 (16.2–19.7)	15.4 (13.8–17.9)	32.2 (27.8–36.8)	73.4 (55.3–91.8)	137 (97.7–224)	1,371
	2003–2004	18.9 (17.1–20.9)	17.1 (15.2–18.6)	32.7 (26.6–41.6)	93.4 (68.8–123)	193 (108–291)	1,250
	2005–2006	23.8 (21.0–26.9)	20.2 (17.5–23.3)	44.8 (38.5–54.1)	129 (92.5–166)	251 (202–352)	1,270
	2007–2008	19.6 (16.8–22.7)	16.8 (14.6–20.0)	34.8 (28.7–41.8)	90.3 (65.2–119)	164 (111–258)	1,294
	2009–2010	13.6 (11.7–15.9)	11.6 (10.5–13.1)	22.9 (17.8–27.4)	52.8 (39.1–74.3)	103 (73.3–173)	1,399
	2011–2012	8.15 (7.67–8.66)	7.80 (7.18–8.21)	12.2 (11.5–13.2)	24.2 (20.9–25.9)	37.2 (29.5–50.2)	1,258
	2013–2014	5.86 (5.38–6.39)	5.68 (5.26–6.12)	8.97 (8.48–9.79)	15.2 (13.9–16.7)	22.3 (19.8–26.3)	1,284
Females	2001–2002	19.7 (17.3–22.4)	17.6 (15.4–19.5)	32.1 (26.8–38.6)	70.5 (57.8–93.7)	156 (93.7–201)	1,411
	2003–2004	21.9 (19.7–24.5)	18.7 (16.8–20.9)	39.3 (33.8–46.9)	94.3 (72.8–136)	171 (146–261)	1,355
	2005–2006	25.9 (23.2–28.8)	22.5 (19.6–25.5)	47.3 (40.5–52.3)	108 (88.8–131)	202 (157–278)	1,278
	2007–2008	25.2 (22.1–28.6)	22.1 (19.6–24.4)	47.4 (38.5–56.1)	112 (84.6–150)	190 (162–268)	1,310
	2009–2010	13.3 (11.4–15.6)	12.4 (10.4–14.6)	21.9 (18.3–26.7)	38.8 (30.9–52.2)	74.0 (46.9–115)	1,350
	2011–2012	9.89 (9.16–10.7)	9.64 (8.33–11.1)	15.6 (14.7–17.3)	27.0 (23.9–28.8)	37.7 (33.2–44.4)	1,229
	2013–2014	7.15 (6.38–8.01)	6.94 (6.25–7.58)	12.0 (10.3–13.7)	20.4 (16.4–23.6)	28.3 (22.9–36.4)	1,400
Race/ethnicity							
Mexican Americans	2001–2002	17.4 (15.9–19.1)	15.7 (14.4–17.5)	30.6 (26.0–34.7)	65.9 (50.6–83.9)	103 (75.5–128)	677
	2003–2004	17.1 (14.3–20.4)	15.4 (13.2–17.7)	29.3 (23.8–36.8)	57.3 (45.7–97.6)	105 (70.1–195)	652
	2005–2006	20.7 (17.2–25.0)	17.5 (15.3–21.3)	37.3 (30.9–45.7)	99.9 (67.6–159)	181 (110–357)	637
	2007–2008	22.1 (17.7–27.7)	18.8 (15.2–23.4)	38.6 (28.3–54.9)	91.4 (61.0–170)	197 (141–282)	531
	2009–2010	15.2 (12.9–17.8)	14.6 (12.8–16.2)	27.0 (21.3–33.3)	60.0 (42.3–77.7)	94.8 (66.4–142)	566
	2011–2012	10.3 (8.35–12.7)	9.10 (7.02–12.5)	18.6 (14.4–21.1)	30.4 (25.1–36.0)	49.6 (30.4–114)	316
	2013–2014	7.86 (7.13–8.66)	7.54 (6.78–8.10)	12.6 (10.8–16.5)	23.5 (19.6–32.1)	37.2 (25.8–54.9)	438
Non- Hispanic blacks	2001–2002	20.9 (18.8–23.3)	19.7 (17.5–21.8)	38.3 (32.1–46.0)	93.5 (69.2–123)	164 (130–183)	703
	2003–2004	21.9 (20.1–23.8)	19.5 (17.3–22.6)	40.1 (35.8–45.3)	102 (75.5–122)	164 (133–269)	699
	2005–2006	24.5 (21.2–28.2)	18.9 (17.1–22.8)	46.1 (37.9–59.7)	128 (100–158)	308 (158–399)	678
	2007–2008	20.0 (18.1–22.0)	18.3 (16.5–19.2)	36.9 (27.9–46.7)	85.5 (65.8–103)	136 (107–227)	597
	2009–2010	11.2 (8.71–14.5)	10.5 (8.26–13.5)	18.9 (13.6–25.3)	32.9 (22.3–58.4)	47.2 (33.6–153)	516
	2011–2012	8.80 (7.84–9.87)	8.12 (7.37–8.96)	14.7 (12.4–17.8)	28.2 (20.7–37.4)	46.2 (29.7–57.5)	665
	2013–2014	5.94 (5.28–6.70)	5.68 (4.84–6.85)	9.94 (9.02–11.4)	17.1 (15.0–20.1)	23.9 (19.2–32.4)	609

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-12. Creatinine-Corrected Urinary MEHHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Non- Hispanic whites	2001–2002	18.9 (17.0–21.0)	16.3 (14.8–18.4)	32.1 (27.3–37.3)	70.8 (56.9–93.7)	177 (98.0–242)	1,216
	2003–2004	20.5 (18.5–22.8)	17.8 (16.2–19.7)	35.3 (29.7–44.9)	96.2 (75.8–136)	211 (136–283)	1,088
	2005–2006	25.6 (23.1–28.2)	22.4 (19.6–24.5)	47.6 (40.7–54.1)	119 (98.2–148)	231 (181–297)	1,038
	2007–2008	22.2 (18.7–26.3)	19.5 (16.7–23.0)	40.4 (32.1–54.8)	99.1 (68.1–145)	179 (129–258)	1,077
	2009–2010	13.7 (11.7–16.1)	11.9 (10.4–13.9)	22.4 (18.0–27.6)	43.0 (34.2–61.0)	84.1 (54.7–142)	1,206
	2011–2012	8.74 (8.02–9.53)	8.36 (7.62–9.58)	13.5 (12.5–14.5)	23.1 (19.7–25.9)	32.7 (27.8–42.9)	811
	2013–2014	6.37 (5.74–7.07)	6.06 (5.56–6.74)	10.0 (8.72–11.6)	16.2 (14.2–20.0)	23.1 (20.8–27.7)	987
All Hispanics	2011–2012	10.4 (9.42–11.5)	9.29 (8.14–10.5)	18.7 (16.7–21.1)	30.5 (27.1–35.6)	49.6 (33.4–67.9)	571
	2013–2014	7.50 (7.04–8.00)	7.19 (6.58–7.77)	12.3 (11.1–14.1)	22.3 (19.0–27.0)	34.9 (27.1–40.6)	690
Asians	2011–2012	9.18 (7.54–11.2)	8.45 (7.34–10.0)	16.2 (12.7–21.0)	33.0 (23.6–42.6)	57.4 (35.8–85.4)	352
	2013–2014	6.06 (5.35–6.86)	5.63 (4.83–6.50)	11.0 (9.79–13.2)	19.6 (16.0–24.5)	26.3 (19.6–41.9)	288

^aThe limit of detection (not corrected for creatinine) for survey years 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.0, 0.3, 0.7, 0.7, 0.2, 0.2, and 0.4 µg/L, respectively.

CI = confidence interval; MEHHP = mono-2-ethyl-5-hydroxyhexyl phthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-13. Uncorrected Urinary MEOHP Concentrations for the U.S. Population NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Total	2001–2002	13.5 (12.0–15.0)	14.0 (12.5–15.1)	29.6 (25.2–34.0)	59.9 (50.4–70.9)	120 (87.2–156)	2,782
	2003–2004	14.5 (13.0–16.1)	14.4 (12.4–16.7)	31.4 (27.4–36.6)	76.7 (59.4–102)	157 (106–232)	2,605
	2005–2006	16.2 (14.6–18.0)	15.1 (13.5–17.1)	35.5 (32.1–40.3)	94.8 (78.5–112)	183 (147–250)	2,548
	2007–2008	12.2 (10.3–14.3)	11.3 (10.0–13.3)	27.1 (21.8–33.8)	64.2 (47.3–93.6)	130 (93.6–177)	2,604
	2009–2010	8.02 (7.11–9.06)	8.02 (7.27–9.04)	16.1 (13.8–19.0)	32.5 (25.2–41.8)	55.7 (41.8–80.9)	2,749
	2011–2012	5.08 (4.78–5.41)	5.30 (4.90–5.80)	10.3 (9.40–11.5)	18.4 (17.0–19.5)	26.5 (23.7–30.2)	2,489
	2013–2014	4.24 (3.94–4.57)	4.70 (4.20–5.00)	8.50 (7.60–9.30)	15.2 (13.3–17.3)	20.2 (18.2–23.7)	2,685
Age group							
6–11 years	2001–2002	23.3 (20.9–26.1)	22.9 (18.5–28.1)	46.5 (38.1–52.0)	81.6 (64.7–109)	142 (93.9–178)	393
	2003–2004	25.1 (19.6–32.3)	25.8 (19.3–31.4)	51.1 (32.1–76.5)	97.9 (58.8–197)	197 (97.6–261)	342
	2005–2006	23.0 (20.3–26.1)	24.5 (19.9–27.6)	44.3 (38.1–52.3)	83.8 (63.6–115)	126 (77.3–253)	356
	2007–2008	16.9 (13.9–20.6)	16.6 (12.4–22.6)	34.5 (26.2–40.9)	64.4 (46.9–129)	137 (63.8–179)	389
	2009–2010	9.78 (8.72–11.0)	11.1 (8.87–12.7)	20.0 (17.1–21.6)	35.4 (24.8–41.4)	48.4 (36.0–74.0)	415
	2011–2012	6.96 (5.86–8.28)	8.10 (6.90–9.70)	14.6 (12.8–17.1)	26.1 (22.6–28.4)	34.7 (27.9–42.5)	396
	2013–2014	6.51 (5.49–7.72)	6.60 (5.10–8.30)	12.4 (10.2–16.4)	23.4 (17.1–30.0)	33.0 (24.2–50.7)	409
12–19 years	2001–2002	17.5 (15.1–20.3)	18.6 (16.2–20.7)	35.0 (27.7–42.1)	70.7 (52.2–104)	118 (74.0–174)	742
	2003–2004	19.5 (16.0–23.7)	20.3 (18.4–23.5)	37.8 (32.6–44.6)	110 (54.6–168)	212 (103–326)	729
	2005–2006	23.0 (18.7–28.4)	22.1 (18.0–26.2)	50.7 (42.7–62.2)	134 (82.3–240)	263 (134–511)	702
	2007–2008	16.9 (12.8–22.3)	15.9 (11.9–19.3)	38.2 (24.0–55.5)	121 (58.1–258)	258 (120–354)	401
	2009–2010	10.0 (8.32–12.1)	9.82 (7.96–12.3)	19.0 (15.9–23.5)	40.0 (26.7–61.5)	68.4 (32.6–154)	420
	2011–2012	5.70 (4.64–7.01)	5.70 (4.60–7.30)	12.2 (10.5–13.7)	22.2 (19.1–29.5)	35.1 (23.0–46.8)	388
	2013–2014	5.34 (4.57–6.24)	5.50 (4.40–7.00)	10.5 (8.10–13.3)	17.5 (14.9–29.5)	25.6 (17.6–33.7)	462
≥20 years	2001–2002	12.0 (10.5–13.9)	12.3 (10.4–14.1)	26.0 (21.6–32.1)	52.3 (41.8–68.3)	116 (74.9–160)	1,647
	2003–2004	12.9 (11.8–14.1)	12.4 (10.9–14.5)	27.0 (25.0–30.9)	68.9 (55.0–86.5)	139 (92.7–216)	1,534
	2005–2006	14.7 (13.2–16.4)	13.4 (12.2–15.1)	31.9 (28.2–36.2)	91.6 (74.6–104)	182 (138–247)	1,490
	2007–2008	11.1 (9.48–13.1)	10.7 (9.30–12.3)	25.5 (20.2–31.4)	59.8 (42.3–88.9)	108 (80.0–155)	1,814
	2009–2010	7.59 (6.64–8.68)	7.55 (6.69–8.58)	15.3 (12.6–18.6)	30.6 (24.1–41.5)	54.9 (41.8–81.0)	1,914
	2011–2012	4.83 (4.45–5.23)	5.10 (4.70–5.60)	9.60 (8.50–10.8)	16.7 (14.5–18.6)	23.0 (20.8–26.5)	1,705
	2013–2014	3.91 (3.65–4.19)	4.40 (3.90–4.80)	7.80 (7.10–8.70)	13.9 (12.1–15.7)	19.1 (17.1–20.8)	1,814

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-13. Uncorrected Urinary MEOHP Concentrations for the U.S. Population NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	2001–2002	14.5 (13.0–16.2)	14.6 (13.1–16.2)	31.6 (25.6–34.7)	60.4 (52.3–71.4)	129 (84.4–167)	1,371
	2003–2004	15.6 (13.6–17.9)	14.7 (12.7–18.1)	31.8 (27.2–39.5)	83.8 (59.4–134)	185 (96.2–277)	1,250
	2005–2006	18.3 (16.0–20.9)	16.3 (14.7–19.7)	39.3 (33.4–48.1)	104 (80.7–140)	258 (180–337)	1,270
	2007–2008	12.5 (10.5–15.0)	11.3 (9.80–13.4)	26.1 (21.8–32.2)	61.8 (46.8–98.1)	139 (83.7–189)	1,294
	2009–2010	9.14 (8.01–10.4)	8.76 (7.87–9.88)	18.2 (14.8–21.0)	39.3 (27.5–50.9)	69.6 (50.0–109)	1,399
	2011–2012	5.50 (5.07–5.95)	5.70 (5.10–6.30)	10.5 (8.90–12.2)	18.7 (16.9–21.2)	28.1 (23.4–34.3)	1,259
	2013–2014	4.45 (4.05–4.89)	4.90 (4.40–5.30)	8.10 (7.30–9.00)	13.7 (12.6–15.9)	19.8 (17.3–23.6)	1,285
Females	2001–2002	12.5 (10.8–14.6)	13.1 (11.2–15.0)	28.1 (23.7–33.5)	57.5 (45.8–72.7)	115 (81.8–147)	1,411
	2003–2004	13.4 (11.9–15.1)	13.7 (11.4–16.4)	29.5 (26.1–36.6)	68.6 (53.7–88.1)	143 (88.2–210)	1,355
	2005–2006	14.4 (12.6–16.5)	13.8 (12.5–15.7)	32.5 (29.3–36.4)	81.7 (68.6–104)	159 (114–182)	1,278
	2007–2008	11.8 (10.0–14.0)	11.7 (9.80–13.5)	27.9 (21.3–37.5)	64.2 (43.9–93.6)	122 (92.0–191)	1,310
	2009–2010	7.09 (6.17–8.14)	7.35 (6.13–8.67)	15.2 (12.1–17.5)	27.1 (21.6–36.9)	48.2 (31.3–67.1)	1,350
	2011–2012	4.71 (4.39–5.07)	5.00 (4.70–5.60)	10.2 (9.20–11.1)	18.1 (15.8–19.3)	25.4 (21.9–29.6)	1,230
	2013–2014	4.06 (3.65–4.50)	4.20 (3.60–4.80)	9.00 (7.70–9.90)	16.2 (13.9–18.2)	21.5 (17.6–25.5)	1,400
Race/ethnicity							
Mexican Americans	2001–2002	13.1 (11.6–14.9)	13.4 (11.6–15.0)	25.5 (21.6–30.8)	56.6 (40.6–70.3)	77.3 (70.5–101)	677
	2003–2004	12.8 (10.5–15.5)	13.6 (11.4–15.6)	25.3 (20.4–29.9)	46.6 (32.3–70.8)	76.0 (51.6–153)	652
	2005–2006	14.8 (11.7–18.8)	12.8 (10.5–16.1)	30.9 (22.6–42.9)	79.1 (51.7–131)	152 (92.9–276)	637
	2007–2008	12.6 (10.5–15.1)	11.4 (10.4–12.9)	25.1 (18.7–37.2)	53.4 (43.2–91.7)	118 (88.4–147)	531
	2009–2010	9.57 (8.10–11.3)	9.65 (8.17–11.3)	19.2 (16.3–23.3)	39.0 (30.9–54.7)	64.6 (43.0–95.3)	566
	2011–2012	5.86 (4.69–7.33)	5.90 (4.40–7.50)	11.6 (8.40–14.4)	20.5 (15.4–25.4)	31.5 (22.5–35.1)	316
	2013–2014	5.03 (4.21–6.01)	5.20 (4.60–5.80)	9.40 (7.70–11.5)	18.3 (12.3–24.2)	25.1 (19.5–34.3)	438
Non- Hispanic blacks	2001–2002	19.6 (17.1–22.5)	20.1 (17.9–22.4)	39.0 (34.8–44.2)	80.5 (71.4–97.4)	153 (102–228)	703
	2003–2004	20.2 (17.7–23.0)	20.1 (17.0–22.5)	40.0 (33.9–46.9)	92.6 (68.8–130)	173 (104–247)	699
	2005–2006	21.8 (18.9–25.2)	18.8 (16.8–21.0)	46.0 (38.0–59.1)	130 (104–168)	243 (159–304)	678
	2007–2008	14.2 (12.7–15.9)	14.0 (12.7–16.3)	30.5 (25.8–35.9)	64.2 (52.1–76.1)	110 (71.9–136)	597
	2009–2010	9.57 (8.17–11.2)	9.64 (8.15–11.1)	19.8 (16.5–22.1)	31.4 (25.5–44.0)	50.9 (31.4–129)	516
	2011–2012	7.30 (6.48–8.22)	7.40 (6.70–8.00)	14.0 (11.5–16.0)	26.0 (21.1–32.1)	36.9 (30.8–48.2)	665
	2013–2014	5.38 (4.58–6.31)	5.80 (5.00–7.00)	10.8 (9.10–12.0)	17.5 (15.2–21.8)	25.3 (21.7–29.7)	609

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-13. Uncorrected Urinary MEOHP Concentrations for the U.S. Population NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Non- Hispanic whites	2001–2002	12.8 (11.2–14.6)	13.2 (11.6–14.6)	28.5 (23.6–34.0)	58.6 (48.8–70.9)	126 (83.7–172)	1,216
	2003–2004	13.8 (12.4–15.4)	13.4 (11.3–16.3)	31.0 (27.0–36.3)	77.6 (59.4–102)	161 (98.7–241)	1,088
	2005–2006	15.5 (13.9–17.3)	15.0 (13.3–16.5)	35.3 (30.1–40.8)	92.6 (74.6–111)	182 (134–247)	1,038
	2007–2008	11.7 (9.66–14.2)	11.0 (9.20–13.6)	26.9 (20.2–35.1)	64.2 (43.4–108)	137 (90.0–197)	1,077
	2009–2010	7.59 (6.60–8.73)	7.55 (6.81–8.63)	15.7 (12.9–18.6)	29.1 (22.9–46.5)	55.7 (41.5–83.5)	1,206
	2011–2012	4.62 (4.31–4.96)	4.90 (4.50–5.50)	9.50 (8.40–10.8)	16.1 (13.8–18.6)	21.2 (19.0–25.9)	813
	2013–2014	3.95 (3.63–4.30)	4.40 (3.90–4.80)	7.90 (7.00–9.10)	14.2 (12.0–16.5)	19.1 (16.5–23.4)	987
All Hispanics	2011–2012	5.94 (5.17–6.83)	6.00 (4.80–7.30)	11.8 (9.70–14.0)	22.5 (19.8–25.8)	34.3 (26.2–39.8)	571
	2013–2014	4.95 (4.46–5.49)	5.30 (4.80–5.70)	9.20 (8.30–10.4)	17.5 (12.9–20.2)	24.2 (19.7–28.5)	690
Asians	2011–2012	4.39 (3.63–5.31)	4.20 (3.30–5.20)	9.40 (7.20–11.3)	19.0 (15.0–27.8)	36.8 (25.4–52.4)	352
	2013–2014	3.25 (2.81–3.76)	3.20 (2.60–4.10)	6.20 (5.10–7.30)	11.0 (9.50–13.7)	16.1 (11.7–23.3)	289

^aThe limit of detection for survey years 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.1, 0.5, 0.7, 0.6, 0.2, 0.2, and 0.2 µg/L, respectively.

CI = confidence interval; MEOHP = mono-2-ethyl-5-oxyhexyl phthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-14. Creatinine-Corrected Urinary MEOHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Total	2001–2002	12.6 (11.5–13.9)	11.2 (10.2–12.3)	21.3 (18.3–23.8)	45.2 (37.1–58.1)	87.0 (68.0–124)	2,782
	2003–2004	13.6 (12.4–14.8)	12.1 (11.0–12.9)	24.3 (20.9–27.8)	63.0 (47.8–75.8)	118 (94.1–153)	2,605
	2005–2006	15.8 (14.2–17.5)	13.5 (12.4–14.7)	28.9 (26.0–33.8)	77.7 (62.4–91.1)	144 (118–172)	2,548
	2007–2008	12.3 (10.7–14.0)	11.0 (9.72–12.2)	22.3 (18.4–27.9)	52.9 (37.9–74.6)	107 (74.8–136)	2,604
	2009–2010	8.37 (7.31–9.59)	7.44 (6.71–8.42)	13.5 (11.5–15.7)	25.9 (21.4–34.6)	47.7 (34.6–67.8)	2,749
	2011–2012	5.78 (5.51–6.06)	5.51 (5.12–5.83)	8.99 (8.41–9.83)	15.6 (14.3–16.8)	23.4 (19.6–28.0)	2,487
	2013–2014	4.25 (3.92–4.61)	4.02 (3.74–4.43)	6.61 (5.93–7.26)	11.1 (9.73–12.7)	16.4 (13.9–18.1)	2,684
Age group							
6–11 years	2001–2002	26.6 (24.0–29.4)	22.8 (20.3–25.0)	43.3 (33.6–47.1)	74.7 (69.0–91.9)	131 (83.0–183)	393
	2003–2004	26.6 (21.4–33.0)	25.3 (17.8–32.4)	43.6 (34.2–63.2)	77.1 (63.0–118)	121 (76.3–435)	342
	2005–2006	25.4 (22.5–28.6)	24.4 (21.4–26.4)	42.7 (36.0–46.9)	70.4 (52.8–117)	136 (77.2–195)	356
	2007–2008	20.8 (17.1–25.4)	18.5 (15.6–21.5)	35.0 (26.4–46.0)	84.1 (47.2–137)	145 (84.8–187)	389
	2009–2010	12.7 (11.4–14.2)	12.6 (11.5–14.1)	20.8 (16.5–25.1)	33.3 (29.5–44.1)	45.5 (33.7–74.5)	415
	2011–2012	9.93 (8.63–11.4)	9.89 (8.25–11.4)	16.5 (14.3–18.9)	25.6 (22.9–30.2)	34.5 (27.9–41.9)	395
	2013–2014	8.23 (7.16–9.44)	7.77 (6.25–9.38)	12.8 (10.7–17.1)	24.7 (17.9–37.1)	37.4 (21.4–80.7)	409
12–19 years	2001–2002	13.5 (12.0–15.2)	12.0 (10.8–14.3)	23.4 (20.0–28.5)	48.4 (39.2–54.9)	70.5 (55.0–97.2)	742
	2003–2004	14.6 (12.6–16.9)	12.7 (11.6–14.4)	25.5 (20.7–33.8)	67.9 (42.3–143)	153 (61.8–209)	729
	2005–2006	17.2 (14.1–20.9)	15.3 (12.5–18.6)	32.5 (25.7–41.3)	84.2 (49.1–147)	163 (93.1–250)	702
	2007–2008	13.2 (10.3–16.7)	11.1 (9.08–13.5)	25.9 (18.9–35.6)	86.3 (39.4–128)	132 (84.3–203)	401
	2009–2010	8.06 (6.57–9.88)	7.05 (6.16–8.25)	13.3 (9.53–18.3)	24.8 (17.8–47.9)	55.2 (23.6–110)	420
	2011–2012	5.55 (4.95–6.23)	5.21 (4.81–5.61)	8.54 (7.09–11.1)	16.3 (12.9–21.3)	31.6 (16.8–35.3)	388
	2013–2014	4.33 (3.82–4.92)	4.07 (3.71–4.69)	6.55 (5.64–7.19)	10.7 (8.02–13.4)	15.3 (11.5–18.6)	462
≥20 years	2001–2002	11.4 (10.2–12.8)	10.1 (8.89–11.4)	17.5 (15.2–21.8)	38.4 (30.5–52.5)	84.3 (53.3–128)	1,647
	2003–2004	12.4 (11.5–13.3)	11.0 (10.0–12.0)	20.9 (18.6–22.8)	53.9 (40.7–70.2)	109 (88.6–130)	1,534
	2005–2006	14.8 (13.3–16.4)	12.4 (11.2–13.5)	26.7 (23.8–31.3)	76.3 (61.0–89.9)	144 (106–170)	1,490
	2007–2008	11.4 (10.0–13.0)	10.0 (9.15–11.2)	20.8 (16.7–25.6)	47.3 (33.2–65.3)	87.4 (62.7–136)	1,814
	2009–2010	8.04 (6.99–9.24)	7.19 (6.36–8.00)	12.7 (10.7–14.7)	24.3 (19.7–34.6)	47.4 (33.7–67.3)	1,914
	2011–2012	5.48 (5.23–5.74)	5.19 (4.89–5.56)	8.39 (7.95–8.98)	13.9 (12.8–15.3)	19.3 (16.6–25.4)	1,704
	2013–2014	3.95 (3.63–4.30)	3.85 (3.54–4.15)	6.15 (5.48–6.75)	9.83 (8.57–11.1)	13.6 (12.2–16.3)	1,813

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-14. Creatinine-Corrected Urinary MEOHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Gender							
Males	2001–2002	11.8 (10.7–13.0)	10.2 (8.93–11.7)	21.2 (18.5–23.3)	46.1 (35.3–58.7)	84.2 (69.6–104)	1,371
	2003–2004	12.3 (11.1–13.5)	11.1 (10.0–12.0)	21.6 (17.6–26.9)	59.1 (45.4–72.0)	120 (72.0–162)	1,250
	2005–2006	14.7 (12.9–16.7)	12.6 (10.9–14.3)	27.1 (23.3–35.6)	79.5 (59.6–96.9)	147 (120–190)	1,270
	2007–2008	10.5 (9.08–12.2)	9.25 (8.08–10.6)	18.9 (15.9–23.1)	46.7 (32.6–62.4)	86.4 (56.5–144)	1,294
	2009–2010	8.19 (7.15–9.38)	7.07 (6.59–7.55)	13.4 (10.8–15.8)	30.2 (21.7–41.2)	52.1 (41.2–82.3)	1,399
	2011–2012	5.14 (4.86–5.43)	4.86 (4.55–5.12)	7.83 (7.29–8.24)	14.7 (12.7–16.1)	22.6 (18.1–29.7)	1,258
	2013–2014	3.74 (3.45–4.06)	3.70 (3.46–3.91)	5.59 (5.13–3.67)	9.03 (8.37–9.74)	14.3 (11.6–16.8)	1,284
Females	2001–2002	13.5 (11.9–15.2)	12.0 (10.8–13.7)	21.5 (18.0–25.6)	44.8 (36.8–61.6)	92.3 (61.0–139)	1,411
	2003–2004	14.9 (13.4–16.7)	12.7 (11.4–14.2)	26.6 (21.8–30.6)	65.6 (48.0–90.1)	118 (97.0–157)	1,355
	2005–2006	16.9 (15.1–19.0)	14.7 (13.2–16.5)	30.3 (26.6–34.7)	76.4 (57.6–97.2)	137 (106–170)	1,278
	2007–2008	14.2 (12.5–16.0)	12.5 (11.5–13.8)	25.9 (21.2–31.0)	61.1 (40.9–84.1)	114 (84.3–142)	1,310
	2009–2010	8.55 (7.37–9.93)	8.06 (6.76–9.33)	13.6 (11.6–16.2)	24.1 (20.0–32.7)	43.2 (27.7–64.5)	1,350
	2011–2012	6.48 (6.07–6.91)	6.34 (5.71–6.91)	10.0 (9.34–11.0)	16.5 (14.7–18.6)	23.5 (19.5–27.9)	1,229
	2013–2014	4.81 (4.31–5.37)	4.58 (4.00–5.16)	7.42 (6.58–8.62)	12.6 (10.7–15.6)	17.2 (14.8–21.3)	1,400
Race/ethnicity							
Mexican Americans	2001–2002	12.4 (11.4–13.5)	11.0 (10.5–12.3)	20.9 (18.5–24.4)	44.6 (33.4–56.2)	65.9 (53.1–83.1)	677
	2003–2004	11.5 (9.81–13.6)	10.7 (9.04–12.3)	18.8 (15.6–24.6)	39.1 (31.8–53.9)	63.0 (47.2–121)	652
	2005–2006	13.3 (11.1–16.1)	11.4 (9.58–13.6)	24.0 (19.4–30.4)	61.2 (45.6–85.9)	102 (69.9–200)	637
	2007–2008	12.3 (10.0–15.0)	10.6 (8.57–14.1)	21.7 (16.1–28.7)	53.1 (32.7–85.2)	113 (79.7–149)	531
	2009–2010	9.50 (8.16–11.1)	8.72 (8.13–9.61)	16.6 (12.9–20.5)	35.4 (24.4–46.6)	56.7 (40.9–82.3)	566
	2011–2012	6.61 (5.43–8.05)	6.00 (4.83–7.45)	11.3 (8.87–13.9)	18.9 (15.8–22.8)	28.1 (18.2–60.8)	316
	2013–2014	5.13 (4.57–5.75)	4.90 (4.42–5.49)	8.51 (6.80–10.7)	14.3 (12.0–19.5)	20.7 (15.6–31.7)	438
Non- Hispanic blacks	2001–2002	13.8 (12.3–15.4)	13.1 (12.0–14.2)	23.9 (20.0–29.3)	58.3 (45.3–79.7)	101 (81.3–124)	703
	2003–2004	14.3 (13.1–15.6)	13.3 (11.3–15.5)	24.8 (21.7–27.7)	61.2 (46.8–76.6)	105 (79.7–152)	699
	2005–2006	15.3 (13.2–17.8)	11.6 (9.87–14.6)	29.1 (24.7–38.5)	77.1 (61.9–113)	172 (99.8–251)	678
	2007–2008	11.0 (9.94–12.2)	9.90 (9.16–11.1)	20.6 (16.6–23.5)	42.2 (33.8–58.6)	75.8 (57.2–124)	597
	2009–2010	6.92 (5.60–8.57)	6.49 (5.12–8.24)	11.4 (9.09–14.1)	19.2 (14.9–27.3)	28.1 (19.8–61.1)	516
	2011–2012	5.68 (5.11–6.32)	5.31 (4.71–5.70)	9.33 (7.79–10.8)	16.9 (13.6–23.4)	29.4 (18.8–39.2)	665
	2013–2014	3.97 (3.55–4.44)	3.79 (3.28–4.42)	6.43 (5.67–7.31)	10.6 (9.55–12.2)	14.1 (11.2–18.6)	609

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-14. Creatinine-Corrected Urinary MEOHP Concentrations for the U.S. Population from NHANES 2001–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Non- Hispanic whites	2001–2002	12.7 (11.4–14.0)	11.1 (9.90–12.3)	20.8 (18.0–23.9)	45.7 (35.9–64.9)	96.0 (68.5–161)	1,216
	2003–2004	13.7 (12.2–15.3)	12.0 (10.5–12.9)	24.9 (20.7–28.6)	69.5 (51.4–95.3)	124 (90.3–182)	1,088
	2005–2006	16.3 (14.8–18.0)	14.0 (12.9–15.7)	30.8 (27.3–34.8)	79.3 (66.0–93.8)	139 (117–163)	1,038
	2007–2008	12.2 (10.3–14.5)	11.0 (9.38–13.1)	22.3 (17.4–30.4)	51.4 (35.7–77.7)	107 (74.6–139)	1,077
	2009–2010	8.53 (7.35–9.90)	7.62 (6.76–8.54)	13.7 (11.2–16.2)	24.9 (20.3–35.7)	47.8 (32.8–76.4)	1,206
	2011–2012	5.61 (5.17–6.08)	5.41 (4.90–5.99)	8.42 (7.84–9.61)	14.3 (12.5–15.8)	20.5 (16.6–25.4)	811
	2013–2014	4.16 (3.75–4.62)	3.96 (3.70–4.41)	6.37 (5.47–7.23)	10.3 (8.57–12.7)	15.6 (12.8–17.3)	987
All Hispanics	2011–2012	6.66 (6.05–7.33)	6.04 (5.49–6.74)	11.3 (10.0–13.5)	18.9 (16.9–21.0)	30.2 (20.8–41.9)	571
	2013–2014	4.91 (4.55–5.31)	4.60 (4.26–5.07)	7.76 (6.94–9.27)	13.6 (11.5–17.0)	20.2 (16.9–25.2)	690
Asians	2011–2012	5.88 (4.96–6.97)	5.30 (4.59–6.36)	10.0 (8.11–12.2)	20.2 (14.7–26.3)	39.3 (23.5–50.1)	352
	2013–2014	4.12 (3.68–4.62)	3.67 (3.20–4.49)	7.02 (5.79–8.62)	11.2 (10.0–13.9)	15.7 (12.1–24.4)	288

^aThe limit of detection (not corrected for creatinine) for survey years 2001–2002, 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 1.1, 0.5, 0.7, 0.6, 0.2, 0.2, and 0.2 µg/L, respectively.

CI = confidence interval; MEOHP = mono-2-ethyl-5-oxyhexyl phthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-15. Uncorrected Urinary MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Total	2003–2004	34.7 (31.0–38.9)	33.0 (29.1–37.4)	71.8 (61.7–84.8)	168 (133–240)	339 (235–506)	2,605
	2005–2006	38.6 (34.7–42.9)	35.6 (31.1–40.3)	79.7 (70.9–92.9)	211 (180–246)	386 (311–484)	2,548
	2007–2008	33.3 (28.7–38.6)	31.2 (27.1–36.0)	69.5 (55.8–86.3)	153 (120–216)	308 (220–397)	2,604
	2009–2010	20.7 (18.5–23.3)	20.4 (18.1–23.5)	39.9 (34.9–45.5)	78.4 (67.2–95.5)	127 (99.5–199)	2,749
	2011–2012	12.9 (12.0–13.9)	13.5 (12.4–14.8)	25.3 (23.6–26.7)	43.9 (40.0–51.1)	68.9 (60.4–75.2)	2,489
	2013–2014	10.5 (9.71–11.5)	11.1 (10.1–12.0)	20.0 (17.9–22.5)	36.4 (31.4–42.0)	50.7 (46.5–58.7)	2,685
Age group							
6–11 years	2003–2004	58.2 (44.7–75.6)	51.6 (39.2–67.6)	112 (71.4–182)	314 (124–524)	391 (238–781)	342
	2005–2006	57.4 (50.2–65.7)	53.5 (42.7–67.2)	94.7 (83.2–108)	200 (154–247)	297 (196–548)	356
	2007–2008	46.6 (39.2–55.3)	44.1 (34.9–54.7)	92.7 (64.7–112)	156 (117–278)	357 (176–441)	389
	2009–2010	27.7 (24.7–31.2)	29.4 (25.4–33.2)	48.5 (39.0–60.2)	87.1 (64.4–102)	118 (87.1–202)	415
	2011–2012	18.8 (16.1–21.9)	21.6 (17.3–24.1)	36.8 (31.5–41.4)	63.5 (51.5–71.5)	81.5 (68.1–95.1)	396
	2013–2014	18.2 (15.5–21.4)	18.1 (14.2–23.4)	33.9 (28.3–39.6)	54.3 (45.5–71.8)	81.1 (58.5–142)	409
12–19 years	2003–2004	44.6 (36.8–54.0)	42.7 (38.4–47.6)	86.5 (67.3–108)	220 (120–397)	448 (235–808)	729
	2005–2006	52.9 (43.0–65.2)	46.7 (39.4–61.6)	114 (85.6–173)	314 (195–515)	560 (324–1180)	702
	2007–2008	44.3 (35.2–55.9)	38.9 (28.9–49.3)	97.3 (64.3–127)	247 (140–456)	476 (231–977)	401
	2009–2010	26.2 (22.4–30.6)	25.7 (21.2–30.0)	50.1 (38.3–57.7)	90.3 (58.2–162)	147 (83.9–349)	420
	2011–2012	14.3 (11.3–18.0)	14.0 (11.6–17.5)	28.4 (23.5–36.8)	59.3 (47.1–70.2)	74.6 (59.3–135)	388
	2013–2014	13.6 (11.8–15.7)	13.8 (11.2–16.2)	25.7 (20.0–29.9)	44.4 (31.2–64.6)	64.9 (48.6–99.5)	462
≥20 years	2003–2004	31.3 (28.6–34.4)	29.2 (26.2–33.0)	63.5 (56.5–73.9)	157 (130–187)	312 (199–457)	1,534
	2005–2006	35.1 (31.5–39.0)	31.3 (28.1–35.7)	72.7 (65.4–82.4)	193 (163–237)	377 (285–460)	1,490
	2007–2008	30.7 (26.4–35.8)	29.2 (24.3–34.4)	63.2 (51.6–80.0)	145 (109–206)	286 (182–378)	1,814
	2009–2010	19.4 (17.0–22.0)	18.8 (15.7–22.3)	37.7 (32.8–44.0)	76.9 (63.1–92.8)	126 (96.1–197)	1,914
	2011–2012	12.2 (11.3–13.3)	13.0 (11.4–14.4)	23.8 (21.8–25.8)	40.2 (36.9–43.9)	61.8 (52.1–73.6)	1,705
	2013–2014	9.57 (8.84–10.4)	10.1 (9.10–11.3)	18.3 (16.6–19.8)	32.1 (27.5–37.8)	46.4 (39.5–51.8)	1,814
Gender							
Males	2003–2004	37.9 (33.1–43.5)	34.7 (30.0–39.5)	73.7 (60.8–91.9)	187 (133–300)	388 (222–660)	1,250
	2005–2006	43.6 (38.1–49.8)	39.7 (31.9–46.2)	87.0 (76.8–103)	260 (188–308)	460 (347–670)	1,270
	2007–2008	34.4 (29.3–40.5)	31.5 (27.3–35.7)	65.1 (52.1–87.4)	161 (120–217)	321 (213–422)	1,294
	2009–2010	23.4 (20.7–26.4)	23.0 (20.2–25.6)	44.9 (40.1–51.0)	87.1 (70.3–110)	162 (107–288)	1,399
	2011–2012	14.3 (13.0–15.6)	14.6 (12.7–16.4)	24.7 (23.1–27.6)	45.7 (40.2–54.9)	71.5 (59.8–88.4)	1,259
	2013–2014	11.1 (9.98–12.3)	11.6 (10.6–12.5)	19.3 (17.1–22.3)	35.1 (29.3–44.0)	50.7 (47.4–58.4)	1,285

5. POTENTIAL FOR HUMAN EXPOSURE

Table 5-15. Uncorrected Urinary MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Females	2003–2004	31.9 (28.1–36.2)	31.3 (27.5–35.8)	69.3 (58.9–81.9)	154 (128–199)	312 (182–441)	1,355
	2005–2006	34.3 (30.4–38.6)	31.7 (28.0–36.6)	71.0 (63.0–85.3)	192 (156–217)	309 (251–386)	1,278
	2007–2008	32.3 (27.8–37.5)	31.0 (26.4–38.3)	73.0 (56.9–90.2)	147 (112–220)	297 (185–420)	1,310
	2009–2010	18.5 (16.2–21.0)	18.8 (15.8–22.4)	34.7 (29.3–41.5)	72.9 (56.3–93.2)	111 (90.3–146)	1,350
	2011–2012	11.8 (10.8–12.9)	12.4 (11.2–13.7)	25.6 (23.4–26.6)	42.3 (37.4–49.5)	64.9 (54.4–76.8)	1,230
	2013–2014	10.1 (9.05–11.2)	10.2 (8.90–1.7)	20.6 (18.4–23.2)	36.9 (31.5–41.8)	51.7 (41.3–64.9)	1,400
Race/ethnicity							
Mexican Americans	2003–2004	31.9 (27.1–37.6)	31.5 (26.8–37.4)	57.4 (45.9–71.8)	116 (86.0–162)	175 (133–355)	652
	2005–2006	39.4 (30.3–51.2)	34.2 (24.1–47.1)	74.6 (54.0–111)	220 (124–338)	394 (222–673)	637
	2007–2008	36.7 (30.6–44.0)	31.3 (27.0–35.7)	71.9 (54.9–91.7)	162 (123–241)	321 (209–477)	531
	2009–2010	26.2 (22.5–30.4)	25.8 (22.3–29.8)	51.0 (41.3–57.4)	92.6 (74.5–122)	160 (113–245)	566
	2011–2012	15.8 (12.7–19.6)	14.9 (12.7–18.3)	28.1 (22.6–34.3)	51.3 (42.5–71.3)	72.6 (56.6–109)	316
	2013–2014	13.4 (11.0–16.2)	13.1 (12.1–14.2)	23.4 (18.0–30.2)	44.4 (31.8–67.9)	67.9 (42.0–95.0)	438
Non- Hispanic blacks	2003–2004	42.6 (37.0–49.2)	38.3 (33.8–46.9)	82.5 (68.7–103)	191 (146–246)	339 (244–468)	699
	2005–2006	46.6 (41.3–52.5)	40.3 (35.6–46.1)	96.3 (76.6–132)	256 (208–347)	455 (328–528)	678
	2007–2008	35.0 (31.1–39.4)	35.6 (30.7–38.8)	71.6 (59.1–84.5)	151 (113–192)	235 (184–338)	597
	2009–2010	21.9 (18.6–25.7)	22.3 (18.2–25.6)	40.5 (35.7–47.1)	77.0 (61.1–97.1)	127 (76.0–268)	516
	2011–2012	16.4 (14.5–18.5)	16.2 (14.4–18.8)	30.7 (26.0–36.6)	60.9 (47.5–69.9)	78.1 (68.2–96.7)	665
	2013–2014	11.5 (9.62–13.8)	12.7 (10.4–14.6)	21.5 (18.5–25.6)	36.4 (30.4–48.6)	58.5 (42.1–71.8)	609
Non- Hispanic whites	2003–2004	33.8 (30.1–37.9)	32.1 (27.6–37.5)	72.4 (62.0–87.7)	167 (133–240)	354 (220–560)	1,088
	2005–2006	37.0 (33.4–41.0)	35.4 (30.4–40.9)	79.7 (70.0–93.3)	203 (174–237)	380 (284–484)	1,038
	2007–2008	32.0 (26.7–38.3)	30.4 (24.5–38.0)	67.6 (52.5–90.9)	145 (105–244)	316 (197–476)	1,077
	2009–2010	19.6 (17.2–22.2)	19.3 (16.4–23.1)	39.3 (33.0–45.3)	75.0 (61.9–95.7)	120 (95.5–197)	1,206
	2011–2012	11.8 (10.9–12.9)	12.5 (11.0–14.6)	23.8 (21.8–25.7)	39.9 (34.8–44.2)	55.8 (47.1–76.8)	813
	2013–2014	9.81 (8.92–10.8)	10.3 (9.20–11.4)	19.3 (16.5–22.0)	34.1 (28.7–41.0)	49.1 (43.1–54.8)	987
All Hispanics	2011–2012	16.0 (14.0–18.2)	15.2 (12.9–18.2)	30.1 (24.5–37.8)	59.8 (50.0–68.9)	76.0 (70.2–104)	571
	2013–2014	13.1 (11.7–14.7)	13.4 (12.3–14.4)	23.1 (20.6–26.5)	41.6 (35.1–54.1)	63.5 (45.5–75.4)	690

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Table 5-15. Uncorrected Urinary MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/L)	Selected percentiles (95% CI) (µg/L)				Sample size
			50 th	75 th	90 th	95 th	
Asians	2011–2012	12.0 (10.2–14.2)	11.7 (10.2–13.7)	23.6 (19.5–27.7)	51.1 (36.5–70.0)	80.5 (58.7–138)	352
	2013–2014	9.01 (7.74–10.5)	9.20 (7.20–11.1)	16.5 (14.3–18.5)	27.3 (22.9–31.4)	39.4 (29.4–63.2)	289

^aThe limit of detection for survey years 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 0.3, 0.6, 0.5, 0.2, 0.2, and 0.4 µg/L, respectively.

CI = confidence interval; MECPP = mono-2-ethyl-5-carboxypentylphthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

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Table 5-16. Creatinine Corrected MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Total	2003–2004	32.6 (29.6–36.0)	27.0 (24.3–30.6)	54.6 (48.0–63.5)	139 (109–186)	251 (192–356)	2,605
	2005–2006	37.6 (33.7–42.0)	32.2 (29.5–37.0)	67.5 (58.2–80.8)	168 (139–209)	290 (261–328)	2,548
	2007–2008	33.6 (29.7–38.0)	29.1 (25.8–32.2)	58.7 (49.6–68.8)	138 (109–164)	233 (178–319)	2,604
	2009–2010	21.6 (19.0–24.7)	19.2 (16.8–22.1)	33.7 (30.0–40.0)	69.4 (54.0–84.3)	121 (85.1–184)	2,749
	2011–2012	14.7 (13.8–15.7)	14.1 (12.9–15.1)	22.7 (21.1–25.3)	38.9 (34.5–44.3)	59.8 (54.5–63.5)	2,487
	2013–2014	10.6 (9.68–11.6)	10.2 (9.32–11.0)	16.2(14.5–18.2)	28.5 (25.3–32.0)	40.3 (35.2–46.0)	2,684
Age group							
6–11 years	2003–2004	61.5 (49.0–77.2)	52.2 (41.6–73.8)	104 (74.2–140)	210 (111–500)	372 (192–988)	342
	2005–2006	63.2 (55.6–71.9)	54.2 (48.1–63.6)	92.8 (82.6–111)	160 (124–247)	312 (172–480)	356
	2007–2008	57.4 (49.2–66.9)	49.6 (42.7–61.5)	94.2 (79.5–122)	185 (138–294)	376 (188–404)	389
	2009–2010	36.1 (32.4–40.3)	33.6 (32.3–36.8)	55.4 (43.9–66.9)	88.7 (69.2–113)	121 (90.6–224)	415
	2011–2012	26.8 (23.6–30.3)	26.2 (22.6–29.4)	41.5 (36.5–47.2)	63.5 (57.5–70.7)	84.4 (71.3–107)	395
	2013–2014	23.0 (20.4–26.0)	21.1 (18.4–24.7)	33.8 (29.6–41.3)	64.7 (41.9–97.3)	97.3 (60.0–180)	409
12–19 years	2003–2004	33.4 (28.7–38.7)	27.1 (23.9–32.0)	55.0 (43.8–83.8)	168 (92.5–289)	294 (159–387)	729
	2005–2006	39.5 (32.5–47.8)	33.9 (27.9–41.0)	72.7 (56.9–96.7)	197 (106–327)	385 (216–531)	702
	2007–2008	34.5 (28.1–42.3)	28.0 (23.2–35.0)	65.9 (47.5–85.9)	159 (90.1–233)	246 (159–489)	401
	2009–2010	21.1 (17.4–25.5)	18.2 (15.6–20.7)	33.2 (25.5–43.3)	64.9 (42.7–158)	158 (58.4–246)	420
	2011–2012	13.9 (11.7–16.5)	12.5 (10.7–15.5)	22.2 (16.5–29.2)	41.4 (30.3–60.8)	70.9 (46.9–92.9)	388
	2013–2014	11.0 (9.68–12.6)	10.2 (8.77–12.2)	16.9 (14.4–20.5)	28.9 (23.2–35.5)	40.7 (33.8–48.8)	462
≥20 years	2003–2004	30.1 (27.7–32.7)	25.1 (22.9–27.6)	49.1 (44.1–55.2)	126 (101–154)	237 (191–315)	1,534
	2005–2006	35.2 (31.2–39.6)	29.8 (26.1–33.3)	60.8 (54.2–74.0)	167 (131–206)	279 (247–322)	1,490
	2007–2008	31.5 (27.8–35.8)	27.8 (24.1–30.8)	53.1 (45.0–65.4)	128 (101–155)	214 (162–319)	1,814
	2009–2010	20.5 (17.9–23.5)	18.2 (15.9–20.5)	31.7 (27.4–36.8)	65.0 (52.1–80.6)	118 (81.3–173)	1,914
	2011–2012	13.9 (13.0–14.8)	13.3 (12.4–14.4)	21.3 (19.5–22.8)	33.9 (30.6–39.0)	53.7 (42.9–63.2)	1,704
	2013–2014	9.65 (8.80–10.6)	9.62 (8.61–10.4)	14.5 (13.0–16.3)	24.4 (21.0–28.3)	34.2 (28.9–39.6)	1,813
Gender							
Males	2003–2004	29.8 (26.8–33.1)	23.5 (21.4–27.1)	50.7 (42.2–61.7)	132 (98.0–191)	248 (159–422)	1,250
	2005–2006	35.0 (30.7–39.9)	29.0 (25.7–32.1)	69.3 (54.2–82.9)	172 (141–210)	301 (249–376)	1,270
	2007–2008	29.0 (25.3–33.2)	25.1 (21.7–28.8)	47.6 (40.8–57.4)	120 (91.7–157)	210 (157–331)	1,294
	2009–2010	21.0 (18.4–23.9)	18.4 (16.1–20.4)	33.1 (29.2–38.5)	70.2 (53.4–94.6)	125 (92.8–210)	1,399
	2011–2012	13.3 (12.6–14.1)	12.5 (11.6–13.3)	20.1 (17.8–21.6)	35.0 (31.0–38.8)	56.4 (46.4–62.4)	1,258
	2013–2014	9.32 (8.50–10.2)	9.05 (8.18–9.88)	14.1 (12.6–15.5)	24.7 (21.0–29.0)	37.1 (33.8–41.9)	1,284

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Table 5-16. Creatinine Corrected MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Females	2003–2004	35.5 (31.6–40.0)	30.6 (26.4–35.5)	58.3 (48.8–71.8)	144 (108–192)	251 (192–349)	1,355
	2005–2006	40.3 (36.2–44.9)	36.7 (31.6–40.0)	66.1 (58.2–83.7)	168 (127–206)	279 (240–341)	1,278
	2007–2008	38.7 (34.5–43.3)	32.8 (30.0–36.4)	67.8 (58.5–80.0)	147 (117–176)	266 (176–379)	1,310
	2009–2010	22.3 (19.3–25.7)	20.5 (17.2–23.9)	34.9 (30.0–41.7)	67.0 (53.5–80.8)	104 (80.8–170)	1,350
	2011–2012	16.2 (14.6–18.0)	15.8 (14.1–17.7)	25.8 (22.2–29.4)	44.1 (37.6–50.8)	63.2 (54.3–72.4)	1,229
	2013–2014	11.9 (10.6–13.4)	11.4 (10.1–12.8)	18.2 (15.9–21.6)	30.1 (27.2–35.2)	42.5 (35.2–57.4)	1,400
Race/ethnicity							
Mexican Americans	2003–2004	28.8 (25.4–32.6)	24.7 (22.4–26.3)	46.7 (39.0–56.3)	94.7 (73.2–137)	152 (118–238)	652
	2005–2006	35.5 (28.7–43.7)	29.8 (25.5–34.9)	61.5 (48.4–86.2)	165 (105–201)	278 (181–501)	637
	2007–2008	35.8 (29.1–44.0)	30.6 (23.2–38.6)	64.1 (47.2–84.8)	151 (92.3–240)	286 (198–402)	531
	2009–2010	26.0 (22.5–30.0)	23.8 (21.3–27.0)	40.1 (35.0–52.3)	88.8 (69.5–104)	148 (100–203)	566
	2011–2012	17.8 (14.6–21.6)	15.3 (12.7–19.8)	28.4 (21.9–39.0)	53.4 (44.7–62.9)	80.0 (52.4–115)	316
	2013–2014	13.6 (12.1–15.3)	12.1 (11.3–13.6)	22.0 (17.3–27.4)	39.5 (30.0–51.6)	55.9 (41.5–66.7)	438
Non- Hispanic blacks	2003–2004	30.3 (27.7–33.2)	27.0 (23.2–30.7)	51.1 (41.6–64.0)	135 (100–161)	212 (173–252)	699
	2005–2006	32.7 (28.9–37.1)	27.1 (22.4–31.5)	62.4 (53.1–76.0)	166 (119–260)	370 (231–429)	678
	2007–2008	27.2 (24.3–30.5)	23.5 (21.9–26.1)	49.1 (37.7–58.6)	103 (85.9–132)	178 (140–259)	597
	2009–2010	15.9 (12.6–19.9)	14.5 (11.5–18.8)	26.3 (19.5–32.9)	48.0 (32.6–66.4)	70.7 (46.1–209)	516
	2011–2012	12.8 (11.3–14.4)	11.8 (10.6–13.0)	20.8 (18.0–25.0)	40.4 (29.6–50.3)	59.4 (46.2–75.6)	665
	2013–2014	8.51 (7.48–9.68)	8.22 (7.07–9.65)	14.2 (12.1–16.4)	23.9 (20.9–27.1)	35.3 (27.3–41.6)	609
Non- Hispanic whites	2003–2004	33.4 (29.5–37.7)	27.0 (23.5–31.6)	56.8 (48.6–69.4)	145 (109–198)	294 (193–385)	1,088
	2005–2006	39.0 (34.9–43.4)	34.3 (30.3–39.1)	69.5 (59.5–83.7)	182 (143–214)	284 (237–324)	1,038
	2007–2008	33.4 (28.4–39.3)	29.2 (25.1–34.6)	57.7 (46.6–72.4)	138 (101–166)	233 (164–338)	1,077
	2009–2010	22.0 (19.1–25.3)	19.4 (16.8–22.9)	34.0 (29.3–42.5)	67.6 (52.5–84.3)	113 (81.3–188)	1,206
	2011–2012	14.3 (13.1–15.7)	13.9 (12.6–15.2)	21.8 (19.7–24.6)	34.3 (30.1–42.2)	54.9 (42.9–60.9)	811
	2013–2014	10.3 (9.23–11.6)	10.0 (9.02–11.1)	15.4 (13.0–18.6)	27.2 (21.6–32.0)	35.9 (30.0–48.2)	987
All Hispanics	2011–2012	17.9 (16.2–19.7)	15.7 (14.1–18.1)	30.0 (25.8–33.4)	53.4 (44.8–64.0)	80.0 (59.0–107)	571
	2013–2014	13.0 (12.0–14.1)	12.1 (11.3–12.9)	20.7 (17.7–23.8)	37.6 (30.9–42.5)	54.1 (42.5–64.4)	690

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Table 5-16. Creatinine Corrected MECPP Concentrations for the U.S. Population from NHANES 2003–2014

	Survey years ^a	Geometric mean (95% CI) (µg/g of creatinine)	Selected percentiles (95% CI) (µg/g of creatinine)				Sample size
			50 th	75 th	90 th	95 th	
Asians	2011–2012	16.1 (13.7–18.9)	14.7 (12.3–17.2)	26.4 (21.7–33.0)	51.2 (41.5–76.9)	90.0 (51.0–142)	352
	2013–2014	11.5 (10.3–12.8)	10.8 (8.94–12.1)	18.2 (15.1–20.9)	30.7 (27.2–38.0)	40.8 (32.2–65.0)	288

^aThe limit of detection (not corrected for creatinine) for survey years 2003–2004, 2005–2006, 2007–2008, 2009–2010, 2011–2012, and 2013–2014 were 0.3, 0.6, 0.5, 0.2, 0.2, and 0.4 µg/L, respectively.

CI = confidence interval; MECPP = mono-2-ethyl-5-carboxypentylphthalate; NHANES = National Health and Nutrition Examination Survey

Source: CDC 2018

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individuals fasting on bottled water only over a 48-hour period showed a rapid decline to levels 5–10 times lower than initial levels within 24 hours of the fast and remained low thereafter; levels rose again after food consumption, showing that food was a significant source of exposure. Some attempts have been made to estimate exposures of DEHP to the general population in the United States (3–30 $\mu\text{g}/\text{kg}$ body weight/day) through ingestion that is based on current use patterns for DEHP (NTP 2000), but more information is still needed. Data obtained from a study in which phthalates were measured in 72 food items purchased in Albany, New York yielded an estimated mean adult intake of approximately 0.673 $\mu\text{g}/\text{kg}/\text{day}$ for DEHP (Schechter et al. 2013).

Much of the current literature on DEHP contamination of foodstuffs comes from outside the United States or does not reflect typical exposures of U.S. consumers; therefore, it is uncertain whether and for which products this information can be used in U.S.-centered exposure or risk calculations. Examples of available data include: migration of DEHP into bottled water, Saudi Arabia (Fayad et al. 1997); migration of DEHP from caps into foods, Italy (Gramiccioni et al. 1990); migration of DEHP from a plastic bag containing contaminated corn in a laboratory (the corn was not intended for consumer use), Canada/France (Cohen et al. 1991); migration of DEHP from PVC gloves to prepared food, Japan (Tsumura et al. 2001); post-secretory migration of DEHP during milk processing and storage, Germany (Bluthgen 2000); and migration of DEHP into food simulants, Brazil (Morelli-Cardoso et al. 1999). Further, while the FDA allows the use of DEHP in food contact applications (e.g., can coatings [FDA 1999g]; adhesives [FDA 1999a]; defoaming agent in paper manufacture [FDA 1999e]; as a flow promoter at no more than 3% in acrylic and modified acrylic single and repeated use containers [FDA 1999c]; in cellophane used for food packaging at a concentration not to exceed 5% [FDA 1999b]; and as a surface lubricant in the processing of metal foil at a concentration not to exceed 0.015 mg/in^2 of metal surface [FDA 1999d]), it is not clear if industry currently uses DEHP in these applications. Thus, the uncertainty associated with current concentrations in food (as outlined above) makes quantifying intakes speculative. This might be especially true given the recent activity (as noted in Section 5.2.3) in eliminating phthalates from some consumer products.

While it is likely that food represents the major, chronic route of exposure to DEHP for the general population, the highest degree of acute exposure to individuals occurs in hospital patients through hospital equipment plastics, such as tubing and intravenous bags made using PVC. The amount of DEHP detected in liquids that have passed through hospital equipment are several orders of magnitude higher than the amounts detected in water and food samples (Inoue et al. 2005; Jaeger and Rubin 1972; Rock et al. 1978)—see Section 5.5.4 for further discussion. However, people who require only occasional medical

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care for conditions that do not require intravenous administration of fluids or medication, the use of medical devices, the use of invasive medical procedures, or instrumentation have a lower risk of exposure than people with chronic conditions who require regular treatment or the use of medical devices. Individuals with chronic conditions are discussed in Section 5.7 (Populations with Potentially High Exposures).

Oral exposure from drinking water is not expected to be a significant route of exposure (Doull et al. 1999; Huber et al. 1996; NTP 2000) based on a mean concentration of 0.55 $\mu\text{g/L}$ for DEHP in drinking water (NTP 2006).

Dermal exposure to DEHP can occur when items containing DEHP as a plasticizer are handled. Schwowe and Reid (1988) noted that DEHP migrated into dry materials in contact with PVC containing DEHP. However, the data available in this study did not indicate how much DEHP will be transferred. A study of the migration of DEHP from PVC film to rat skin found that the mean dermal uptake of DEHP was small, only 0.24 $\mu\text{g/cm}^2\text{-hour}$ (Deisinger et al. 1998), a rate that is likely to be 2–4 times faster than is expected for human skin (Barber et al. 1992; Scott et al. 1987). In a study measuring the levels of phthalates in skin wipe samples from 20 Chinese adults not deliberately exposed to phthalates, mean DEHP concentrations collected from the skin were 678 $\mu\text{g/m}^2$ for the forehead, 867–884 $\mu\text{g/m}^2$ for the left and right forearm, 1,725–1,840 $\mu\text{g/m}^2$ for the left and right back-of-hand, and 4,104–4,155 $\mu\text{g/m}^2$ for the left and right palm (Gong et al. 2014). From this study, an estimated median total dermal adsorption from skin surface lipids of 0.66 $\mu\text{g/kg/day}$ was determined for DEHP, accounting for roughly 10–20% of total daily uptake. Repeated sampling over a month for a subsample (six adults) showed that levels at measured body locations did not significantly change. Washing hands with soap and water reduced palm levels to about half.

Inhalation exposure can occur from breathing ambient air and indoor air and is not considered to be a primary or significant route of exposure to DEHP. Huber et al. (1996) and Doull et al. (1999) have suggested, based on monitoring studies from the 1970s and 1980s, that inhalation exposures from breathing ambient air are low. During a study in which 96 women living in New York City wore personal ambient air samplers for 2 consecutive days, DEHP was detected in all air samples at a mean concentration of 0.18 $\mu\text{g/m}^3$ (Adibi et al. 2008). Ambient air studies found in the available literature reported concentrations that span a relatively narrow range, even in industrialized areas (Section 5.5.1); although industrial areas appeared to have higher concentrations in some cases. Thurén and Larsson (1990) reported that higher concentrations of DEHP were seen adjacent to a facility using DEHP, but

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these concentrations fell off rapidly. Thus, it is anticipated that people living near DEHP use and disposal areas might be exposed to elevated levels, but it is unclear how much higher these concentrations might be. It is further anticipated that use facilities where DEHP is actively used, such as DEHP production or PVC manufacturing facilities, will emit more DEHP into the ambient environment (e.g., through air-borne particulates or water) than storage or disposal facilities because of the tendency of DEHP to sorb to organic matter in the soil or sediment.

Occupational exposure to DEHP might be important during the manufacture and processing of this compound, mostly via inhalation, essentially in the form of an aerosol (IARC 2012). Workers might be exposed to relatively high concentrations of DEHP during the compounding of this plasticizer with resins and the manufacture of PVC plastic products. The National Institute for Occupational Safety and Health (NIOSH) estimated that about 340,000 workers (of which approximately 106,900 were female) were potentially exposed to DEHP in the early 1980s (NOES 1990). Workplace air levels of DEHP ranging from 0.02 to 4.1 mg/m³ were reported at facilities using or manufacturing the compound (Hill et al. 2001; IARC 1982; Liss et al. 1985). These levels are below the current OSHA Permissible Exposure Limit (PEL) for DEHP for an 8-hour workday of 5 mg/m³ (OSHA 2019a, 2019b, 2019c).

Exposures of phthalate and PVC production workers to DEHP are estimated to be typically less than 143 and 286 µg/kg body weight/workday, respectively (NTP 2000). Hines et al. (2009b, 2011) studied four DEHP urinary phthalate metabolite concentrations among 156 workers in 2003–2005 from eight industry sectors. Mean end-shift concentrations in plastic industries in µg/g creatinine were 3.75–25.4 (phthalate manufacturing), 16.7–158 (PVC film), 10.2–34.6 (vehicle filters), 12.1–124 (PVC compounding), 5.41–36.2 (rubber hoses), 5.37–69.3 (rubber boots), and 12.1–54.6 (rubber gaskets). In nail salons, mean end-shift concentrations were 17.9–34.4 µg/g creatinine. Mean end-shift concentrations of urinary DEHP metabolites in workers exceeded general population levels by 8-, 6-, and 3-fold in PVC film manufacturing, PVC compounding, and rubber boot manufacturing, respectively, where occupational exposure to DEHP was strongest (Hines et al. 2009b). Daily DEHP intake estimates were 0.6–850 µg/kg/day, where the highest mean intakes occurred in PVC film manufacturing (17 µg/kg/day) and PVC compounding (12 µg/kg/day) (Hines et al. 2011).

Children may be exposed to DEHP orally from mouthing toys and other soft PVC products and from ingestion of food, via inhalation from ambient and indoor air and from ingestion of house dust, and dermally from handling materials containing DEHP. In addition, children are potentially exposed from medical devices via the inhalation, dermal, oral, and intravenous routes. Exposures from medical devices

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will be treated separately in this section. It has been predicted that toddlers and infants are exposed to higher levels of DEHP than adults, with a major portion (as much as 35%) of this exposure resulting from the ingestion of contaminated dust (NTP 2006). It should be noted that assessing exposures to DEHP, and especially children's exposures, is difficult because the uses of DEHP, while constant for many years, have changed over the last 20 years (CPSC 1999; CPSIA 2008; Wilkinson and Lamb 1999). For example, manufacturers stopped using phthalates in teething rings and rattles in early 1999 (CPSC 1999). Further, in 2008, Congress permanently banned DEHP in any amount >0.1% in children's toys and certain child care articles, such as those to help sleeping, feeding, sucking, or teething of children ≤ 3 years old (CPSIA 2008). This change, and others that might be made in the near future, makes an assessment of a child's exposure to DEHP more difficult than would otherwise be the case.

Just as is the case with the general population, food is likely the dominant source of oral exposure to DEHP for children. A Danish study published by Petersen and Breindahl (2000) estimated the dietary intake of DEHP in infants (based on measurements of DEHP in baby food and formula) to be between 0.005 and 0.010 mg/kg body weight. Drinking water is not anticipated to be a significant source of DEHP exposure. DEHP concentrations in human breast milk of 70–160 $\mu\text{g}/\text{kg}$ milk (mean concentration of 93 ± 37.5 $\mu\text{g}/\text{kg}$ milk) and 0–110 $\mu\text{g}/\text{kg}$ milk (mean concentration of 0.034 ± 0.043 $\mu\text{g}/\text{kg}$ milk) have been reported (FDA 2001). Calafat et al. (2004) reported a mean concentration of 7.8 ng/mL milk for MEHP, a DEHP metabolite, in three pooled breast milk samples. However, no information is available relating the concentration of DEHP in human breast milk obtained from women with high occupational exposures to DEHP or exposures that result from medical treatments (e.g., hemodialysis). One study explored the relationship of phthalate metabolites, including those of DEHP, in urine, serum, saliva, and breast milk and potential routes of exposure using samples collected from 33 lactating mothers in North Carolina (Hines et al. 2009a); however, phthalates were detected in <50% of the samples collected across matrices, so a correlation could not be made. Of the total milk samples, only 8, 5, and 2% contained detectable levels of DEHP metabolites MECPP, MEHHP, and MEOHP, respectively, in low ppb concentrations (up to 0.4 $\mu\text{g}/\text{L}$). As previously noted, this study is limited by small sample size and low detection rate.

A source of DEHP exposure for young children by the oral route might be plastic toys. The exposure will be dependent on the time that a child spends mouthing a toy and the DEHP content of the toy. Information on children's mouthing behavior is available and indicates that the behavior is dependent on the age of the child and the items mouthed (CPSC 2001; Juberg et al. 2001). Juberg et al. (2001) found that children spend an average of 23 minutes/day (children between the ages of 0 and 18 months) and 5 minutes/day (children between the ages of 19 and 36 months) mouthing toys and teething rings; these times

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are shorter than the estimated mouthing times (e.g., 1–3 hours) found elsewhere (Health Canada 1998). These average mouthing times provided by Juberg et al. (2001) included children who did not exhibit mouthing behavior. If the averages included only children exhibiting mouthing behavior, then the time spent by these children mouthing teethingers and toys increases to 48 minutes/day (children between the ages of 0 and 18 months) and 41 minutes/day (children between the ages of 19 and 36 months). Juberg et al. (2001) also reported pacifier use to average 108 ± 187 (mean ± 1 standard deviation [SD]) minutes/day for children ages 0–18 months and 126 ± 246 minutes/day for children ages 19–36 months. However, manufacturers have discontinued the use of DEHP in pacifiers, teethingers, rattles, and toys designed for very young children (CPSC 1999). Therefore, the mouthing of pacifiers, teethingers, and toys is not expected to be a significant route of exposure of young children to DEHP. Yet, families might hand down toys containing DEHP from older children rather than buy new toys that contain no DEHP. At the present time, however, sufficient information is not available to quantify these exposures.

Some research has been conducted to examine the migration of DEHP and other plasticizers from PVC into saliva. Steiner et al. (1998) reported that migration of DEHP from PVC into a saliva simulant was dependent on the contact time and agitation of the test matrix. *In vivo* studies of the migration of DEHP into human saliva from four adult volunteers chewing PVC balls (185 mg DEHP/g) showed a migration rate of $44.4 \mu\text{g}/10 \text{ cm}^2/\text{hour}$ (Niino et al. 2001). However, no other studies, especially in children, are available evaluating DEHP migration rates in toys.

Other potential sources of oral exposure for young children, as well as dermal exposure to all children, include general household items made from PVC including dolls, furniture upholstery, floor tiles, shower curtains, and tablecloths (all of which are available for mouthing by children in addition to touching). In addition, young children might be exposed to DEHP when wearing such items as rainwear and shoes made from PVC. Dermal uptake of DEHP from PVC film to rat skin was found to be low, only $0.24 \mu\text{g}/\text{cm}^2\text{-hour}$ (Deisinger et al. 1998), but is expected to be 2–4 times lower for human skin (Barber et al. 1992; Scott et al. 1987). Gong et al. (2014) reported an estimated median total dermal adsorption from skin surface lipids of $0.66 \mu\text{g}/\text{kg}/\text{day}$ for DEHP for adults. Oral exposure also might occur when PVC items containing DEHP are handled by children, and then the children's hands are mouthed. However, no specific reference to DEHP transfer from items to skin was found in the available literature. Therefore, sufficient information is not available to assess this route of exposure to DEHP.

Children might have inhalation exposures from both vapor and particle bound DEHP as well as oral exposure to DEHP from inhalation of large particles containing DEHP followed by deposition in the

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upper airways and swallowing (Hill et al. 2001). Øie et al. (1997) reported that sedimented dust samples from 38 dwellings in Oslo, Norway (including samples taken from sheets in a child's bed and floor in a child's bedroom) contained an average of 640 µg/g sedimented dust (100–1,610 µg/g), while suspended particulate matter from six dwellings contained an average of 600 µg/g (24–94 µg/g). The authors noted that exposure to particle-bound DEHP is 0.4–1.2 µg/day for adults, but suggested that children, and especially small children, are “subject to the highest exposure risk” because they usually have small rooms that have higher surface to volume ratios and few doors or windows. In a study of 390 homes in Sweden, DEHP was found in nearly all dust samples collected (99.1%) from 346 children's bedrooms at mean and median concentrations of 1.31 and 0.77 mg/g dust, respectively (Bornehag et al. 2005). The authors found an association between DEHP concentrations in dust and the amount of PVC used as flooring and wall material, where bedrooms with PVC flooring (n=186) had a median DEHP concentration of 0.868 mg/g dust as opposed to a median concentration of 0.70 mg/g dust in bedrooms with no PVC flooring (n=157). Children's exposures to DEHP from inhalation of outdoor air is likely small because of the relatively low ambient concentrations (Doull et al. 1999; Huber et al. 1996). While the database of outdoor concentrations is dated (1970s through the 1980s), the concentrations appear to be very consistent both spatially and temporally.

A possible exception to the anticipated low exposure from inhalation to outdoor air might be in the vicinity of hazardous waste sites containing large concentrations of DEHP or use facilities. DEHP has a low volatility and is not expected to enter the air extensively; nonetheless, Thurén and Larsson (1990) noted higher concentrations of DEHP near a facility that used it, indicating that somewhat higher concentrations might be anticipated near use or storage facilities. Children living near the vicinity of one of these facilities might be exposed to somewhat elevated concentrations of DEHP, although exact concentrations are not known.

Children's exposures to DEHP during medical procedures have been reported (Hill et al. 2001; Karle et al. 1997; Latini and Avery 1999; NTP 2000; Plonait et al. 1993; Shneider et al. 1991). Shneider et al. (1991) reported that serum DEHP concentrations varied depending on the nature of the treatment. They reported that for an infant cardiopulmonary bypass, pediatric hemodialysis, exchange transfusion, and ECMO, serum DEHP concentrations ranges were 1.1–5.1, 0.4–4.2, 5.4–21.5, and 18–98 µg/mL, respectively. Karle et al. (1997) confirmed this study but reported lower concentrations. The authors reported the results of blood DEHP concentrations using three different ECMO circuit designs (small surface area, larger surface area, and small surface area but with heparin-bonded tubing). The results indicated that DEHP leaches from ECMO circuits and that the exposure potential is correlated with the

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surface area of the tubing. There was almost no exposure for patients using the heparin-bonded circuit. After 3 days, DEHP concentrations in 18 infants averaged 4.9 µg/mL; the highest level seen was 8.3±5.7 µg/mL. Karle et al. (1997) calculated that DEHP exposures during ECMO therapy averaged 1.2 mg/kg (2.0 mg/kg maximum) for a 3-day exposure, based on an average patient weight of 3.3 kg and an average blood volume of 800 mL for the 18 infants studied. Patients treated for longer periods did not have higher DEHP concentrations during treatment. The study authors also reported that DEHP concentrations were below the detection limit in all patients before and after decannulation.

Latini and Avery (1999) reported that 60–120 mg of DEHP/g of tube was removed from endotracheal tubes during use (range of 44 samples). Plonait et al. (1993) studied 16 newborn infants receiving blood exchange transfusions. The authors calculated exposures of 1.2–22.6 mg/kg-body weight, based on the volume of blood transfused and the mean DEHP concentration in the plasma of the blood units. The study authors reported that for three infants, DEHP eliminated in the waste (exchanged) blood accounted for 12.5, 22.9, and 26.5% of the DEHP accumulated during transfusions, respectively (further details on this analysis were not available). The authors reported that no correlation was found between the volume of blood transfused and the serum DEHP concentration immediately after the transfusion. There was also no correlation between the concentration of DEHP in the plasma and the storage time of the red cell bag. The authors reported that serum DEHP concentrations decreased rapidly after the transfusion was complete. Plonait et al. (1993) also reported that ethylhexanoic acid concentrations in the urine of infants undergoing transfusion therapy was below the detection limit (45 ng/mL) before or during the transfusion but ranged from 50 to 416 ng/mL (median 130 ng/mL) in six infants 6 hours after the transfusion. Peak levels occurred within the first 18 hours, and then declined to close to the detection limit where they remained for 96 hours. Finally, these authors noted that for two infants, DEHP concentrations appeared to accumulate, resulting in higher concentrations in the post-exchange serum than the average DEHP concentration in the blood received by the patients.

5.7 POPULATIONS WITH POTENTIALLY HIGH EXPOSURES

Several population subgroups might have above-average exposure to DEHP. These include hemophiliacs and others who require frequent blood transfusions, dialysis patients who might be exposed to DEHP leached from the dialysis tubing (Section 5.5.4), and preterm infants (Doull et al. 1999; FDA 2001; Huber et al. 1996; Latini 2000; NTP 2000; Tickner et al. 2001). Estimates of exposure levels indicate that hemophiliacs might be exposed up to 1–2 mg/day and dialysis patients might receive average doses of 40 mg/day (Pollack et al. 1985b; Wams 1987). Faouzi et al. (1999) estimated that dialysis patients

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received an average of 75 mg of DEHP per treatment and an average of almost 12 g of DEHP over a 1-year period (assuming dialysis treatments 3 times a week). Adult exposures to DEHP from hemodialysis have been estimated at <5–155 mg/day or <0.1–3.1 mg/kg/day and can vary considerably between patients (Dine et al. 2000; FDA 2001; Huber et al. 1996; NTP 2000). Infants receiving exchange transfusions might be exposed to >4 mg/kg/day (FDA 2001; Sjöberg et al. 1985c), based on a worst-case scenario. Plonait et al. (1993) reported higher plasma concentrations than those in the Sjöberg et al. (1985c) study, but the blood units used had a lower initial DEHP concentration. Plonait et al. (1993) suggest that this can be explained by pauses during the exchange transfusion during the Sjöberg et al. (1985c) study, which resulted in a lowering of the DEHP concentration. Faouzi et al. (1994) reported that administration of teniposide is sometimes associated with a nonionic surfactant polyoxyethylated castor oil. The presence of this surfactant increases the concentration of DEHP that is leached from the PVC bags into the administered solution. The authors reported that 52 mg was extracted at 48-hour room temperature storage. Preterm infants can be exposed to DEHP at levels estimated to be as high as 10–20 mg/day during the course of their care (Loff et al. 2000). Measured concentrations of DEHP in TPN solutions (423 ± 47 $\mu\text{g/mL}$), blood products (platelet-rich plasma, 13.9 ± 2.5 $\mu\text{g/mL}$; fresh frozen plasma, 24.9 ± 17 $\mu\text{g/mL}$), and selected drugs (propofol, 655 ± 96 $\mu\text{g/mL}$) have been obtained in these solutions/products as a consequence of contact with PVC bags and tubing. Inoue et al. (2005) reported that the maximum exposure to DEHP released from blood bags would be 0.7 mg/kg body weight/day. Exposures to DEHP can be especially high for infants receiving TPN solutions (contains approximately 20% lipid emulsions), where a 24-hour infusion can deliver up to an estimated 10 mg of DEHP (Loff et al. 2000). It has been estimated that newborns and infants undergoing medical procedures, such as transfusions, ECMO, and TPN might be exposed to DEHP levels ranging from 0.13 to 6.0 mg/kg/day (NTP 2006). Kaestner et al. (2020) measured DEHP blood levels of ECMO patients hospitalized between May 2015 and December 2016. DEHP levels of patients receiving ECMO ranged from 31.5 to 1,009 $\mu\text{g/L}$ (median 156.0 $\mu\text{g/L}$) while DEHP levels of a control group ranged from 19.4 to 75.3 $\mu\text{g/L}$ (median 36.4 $\mu\text{g/L}$). The FDA's DEHP exposure estimates resulting from various medical treatments are presented in Table 5-17.

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Table 5-17. FDA Estimates of DEHP Exposures Resulting from Medical Treatments

Medical procedure	Estimated DEHP dose (mg/kg body weight/day)	
	70 kg adult	4 kg neonate
Crystalloid IV solution infusion	0.005	0.03
Infusion of pharmaceuticals with solubilization vehicles		
Administered according to manufacturer instructions	0.04	0.03
Mixed and stored at room temperature for 24 hours	0.15	
TPN administration		
Without added lipid	0.03	0.03
With added lipid	0.13	2.5
Administered via ethyl vinyl acetate bag and PVC tubing	0.06	
Blood transfusion		
Trauma patient	8.5	
Transfusion/ECMO in adult patients	3.0	
Exchange transfusion in neonates		22.6
Replacement transfusions in neonates in NICU		0.3
Replacement transfusions to treat anemia in chemotherapy and sickle cell disease patients	0.09	
Replacement transfusions in patients undergoing coronary artery bypass grafting	0.28	
Treatment of cryodisorders with cryoprecipitate	0.03	
Cardiopulmonary bypass		
Coronary artery bypass grafting	1	
Orthotopic heart transplant	0.3	
Artificial heart transplant	2.4	
ECMO		14
Apheresis	0.03	
Hemodialysis	0.36	
Peritoneal dialysis	<0.01	
Enteral nutrition	0.14	0.14
Aggregate exposures of NICU infants undergoing IV administration of sedatives, IV administration of TPN, and replacement transfusion		2.83

DEHP = di(2-ethylhexyl)phthalate; ECMO = extracorporeal membrane oxygenation; FDA = Food and Drug Administration; IV = intravenous; NICU = neonatal intensive care unit; PVC = polyvinyl chloride; TPN = total parenteral nutrition

Source: NTP 2006

Since the permanent ban of DEHP in children's toys or clothing articles, the main source of exposures are food, beverages, and drugs via direct ingestion (CPSC 2014; Liroy et al. 2015).

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As discussed in Section 5.6, workers in industries manufacturing or using DEHP plasticizer might be frequently exposed to above-average levels of this compound. Firefighters and other emergency workers are also at a greater risk of DEHP exposure during structural fires due to potential release of DEHP from burning materials (Alexander and Baxter 2016; Lacey et al. 2014). Those living near industrial facilities or hazardous waste sites with higher than average levels of DEHP in water might also have potential above-average exposure (Section 5.5).