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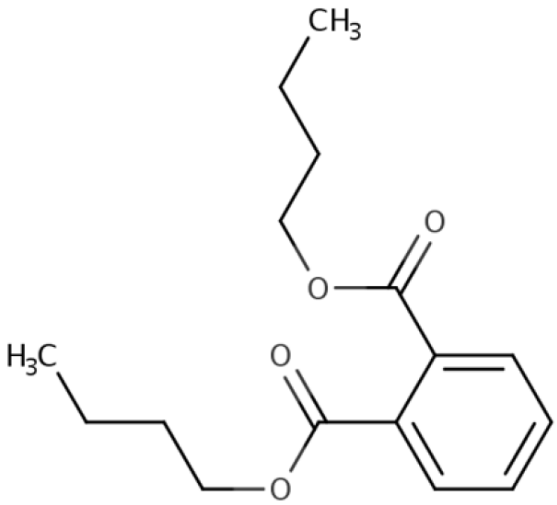
Draft Environmental Media, General Population, and Environmental Exposure for Dibutyl Phthalate (DBP)

Technical Support Document for the Draft Risk Evaluation

CASRN 84-74-2



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KEY ABBREVIATIONS AND ACRONYMS

7Q10	Lowest 7-day flow in a 10-year period
ADD	Average daily dose
ADR	Acute dose rate
AERMOD	American Meteorological Society (AMS)/EPA Regulatory Model
BAF	Bioaccumulation factor
BCF	Bioconcentration factor
CDC	Centers for Disease Control and Prevention (U.S.)
CEM	Consumer Exposure Model
COU	Condition of use
DAD	Dermal absorbed dose
DBP	Dibutyl phthalate
DI	Daily intake
EPA	Environmental Protection Agency (U.S.)
dw	Dry weight
ECHO	EPA's Enforcement and Compliance History Online Database
F_{ue}	Fractional urinary excretion
IIOAC	Integrated Indoor-Outdoor Air Calculator (model)
EPA	Environmental Protection Agency (U.S.)
HEC	Human equivalent concentration
HED	Human equivalent dose
HM	Harmonic mean
IIOAC	Integrated Indoor/Outdoor Air Calculator (IIOAC) (Model)
K_{OA}	Octanol:air partition coefficient
K_{OC}	Organic carbon:water partition coefficent
K_{OW}	Octanol:water partition coefficient
K_p	Dermal permeability coefficient
LADD	Lifetime average daily dose
MCNP	Mono-(carboxynonyl) phthalate
MHBP	Mono-3-hydroxybutyl phthalate
MnBP	Mono-n-butyl phthalate
MOE	Margin of exposure
NAICS	North American Industry Classification System
NHANES	National Health and Nutrition Examination Survey
NPDES	National Pollutant Discharge Elimination System
OCSPP	Office of Chemical Safety and Pollution Prevention
OES	Occupational exposure scenario
OPPT	Office of Pollution Prevention and Toxics
PESS	Potentially exposed or susceptible subpopulation(s)
POD	Point of departure
RCRA	Resource Conservation and Recovery Act
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
U.S.	United States
ww	Wet weight
WWTP	Wastewater treatment plant

SUMMARY

DBP – Environmental Media Concentration and General Population Exposure: Key Points

EPA evaluated the reasonably available information for various environmental media concentrations and estimated exposure using a conservative scenario as a screening level approach. The conservative high-end exposure was assumed to result from the highest DBP releases associated with the corresponding Toxic Substances Control Act (TSCA) condition of use (COU) via different exposure pathways. The key points are summarized below:

- EPA conducted a screening level assessment of general population and environmental exposure through air, water, and land (*i.e.*, soil, biosolids, and groundwater).
  - For the land pathway, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered. However, based on high-quality physical and chemical property data, EPA determined that DBP will have low persistence potential and mobility in soils. Therefore, groundwater concentrations resulting from releases to the landfill or to agricultural lands via biosolids applications were not quantified but are discussed qualitatively.
  - For the water pathway, DBP in water releases is expected to predominantly partition into sediment and suspended particles in the water column. The high-end modeled total water column concentration of DBP for the acute human exposure scenarios was 885 µg/L. The modeled value was several orders of magnitude above any monitored concentration likely due to conservative inputs. Therefore, EPA is confident that the use of the modelled concentration to estimate risk is protective.
  - For the ambient air pathway, the modeled DBP concentrations are several orders of magnitude above any monitored concentration likely due to use of high end releases and conservative meteorological data. Therefore, EPA is confident that the use of the modelled concentration to estimate risk is protective.
- Screening level risk estimates using high-end modeled water concentrations exceeded the benchmark (therefore no refinement necessary) for incidental dermal contact, incidental ingestion from swimming, and ingestion of drinking water. The same is true using high-end modeled air concentrations for inhalation of ambient air. EPA concluded that these exposure pathways are not of concern for the general population for DBP.
- EPA used a refined screening-level approach to determine that human exposure to DBP through ingestion of potentially contaminated fish is not expected to be a pathway of concern for the general population, subsistence fishers, or Tribal populations.
- DBP is not readily found in aquatic or terrestrial organisms and has low bioaccumulation and biomagnification potential. Therefore, DBP has low potential for trophic transfer through food webs.

1 ENVIRONMENTAL MEDIA CONCENTRATION OVERVIEW

This technical support document (TSD) accompanies the *Draft Risk Evaluation for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025d). DBP is a diester of phthalic acid (CASRN 84-74-2). It is a member of the phthalate class of chemicals that are widely used as adhesives and sealants in the construction and automotive sectors. DBP is also commonly used in electronics, children's toys, and plastic and rubber materials.

This draft TSD describes the use of reasonably available information to estimate environmental concentrations of DBP in different environmental media and the use of the estimated concentrations to evaluate exposure to the general population from releases associated with TSCA conditions of use (COUs). EPA evaluated the reasonably available information for releases of DBP from facilities that use, manufacture, or process DBP under industrial and/or commercial COUs as detailed in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b). Table 1-1 provides a crosswalk between COUs and occupational exposure scenarios (OESs). Table 1-2 shows the types of releases to the environment by OES.

Table 1-1. Crosswalk of Conditions of Use to Assess Occupational Exposure Scenarios

Life Cycle Stage	Category	Subcategory	OES
Manufacturing	Domestic manufacturing	Domestic manufacturing	Manufacturing
	Importing	Importing	Import and repackaging
Processing	Repackaging	Laboratory chemicals in wholesale and retail trade	Import and repackaging
		Plasticizers in wholesale and retail trade	Import and repackaging
	Processing as a reactant	Intermediates in all other basic organic chemical manufacturing	Incorporation into formulation, mixture, or reaction product
		Plasticizers in wholesale and retail trade	Incorporation into formulation, mixture, or reaction product
	Incorporation into formulation, mixture, or reaction product	Solvents (which become part of product formulation or mixture) in all other chemical product and preparation manufacturing	Incorporation into formulation, mixture, or reaction product
		Solvents in soap, cleaning compound, and toilet preparation manufacturing	Incorporation into formulation, mixture, or reaction product
		Adhesive and sealant chemicals in construction	Incorporation into adhesives and sealants
		Plasticizer (paint and coating manufacturing; plastic material and resin manufacturing; plastics product manufacturing; soap, cleaning compound, and toilet preparation manufacturing; textiles, apparel, and leather manufacturing	Incorporation into formulation, mixture, or reaction product; PVC plastics compounding; non-PVC material compounding
		Intermediates (asphalt paving, roofing,	Incorporation into formulation,

Life Cycle Stage	Category	Subcategory	OES
Processing		and coating materials manufacturing; petrochemical manufacturing; rubber product manufacturing)	mixture, or reaction product
		Functional fluids (closed systems) in printing and related support activities	Incorporation into formulation, mixture, or reaction product
	Incorporation into articles	Plasticizer (adhesive manufacturing; plastic product manufacturing; rubber product manufacturing)	PVC plastics converting; non-PVC material converting; incorporation into adhesives and sealants
	Recycling	Recycling	Recycling
Distribution	Distribution in commerce	Distribution in commerce	Distribution in commerce
Industrial Uses	Non-incorporative activities	Solvent in Huntsman's maleic anhydride manufacturing technology	Industrial process solvent use
		Solvent	Industrial process solvent use
Commercial Uses	Adhesives and sealants	Adhesives and sealants	Application of adhesives and sealants
	Cleaning and furnishing care products	Cleaning and furnishing care products	Fabrication of final product from articles
	Explosive materials	Explosive materials	Non-TSCA
	Floor coverings	Floor coverings	Application of paints and coatings; fabrication of final product from articles
	Furniture and furnishings not covered elsewhere	Furniture and furnishings not covered elsewhere	Fabrication of final product from articles
	Inks, toner and colorant products	Inks, toner and colorant products (*e.g.*, screen printing ink)	Application of paints and coatings
	Laboratory chemical	Laboratory chemical	Use of laboratory chemicals
	Paints and coatings	Paints and coatings	Application of paints and coatings
	Personal care products	Personal care products	Non-occupational use
	Plastic and rubber products not covered elsewhere	Plastic and rubber products not covered elsewhere	Fabrication of final product from articles
	Miscellaneous	Laboratory chemical; chemiluminescent	Use of laboratory chemicals; use of

Life Cycle Stage	Category	Subcategory	OES
	uses	light sticks; inspection penetrant kit; lubricants;	lubricants and functional fluids; use of penetrants and inspection fluids
Disposal	Disposal	Disposal	Waste handling, treatment, and disposal

Table 1-2. Type of Release to the Environment by Occupational Exposure Scenario

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d] – Data Sources[e]
Manufacturing	Fugitive air
	Stack air
	Water, incineration, or landfill
Import and repackaging	Fugitive or stack air – Toxics Release Inventory (TRI) and National Emissions Inventory (NEI)
	Land releases (includes both Resource Conservation and Recovery Act [RCRA] Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI
	Surface water, direct – TRI
	Surface water, indirect transfer to POTW – TRI
	Surface water, indirect transfer to non-POTW – TRI
	Surface water, with or without on-site treatment – Discharge Monitoring Report (DMR)
Incorporation into formulations, mixtures, and reaction products	Fugitive or stack air – TRI and NEI
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI
	Surface water, direct – TRI
	Surface water, indirect transfer to POTW – TRI
	Surface water, indirect transfer to non-POTW – TRI
PVC plastics compounding	Fugitive or stack air – TRI and NEI
	Surface water, with or without on-site treatment – DMR
PVC plastics converting	Fugitive or stack air – TRI and NEI
	Surface water, direct – TRI (PVC compounding as a surrogate OES)
	Surface water, indirect transfer to POTW – TRI
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI (non-PVC material manufacturing as a surrogate OES)
Non-PVC material compounding and converting	Fugitive or stack air – TRI and NEI
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI
	Surface water, direct – TRI
	Surface water, indirect transfer to POTW – TRI

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d] – Data Sources[e]
Application of adhesives and Sealants	Fugitive air
	Water, incineration, or landfill
	Incineration, or landfill
Application of paints and coatings – no spray control	Fugitive air
	Stack air
	Wastewater, incineration, or landfill
	Incineration, or landfill
	Air, water, incineration, or landfill [unknown]
Application of paints and coatings – spray control	Fugitive air
	Stack air
	Wastewater, incineration, or landfill
	Incineration, or landfill
Application of paints, coatings, adhesives, and sealants	Fugitive or stack air – TRI and NEI
Industrial process solvent use	Fugitive or stack air – TRI and NEI
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI (incorporation into formulation, mixture, or reaction product)
Use of laboratory chemicals – liquid	Fugitive or stack air
	Wastewater, incineration, or landfill
Use of laboratory chemicals – solid	Stack air
	Air, water, incineration, or landfill [unknown]
	Water, incineration, or landfill
	Incineration or landfill
Use of lubricants and functional fluids	Wastewater
	Landfill
	Recycling
	Fuel blending (incineration)
Use of penetrants and inspection fluids – aerosol based	Fugitive air
	Wastewater, incineration, or landfill
Use of penetrants and inspection fluids – non-aerosol based	Fugitive air
	Wastewater, incineration, or landfill
Fabrication of final product from articles	Fugitive or stack air, water, incineration, or landfill (dust generation from cutting, grinding, shaping, drilling, abrading, and similar activities)
	Fugitive or stack air (vapor generation from heating/plastic welding activities)

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d] – Data Sources[e]
Recycling	Fugitive or stack air – TRI and NEI (from PVC compounding and converting OES)
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI (from Non-PVC material manufacturing)
	Surface water, with or without on-site treatment – DMR (from PVC plastics compounding OES)
Waste handling, treatment, and disposal	Fugitive or stack air – TRI and NEI
	Land releases (includes both RCRA Subtitle C landfills and those classified as other, underground injection, and Land Treatment) – TRI
	Surface water, with or without on-site treatment – DMR
	Surface water, indirect transfer to POTW – TRI

[a] Table 1-1 provides the crosswalk of OES to COUs
[b] Direct discharge to surface water; indirect discharge to non-POTW; indirect discharge to POTW
[c] Emissions via fugitive air or stack air, or treatment via incineration
[d] Transfer to surface impoundment, land application, or landfills
[e] Discharge, release or emission database source(s) (*i.e.,* TRI, DMR, or NEI). If none listed, a modeled scenario was leveraged. See the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b) for additional information on sources and model details.

Releases from all OESs were considered, but EPA focused on estimating high-end concentrations of DBP from the largest estimated releases for its screening level assessment of environmental and general population exposures. This means that the Agency considered the concentration of DBP in a given environmental media resulting from the OES that had the highest release to that media compared to the other OES(s). The OES resulting in the highest concentration of DBP varied by environmental media as shown in Table 2-1. Additionally, EPA relied on its fate assessment to determine which environmental pathways to consider. Details on the environmental partitioning and media assessment can be found in the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g). Briefly, based on DBP's fate parameters and behavior (*e.g.,* Henry's Law constant, log K_{OC}, water solubility, fugacity modeling), EPA anticipates DBP to be predominantly in water and soil, although the chemical may also be present in air and sediments. Moreover, because DBP is released to the ambient air from industrial facilities and processes, inhalation of ambient air is a possible exposure pathway. EPA thus quantitatively assessed concentrations of DBP in surface water, sediment, and ambient air. Soil concentrations of DBP from land application of biosolids were not quantitatively assessed as DBP was expected to have limited persistence potential and mobility in soils receiving biosolids.

Environmental exposures using the predicted media concentrations of DBP are presented in Section 12. As DBP fate and exposure from groundwater, biosolids, and landfills were not quantified, EPA performed a qualitative assessment for all these land exposure scenarios (U.S. EPA, 2024g). Additionally, EPA discusses the potential DBP dietary exposures to aquatic and terrestrial organisms in the environment in Section 12. EPA did not conduct a quantitative analysis of DBP trophic transfer, as DBP is expected to have low bioaccumulation potential, no apparent biomagnification potential, and thus low potential for uptake overall. For further information on the bioaccumulation and biomagnification of DBP, please see the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g).

General population exposure is discussed using a risk screening approach detailed in Section 0. EPA used a margin of exposure (MOE) approach discussed in Section 2.2 using high-end exposure estimates (Section 2.1) to screen for potential non-cancer risks. EPA assumed that if there is no unreasonable risk for an individual identified as having the potential for the highest exposure associated with a COU for a given exposure pathway, then that pathway was determined not to be a pathway of concern for general population exposure and not pursued further. If any pathways were identified as a pathway of concern for the general population, further exposure assessments for that pathway would be conducted to include higher tiers of modeling when available, refinement of exposure estimates, and exposure estimates for additional subpopulations and COUs/OES.

Table 1-3 summarizes the exposure pathways assessed for the general population. For DBP, exposures to the general population via surface water, drinking water, fish ingestion, and ambient air were quantified, and modeled concentrations were compared to environmental monitoring data when possible. Exposures via the land pathway (*i.e.*, biosolids and landfills) were qualitatively assessed because DBP is not expected to be persistent or mobile in soils. Concentrations of DBP in soil following agricultural application of municipal biosolids were not identified during systematic review. Further description of the qualitative and quantitative assessments for each exposure pathway can be found in the sections linked in Table 1-3. As summarized in Table 1-3, biosolids, landfills, surface water, drinking water, ambient air, and fish ingestion are not pathways of concern for DBP for highly exposed populations based on the OES leading to the highest concentrations of DBP in environmental media.

Table 1-3. Exposure Pathways Assessed for General Population Screening Level Assessment

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Pathway of Concern[b]
All	Biosolids (Section 3.1)	All considered qualitatively		No
All	Landfills (Section 3.2)	All considered qualitatively		No
Manufacturing	Surface water	Dermal	Dermal exposure to DBP in surface water during swimming (Section 5.1.1)	No
		Oral	Incidental ingestion of DBP in surface water during swimming (Section 5.1.2)	No
Manufacturing	Drinking water	Oral	Ingestion of drinking water (Section 6.1.1)	No
Manufacturing; waste handling, treatment, disposal	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	No
			Ingestion of fish for subsistence fishers (Section 7.2)	No
			Ingestion of fish for tribal populations (Section 7.3)	No
Waste handling, treatment, disposal (stack)	Ambient air	Inhalation	Inhalation of DBP in ambient air resulting from industrial releases (Section 9)	No
Application of paints, coatings, adhesives, and sealants		Oral	Ingestion from air to soil deposition resulting from industrial releases (Section 9)	No

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Pathway of Concern[b]
(fugitive)				

[a] Table 1-1 provides a crosswalk of industrial and commercial COUs to OES.
[b] Using the MOE approach, an exposure pathway was determined to not be a pathway of concern if the MOE was equal to or exceeded the benchmark MOE of 30.
[c] Used in assessment presented in *Draft Environmental Hazard Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024c).

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2 SCREENING LEVEL ASSESSMENT OVERVIEW

EPA began its DBP exposure assessment using a screening level approach that relies on conservative assumptions. Conservative assumptions, including default input parameters for modeling environmental media concentrations, help to characterize exposure resulting from the high-end of the expected distribution. Most of the OESs presented in Table 1-1 report facility location data and releases in the TRI and Discharge Monitoring Report (DMR) databases. When facility location- or scenario-specific information are unavailable, EPA used generic EPA models and default input parameter values as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b). Details on the use of screening level analyses in exposure assessment can be found in EPA's *Guidelines for Human Exposure Assessment* (U.S. EPA, 2019b).

High-end exposure estimates used for screening level analyses were defined as those associated with the industrial and commercial releases from a COU and OES that resulted in the highest environmental media concentrations. Additionally, individuals with the greatest intake rate of DBP per body weight were considered to be those at the upper end of the exposure distribution. Taken together, these exposure estimates are conservative because they were determined using the highest environmental media concentrations and greatest intake rate of DBP per kilogram of body weight. These exposure estimates are also protective of individuals having less exposure either due to lower intake rate or exposure to lower environmental media concentration. This is explained further in Section 2.1.

For the general population screening level assessment, EPA used an MOE approach based on high-end exposure estimates to determine which exposure pathways were of potential concern for non-cancer risks. Using the MOE approach, an exposure pathway associated with a COU was determined to not be a pathway of concern if the MOE was equal to or exceeded the benchmark MOE of 30 (U.S. EPA, 2024f). Further details of the MOE approach are described in Section 2.2.

If there is no unreasonable risk for an individual identified as having the potential for the highest exposure associated with a COU, then that pathway was determined not to be a pathway of concern. If any pathways were identified as having potential for risk to the general population, further exposure assessments for that pathway would be conducted to include higher tiers of modeling, additional subpopulations, and additional OES/COUs.

2.1 Estimating High-End Exposure

General population exposures occur when DBP is released into the environment and the environmental media is then a pathway for exposure. As described in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b) and summarized in Table 1-1 releases of DBP are expected to occur to air, water, and land. Figure 2-1 provides a graphical representation of where and in which media DBP is expected to be found due to environmental releases and the corresponding route of exposure.

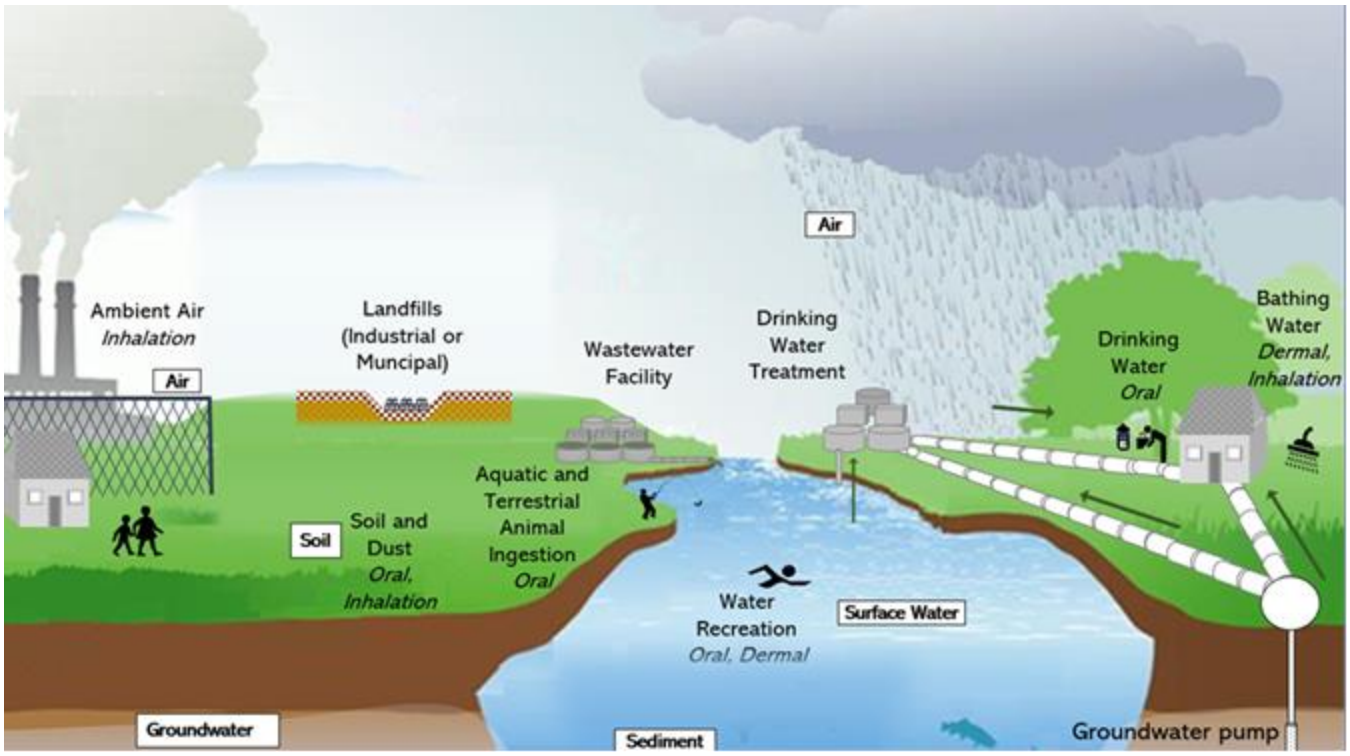


Figure 2-1. Potential Human Exposure Pathways for the General Population
The diagram presents the media (white text boxes) and routes of exposure (italics for oral, inhalation, or dermal) for the general population. Sources of drinking water from surface or water pipes are depicted with grey arrows.

For a screening level analysis, high-end exposures were estimated for each exposure pathway assessed. EPA's *Guidelines for Human Exposure Assessment* defined high-end exposure estimates as a "plausible estimate of individual exposure for those individuals at the upper- end of an exposure distribution, the intent of which is to convey an estimate of exposure in the upper range of the distribution while avoiding estimates that are beyond the true distribution" (U.S. EPA, 2019b). If risk is not found for these individuals with high-end exposure, no unreasonable risk is anticipated for central tendency exposures, which is defined as "an estimate of individuals in the middle of the distribution."

Identifying individuals at the upper end of an exposure distribution included consideration of high-end exposure scenarios defined as those associated with the industrial and commercial releases from a COU and OES that resulted in the highest environmental media concentrations. Additionally, individuals with the greatest intake rate of DBP per body weight were considered to be those at the upper end of the exposure. Intake rate and body weight are dependent on lifestage as shown in Appendix A.

Table 2-1 summarizes the high-end exposure scenarios that were considered in the screening level analysis including the lifestage assessed as the most potentially exposed population based on intake rate and body weight. Exposure scenarios were assessed quantitatively only when environmental media concentrations were quantified for the appropriate exposure scenario. Because DBP environmental releases from biosolids and landfills (and therefore, resulting soil concentrations) were not quantified, exposure from soil or groundwater resulting from DBP release to the environment via biosolids or landfills was not quantitatively assessed. Instead, the scenarios were assessed qualitatively for exposures potentially resulting from biosolids and landfills.

Table 2-1. Exposure Scenarios Assessed in Risk Screening

OES	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Analysis (Quantitative or Qualitative)
All	Biosolids	All considered qualitatively			Qualitative, Section 3.1
All	Landfills	All considered qualitatively			Qualitative, Section 3.2
PVC plastics compounding	Surface water	Dermal	Dermal exposure to DBP in surface water during swimming	All	Quantitative, Section 5.1.1
		Oral	Incidental ingestion of DBP in surface water during swimming	All	Quantitative, Section 5.1.2
PVC plastics compounding	Drinking water	Oral	Ingestion of drinking water	All	Quantitative, Section 6.1.1
PVC plastics compounding	Fish ingestion	Oral	Ingestion of fish for general population	Adults and young toddlers (1–2 years)	Quantitative, Section 7.1
			Ingestion of fish for subsistence fishers	Adults (16 to <70 years)	Quantitative, Section 7.2
			Ingestion of fish for tribal populations	Adults (16 to <70 years)	Quantitative, Section 7.3
Waste handling, treatment, disposal (stack)	Ambient air	Inhalation	Inhalation of DBP in ambient air resulting from industrial releases	All	Quantitative, Section 9
Application of paints, coatings, adhesives, and sealants (fugitive)		Oral	Ingestion from air to soil deposition resulting from industrial releases	Infant and children (6 months to 12 years)	

As part of the general population exposure assessment, EPA considered fenceline populations in proximity to releasing facilities as part of the ambient air exposure assessment by utilizing pre-screening methodology described in EPA's *Draft TSCA Screening Level Approach for Assessing Ambient Air and Water Exposures to Fenceline Communities (Version 1.0)* (U.S. EPA, 2022b). For other exposure pathways, EPA's screening method assessing high-end exposure scenarios used release data that reflects exposures expected to occur in proximity to releasing facilities, which would include fenceline populations.

Modeled and monitored surface water concentrations (Section 4.1) were used to estimate oral drinking water exposures (Section 6), incidental dermal exposures (Section 5.1.1), and incidental oral exposures (Section 5.1.2) for the general population. Modeled ambient air concentrations (Section 8.1) were used to estimate inhalation exposures.

If any pathways were identified as an exposure pathway of concern for the general population, further exposure assessments for that pathway would be conducted to include higher tiers of modeling when available and exposure estimates for additional subpopulations and COUs.

2.2 Margin of Exposure Approach

EPA used an MOE approach using high-end exposure estimates to determine if the pathway analyzed is a pathway of concern. The MOE is the ratio of the non-cancer hazard value (or point of departure [POD]) divided by a human exposure dose. Acute, intermediate, and chronic MOEs for non-cancer inhalation and dermal risks were calculated using the following equation:

Equation 2-1. Margin of Exposure Calculation

$$MOE = \frac{Non-cancer\ Hazard\ Value\ (POD)}{Human\ Exposure}$$

Where:

MOE	=	Margin of exposure for acute, short-term, or chronic risk comparison (unitless)
$Non-cancer\ Hazard\ Value\ (POD)$	=	Human equivalent concentration (HEC, mg/m^3) or human equivalent dose (HED, in units of mg/kg-day)
$Human\ Exposure$	=	Exposure estimate (mg/m^3 or mg/kg-day)

MOE risk estimates may be interpreted in relation to benchmark MOEs. Benchmark MOEs are typically the total uncertainty factor for each non-cancer POD. The MOE estimate is interpreted as a human health risk of concern if the MOE estimate is less than the benchmark MOE (*i.e.*, the total uncertainty factor). On the other hand, for this screening level analysis, if the MOE estimate is equal to or exceeds the benchmark MOE, the exposure pathway is not analyzed further. Typically, the larger the MOE, the more unlikely it is that a non-cancer adverse effect occurs relative to the benchmark. When determining whether a chemical substance presents unreasonable risk to human health or the environment, calculated risk estimates are not "bright-line" indicators of unreasonable risk, and EPA has the discretion to consider other risk-related factors in addition to risks identified in the risk characterization.

The non-cancer hazard values used to screen for risk are described in detail in the *Draft Non-Cancer Human Health Hazard Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024f). Briefly, after considering hazard identification and evidence integration, dose-response evaluation, and weight of the scientific evidence of POD candidates, EPA chose one non-cancer POD for acute, intermediate, and chronic exposure scenarios (Table 2-2). Human equivalent concentrations (HECs) are based on daily continuous (24-hour) exposure, and human equivalent doses (HEDs) are daily values.

Table 2-2. Non-Cancer Hazard Values Used to Estimate Risks

Target Organ System	Species	Duration	POD (mg/kg-day)	Effect	HED[a] (mg/kg-day)	HEC (mg/m^3) [ppm]	Benchmark MOE	Reference
Development/ Reproductive	Rat	5–14 days throughout gestation	$BMDL_5$ = 9	↓ fetal testicular testosterone	2.1	12 [1.0]	UF_A = 3 UF_H = 10 *Total UF = 30*	–[b]

POD = point of departure; HEC = human equivalent concentration; HED = human equivalent dose; MOE = margin of exposure; UF = uncertainty factor; BMDL5 = Benchmark dose (lower confidence limit) associated with a 5% response level

[a] EPA used allometric body weight scaling to the three-quarters power to derive the HED. Consistent with EPA Guidance (U.S. EPA, 2011b), the interspecies uncertainty factor (UF_A), was reduced from 10 to 3 to account remaining uncertainty associated with interspecies differences in toxicodynamics. EPA used a default intraspecies (UF_H) of 10 to account for variation in sensitivity within human populations.

[b] The $BMDL_5$ was derived through meta-regression and BMD modeling of fetal testicular testosterone data from eight studies of DBP with rats (Gray et al., 2021; Furr et al., 2014; Johnson et al., 2011; Struve et al., 2009; Howdeshell et al., 2008; Martino-Andrade et al., 2008; Johnson et al., 2007; Kuhl et al., 2007).

Using the MOE approach in a screening level analysis, an exposure pathway associated with a COU was determined to not be a pathway of concern for non-cancer risk if the MOE was equal to or exceeded the benchmark MOE of 30.

3 LAND PATHWAY

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data identified during systematic review to obtain concentrations of DBP in terrestrial land pathways (*i.e.*, biosolids, wastewater sludge, agricultural soils, landfills, and landfill leachate). No monitoring data was available from a review of government regulatory and reporting databases related to soil, landfills, or biosolids (*e.g.*, California Environmental Data Exchange Network [CEDEN], Water Quality Portal [WQP]). Several academic experimental and field studies, however, have identified DBP in various relevant compartments including leachate, activated sludge, and biosolids. EPA cannot correlate monitoring levels from the reviewed studies with any specific releases associated with DBP TSCA COUs. That is, EPA does not have any facility specific DBP release data since facilities do not report releases of DBP to surface waters from TSCA COUs. As such, the present assessment of DBP exposure via potential land pathways is qualitative in nature relying on the fate and physical-chemical characteristics of DBP. When possible, data from the existing literature including experimental and field data was used to support the qualitative assessment.

The monitoring studies and analysis presented in the following land pathway sections are for informational purposes and were not used as part of the analysis for quantifying exposure estimates or exposure risk. DBP was not anticipated to pose a substantial risk of exposure for the general population through the biosolids or land pathways due to the low quantity of DBP released and the high sorption causing significant retardation in either of the terrestrial system. As such, the assessments were qualitative in nature and were not used to quantitatively determine exposure estimates. The monitoring studies and application estimates presented here were not used as part of the analysis for quantifying exposure estimates and are included for informational and contextual purposes.

3.1 Biosolids

The term “biosolids” refers to treated sludge that meet the EPA pollutant and pathogen requirements for land application and surface disposal and can be beneficially recycled (40 CFR Part 503) (U.S. EPA, 1993). Biosolids generated during the treatment of industrial and municipal wastewater may be applied to agricultural fields or pastures as fertilizer in either its dewatered form or as a water-biosolid slurry. Biosolids that are not applied to agricultural fields or pastures may be disposed of by incineration or landfill disposal. Landfill disposal will be discussed in further depth in Section 3.2. DBP may be introduced to biosolids by the absorption or adsorption of DBP to particulate or organic material during wastewater treatment. Based on the available information, the main mechanisms for the removal of DBP in conventional municipal wastewater treatment plants are sorption to suspended organic matter, biodegradation during activated sludge treatment, or a combination of sorption and biodegradation. These removal mechanisms are influenced by DBP’s physical-chemical properties and treatment time. Monitoring wastewater treatment studies have reported removal ranging from 38 to 99 percent of DBP during wastewater treatment with a representative removal of 65 to 98 percent (Wu et al., 2019; Salaudeen et al., 2018a, b; Wu et al., 2017; Gani and Kazmi, 2016; Saini et al., 2016; Tran et al., 2014; Huang et al., 2013b; Shao and Ma, 2009; Roslev et al., 2007; Peterson and Staples, 2003). The primary removal mechanism of DBP in wastewater treatment is sorption to biosolids, with up to 90 percent of removal due to sorption (Wu et al., 2019; Wu et al., 2017; Gani and Kazmi, 2016; Huang et al., 2013b; Shao and Ma, 2009; Peterson and Staples, 2003). The STPWIN™ model in EPI Suite™ predicts 56 percent removal of DBP removal in wastewater treatment with 55.5 percent of removal (out of 56 percent overall removal) resulting from sorption to activated sludge and solids assuming negligible biodegradation (U.S. EPA, 2017a). However, STPWIN™ is conservative estimate of overall removal and may underestimate overall DBP removal across in wastewater treatment plants depending on the specific technologies and processes implemented.

Overall removal of DBP from various wastewater treatment plant trains ranged from 38 to over 99 percent (Tomei et al., 2019; Salaudeen et al., 2018a, b; Wu et al., 2017; Gani and Kazmi, 2016; Saini et al., 2016; Tran et al., 2014; Huang et al., 2013b; Shao and Ma, 2009; Roslev et al., 2007; Peterson and Staples, 2003). A survey of 50 wastewater plants in the United States saw a median removal of DBP ranging from 68 to 98 percent (U.S. EPA, 1982). Approximately 27 to 59 percent of the overall removal was attributed to biodegradation during primary and secondary treatment while the remainder of the DBP removed being the result of adsorption or absorption to biosolids and organic matter (Salaudeen et al., 2018a, b; Wu et al., 2017; Tran et al., 2014; Huang et al., 2013b; Shao and Ma, 2009; Peterson and Staples, 2003). See the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* for additional detail regarding DBP wastewater treatment and removal (U.S. EPA, 2024g).

DBP has been identified in several U.S.-based and international surveys of wastewater sludge, composted biosolids, and otherwise stabilized biosolids. A 2012 survey of North American wastewater plants (Canada and United States) identified DBP in sludge at concentrations ranging from 1.7 to 1,260 ng/g dry weight (dw) (Ikonomou et al., 2012). Post-aerobic treatment (*e.g.* aerobic, anaerobic digestion) of activated sludges may reduce the concentration of DBP (100% removal) and other phthalates (11–100% removal) in treated biosolids, however, current research is limited to a single 2019 study (Tomei et al., 2019).

No U.S.-based studies were identified evaluating the effects land application of DBP-containing biosolids. Sludge and biosolids containing DBP have not been reported for use in surface land disposal or agricultural application. As such, no data was identified directly evaluating the fate, persistence, degradation, or exposure profiles of DBP in soil resulting from land application.

DBP is not expected to be persistent in topsoil if it is applied to land through biosolids applications. Several academic studies have reported on degradation of DBP in aerobic soils. The half-life of DBP in aerobic soils range from less than 1 to 19 days (Cheng et al., 2018; Zhao et al., 2016; Yuan et al., 2011; Xu et al., 2008; Wang et al., 1997; Russell et al., 1985; Shanker et al., 1985). In mixed aerobic and anaerobic conditions in which oxygen or terminal electron acceptors may not be readily replaced, the degradation of DBP may be slower. Current research suggests that the half-life of DBP may be extended to as long as 65 days under evolving aerobic conditions (Inman et al., 1984). In strictly anaerobic soil conditions, DBP appears to degrade under comparable rates to aerobic or evolutionary conditions with half-lives reported from 19 to 36 days (Shanker et al., 1985; Inman et al., 1984).

Other sources of DBP in biosolids-amended soils may include atmospheric deposition to soil. While long-range transport and deposition of DBP in the atmosphere has not been directly monitored, Net et al. (Net et al., 2015) noted possible atmospheric deposition of similar phthalates in agricultural settings. A 2008 study noted concentrations up to 1,173 ng/L of DBP in precipitation samples (Peters et al., 2008) while a 2010 study on atmospheric deposition of phthalates notes bulk wet and dry deposition of DBP and other phthalates from the atmosphere (Zeng et al., 2010).

DBP present in soil through the application of biosolids or otherwise introduced to topsoil has limited mobility within the soil column. Due to the tendency of DBP to sorb strongly to organic media and soil (log K_{OW} = 4.5; log K_{OC} = 3.14–3.94), potential leaching is limited. Any leaching which does occur in the uppermost soil layers will sorb to soil lower in the column and show minimal potential to interact with groundwater systems. DBP is not readily taken up by agricultural crops or cover crops planted in soils fertilized with biosolids. One study evaluating the potential for DBP to be taken up by crops observed the largest concentrations of DBP on the surface of crops caused by the volatilization of DBP

from soil particulate and subsequent deposition onto the surface of plant shoots and leaves (Müller and Kördel, 1993). Exposed plants do not readily absorb DBP from the soil nor do they incorporate DBP into the roots, shoots, leaves, or fruiting bodies (Müller and Kördel, 1993). DBP can be present on the surface of any plants growing in the vicinity resulting from localized atmospheric deposition of DBP blown up by the wind or volatizing out of the top layer of soil. While possible, no studies identified thus far in systematic review have reported that DBP is susceptible to longer range atmospheric transport resulting in land application of DBP containing biosolids beyond the immediate region of initial application.

Concentrations of DBP in soil following agricultural application of municipal biosolids were not identified in any monitoring databases, release databases, or in a survey of the existing literature identified during systematic review. As such, DBP concentrations in soil were estimated using the concentrations identified in sludge, ranging from 1.7 to 1,260 ng/g dw (Ikonomou et al., 2012). Biosolids application rates and frequencies were selected using EPA's recommendation to the public in the *Land Application of Biosolids* (
Table 3-1) (U.S. EPA, 2000a). Annual application rates ranged from 2 to 100 tons of dry biosolids per application per acre, with frequency ranging from three times a year to once every 5 years.

Table 3-1. Typical Biosolids Application Scenarios

Vegetation	Application Frequency ($year^{-1}$)	Application Rate (tons/acre)
Corn	1	5–10
Small grain	1–3	2–5
Soybeans	1	2–20
Hay	1–3	2–5
Forested land	0.2–0.5	5–100
Range land	0.5–1	2–60
Reclamation sites	1	60–100

Soil surface concentrations and incorporated concentrations were calculated from the minimum and maximum recommended application rates for each agricultural crop cover (Table 3-2). Minimum (1.7 ng/g) and maximum (1,260 ng/g) concentrations of DBP in biosolids were selected from the observed concentrations in biosolids during the 2008 EPA National Sewage Survey (U.S. EPA, 2009).

Table 3-2. Estimated DBP Soil Concentrations Following Application of Biosolids

Crop	Sludge Concentration (mg/kg) [a]	Application Rate (kg/acre) [b]	Frequency ($year^{-1}$) [b]	Surface Concentration (mg/m^2)	Topsoil Concentration (mg/kg)
Corn	1.7	5,080	1	0.00	0.000
Corn	1.7	10,161	1	0.00	0.000
Corn	1260	5,080	1	1.58	0.01
Corn	1260	10,161	1	3.16	0.01

Crop	Sludge Concentration (mg/kg) [a]	Application Rate (kg/acre) [b]	Frequency ($year^{-1}$) [b]	Surface Concentration (mg/m^2)	Topsoil Concentration (mg/kg)
Hay	1.7	2,032	1	0.00	0.000
Hay	1.7	5,080	3	0.01	0.000
Hay	1,260	2,032	1	0.63	0.00
Hay	1,260	5,080	3	4.75	0.02
Small grains	1.7	2,032	1	0.00	0.000
Small grains	1.7	5,080	3	0.01	0.000
Small grains	1,260	2,032	1	0.63	0.00
Small grains	1,260	5,080	3	4.75	0.02
Soybeans	1.7	5,080	1	0.00	0.000
Soybeans	1.7	20,321	1	0.01	0.000
Soybeans	1,260	5,080	1	1.58	0.01
Soybeans	1,260	20,321	1	6.33	0.03

[a] Targeted National Sewage Sludge Survey Sampling and Analysis Technical Report (U.S. EPA, 2009).
[b] EPA Recommended Application Rates were taken from EPA 832-F-00-064, Biosolids Technology Fact Sheet: Land Application of Biosolids (U.S. EPA, 2000a).
[c] Recommended incorporation depth of 7 inches (18 cm) as outlined in 40 CFR Part 503.
[d] An average topsoil bulk density value of 2,530 lb/yd3 (1,500 kg/m3) was selected from NRCS Soil Quality Indicators (USDA, 2008).

Using the generic application scenarios and biosolids concentrations collected from national surveys, the typical concentration of DBP in biosolids may range by several orders of magnitude depending largely on the source material and method of application. The surface loading rate for spray or near surface injection applications range from 9×10^{-5} to 6.3 mg/m^2 while mixing applications (assuming a 7-inch tilling depth) may range from 3×10^{-6} to 0.03 mg/m^3—depending on the application rate, frequency, and applied biosolids concentration.

Once in the soil, DBP is expected to have a high affinity to soil and sediment (log K_{OC} = 3.14–3.94) and organic media (log K_{OW} = 4.5), which would limit mobility from biosolids or biosolid amended soils. Similarly, high sorption to particulate and organics would likely lead to high retardation which would limit infiltration to and mobility within surrounding groundwater systems. DBP is slightly soluble in water (11.2 mg/L) and does have limited potential to leach from biosolids and infiltrate into deeper soil strata. Since DBP does have high hydrophobicity and a high affinity for soil sorption, it is unlikely that DBP will migrate from potential biosolids-amended soils via groundwater infiltration. DBP has been detected in surface runoff originating from landfills containing DBP (IARC, 2013). However, the limited mobility and high sorption to soil suggests that infiltration of such stormwater runoff would be of minimal concern to deeper groundwater systems.

There is limited information available related to the uptake and bioavailability of DBP in land applied

soils. DBP's solubility and sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms. Similarly, no studies were identified evaluating the bioaccumulation potential of DBP. Based on the solubility (11.2 mg/L) and hydrophobicity (log K_{OW} = 4.5; log K_{OC} = 3.14–3.94), DBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of DBP into crops planted in DBP containing soils found that DBP was not found in any of the plant tissues (*i.e.*, roots, shoots, leaves) resulting from uptake via soil or water. DIBP, a DBP isomer, was found, however, on the surface of the plants due to localized atmospheric transport and deposition but is not readily absorbed by plants directly through the soil (Müller and Kördel, 1993). BAF and BCF were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 2.02 to 2.35 (upper-lower trophic levels) and log BAF ranging from 2.20 to 2.37 (upper-lower trophic levels) (U.S. EPA, 2017a).

There is limited measured data on concentrations of DBP in biosolids or soils receiving biosolids, and there is uncertainty that concentrations used in this analysis are representative of all types of environmental releases. However, the high-quality biodegradation rates and physical and chemical properties suggest that DBP will have limited persistence potential and mobility in soils receiving biosolids.

3.1.1 Weight of Scientific Evidence Conclusions

There is considerable uncertainty in the applicability of using generic release scenarios and wastewater treatment plant modeling software to estimate concentrations of DBP in biosolids. There is currently no direct evidence that biosolids containing DBP are being consistently applied agricultural fields in any part of the United States. However, this may be due to lack of testing and monitoring data, as DBP has been identified in various wastewater sludges as previous stated. There is currently limited evidence that biosolids containing appreciable concentrations of DBP is being incorporate into soils for agricultural or disposal purposes. Consequentially, while theoretically possible, there is currently no direct, observed evidence demonstrating the update of DBP from soil into plants in a manner which would cause significant exposure to those individuals consuming or coming into contact with such plants. However, the lack of direct observations does not filter out the possibility of such an exposure mechanism, but instead reflects the limited data available for DBP in stabilized biosolids and its land application to soil.

Additionally, there is uncertainty in the relevancy of the biosolids monitoring data to the COUs considered in this evaluation. However, due to the high confidence in the biodegradation rates and physical and chemical data, there is robust confidence that DBP in soils will not be mobile and will have low persistence potential. The existing literature suggests that DBP present in biosolid amended soils will likely not be absorbed by any plants or crops growing in the soil. While field and experimental data are limited, soil dwelling organisms may be exposed to DBP through soils which have been amended with DBP containing biosolids applied as fertilizers but are not expected to readily accumulate DBP through ingestion or absorption.

3.2 Landfills

For this assessment, landfills will be considered to be divided into two zones: (1) "upper-landfill" zone with typical environmental temperatures and pressures (*i.e.*, 1 atm, 20–25 °C, aerobic conditions), where biotic processes are the predominant route of degradation for DBP; and (2) "lower-landfill" zone where elevated temperatures and pressures exist, and abiotic degradation is the predominant route of degradation. In the upper-landfill zone where oxygen might still be present in the subsurface, conditions may still be favorable for aerobic biodegradation. However, photolysis is not considered to be a significant source of degradation in this zone. In the lower-landfill zone, conditions are assumed to be

anoxic, and temperatures present in this zone are likely to inhibit aerobic and anaerobic biodegradation of DBP. Temperatures in lower landfills may be as high as 70 °C; At temperatures at and above 60 °C, biotic processes are significantly inhibited and are likely to be completely irrelevant at 70 °C (Huang et al., 2013a). Hydrolysis may still degrade DBP in the lower landfill even with the elevated temperatures. Photolysis, however, will only impact degradation on the outermost surface of the landfill where DBP may be exposed to sunlight prior to daily capping. Once the daily cap has been applied, the lack of light penetration would prevent further photolysis.

DBP may be deposited into the landfill through various waste streams including consumer waste, residential waste, industrial waste, and municipal waste—including dewatered wastewater biosolids. No studies were identified in systematic review determining the concentration of DBP in waste entering landfills in the United States. A 1997 study of German refuse, however, identified phthalates in residential refuse; DBP was identified in residential refuse with the highest concentrations of DBP present in compound materials (*e.g.*, plastic products) (610–2,160 µg/g) and other plastics (36–763 µg/g) (Bauer and Herrmann, 1997). All other tested fractions (Food waste, paper, cardboard, plastic films, textiles, compound packaging, and diapers) had DBP contents ranging from 1.8 to 121 µg/g (Bauer and Herrmann, 1997). Combined, refuse contained approximately 11.4 to 105 µg of DBP per gram waste.

Several facilities have reported annual releases of DBP to landfill facilities through the TRI. Major OESs include Repackaging into large and small containers, Incorporation into formulation, mixture, or reaction product, non-PVC material manufacturing (compounding or converting), and waste handling, treatment, and disposal. Waste handling, treatment, and disposal makes up the majority of OESs contributing to DBP releases, sixty percent of contributing facilities (12 of 20) and 85 percent of overall contributions (by mass). DBP releases to Resource Conservation and Recovery Act (RCRA) Subtitle C landfills include 265,000 kg (on-site) and 54,500 kg (off-site) annually. Approximately 91,000 kg are released annually to other off-site landfills (U.S. EPA, 2025b).

One of the potential disposal methods for biosolids following stabilization is landfilling. and contribute to the presence of DBP in landfills. No data directly measuring DBP in dewatered or stabilized biosolids was identified during systematic review. A 2012 survey of North American wastewater plants (Canada and United States), however, identified DBP in sludge at concentrations ranging from 1.7 to 1,260 ng/g dw (Ikonomou et al., 2012). Beyond North America, DBP has been identified in sludge at various concentrations in wastewater plants located in China (Zhu et al., 2019; Meng et al., 2014).

DBP is capable of leaching from bioreactors simulating landfill conditions using residential waste. One 1997 study evaluating a variety of phthalates, including DBP, estimated a leaching potential over 90 days using 50 kg of unaltered refuse. The refuse leached 1.1 g of total phthalates per 1 ton of refuse with DBP making up approximately 6.0 to 6.7 percent of total phthalates (66 to 74 mg of DBP per 1 ton of residential refuse) (Bauer and Herrmann, 1997). No studies have directly evaluated the presence of DBP in leachate collected directly from landfills *in situ*. However, DBP is expected to have a high affinity to particulate (log K_{OC} = 3.14–3.94) and organic media (log K_{OW} = 4.5), which would cause significant retardation in groundwater and limit leaching to groundwater. Because of its high hydrophobicity and high affinity for soil sorption, it is unlikely that DBP will migrate from landfills via groundwater infiltration. Nearby surface waters, however, can be susceptible to DBP contamination via surface water runoff if it is not captured before interacting with surface water.

While persistence in landfills has not been directly measured, DBP can undergo abiotic degradation via carboxylic acid ester hydrolysis to form 2-butyl phthalate and 1-butanol (U.S. EPA, 2024a). DBP can then by further hydrogenated to form phthalic acid (Huang et al., 2013a). The phthalic acid product has

been noted accumulate in landfills, particularly in the lower landfill, where further degradation may be limited due to acidic conditions preventing reactions with the free aromatic acid (Huang et al., 2013a). Hydrolysis is not expected to be a significant degradation pathway in landfills with an estimated half-life of 3.4 years under standard environmental conditions (at pH 7 and 20 °C) (U.S. EPA, 2017a). Temperature in lower landfills, however, often exceed 70 °C in very complex matrices. In such matrices, temperature, pressure, ionic strength, and chemical activity may all effect the hydrolysis rate of DBP. With the very limited data available, the hydrolysis rate of DBP cannot reliably be estimated in the complex conditions present in lower landfills. Chemical rates of reaction, in general, tend to increase as temperature, pressure, and chemical activity increase. In both the upper and lower landfills, DBP is shielded from light and photolysis is not considered a significant abiotic degradation pathway.

DBP may be degrade biologically; The biological degradation pathway for DBP includes the primary degradation of DBP to a monoester form, such as 2-butyl phthalate, followed by hydrogenation to phthalic acid; Phthalic acid may ultimately be degraded to CO_2 and/or CH_4 under aerobic or anaerobic conditions, respectively (Huang et al., 2013a). In the lower landfill, high temperatures (>60 °C) and low water content can partially or completely inhibit biological degradation (Huang et al., 2013a). Aerobic and anaerobic degradation of DBP, however, has not been directly measured in landfills. Aerobic degradation of DBP; however, has been measured experimentally. DBP is readily degradable in aerobic soil conditions with a half-life ranging less than 4 hours to 19 days (Cheng et al., 2018; Zhao et al., 2016; Yuan et al., 2011; Xu et al., 2008; Wang et al., 1997; Russell et al., 1985; Shanker et al., 1985). DBP might also degrade under anaerobic conditions such as those that would exist in lower landfills. Anaerobic biodegradation of DBP in soil has been measured with a half-life extending up to 65 days (Shanker et al., 1985; Inman et al., 1984). DBP can be more persistent in areas with high leachate production, such as in the lowest sections of the lower landfill, where temperature, pressure, pH, and ionic strength may exceed bacteria's habitable zones thereby limiting biotic degradation of DBP (Huang et al., 2013a).

DBP's sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms adjacent to landfills. DBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of DBP into crops planted in DBP containing soils found that DBP was not found in any of the plant tissues (*i.e.*, roots, shoots, and leaves) resulting from uptake via soil or water. DBP was found, however, on the surface of the plants due to localized atmospheric transport and deposition, but it is not readily absorbed by plants directly through the soil (Müller and Kördel, 1993).
BAF and BCF were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 2.02 to 2.35 (upper-lower trophic levels) and log BAF ranging from 2.20 to 2.37 (upper-lower trophic levels) (U.S. EPA, 2017a).

3.2.1 Weight of Scientific Evidence Conclusions

There is uncertainty in the relevancy of the landfill leachate monitoring data to the COUs considered in this evaluation. While there is evidence that DBP is present in refuse and may be present in biosolids disposed of in a landfill, the examined refuse did not originate in United States and is from 1997. Although the data demonstrates that DBP might exist in and leach from landfill refuse, there is uncertainty as to if the presented study accurately reflects the current state of refuse and landfill DBP with respect to landfills operating within the United States.

Based on the biodegradation and hydrolysis data for conditions relevant to landfills, there is high confidence that DBP will be persistent in landfills. There is currently no direct evidence that the general populus or surrounding fauna have been directly exposed to DBP through refuse or waste disposed of

through landfills. Although possible, there has been no data to suggest that DBP is present in environmental compartment adjacent to landfills as the direct result of landfill operations.

Overall, due to high-quality physical and chemical property data, there is robust confidence that DBP is unlikely to be present in landfill leachates. The existing literature suggests that if DBP is disposed of in a landfill, it will likely not be absorbed by any nearby plants. Although experimental data are limited, the available data does not support the likelihood that soil dwelling organisms will be exposed to DBP, nor does it show that DBP will accumulate in landfills as a result of the disposal of biosolids or refuse.

4 SURFACE WATER CONCENTRATION

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of DBP in surface water and aquatic sediments. Although the available monitoring data were limited, DBP was found in detectable concentrations in ambient surface waters, finished drinking water, and in aquatic sediments. TSCA industrial releases of DBP to surface waters were reported to EPA via the TRI and DMR databases and are described in *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b). The Agency conducted modeling of industrial releases to surface water to assess the expected resulting environmental media concentrations from TSCA COUs presented in Table 1-1. Section 4.1 presents EPA modeled surface water concentrations and modeled sediment concentrations. Section 4.2.1 includes a summary of monitoring concentrations for ambient surface water, and Section 4.2.2 includes monitoring concentrations for sediment found from the systematic review process.

4.1 Modeling Approach for Estimating Concentrations in Surface Water

EPA conducted modeling using the EPA's Variable Volume Water Model (VVWM) in Point Source Calculator (PSC) tool (U.S. EPA, 2019c) to estimate surface water and sediment concentrations of DBP resulting from TSCA COU releases. PSC inputs include physical and chemical properties of DBP (*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and reported or estimated DBP releases to water (U.S. EPA, 2025b), which are used to predict receiving water column concentrations and partitioning to pore water and sediment in the benthic region of streams.

Site-specific parameters influence how partitioning occurs over time. For example, the concentration of suspended sediments, water depth, and weather patterns all influence how a chemical may partition between compartments. However, the physical and chemical properties of the chemical itself also have major influences on partitioning and half-lives in aqueous environments. DBP has a log K_{OC} range of 3.14 to 3.94, indicating a high potential to sorb to suspended solids in the water column and settled sediment in the benthic environment (U.S. EPA, 2017a).

Physical, chemical, and environmental fate properties selected by EPA for this assessment were applied as inputs to the PSC model (Table 4-1). Selected values are described in detail in the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g). The PSC Model relies on the Heat of Henry parameter, which was estimated from temperature variation of the Henry's Law constant calculated by HENRYWIN™ in EPI Suite™ (U.S. EPA, 2015b).

Table 4-1. PSC Model Inputs (Chemical Parameters)

Parameter	Value
K_{OC}	4,898 mL/g
Water Column Half-Life	10 days at 25 °C
Photolysis Half-Life	1.15 days at 30N
Hydrolysis Half-Life	8,030 days at 25 °C
Benthic Half-Life	2.9 days at 25 °C
Molecular Weight	278.35 g/mol
Vapor Pressure	0.0000201 torr
Water Solubility	11.2 mg/L

Parameter	Value
Henry's Law Constant	0.00000181 atm·m^3/mol
Heat of Henry	74,826 J/mol
Reference Temp	25 °C
[a] For details on selected values, see *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g).	

A common setup for the model environment and media parameters was applied consistently across all PSC runs. The standard EPA "farm pond" waterbody characteristics were used to parameterize the water column and sediment parameters (Table 4-2), which is applied consistently as a conservative screening scenario. Standardized waterbody geometry was also applied consistently across runs, with a standardized width of 5 m, length of 40 m, and depth of 1 m. Only the release parameters (daily release amount and days of release) and the hydrologic flow rate were changed between model runs for this chemical to reflect facility-specific release conditions.

Table 4-2. Standard EPA "Farm Pond" Waterbody Characteristics for PSC Model Inputs

Parameter	Value
DFAC (represents the ratio of vertical path lengths to depth as defined in EPA's exposure analysis modeling system [EXAMS] (U.S. EPA, 2019c))	1.19
Water column suspended sediment	30 mg/L
Chlorophyll	0.005 mg/L
Water column f_{oc} (fraction of organic carbon associated with suspended sediment)	0.04
Water column dissolved organic carbon (DOC)	5.0 mg/L
Water column biomass	0.4 mg/L
Benthic depth	0.05 m
Benthic porosity	0.50
Benthic bulk density	1.35 g/cm³
Benthic f_{oc}	0.04
Benthic DOC	5.0 mg/L
Benthic biomass	0.006 g/m²
Mass transfer coefficient	0.00000001 m/s

A required input for the PSC model is the hydrologic flow rate of the receiving water body. For facilities reporting releases to TRI, relevant flow data from the associated receiving waterbody were collected. Databases that were queried to estimate a flow rate include EPA's Enforcement and Compliance History Online (ECHO) that contains facilities with a National Pollutant Discharge Elimination System (NPDES) permit, National Hydrography Dataset Plus (NHDPlus), and NHDPlus V2.1 Flowline Network Enhanced Runoff Method (EROM) Flow. The complete methods for retrieving and processing flow data are detailed in Appendix B. For OESs where releases were estimated using a generic scenario, there were no reported data from available sources (*e.g.,* TRI and DMR). Without TRI and DMR data, EPA cannot identify the receiving water bodies and their location-specific hydrological flow data. Thus, the Agency generated a distribution of flow metrics by collecting flow data for facilities across a North American Industry Classification System (NAICS) code associated with each COU for a DIBP-releasing facility. Databases that were queried to develop the distribution include EPA's ECHO, which includes facilities with an NPDES permit, as well as NHDPlus and NHDPlus V2.1 EROM Flow. Although this

modeled distribution of hydrological flow data is specific to an industry sector rather than a facility, it provides a reasonable estimate of the distribution of location-specific values. The complete methods for retrieving and processing flow data by NAICS code are also provided in Appendix B.

Different hydrological flow rates were used for different exposure scenarios. The 30Q5 flows (*i.e.*, the lowest 30-day average flow that occurs in a 5-year period) are used to estimate acute, incidental human exposure through swimming or recreational contact. The annual average flow represents long-term flow rates, but a harmonic mean provides a more conservative estimate and is preferred for assessing potential chronic human exposure via drinking water. The harmonic mean is also used for estimating human exposure through fish ingestion because it takes time for chemical concentrations to accumulate in fish. Lastly, for aquatic or ecological exposure, a 7Q10 flow (*i.e.*, the lowest 7-day average flow that occurs in a 10-year period) is used to estimate exceedances of concentrations of concern for aquatic life (U.S. EPA, 2007). The regression equations for deriving the harmonic mean and 7Q10 flows are provided in Appendix B. Hydrologic flows in the receiving waterbodies were added to facility effluent flows as the rate of effluent contributes a substantial amount of flow to receiving waterbodies in many cases. The median, 75th percentile, and 90th percentile (P50, P75, P90, respectively) flows from the distribution were applied to represent variation in the potential receiving waterbodies for OESs in which releases were estimated using generic scenarios.

Manufacturing OES was chosen as an appropriate OES for a screening level assessment based on it resulting in a conservatively high surface water concentration based on high volumes of releases paired with an assumption of a low flow (P50) in the receiving water body, with environmental concentrations exceeding those estimated in all other OES. Additionally, the generic release scenario for the Manufacturing OES estimates a combined release to wastewater, incineration, or landfill. Because the proportion of the release from Manufacturing OES to just surface water could not be determined from reasonably available information, and the discharge as wastewater includes the possibility of direct discharge without further treatment, for screening purposes EPA assumed that all of the release would be directly discharged to surface water, to represent an upper-bound of surface water concentrations. The tiered exposure approach utilized the highest resulting environmental concentrations from this release scenario as the basis of a screening analysis for general population exposure. Table Table 4-3 and Table 4-4 presents the surface water concentrations associated with the Manufacturing OES modeled with median, 75th percentile, and 90th percentile (P50, P75, P90, respectively) flows. The hydrologic flow distribution for the generic scenario was developed from receiving waterbody flows from relevant facilities with NPDES permits, and this process is described in more detail in 13.4Appendix B.

Although Manufacturing OES was utilized for screening purposes, EPA prioritized use of programmatic data with actual release data from reporting facilities where overall confidence in the estimates would be higher. For estimating surface water concentrations from releases, the Agency prioritized the use of TRI annual release reports over DMR monitoring data, reviewing DMR period data as supporting information for the releases reported to TRI. Therefore, EPA estimated surface water concentrations from Waste handling, treatment, and disposal OES that had release data collected from TRI and DMR databases. Surface water concentrations associated with Waste handling, treatment, and disposal OES are presented in Table 4-3 and Table 4-4.

Receiving water body DBP concentrations were estimated at the point of release (*i.e.*, stream DBP concentration at the location where DBP-containing effluent is discharging). Release data were collected from TRI and DMR databases, which represent effluent loading after any on-site treatment; therefore, no further treatment or removal is estimated in this high-end release estimate screening assessment. For

releases estimated using generic scenarios, EPA also assumed no treatment or removal for a high-end release estimate screening assessmnt. Due to the partitioning of the compound to solids (in addition to some expected biodegradation), wastewater treatment is expected to be effective at removing DBP from the water column prior to discharge, with treated effluent showing up to a 96.6 percent reduction in one study (Tran et al., 2014), and an EPA review finding a typical removal efficiency of 68 percent (U.S. EPA, 1982).

Release modeling values shown in Table 4-3 are carried through to the ecological risk assessment for further evaluation as a conservative high-end approach to screen for ecological risk as discussed in the *Draft Environmental Hazard Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024c), following the screening approach as described in Section 5.3.1 of the *Draft Risk Evaluation for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025d).

Table 4-3. PSC Modeling Results for Water and Benthic Sediment Using 7Q10 Flow

OES	Number of Operating Days Per Year	Daily Release (kg/day) [a]	Flow Distribution Percentile [b]	7Q10 Total Water Column Concentration (µg/L)	7Q10 Benthic Pore Water Concentration (µg/L)	7Q10 Benthic Sediment Concentration (mg/kg)
Waste handling, treatment, and disposal (TRI-reported release)	286	0.043	N/A (Reported water body flow obtained from NHDPlus)	14.40	6.01	0.335
Manufacturing (generic multimedia release)	300	43	P50	1,160	484.0	27
			P75	67.8	28.2	1.58
			P90	4.00	1.67	0.093
Application of paints and coatings (no spray control) (generic multimedia release)	287	34	P50	920	383	21.4
			P75	53.6	22.3	1.25
			P90	3.17	1.32	0.074
Use of lubricants and fluids (generic wastewater release)	4	26	P50	703	34.20	1.91
			P75	41	2.61	0.146
			P90	2.42	0.12	0.0066

[a] Details on operating days and daily releases are provided in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b)
[b] The P50, P75, and P90 flows refer to the 50th, 75th, and 90th percentiles of the distribution of water body flow rates in generic release scenarios; see Appendix B.

For the purpose of a screening analysis as described in Section 2, EPA modeled high-end surface water concentrations using releases associated with OESs leading to the highest surface water concentrations. The OES with the highest total water column concentrations (Manufacturing) was additionally run under harmonic mean and 30Q5 flow conditions. Surface water concentrations shown in Table 4-4 are carried through to the human health risk assessment for further evaluation as a conservative high-end approach to screen for human health risk as discussed in the screening approach detailed in Section 2.

Table 4-4. PSC Modeling Results for Total Water Column Using Harmonic Mean Flow and 30Q5 Flow

OES	Flow Distribution Percentile [b]	Release Estimate (kg/day) [a]	Harmonic Mean Flow (m^3/d)	30Q5 Flow (m^3/d)	Harmonic Mean Concentration (µg/L)	30Q5 Concentration (µg/L)
Manufacturing (generic multimedia release)	P50	43	69,800	13,821	616.0	885.0
	P75	43	1,763,000	926,000	24.4	46.6
	P90	43	25,240,000	14,320,000	1.7	3.0
Waste handling, treatment, and disposal (TRI reported release)	N/A (Reported water body flow obtained from NHDPlus)	0.132	9,139	9,139	14.5	14.5

[a] Details on operating days and daily releases are provided in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b)
[b] The P50, P75, and P90 flows refer to the 50th, 75th, and 90th percentiles of the distribution of water body flow rates in generic release scenarios; see Appendix B.

4.2 Measured Concentrations

EPA identified monitoring studies through systematic review to provide context to modelling results. The monitoring studies presented here were not used as part of the analysis for quantifying exposure estimates. Measured concentrations of DBP in surface water and sediment are presented in Section 4.2.1 and 4.2.2, respectively.

4.2.1 Measured Concentrations in Surface Water

A total of three references were identified from the United States that reported DBP in surface water (NWQMC, 2021; Li et al., 2019; Liu et al., 2013) (Table 4-5). EPA STOrage and RETrieval (STORET) data were obtained through the Water Quality Portal (WQP), which houses publicly available water quality data from the U.S. Geological Survey (USGS), EPA, and state, federal, Tribal, and local agencies (NWQMC, 2021). Since 2004, the maximum level reported in water was 40 µg/L. Where the media subdivision was specified as surface water, the maximum level reported was 8.2 µg/L.

In March 2008 through June 2009, Liu et al. (2013) assessed the spatial distribution of phthalates in Lake Pontchartrain, LA, before, during, and after the opening of the Bonnet Carré Spillway that occurred April to May 2008. Forty-two freshwater samples were collected from the Bonnet Carré Spillway at 6 sites located about 1 mile apart. DBP was detected in 95 percent of these samples with concentrations ranging from nondetect to 5.9 µg/L. Fifty-four samples were also collected from the central lake area at 6 sites located near Lake Maurepas to the Causeway Bridge, with 1 site near the Manchac Pass. DBP was detected in 80 percent of these samples with concentrations up to 3.9 µg/L.

For the central lake area, authors reported that concentrations of phthalates, including DBP, were close to zero before opening of the spillway, increased significantly after opening of the spillway, and dropped back down to almost zero 1 year following the spillway opening. For the Bonnet Carré Spillway area, authors reported that phthalate levels were high even before the spillway opened due to freshwater flows from the Mississippi River, but levels dropped close to zero 1 year following the spillway opening. Samples collected in June 2009 showed phthalate increases, once again likely from a combination of

rain/stormwater, industrial discharges, and inputs from the Mississippi River (Liu et al., 2013).

Li et al. (2019) evaluated chemical emissions and residuals associated with the installation of UV-cured in-place pipes (CIPPs) for stormwater culverts at three sites in Syracuse, New York, and one site in Fairfax, Virginia. Standing water at culvert inlets and outlets, truck water, and rinse water exiting each CIPP were sampled and analyzed at New York sites whereas truck water and rinse water were sampled and analyzed in Virginia. A maximum DBP concentration of 12.5 µg/L was found in rinse water at New York Site #3. No DBP was detected in samples of truck water or rinse water in Virginia.

Four additional studies, three from France and one from South Korea, reported levels of DBP in surface water. Valton et al. (2014) examined levels of phthalates in the Orge River, a suburban tributary of the Seine River. The authors reported that the Orge River basin is characterized by intense human impact associated with agricultural areas upstream and urbanized and industrialized areas downstream. They collected freshwater samples from the outlet of the Orge River basin and found DBP at an average concentration of 120 ng/L (0.12 µg/L). Sampling year, number of samples, and detection frequency were not reported.

From 2015 to 2016, Bach et al. (2020) conducted a national sampling campaign in France of drinking water networks supplied by groundwater, surface water, or a mixture of both. As part of this sampling campaign, 114 raw surface water samples were collected. DBP was detected once at a concentration of 768 ng/L (0.768 µg/L).

A study conducted by Schmidt et al. (2020) in 2017 to 2018 quantified phthalate concentrations in the Rhône River in Arles city, France. This river exports water to the Gulf of Lion, the main freshwater source of the Mediterranean Sea. Surface water samples were collected monthly in duplicate at an arm's length from the dock in the Rhône River. DBP was detected in all samples with a mean concentration of 32.8 ng/L (0.328 µg/L).

From 2016 to 2017, Lee et al. (2019) assessed the seasonal and spatial distribution of phthalate esters in air, surface water, sediments, and fish in the Asan Lake in South Korea. Asan Lake is one of the largest artificial lakes in Korea and is mainly used for agricultural and industrial purposes and discharges to Asan Bay. Forty-seven surface water samples were collected at 12 sampling locations. DBP was detected in approximately 53 percent of samples at a mean concentration of 0.03 µg/L and maximum concentration of 0.34 µg/L.

Table 4-5. Summary of Measured DBP Concentrations in Surface Water

Reference	Sampling Location	DBP Concentration	Sampling Notes
Water Quality Portal (WQP) (NWQMC, 2021)[a]	United States	Overall: ND–40 µg/L Maximum levels by media subdivision (µg/L): 26.8 (unspecified); 40 (groundwater); 8.2 (surface water); 15 (stormwater); 14 (wastewater)	U.S. STOrage and RETrieval (STORET) water quality data, 2004 and after
Liu et al. (2013)	United States	Bonnet Carré Spillway (6 locations; n = 42) FOD: 95%	Freshwater samples from Lake Pontchartrain, LA, before, during, and after opening of the Bonnet

Reference	Sampling Location	DBP Concentration	Sampling Notes
		<0.03–5.9 µg/L Central lake area (6 locations; n = 54) FOD: 80% <0.03–3.9 µg/L	Carré Spillway that occurred April/May 2008, March 2008–June 2009
Li et al. (2019)	United States	Standing water (µg/L) NY sites: 4.8–9.6; VA site: not evaluated Rinse water (µg/L) NY sites: 6.3–12.5; VA site: ND Truck water (µg/L) NY sites: 4.8–6.5; VA site: ND	Water sampling conducted before and after installation of CIPPs, including standing water at culvert inlets and outlets, truck water, and rinse water, 2017
Valton et al. (2014)	France	FOD and sample number NR mean ± SD = 120 ± 80 ng/L	Freshwater samples from the outlet of the Orge River basin, date NR
Bach et al. (2020)	France	FOD = 0.88%* (n = 114), <500–768 ng/L LOQ = 500 ng/L *Calculated	National screening study to examine phthalates in raw surface water (prior to treatment for use as drinking water), November 2015–July 2016
Schmidt et al. (2020)	France	FOD 100% (n = 22) Median, mean ± SD (range) = 19.0, 32.8 ± 31.0 (7.3–107.7) ng/L LOQ = 0.03 ng/L	Monthly Rhône River samples, May 2017–April 2018
Lee et al. (2019)	South Korea	FOD = 53.2% (n = 47) Mean, median (range) = 0.03, 0.01 (ND–0.34) µg/L *A value of zero was used for nondetects. LOD and LOQ were 0.00 and 0.01 µg/L, respectively.	Freshwater samples from Asan Lake collected at 12 sampling locations, 2016–2017

FOD = frequency of detection; ND = non-detect; LOD = limit of detection; SD = standard deviation; LOQ = limit of quantification

[a] Represents samples dated 2004 and after. Values where "result sample fraction" is "total," and "result status identifier" is "final." Results presented by media subdivision if media subdivision was specified. Results may be estimated or actual results.

4.2.2 Measured Concentrations in Sediment

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data

to obtain concentrations of DBP in sediment. One reference from the United States was available. EPA STORET sediment data (surface, subsurface, or unspecified matrices) were obtained through the WQP (NWQMC, 2021). Since 2004, the maximum level in sediment (59,900 µg/kg dw) came from a sample where media subdivision was unspecified (Table 4-6).

From 2016 to 2017, Lee et al. (2019) assessed the seasonal and spatial distribution of phthalate esters in air, surface water, sediments, and fish in the Asan Lake in South Korea. Asan Lake is one of the largest artificial lakes in Korea and is mainly used for agricultural and industrial purposes and discharges to Asan Bay. It is likely affected by pollution coming from an industrial complex and two nearby cities. Forty-seven sediment samples were collected at 12 sampling locations. DBP was detected in approximately 64 percent of samples at a mean concentration of 73.6 µg/kg dw.

Table 4-6. Summary of Measured DBP Concentrations in Sediment

Reference	Sampling Location	DBP Concentration	Sampling Notes
Water Quality Portal (WQP) (NWQMC, 2021) [a]	United States	Overall: 59,900 µg/kg dw Maximum levels by media subdivision (µg/kg): 59,900 (unspecified, dw); 6,610 (surface); 200 (subsurface, dw)	U.S. STOrage and RETrieval (STORET) water quality data, 2004 and after
Lee et al. (2019)	South Korea	FOD 63.8% (n = 47) Mean, median (range) = 73.6, 13.3 (ND*–535) µg/kg dw *A value of zero was used for nondetects. LOD and LOQ were 0.40 and 1.21 µg/kg dw, respectively	Freshwater samples from Asan Lake collected at 12 sampling locations, 2016–2017

dw = dry weight; FOD = frequency of detection; ND = non-detect; LOD = limit of detection; LOQ = limit of quantification

[a] Represents samples dated 2004 and after. Values where "result sample fraction" is "total" and "result status identifier" is "final." Results presented by media subdivision if media subdivision was specified. Results may be estimated or actual results.

4.3 Evidence Integration for Surface Water and Sediment

4.3.1 Strengths, Limitations, and Sources of Uncertainty for Modeled and Monitored Surface Water Concentration

EPA used PSC to estimate concentrations of DBP within surface water and sediment. PSC considers model inputs of physical and chemical properties of DBP (*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and allows EPA to estimate sediment concentrations in addition to surface water concentrations. The use of physical and chemical properties of DBP refined through the systematic review process and supplemented by EPA models increases confidence in the application of the PSC model. A standard EPA waterbody geometry and sediment characteristics were used to represent a consistent and conservative receiving waterbody scenario, with chemical-specific release amounts and receiving waterbody hydrologic flow rates.

The modeled data represent estimated concentrations near actual facilities that are actively releasing DBP to wastewater, while the measured concentrations presented above in Table 4-5 represent sampled ambient water concentrations of DBP. However, measured concentrations are not necessarily associated with TSCA COUs, and the source or sources of these concentrations are unknown. Furthermore, the measured data may not represent locations where the general population may be exposed, either

incidentally or via drinking water. Measured DBP data are included in the exposure assessment as a point of reference and comparison with the modeled release estimates to verify that exposure estimates from modeled releases are not underestimating environmental concentrations reported in monitoring data. Differences in magnitude between modeled and measured concentrations may be due to measured concentrations not being geographically or temporally close to known releases of DBP. Monitoring data did not specifically target industrial releases and may reflect concentrations from sources not regulated under TSCA. While monitoring data locations are known, these data were not evaluated for proximity to known industrial releases.

Concentrations of DBP within the sediment were estimated using the highest 2015 to 2020 annual releases and estimates of 7Q10 hydrologic flow data for the receiving water body that were derived from the NHDPlus V2.1 EROM flow data, for the specific reach codes associated with releasing facilities as listed on their NPDES permits. The 7Q10 flow represents the lowest 7-day flow in a 10-year period and is a conservative approach for examining a condition where a potential contaminant may be predicted to be elevated due to periodic low flow conditions. Flow data collected via the EPA ECHO API and the NHDPlus V2.1 EROM flow database include self-reported hydrologic reach codes on NPDES permits and the best available flow estimations from the EROM flow data. Additionally, a regression-based calculation was applied to estimate flow statistics from NHD-acquired flow data, which introduces some uncertainty. The confidence in the flow values used, with respect to the universe of facilities for which data were pulled, should be considered moderate-to-robust, given the self-reported linkages to actual releasing facilities.. EPA assumes that the results presented in this section include a bias toward overestimation of resulting environmental concentrations due to conservative assumptions made in light of the uncertainties.

Release data were collected from TRI and DMR databases for use in this assessment, as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b). While TRI includes total annual reported loadings, DMR reporting includes monitoring summaries over shorter periods, such as weekly or monthly average concentrations. EPA's Pollutant Loading Tool is used to extrapolate DMR monitoring data and estimate annual total release. EPA reviews the period monitoring data from DMR reporting to verify annual load estimates from the Pollutant Loading Tool. In this assessment, two releasing facilities within the Waste handling, treatment, and disposal – POTW OES were identified as having erroneously high annual release amounts estimated by the Pollutant Loading Tool. Inspection of the DMR period data showed reports of DBP below the detection limit for all but one sample between the two facilities, with that single daily maximum sample reporting a concentration of 0.28 µg/L. Based on these records, EPA excluded the release estimates from these two facilities from the consideration of the high-end of the Waste handling, treatment, and disposal – POTW OES, and the next highest release was considered.

4.4 Weight of Scientific Evidence Conclusions

Modeled inputs were derived from reasonably available literature collected and evaluated through EPA's systematic review process for this TSCA risk evaluation. All monitoring and experimental data included in this analysis were from articles rated "medium" or "high" quality from this process. Monitoring data demonstrate that DBP can be detected in various types of water and sediment around the country. While monitoring data are limited and may not specifically target peak concentrations in the environment resulting from facility effluent, environmental monitoring data show generally low concentrations within the water column, and notable partitioning to sediment. The high-end modeled concentrations, based on industrial release data, for surface water and sediment exceeded the highest values available from monitoring studies by one to two orders of magnitude. This supports EPA's approach in conducting a screening evaluation using the highest modeled DBP concentrations.

5 SURFACE WATER EXPOSURE TO GENERAL POPULATION

Concentrations of DBP in surface water resulting from TSCA COU releases can lead to different exposure scenarios, including dermal exposure (Section 5.1.1) or incidental ingestion exposure (Section 5.1.2) to the general population swimming in affected waters. Additionally, DBP surface water concentrations may impact drinking water exposure (Section 6) and fish ingestion exposure (Section 7).

For the purpose of risk screening, exposure scenarios were assessed for various lifestages (*e.g.*, adult, youth, children) using the highest concentration of DBP in surface water based on the highest releasing OES (PVC plastics compounding) as estimated in Section 4.1.

5.1 Modeling Approach

5.1.1 Dermal Exposure

The general population may swim in surface waters (streams and lakes) that are affected by DBP contamination. Modeled surface water concentrations estimated in Section 4.1 were used to estimate acute doses (ADR) and average daily doses (ADD) from dermal exposure while swimming. The following equations were used to calculate incidental dermal (swimming) doses for adults, youth, and children:

Equation 5-1. Acute Incidental Dermal Calculation

$$ADR = \frac{(SWC \times K_p \times SA \times ET \times CF1 \times CF2)}{BW}$$

Where:

ADR	=	Acute dose rate (mg/kg-day)
SWC	=	Surface water concentration (ppb or µg/L)
K_p	=	Permeability coefficient (cm/h)
SA	=	Skin surface area exposed (cm^2)
ET	=	Exposure time (h/day)
$CF1$	=	Conversion factor (1.0×10^{-3} mg/µg)
$CF2$	=	Conversion factor (1.0×10^{-3} L/cm^3)
BW	=	Body weight (kg)

Equation 5-2. Average Daily Incidental Dermal Calculation

$$ADD = \frac{(SWC \times K_p \times SA \times ET \times RD \times ED \times CF1 \times CF2)}{(BW \times AT \times CF3)}$$

Where:

ADD	=	Average daily dose (mg/kg-day)
SWC	=	Chemical concentration in water (µg/L)
K_p	=	Permeability coefficient (cm/h)
SA	=	Skin surface area exposed (cm^2)
ET	=	Exposure time (h/day)
RD	=	Release days (days/year)
ED	=	Exposure duration (years)
BW	=	Body weight (kg)

AT = Averaging time (years)
$CF1$ = Conversion factor (1.0×10^{-3} mg/µg)
$CF2$ = Conversion factor (1.0×10^{-3} L/cm^3)
$CF3$ = Conversion factor (365 days/year)

A summary of inputs utilized for these exposure estimates are provided in Appendix A. EPA used the DBP dermal permeability coefficient (K_p) of 0.016 cm/h (U.S. EPA, 2024b) and Consumer Exposure Model (CEM) (U.S. EPA; ICF Consulting, 2022) to estimate the steady-state aqueous permeability coefficient of DBP.

Table 5-1 shows a summary of the estimates of ADRs and ADDs due to dermal exposure while swimming for adults, youth, and children. Doses are calculated using Equation 5-1 and Equation 5-2, using the highest surface water concentration from the Manufacturing OES. Dermal doses were also calculated using the highest monitored surface water concentration from the WQP ((NWQMC, 2021); Section 4.2.1) as the surface water concentration. Doses calculated using the surface water monitoring data are on the same order of magnitude as corresponding doses modeled using the high-end Manufacturing OES.

Releases associated with the Manufacturing OES resulted in the highest total water column concentrations among reported releases, with water concentrations of 885 µg/L using 30Q5 flow (the lowest 30-day average flow in a 5-year period). Because of relevance to the exposure route, acute incidental surface water exposures and acute drinking water exposures were derived from the 30Q5 flow concentrations, and chronic drinking water exposures were derived from the harmonic mean (HM) flow concentrations. COUs mapped to the Manufacturing OES are shown in Table 1-1. Manufacturing OES was chosen as an appropriate OES for a screening level assessment based on it resulting in a conservatively high surface water concentration based on high volumes of releases associated with low flow metrics (P50). Additionally, the generic release scenario for the Manufacturing OES estimates a combined release to wastewater, incineration, or landfill. The proportion of the release from Manufacturing OES to just surface water could not be determined from reasonably available information, so for screening purposes EPA assumed that all of the release would be to wastewater to represent an upper-bound of surface water concentrations and no wastewater treatment was assumed.

Table 5-1. Dermal (Swimming) Doses Across Lifestages[a]

Scenario	Water Column Concentrations		Adult (21+ years)		Youth (11–15 years)		Child (6–10 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing[b]	885	616	1.04E−02	1.97E−05	7.93E−03	1.51E−05	4.81E−03	9.17E−06
Highest monitored surface water (NWQMC, 2021)	26.8	26.8	3.14E–04	8.59E–07	2.40E–04	6.58E–07	1.46E–04	3.99E–07

30Q5 = 30 consecutive days of lowest flow over a 5-year period; POT = potential
[a] Doses calculated using Equation 5-1 and Equation 5-2.
[b] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.

5.1.2 Oral Exposure

The general population may swim in surfaces waters (streams and lakes) that are affected by DBP contamination. Modeled surface water concentrations estimated in Section 4.1 were used to estimate ADR and ADD due to ingestion exposure while swimming.

The following equations were used to calculate incidental oral (swimming) doses for adults, youth, and children using the Manufacturing OES that resulted in the highest modeled surface water concentrations, as well as calculated using the highest monitored surface water concentration from the WQP (NWQMC, 2021):

Equation 5-3. Acute Incidental Ingestion Calculation

$$ADR = \frac{(SWC \times IR \times CF1)}{BW}$$

Where:

ADR	=	Acute dose rate (mg/kg-day)
SWC	=	Surface water concentration (ppb or µg/L)
IR	=	Daily ingestion rate (L/day)
$CF1$	=	Conversion factor (1.0×10^{-3} mg/µg)
BW	=	Body weight (kg)

Equation 5-4. Average Daily Incidental Calculation

$$ADD = \frac{(SWC \times IR \times ED \times RD \times CF1)}{(BW \times AT \times CF2)}$$

Where:

ADD	=	Average daily dose (mg/kg-day)
SWC	=	Surface water concentration (ppb or µg/L)
IR	=	Daily ingestion rate (L/day)
ED	=	Exposure duration (years)
RD	=	Release days (days/yr)
$CF1$	=	Conversion factor (1.0×10^{-3} mg/µg)
BW	=	Body weight (kg)
AT	=	Averaging time (years)
$CF2$	=	Conversion factor (365 days/year)

A summary of inputs utilized for these estimates are presented in Appendix A.1. Incidental ingestion doses derived from the modeled concentration presented in Section 4.1 and the above exposure equations are presented in Table 5-2.

Table 5-2. Incidental Ingestion Doses (Swimming) Across Lifestages

Scenario	Water Column Concentrations		Adult (21+ years)		Youth (11–15 years)		Child (6–10 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing(P50) [a]	885	616	3.05E−03	5.82E−06	4.74E−03	9.03E−06	2.67E−03	5.09E−06
Highest monitored surface water (NWQMC, 2021)	26.8	26.8	9.25E−05	2.53E−07	1.43E−04	3.93E−07	8.09E−05	2.22E−07

30Q5 = 30 consecutive days of lowest flow over a 5-year period; POT = potential
[a] Only this OES paired with low flow assumptions was used in the screening assessment because it resulted in the highest surface water concentrations.

5.2 Weight of Scientific Evidence Conclusions

Surface water and sediment concentrations of DBP were modeled using facility release data reported to TRI and DMR databases. As such, EPA has moderate to robust confidence in the release data and the resulting modeled surface water concentrations at the point of release in the receiving waterbody. The high end of those resulting concentrations and exposure estimates are presented in this document. Screening level risk estimates derived from the exposures modeled in this section are discussed in Appendix C and demonstrate no risk estimates for the general population below the benchmark. The screening approach applied for modeling, in conjunction with the available monitoring data showing lower concentrations than those modeled, provide multiple lines of evidence and robust confidence that releases to surface water will not exceed the release concentrations presented in this assessment, which do not appear to pose risk to human health.

Swimming Ingestion/Dermal Estimates

Two scenarios (youth being exposed dermally and through incidental ingestion while swimming in surface water) were assessed as high-end potential exposures to DBP in surface waters. EPA's *Exposure Factors Handbook* provided detailed information on the youth skin surface areas and event per day of the various scenarios (U.S. EPA, 2017b). Non-diluted surface water concentrations were used when estimating dermal exposures to youth swimming in streams and lakes. DBP concentrations will dilute when released to surface waters but it is unclear what level of dilution will occur when the general population swims in waters with DBP releases.

6 DRINKING WATER EXPOSURE TO GENERAL POPULATION

Drinking water in the United States typically comes from surface water (*i.e.*, lakes, rivers, and reservoirs) and groundwater. The source water then flows to a treatment plant where it undergoes a series of water treatment steps before being dispersed to homes and communities. In the United States, public water systems often use conventional treatment processes that include coagulation, flocculation, sedimentation, filtration, and disinfection, as required by law.

Very limited information is reasonably available on the removal of DBP in drinking water treatment plants. As stated in the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate* (U.S. EPA, 2024g), no data were identified by the EPA for DBP in U.S. drinking water. Based on the low water solubility and log K_{OW}, DBP in water is expected to mainly partition to suspended solids present in water. The reasonably available information suggests that the use of flocculants and filtering media could potentially help remove DBP during drinking water treatment by sorption into suspended organic matter, settling, and physical removal.

6.1 Modeling Approach for Estimating Concentrations in Drinking Water

6.1.1 Drinking Water Ingestion

Drinking Water Intake Estimates via Modeled Surface Water Concentrations

Modeled surface water concentrations estimated in Section 4.1 were used to estimate drinking water exposures. For this screening exercise, only the highest modeled facility release was included in the drinking water exposure analysis, alongside the highest monitored surface water concentration. The estimated exposure concentrations presented in this section reflect releases reported by a facility as actual effluent loading (after any wastewater treatment). A range of wastewater and drinking water treatment removal efficiencies for DBP are discussed in *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate* (U.S. EPA, 2024g), and the high-end exposure from a modeled facility release presented here does not include any additional calculated removal from drinking water treatment. The drinking water scenario presented here is expected to be the scenario most representative of a possible upper-bound for drinking water exposure in the general population.

Drinking water doses were calculated using the following equations:

Equation 6-1. Acute Drinking Water Ingestion Calculation

$$ADR_{POT} = \frac{(SWC \times \left(1 - \frac{DWT}{100}\right) \times IR_{dw} \times RD \times CF1)}{(BW \times AT)}$$

Where:

ADR_{POT}	=	Potential acute dose rate (mg/kg/day)
SWC	=	Surface water concentration in receiving waterbody (ppb or µg/L; 30Q5 conc for ADR, harmonic mean for ADD, LADD, LADC)
DWT	=	Removal during drinking water treatment (%) (not applied for this analysis)
IR_{dw}	=	Drinking water intake rate (L/day)
RD	=	Release days (days/yr for ADD, LADD, and LADC; 1 day for ADR)
$CF1$	=	Conversion factor (1.0×10^{-3} mg/µg)
BW	=	Body weight (kg)

AT = Exposure duration (years for ADD, LADD, and LADC; 1 day for ADR)

Equation 6-2. Average Daily Drinking Water Ingestion Calculation

$$ADD_{POT} = \frac{(SWC \times \left(1 - \frac{DWT}{100}\right) \times IR_{dw} \times ED \times RD \times CF1)}{(BW \times AT \times CF2)}$$

Where:

ADD_{POT} = Potential average daily dose (mg/kg/day)
SWC = Surface water concentration in receiving waterbody (ppb or µg/L; 30Q5 conc for ADR, harmonic mean for ADD, LADD, LADC)
DWT = Removal during drinking water treatment (%) (not applied for this analysis)
IR_{dw} = Drinking water intake rate (L/day)
ED = Exposure duration (years for ADD, LADD, and LADC; 1 day for ADR)
RD = Release days (days/yr for ADD, LADD, and LADC; 1 day for ADR)
BW = Body weight (kg)
AT = Exposure duration (years for ADD, LADD, and LADC; 1 day for ADR)
$CF1$ = Conversion factor (1.0×10^{-3} mg/µg)
$CF2$ = Conversion factor (365 days/year)

The ADR and ADD from drinking water for chronic non-cancer were calculated using the 95th percentile ingestion rate for drinking water. The lifetime average daily dose (LADD) was not estimated because available data are insufficient to determine the carcinogenicity of DBP (U.S. EPA, 2024f). Therefore, EPA is not evaluating DBP for carcinogenic risk. Table 6-1 summarizes the drinking water doses for adults, infants, and toddlers. These estimates do not incorporate additional dilution beyond the point of discharge, and in this case, it is assumed that the surface water outfall is located very close (within a few km) to the drinking water intake location. Applying dilution factors would decrease the concentration at the intake as well as the dose for all scenarios.

Table 6-1. Drinking Water Doses Across Lifestages

Scenario	Surface Water Concentrations		Adult (21+ years)		Infant (Birth to <1 year)		Toddler (1–5 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing[a]	885	616	3.56E−02	1.86E−05	1.25E−01	4.74E−05	4.44E−02	2.03E−05
Highest monitored surface water (NWQMC, 2021)	26.8	26.8	1.08E–03	8.07E–07	3.78E–03	2.06E–06	1.35E–03	8.84E–07

30Q5 = 30 consecutive days of lowest flow over a 5-year period; POT = potential
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.

6.2 Measured Concentrations in Drinking Water

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of DBP in drinking water. EPA identified monitoring studies through systematic review to provide context to modelling results. The monitoring studies presented here were

not used as part of the analysis for quantifying exposure estimates. No studies conducted in the United States or Canada were identified that reported concentrations of DBP in drinking water. Drinking water quality data from 2011 through 2022 were obtained from the California Water Boards (2022) for 39 counties in the state (Table 6-2). For the more than 200 active, inactive, or proposed water systems and facilities, DBP was detected in approximately two percent of samples at levels up to 3.1 µg/L. The highest level of DBP was detected in a 2015 sample from an active Arvin Community Services water system in Kern County. Table 6-2 also presents DBP levels in drinking water from two studies conducted in high-income foreign countries. Bach et al. (2020) conducted a national screening study in France to examine levels of phthalates in raw and treated tap water. From 2015 to 2016, 283 treated water samples were examined: 166 supplied by groundwater, 89 supplied by surface water, and 28 supplied by a mixture of surface and groundwater. DBP was detected once for each of the three supply types at a maximum level of 1,340 ng/L. In a second study conducted in Romania in 2017, phthalates were measured in municipal drinking water and consumed bottled water (Sulentic et al., 2018). Ten tap water samples and sixteen bottled water samples that combined brand, type (still or gas), and storage conditions (room temperature or refrigerated) were collected and analyzed for four phthalates. DBP was not detected in the tap water samples. Overall, the median level of DBP in bottled water was 3.23 µg/L. Still water (5.61 µg/L) had a higher median concentration of DBP than gas water (2.16 µg/L). Bottled water at room temperature (3.87 µg/L) had a higher median concentration of DBP than bottled water that was refrigerated (3.05 µg/L).

Table 6-2. Summary of Measured DBP Concentrations in Drinking Water

Reference	Sampling Location	DBP Concentration	Sampling Notes
CA Water Board (2022)	United States	FOD: 1.9% (3 detects in raw (untreated) water [2 inactive, 1 active wells] from Arvin Community Services in Kern County) Overall: <1–3.1 µg/L	Over 1,500 records of DBP levels in drinking water, 2011–2022
Bach et al. (2020)	France	FOD = 1.2% (n = 283) Level by supply type (ng/L) Surface water (n = 89): <LOQ–951 Groundwater (n = 166): <LOQ–1,340 Mixture of surface and groundwater (n = 28): <LOQ–1,114 LOQ = 500 ng/L	National screening study to examine phthalates in treated tap water, November 2015–July 2016
Sulentic et al. (2018)	Romania	Tap water (n = 10) (µg/L) FOD 0%*, median (IQR) = ND (ND, ND) Bottled water (n = 16) (µg/L) FOD NR, median (IQR) = 3.23 (ND, 6.15) LOD = 0.015 µg/L	Tap and bottled water samples were collected as part of an exposure assessment in Romanian adolescents, 2017
FOD = frequency of detection LOD = level of detection; LOQ = limit of quantification; ND = non-detect			

6.3 Evidence Integration for Drinking Water

EPA estimates low potential exposure to DBP via drinking water, with or without considering expected treatment removal efficiencies, even under high-end release scenarios. These exposure estimates also assume that the drinking water intake location is very close (within a few km) to the point of discharge and do not incorporate any dilution beyond the point of discharge. Actual concentrations in raw and finished water are likely to be lower than these conservative estimates as applying dilution factors will decrease the exposure for all scenarios, and additional distances downstream would allow further partitioning and degradation. Monitoring data from finished drinking water in the United States are mostly non-detect for DBP, with a highest reported concentration of 3.1 µg/L, corroborating the expectation of very little exposure to the general population via treated drinking water. Monitoring data also present evidence for generally low concentrations in ambient waters beyond direct points of release. Screening level risk estimates derived from the exposures discussed in this section are presented in Appendix 13.4C.2 and screening level risk estimates were above the benchmark MOE at the upper-bound of exposure for all but the most extreme and unlikely release and exposure scenarios.

6.4 Weight of Scientific Evidence Conclusions

EPA has moderate to high confidence in the surface water as drinking water exposure scenario due to the site-specific uncertainty presented in this section and robust evidence of presenting an upper-bound of exposure with risk beyond the benchmark. As described in Section 3.2, EPA did not assess drinking water estimates as a result of leaching from landfills to groundwater and subsequent migration to drinking water wells.

7 FISH INGESTION EXPOSURE TO GENERAL POPULATION

To estimate exposure to humans from fish ingestion, EPA used multiple surface water concentrations in its assessment: the water solubility of 11.2 mg/L (U.S. EPA, 2024g), the maximum modeled concentration based on reported and estimated releases, and the measured concentrations from monitoring data. Incorporating multiple surface water concentrations accounts for the variation shown in Table 7-1, such as when an OES may result in concentrations exceeding the water solubility limit. The selected surface water concentrations are also the highest among modeled and monitored values, facilitating their use in a screening level analysis that incorporates conservative assumptions.

Another important parameter in estimating human exposure to a chemical through fish ingestion is the bioaccumulation factor (BAF). BAF is preferred over bioconcentration factor (BCF) because it considers the animal's uptake of a chemical from both diet and the water column. For DBP, one high-quality study reporting BAF values for fish was identified during systematic review. Li et al. (2024) reported BAF values of 410 L/kg for tilapia and 314 L/kg for common carp (see *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g)). The BAFs of both fish species were included in this risk evaluation since tilapia is primarily herbivorous and is at a lower trophic level, while common carp reside at the bottom of the water column where DBP is expected to partition and would represent exposure at a higher trophic level. Table 7-1 compares the fish tissue concentration calculated using empirical BAFs with the measured fish tissue concentrations obtained from literature. Fish tissue concentrations calculated with empirical BAFs and water solubility limit were two to three orders of magnitude higher than empirical levels reported within published literature. This indicates that calculated fish tissue concentrations with the water solubility limit are likely overestimated.

The Manufacturing OES resulted in the highest concentration of DBP in receiving waters across all OESs (Section 4.1). The concentration was modeled using VVWM-PSC and represents the harmonic mean based on the highest modeled 95th percentile release to water. Surface water concentrations were estimated for various flows (*i.e.*, P50, P75, and P90). However, EPA does not expect waterbodies with P50 flow rates to receive high-end industrial and commercial releases and thus did not consider modeled surface water concentrations based on P50 flows. For OESs with TRI reported releases, the Waste handling, treatment, and disposal OES had the highest release to surface water. The surface water concentrations for this OES were also modeled using VVWM-PSC and represents the harmonic mean. Fish tissue concentrations calculated with the modeled surface water concentration were within the same order of magnitude or one order lower than empirical levels reported within published literature (Table 7-1).

In addition, EPA calculated fish tissue concentrations using the highest measured DBP concentrations in surface water. As described in Section 4.2.1, the maximum concentration was 8.2 µg/L (8.2×10^{-3} mg/L) from the WQP (NWQMC, 2021). Fish tissue concentrations calculated with empirical BAFs and monitored water surface concentrations are similar to the measured fish tissue concentrations obtained from literature (Table 7-1).

Table 7-1. Fish Tissue Concentrations Calculated from Modeled Surface Water Concentrations and Monitoring Data

Approach	Data Description	Surface Water Concentration	Fish Tissue Concentration
Water solubility limit	Empirical BAF values of 410 L/kg for tilapia and 314 L/kg for common carp (Li et al., 2024)	Estimates of the water solubility limit for DBP, which is approximately 11.2 mg/L (Howard et al., 1985)	4.59E03 mg/kg ww (tilapia) 3.52E03 mg/kg ww (common carp)
Modeled surface water concentrations	Empirical BAF values of 410 L/kg for tilapia and 314 L/kg for common carp (Li et al., 2024)	2.24E–02 mg/L for Manufacturing OES, P75, HE (generic scenario)	10.1 mg/kg ww (tilapia) 7.66 mg/kg ww (common carp)
		1.7E–03 mg/L for Manufacturing OES, P90, HE (generic scenario)	0.70 mg/kg ww (tilapia) 0.53 mg/kg ww (common carp)
		1.45E–02 mg/L for Waste Handling, Treatment, Disposal-POTW (TRI reported release)	5.95 mg/kg ww (tilapia) 4.55 mg/kg ww (common carp)
Monitored surface water concentration	Highest measured concentration from WQP (NWQMC, 2021) and empirical BAF values of 410 L/kg for tilapia and 314 L/kg for common carp (Li et al., 2024)	8.2E–03 mg/L	3.36 mg/kg ww (tilapia) 2.57 mg/kg ww (common carp)
Fish tissue monitoring data (wild-caught)	19 studies from over 70 different species, including four U.S. and two Canadian studies	N/A	Range for U.S. and Canadian studies: ND–35 mg/kg ww Range for other studies: ND–3.9 mg/kg ww
HE = high-end; ND = non-detect; ww = wet weight			

7.1 General Population Fish Ingestion Exposure

EPA estimated exposure from fish consumption using age-specific fish ingestion rates (Table_Apx A-2). Adults have the highest 50th percentile fish ingestion rate (IR) per kilogram of body weight for the general population, as shown in Table_Apx A-2. A young toddler between 1 and 2 years has the highest 90th percentile fish IR per kilogram of body weight. This section estimates exposure and risks for adults and toddlers aged 1 to 2 years who have those two lifestages with the highest fish IR per kilogram of body weight among all lifestages in this used as a screening level approach.

The ADR and ADD for chronic non-cancer estimates were calculated using the 90th percentile and central tendency IR, respectively. Cancer exposure (LADD, lifetime average daily dose) and risks were not characterized because there is insufficient evidence of DBP's carcinogenicity (U.S. EPA, 2024f). Estimated exposure to DBP from fish ingestion were calculated using the following equation:

Equation 7-1. Fish Ingestion Calculation

$$ADR\ or\ ADD = \frac{(SWC \times BAF \times IR \times CF1 \times CF2 \times ED)}{AT}$$

Where:

ADR	=	Acute dose rate (mg/kg/day)
ADD	=	Average daily dose (mg/kg/day)
SWC	=	Surface water (dissolved) concentration (µg/L)
BAF	=	Bioaccumulation factor (L/kg wet weight)
IR	=	Fish ingestion rate (g/kg-day)
$CF1$	=	Conversion factor (0.001 mg/µg)
$CF2$	=	Conversion factor for kg/g (0.001 kg/g)
ED	=	Exposure duration (year)
AT	=	Averaging time (year)

The inputs to this equation can be found in *Draft Fish Ingestion Risk Calculator for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025c). The years within an age group (*i.e.*, 62 years for adults) was used for the exposure duration and averaging time to estimate non-cancer exposure. The exposures calculated using the water solubility limit and maximum modeled and monitored surface water concentrations, with empirical BAFs, are presented in Table 7-2. Corresponding screening level risk estimates are shown in Appendix E.11. Fish ingestion is not expected to be a pathway of concern for the general population based on the conservative screening level risk estimates using an upper-bound of exposure.

Table 7-2. General Population Fish Ingestion Doses by Surface Water Concentration

Surface Water Concentration and Scenario	Adult ADR (mg/kg-day)	Young Toddler ADR (mg/kg-day)	Adult ADD (mg/kg-day)
Water solubility limit (11.2 mg/L)	1.27 (tilapia) 9.76E–01 (common carp)	1.89 (tilapia) 1.45 (common carp)	2.89E–01 (tilapia) 2.22E–01 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	2.78E–03 (tilapia) 2.13E–03 (common carp)	4.12E–03 (tilapia) 3.16E–03 (common carp)	6.30E–04 (tilapia) 4.83E–04 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	9.33E–04 (tilapia) 7.15E–04 (common carp)	1.39E–03 (tilapia) 1.06E–03 (common carp)	2.12E–04 (tilapia) 1.62–04 (common carp)
HE – high end			

7.2 Subsistence Fish Ingestion Exposure

Subsistence fishers represent a potentially exposed or susceptible subpopulation(s) (PESS) group due to their greatly increased exposure via fish ingestion (average of 142.4 g/day of fish consumed compared to a 90th percentile of 22.2 g/day for the general population) (U.S. EPA, 2000b). The ingestion rate for subsistence fishers applies only to adults aged 16 to less than 70 years. EPA calculated exposure for subsistence fishers using Equation 7-1 and the same inputs as the general population, with the exception of the increased ingestion rate. EPA is unable to determine subsistence fishers' exposure estimates specific to younger lifestages based on lack of reasonably available information. Furthermore, unlike the general population fish ingestion rates, there is no central tendency or 90th percentile ingestion rate for subsistence fishers. The same value was used to estimate both the ADD and ADR.

Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark as described in Appendix E.2. Therefore, EPA refined its evaluation by

using the OES that resulted in the highest modeled surface water concentrations based on releases to water combined with the flow rate of the receiving water body (Section 4.1). This refined analysis did not result in screening level risk estimates below the benchmark. Therefore, ingestion of fish potentially contaminated with DBP is not expected to be a pathway of concern for the subsistence fisher.

Table 7-3. Adult Subsistence Fisher Doses by Surface Water Concentration

Surface Water Concentration and Scenario	ADR/ADD (mg/kg-day)
Water solubility limit (11.2 mg/L)	8.17 (tilapia) 6.26 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	1.78E–02 (tilapia) 1.36E–02 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	5.98E–03 (tilapia) 4.58E–03 (common carp)
HE – high end	

7.3 Tribal Fish Ingestion Exposure

Tribal populations represent another PESS group. In the United States there are a total of 574 federally recognized American Indian Tribes and Alaska Native Villages and 63 state recognized tribes. Tribal cultures are inextricably linked to their lands, which provide all their needs from hunting, fishing, food gathering, and grazing horses to commerce, art, education, health care, and social systems. These services flow among natural resources in continuous interlocking cycles, creating a multi-dimensional relationship with the natural environment and forming the basis of *Tamanwit* (natural law) (Harper et al., 2012). Such an intricate connection to the land and the distinctive lifeways and cultures between individual tribes create many unique exposure scenarios that can expose tribal members to higher doses of contaminants in the environment. EPA used the reasonably available information to quantitatively evaluate the tribal fish ingestion pathway for DBP but lacks reasonably available data to assess other exposure scenarios unique to tribal populations.

U.S. EPA (2011a) (Chapter 10, Table 10-6) summarizes relevant studies on current tribal-specific fish ingestion rates that covered 11 tribes and 94 Alaskan communities. The highest central tendency value (a mean) ingestion rate per kilogram of body weight is reported in a 1997 survey of adult members (16+ years) of the Suquamish Tribe in Washington. Adults from the Suquamish Tribe reported a mean ingestion rate of 2.7 g/kg-day, or 216 g/day assuming an adult body weight of 80 kg. In comparison, the ingestion rates for adult subsistence fishers and the general population are 142.2 and 22.2 g/day, respectively. A total of 92 adults responded to the survey funded by the Agency for Toxic Substances and Disease Registry (ATSDR) through a grant to the Washington State Department of Health, of which 44 percent reported consuming less fish/seafood today compared to 20 years ago. One reason for the decline is restricted harvesting caused by increased pollution and habitat degradation (Duncan, 2000).

In addition to the current mean fish ingestion rate, EPA reviewed literature and surveys to identify a high-end (*i.e.,* 90th or 95th percentile) fish ingestion rate. The surveys asked participants to estimate their daily fish consumption over the course of a year by meal size and meal frequency. The highest 95th percentile fish and shellfish ingestion rate was 874 g/day, or 10.9 g/kg-day assuming a body weight of 80 kg, for male adults (18+ years) of the Shoshone-Bannock Tribes in Idaho (Polissar et al., 2016). The 95th percentile ingestion rate for males and females combined was similar at 10.1 g/kg-day. The Suquamish Tribe also reported similar high-end (90th percentile) ingestion rates for adults ranging from 8.56 to 9.73 g/kg-day (Duncan, 2000). Estimated high-end fish ingestion rates were lower for other

tribes in Alaska, the Pacific Northwest, Great Lakes region, and northeastern North America. To evaluate a current high-end exposure scenario, EPA used the highest 95th percentile ingestion rate of 10.9 g/kg-day.

Because current fish consumption rates are suppressed by contamination, degradation, or loss of access, EPA reviewed existing literature for ingestion rates that reflect heritage rates. Heritage ingestion rates refer to typical fish ingestion prior to non-indigenous settlement on tribal fisheries resources as well as changes in culture and lifeways (U.S. EPA, 2016). Heritage ingestion rates were identified for four tribes, all located in the Pacific Northwest region. The highest heritage ingestion rate was reported for the Kootenai Tribe in Idaho at 1,646 g/day, or 20.6 g/kg-day assuming an adult body weight of 80 kg (RIDOLFI, 2016; Northcote, 1973). Northcote (1973) conducted a comprehensive review and evaluation of ethnographic literature, historical accounts, harvest records, archaeological and ecological information, as well as other studies of heritage consumption. The heritage ingestion rate is estimated for Kootenai members living in the vicinity of Kootenay Lake in British Columbia, Canada; the Kootenai Tribe once occupied territories in parts of Montana, Idaho, and British Columbia. It is based on a 2,500 calorie per day diet, assuming 75 percent of the total caloric intake comes from fish which may overestimate fish intake. However, the higher ingestion rate also accounted for salmon fat loss during migration to spawning locations by using a lower caloric value for whole raw fish. Northcote (1973) assumed a caloric content of 113.0 cal/100 g wet weight. In comparison, the U.S. Department of Agriculture's Agricultural Research Service (1963) estimates a caloric content for fish sold in the United States to range from 142 to 242 cal/100 g of fish.

EPA calculated exposure via fish consumption for tribes using Equation 7-1 and the same inputs as the general population except for the ingestion rate. Three ingestion rates were used: 216 g/day (2.7 g/kg-day) for a central tendency current consumption rate; 874 g/day (10.9g/kg-day) as a high-end current tribal fish ingestion rate; and 1,646 g/day (20.58 g/kg-day) for heritage consumption. Similar to subsistence fishers, EPA used the same ingestion rate to estimate both the ADD and ADR. The heritage ingestion rate is assumed to be applicable to adults. For current ingestion rates, U.S. EPA (2011a) provides values specific to younger lifestages, but adults still consume higher amounts of fish per kilogram of body weight. An exception is for the Squaxin Island Tribe in Washington that reported an ingestion rate of 2.9 g/kg-day for children under 5 years. That ingestion rate for children is nearly the same as the adult ingestion rate of 2.7 g/kg-day for the Suquamish Tribe. As a result, exposure estimates based on current ingestion rates (IR) focused on adults (Table 7-4).

Table 7-4 presents multiple exposure estimates for the tribal populations. Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark as described in Appendix E.3. As a result, EPA refined its evaluation by using the two OESs that resulted in the highest modeled surface water concentrations. The surface water releases were estimated based on generic scenarios for one of the OESs and reported in TRI for the other OES. (Section 4.1). This refined analysis resulted in screening level risk estimates below the benchmark for the Manufacturing OES at the P75 flow rate and the current 95th percentile fish ingestion rate and heritage fish ingestion rate. However, EPA has slight confidence in the modeled surface water concentrations for the Manufacturing OES because the estimated release did not provide sufficient information to determine the fraction that discharges to water only. As such, EPA relied on reported TRI data for the Waste handling, treatment, and disposal OES where EPA has moderate-to-robust confidence in the risk estimates. Screening -level risk estimates for the Waste handling, treatment, and disposal OES were above benchmark for all scenarios. Therefore, ingestion of fish potentially contaminated with DBP is not a pathway of concern for tribal populations.

Table 7-4. Adult Tribal Fish Ingestion Doses by Surface Water Concentration

Surface Water Concentration and Scenario	ADR/ADD (mg/kg-day)		
	Current Tribal IR, Mean	Current Tribal IR, 95th Percentile	Heritage IR
Water solubility limit (11.2 mg/L)	1.24E01 (tilapia) 9.50 (common carp)	5.01E01 (tilapia) 3.83E01 (common carp)	9.45E01 (tilapia) 7.24E01 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	2.70E–02 (tilapia) 2.07E–02 (common carp)	1.09E–01 (tilapia) 8.35E–02 (common carp)	2.06E–01 (tilapia) 1.58E–01 (common carp)
Manufacturing OES, P90, HE (generic scenario) (1.7E–03 mg/L)	1.88E–03 (tilapia) 1.44E–03 (common carp)	7.60E–03 (tilapia) 5.82E–03 (common carp)	1.43E–02 (tilapia) 1.10E–02 (common carp)
Waste Handling, Treatment, Disposal-POTW (TRI reported release) (1.45E–02 mg/L)	1.61E–02 (tilapia) 1.23E–02 (common carp)	6.48E–02 (tilapia) 4.96E–02 (common carp)	1.22E–01 (tilapia) 9.37E–02 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	9.08E–03 (tilapia) 6.95E–03 (common carp)	3.66E–02 (tilapia) 2.81E–02 (common carp)	6.92E–02 (tilapia) 5.30E–02 (common carp)
CT – central tendency; HE – high end			

7.4 Weight of Scientific Evidence Conclusions

7.4.1 Strength, Limitations, Assumptions, and Key Sources of Uncertainty

To account for the variability in fish consumption across the United States, fish intake estimates were considered for general population, subsistence fishing populations, and tribal populations. A conservative screening analysis using the water solubility limit and the highest modeled surface water concentrations did not result in screening level risk estimates to be below the benchmark for the general population and subsistence fishers. However, for the tribal populations consuming fish at the 95th ingestion rate and heritage rate, risk estimates were below the benchmark for the highest modeled surface water concentration from the Manufacturing OES and P75 flow rate. EPA has only slight confidence in those risk estimates because the Manufacturing OES had modeled releases from generic scenarios discharging to multiple environmental media, and there is insufficient information to determine the fraction going to each of the media types. As such, EPA relied on reported TRI data for the Waste handling, treatment, and disposal OES where EPA has moderate-to-robust confidence in the risk estimates. Screening-level risk estimates for the Waste handling, treatment, and disposal OES were above benchmark for all scenarios. Therefore, ingestion of fish potentially contaminated with DBP is not a pathway of concern for tribal populations.

8 AMBIENT AIR CONCENTRATION

EPA considers both modeled and monitored concentrations in the ambient air for this draft ambient air exposure assessment for DBP. The Agency's modeling estimates both short- and long-term concentrations in ambient air as well as dry, wet, and total deposition rates. EPA considers monitoring data from published literature for additional insight into ambient air concentrations of DBP.

8.1 Approach for Estimating Concentrations in Ambient Air

EPA uses the Integrated Indoor/Outdoor Air Calculator (IIOAC) Model to estimate daily- and annual-average concentrations of DBP in the ambient air as well as annual average wet, dry, and total deposition rates of DBP from the ambient air. IIOAC is a spreadsheet-based tool that estimates outdoor air concentrations using pre-run results from a suite of dispersion scenarios in a variety of meteorological and land-use settings within EPA's American Meteorological Society/Environmental Protection Agency Regulatory Model (AERMOD). Additional information on IIOAC can be found in the user guide (U.S. EPA, 2019d).

In line with previously peer-reviewed methodology (U.S. EPA, 2022b), EPA's analysis with IIOAC estimates ambient concentrations of DBP at three distances (*e.g.*, 100; 100–1,000, and 1,000 ms) from the releasing facility. EPA considers three different datasets for DBP releases including EPA estimated releases based on production volumes of DBP from facilities that manufacture, process, repackage, or dispose of DBP estimated by EPA methods (U.S. EPA, 2025b), releases reported to TRI by industry (2017 to 2022 reporting years), and releases reported to the NEI (U.S. EPA, 2025b) by industry (2017 and 2020 reporting years). The maximum fugitive release value used in this assessment was reported to the 2017 NEI dataset and is associated with the Application of paints, coatings adhesives, and sealants OES. The maximum stack release value used in this assessment was reported to the TRI dataset and is associated with the Waste handling, treatment, and disposal OES. Both maximum release values represent the maximum release reported across all facilities and COUs and are used as direct inputs to the IIOAC model to estimate concentrations and deposition rates.

8.1.1 Release and Exposure Scenarios Evaluated

The release and exposure scenarios evaluated for this analysis are summarized below.

- Release: Maximum Release (kg/site-day)
- Release Dataset:
  - Fugitive: 2017 NEI
  - Stack: TRI
- Release Type: Stack and Fugitive
- Release Pattern: Consecutive
- Distances Evaluated: 100, 100–1,000, and 1,000 m
- Meteorological Station:
  - South (Coastal): Surface and Upper Air Stations at Lake Charles, Louisiana
- Operating Scenario: 250 days per year; 24 h/day and 8 hours per day to identify the scenario resulting in the maximum ambient air concentration. This is the operating scenario associated with the releases modeled.
- Topography: Urban and Rural
- Particle Size:
  - Coarse (PM_{10}): Particulate matter with an aerodynamic diameter of 10 microns
  - Fine ($PM_{2.5}$): Particulate matter with an aerodynamic diameter of 2.5 microns

EPA used default release input parameters integrated within the IIOAC Model for both stack and fugitive releases along with a user-defined length and width for fugitive releases as listed in

Table 8-1.

Table 8-1. IIOAC Input Parameters for Stack and Fugitive Air Releases

Stack Release Parameters	Value
Stack height (m)	10
Stack diameter (m)	2
Exit velocity (m/sec)	5
Exit temperature (K)	300
Fugitive Release Parameters	**Value**
Length (m)	10
Width (m)	10
Angle (degrees)	0
Release height (m)	3.05

8.1.2 IIOAC Model Output Values

The IIOAC Model provides multiple output values (see *Draft Ambient Air IIOAC Exposure Results and Risk Calculations for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025a)). A description of select outputs relied upon in this draft assessment are provided below. These outputs were relied upon because they represent a more conservative exposure scenario where modeled concentrations are expected to be higher, thus more protective of exposed populations and ensuring potential high-end exposures are not missed during screening for the ambient air pathway.

Fenceline Average: represents the daily-average and annual-average concentrations at 100 m distance from a releasing facility.

High-End, Daily-Average: represents the 95th percentile daily average of all modeled hourly concentrations across the entire distribution of modeled concentrations at 100 m.

High-End, Annual-Average: 95th percentile annual-average concentration across the entire distribution of modeled concentrations at 100 m.

High-End, Annual Average Deposition Rate: 95th percentile annual-average deposition rate across the entire distribution of modeled deposition rates at 100 m.

8.1.3 Modeled Results from IIOAC

All results for each scenario described in Section 8.1.1 are included in the *Draft Ambient Air IIOAC Exposure Results and Risk Calculations for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025a). EPA utilized the highest estimated concentrations and deposition rates across all modeled scenarios to evaluate exposures and deposition rates near a releasing facility. This exposure scenario represents a national level exposure estimate inclusive of sensitive and locally impacted populations who live next to a releasing facility.

The IIOAC model provides source apportioned concentrations and deposition rates (fugitive and stack) based on the respective releases. To evaluate exposures and total deposition rates for this ambient air assessment, EPA assumes the fugitive and stack releases occur simultaneously throughout the day and year. Therefore, the total concentration and deposition rate used to evaluate exposures and derive risk estimates in this ambient air assessment is the sum of the separately modeled fugitive and stack concentrations and total deposition rates at 100 m from a releasing facility. The source apportioned concentrations and the total concentrations for the scenario used are provided in Table 8-2.

Table 8-2. Source Apportioned and Total Daily-Average and Annual-Average IIOAC-Modeled Concentrations at 100 m from Releasing Facility

Source Type	Daily-Average Concentration (µg/m^3)	Annual-Average Concentration (µg/m^3)
Fugitive	16.73	11.46
Stack	0.53	0.37
Total	17.26	11.82

The source apportioned wet and dry deposition rates and the total deposition rates for the scenario used in the *Draft Environmental Hazard Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024c) are provided in Table 8-3.

Table 8-3. Source Apportioned and Total Annual-Average IIOAC-Modeled Wet, Dry, and Total Air to Soil Deposition Rates at 100 m from Releasing Facility

Source Type	Total Annual-Average Air to Soil Deposition Rates (g/m^2)		
	Total	Wet	Dry
Fugitive	1.96E–04	1.94E–04	2.80E–06
Stack	2.75E–05	2.67E–05	1.48E–06
Total	2.23E–04	2.21E–04	4.28E–06

8.2 Measured Concentrations in Ambient Air

EPA identified monitoring studies through systematic review to provide context to modelling results. The monitoring studies presented here were not used as part of the analysis for quantifying exposure estimates. EPA reviewed published literature as described in the *Draft Systematic Review Protocol for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025e) to identify studies where ambient concentrations of DBP were measured. The available data found include data from a Chinese study (Zhu et al., 2016), which measured concentrations of several phthalates including DBP. A simple plot of the measured concentrations is provided in Appendix F.

EPA also identified a single U.S. study through its systematic review process where DBP concentrations were measured at three New York City air sampling stations (Bove et al., 1978). Findings from this study are summarized in Appendix F. Measured concentrations of DBP in these two studies were low, generally in the ng/m^3 range. How these data do or do not reflect conditions in the United States (in relation to the foreign study) or TSCA COUs (in relation to both the foreign study and U.S. study) is unknown, limiting the utility of these data to this assessment.

Uncertainties associated with monitoring data from other countries limit their applicability to this risk assessment. It is unknown how these data do or do not reflect conditions in the United States or TSCA COUs. Information needed to link the monitoring data to foreign industrial processes and crosswalk those to TSCA COUs is not available. The proximity of the monitoring site to a releasing facility associated with a TSCA COU is also unknown. Furthermore, regulation of emissions standards often vary between the United States and foreign countries.

EPA also reviewed EPA's Ambient Monitoring Technology Information Center (AMTIC) database but did not find any monitored DBP concentrations (U.S. EPA, 2022a).

8.3 Evidence Integration

EPA relied on the IIOAC-modeled concentrations and deposition rates to characterize human and ecological exposures for the ambient air exposure assessment. Modeled DBP ambient air concentrations were estimated using the maximum ambient air release, conservative meteorological data, and a distance of 100 m from a releasing facility. The modeled concentrations are higher than measured concentrations (Sections 8.1 and 8.2, respectively). Caution is needed when interpreting such a comparison, however, because modeled concentrations are near a releasing facility (100 m), and it is unknown if the sampling sites are located at a similar distance from a site.

8.3.1 Strengths, Limitations, and Sources of Uncertainty for Modeled Air and Deposition Concentrations

The approach and methodology used in this ambient air exposure assessment replicates previously peer-reviewed approaches and methods, as well as incorporates recommendations provided during peer review of other ambient air exposure assessments.

A strength of the IIOAC modeling includes use of environmental release data from multiple databases across multiple years (including data that are required by law to be reported by industry). These databases undergo repeatable quality assurance and quality control reviews (U.S. EPA, 2025b). These release data are used as direct inputs to EPA's peer-reviewed IIOAC Model to estimate concentrations at several distances from releasing facilities where individuals may reside for many years. The specific maximum release value used for this assessment came from an industry reported release value and was the highest value across multiple datasets considered. For OESs that had no facility-reported release data (*e.g.,* TRI or NEI), DBP releases were estimated and used as a direct input to the IIOAC model. Any limitations and uncertainties of these estimated releases, as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b), are carried over to this ambient air exposure assessment.

The IIOAC Model also has limitations in what inputs can and cannot be changed. Since it is based on pre-run scenarios within AERMOD, default input parameters (*e.g.,* stack characteristics and 2011–2015 meteorological data) are already predefined. Site-specific information like building dimensions, stack heights, elevation, and land use cannot be changed in IIOAC and therefore presents a limitation on the modeled results for DBP. This is in addition to the data gap EPA has on certain parameters like building dimensions, stack heights, and release elevation since such information has not been provided by industry to EPA for consideration which creates additional limitations on using other models to their full potential. Furthermore, IIOAC does not consider the presence or location of residential areas relative to the 100 m distance from releasing facilities, the size of the facility, and the release point within a facility. For larger facilities, 100 m from a release point may still fall within the facility property where individuals within the general population are unlikely to live or frequent. In contrast, for smaller facilities, there may be individuals within the general population living 100 m away from the release

point and therefore could be exposed continuously. However, most individuals may not stay within their residences 24 hours per day, 7 days per week throughout the year.

The use of estimated annual release data to calculate daily average releases can underestimate exposure. Since the maximum annual release value (for stack and fugitive emissions) from each release point is used in this assessment, EPA assumes operations are continuous and releases are the same for each day of operation when calculating daily average concentrations. This assumption may result in modeled concentrations missing true peak releases (and associated exposures). However, EPA utilized multiple conservative assumptions leading to a high ambient air concentrations appropriate for a screening level assessment.

8.4 Weight of Scientific Evidence Conclusions

EPA has moderate confidence in the IIOAC-modeled results used to characterize exposures and deposition rates. Despite the limitations and uncertainties (Section 8.3) potentially under- or overestimating ambient air exposure, this screening level analysis presents a reasonable upper-bound of exposure. Multiple conservative inputs (*e.g.*, maximum estimated ambient air release) and assumptions (*e.g.*, an individual lives at the same location 100 m from a facility for their entire lifetime and spends the entirety of their day every day at that location) bias the resulting exposure estimates toward overestimation. These exposure estimates are thus protective, and ambient air exposure is not a pathway of concern.

9 AMBIENT AIR EXPOSURE TO GENERAL POPULATION

9.1 Exposure Calculations

Modeled ambient air concentration outputs from IIOAC need to be converted to estimates of exposure to derive risk estimates. For this exposure assessment, EPA assumes the general population is continuously exposed (*i.e.*, 24 hours per day, 365 days per year) to outdoor ambient air concentrations. Therefore, daily average modeled ambient air concentrations are equivalent to acute exposure concentrations, and annual average modeled ambient air concentrations are equivalent to chronic exposure concentrations used to derive risk estimates (Section 8.1.3). Calculations for general population exposure to ambient air via inhalation and ingestion from air to soil deposition for lifestages expected to be highly exposed based on exposure factors can be found in *Draft Ambient Air IIOAC Exposure Results and Risk Calculations For Dibutyl Phthalate (DBP)* (U.S. EPA, 2025a).

9.2 Overall Findings

Based on the results from the analysis of the maximum estimated release and high-end exposure concentrations presented in this document and the *Draft Non-cancer Human Health Hazard Risk Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024f), EPA does not expect an inhalation risk from ambient air nor ingestion risk from air to soil deposition to result from exposures to DBP from industrial releases. Because no exposures of concern were identified at the maximum release scenario, EPA does not expect a different finding for smaller releases and therefore additional or more detailed analyses for exposure to DBP through inhalation of ambient air or ingestion from air to soil deposition is not necessary.

10 HUMAN MILK EXPOSURES TO GENERAL POPULATION

Infants are potentially more susceptible for various reasons, including their higher exposure per body weight, immature metabolic systems, and the potential for chemical toxicants to disrupt sensitive developmental processes. Reasonably available information from oral studies of experimental animal models (i.e., rats and mice) also indicates that DBP is a developmental and reproductive toxicant (U.S. EPA, 2024f). EPA considered exposure (Section 10.1) and hazard (Section 10.3) information, as well as pharmacokinetic models (Section 10.2), to determine the most scientifically supportable appropriate approach to evaluate infant exposure to DBP from human milk ingestion. The Agency concluded that the most appropriate approach is to use human health hazard values that are based on fetal and infant effects following maternal exposure during gestational and/or perinatal period. In other words, infant exposure and risk estimates from maternal exposure are expected to also be protective of nursing infants.

10.1 Biomonitoring Information

DBP has the potential to accumulate in human milk because of its small mass (278.34 Daltons or g/mol) and lipophilicity (log K_{OW} = 4.5). EPA identified 13 biomonitoring studies, of which 1 is from the United States, from reasonably available information that investigated if DBP or its metabolites were present in human milk. DBP or its metabolites were detected in human milk samples in each of these studies. A summary of the biomonitoring studies is provided in Figure 10-1. None of the studies characterized if any of the study participants may be occupationally exposed to DBP.

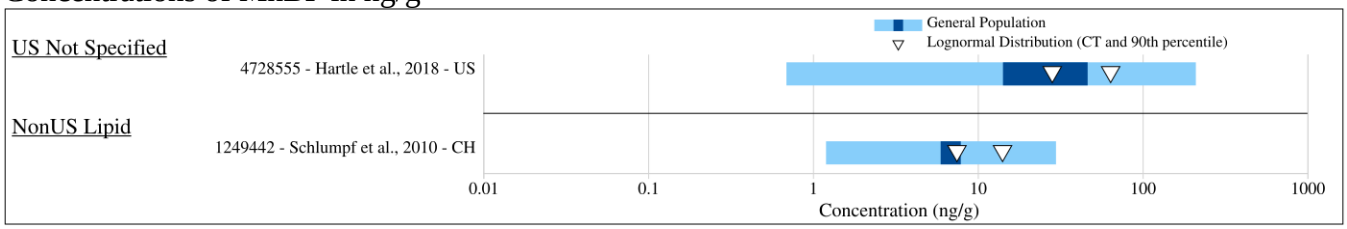
DBP's primary metabolite, mono-n-butyl phthalate (MnBP), was measured in 21 samples collected from the Mother's Milk Bank in California. The concentrations ranged from 0.69 to 210.24 ng/g lipid weight (lw) with a median of 14.2 ng/g (Hartle et al., 2018). The highest lipid weight concentration among eight non-U.S. studies was nearly the same (211.2 ng/g lw) (Brucker-Davis et al., 2008). For wet weight among the non-U.S. studies, the maximum concentration was 10,900 µg/L (median 9.6 µg/L, minimum 0.6 µg/L, n=130) among 130 Finnish and Danish mothers (Main et al., 2006). The authors reported that the interindividual variation for MnBP is extreme and that contamination may have occurred during collection of the human milk samples at home (e.g., from air particles, breast pumps). The other six studies had concentrations that ranged from 0.4 to 32.03 µg/L (Kim et al., 2018; Fromme et al., 2011; Lin et al., 2011; Schlumpf et al., 2010; Latini et al., 2009; Hogberg et al., 2008).

Six non-U.S. studies measured DBP concentrations in human milk. The highest was observed in a cohort of 125 French mothers, (range: 11.8–529.4 ng/g; mean: 81.2 ng/g) (Brucker-Davis et al., 2008). Six other studies measured DBP concentrations that ranged from less than 0.1 to 11 ng/g lw and less than 0.28 to 173.6 ng/mL wet weight (ww) (Kim et al., 2020; Zimmermann et al., 2012; Fromme et al., 2011; Chen et al., 2008; Hogberg et al., 2008; Zhu et al., 2006).

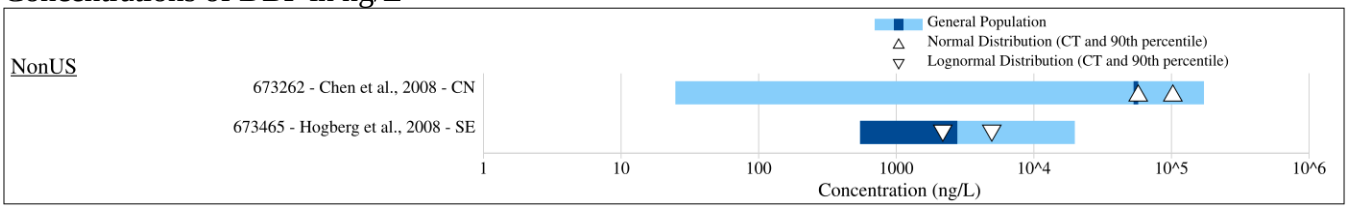
Although biomonitoring studies consistently detect DBP in human milk, concentrations reported in these studies reflect total infant exposure. Biomonitoring data do not distinguish between exposure routes or pathways and do not allow for source apportionment. In other words, the contribution of specific TSCA COUs to overall exposure cannot be determined.

Concentrations of DBP in ng/g

Concentrations of MnBP in ng/g



Concentrations of DBP in ng/L



Concentrations of MnBP in ng/L

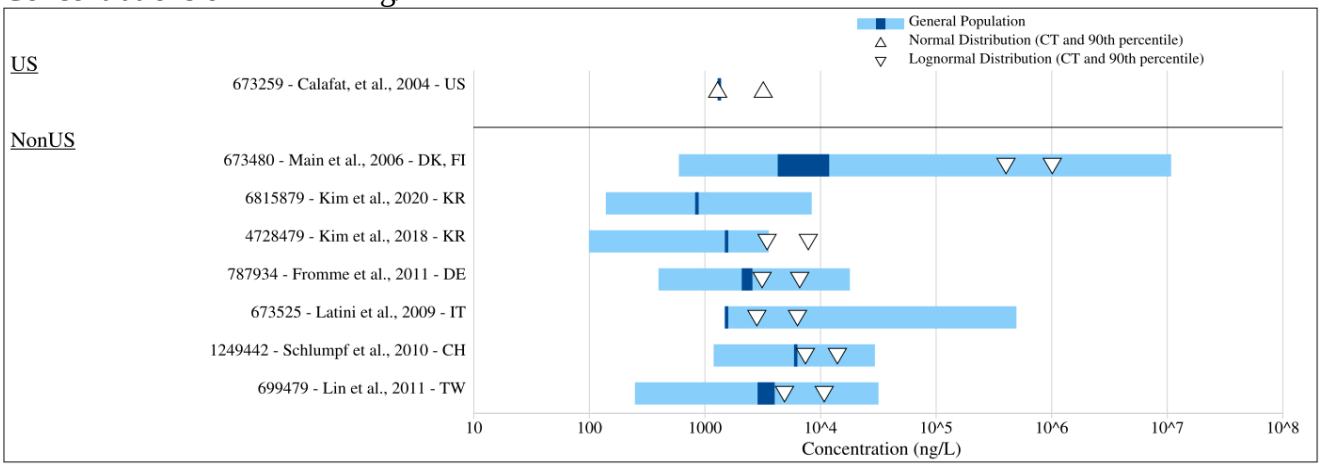


Figure 10-1. Concentrations of DBP or MnBP in Human Milk in Either Lipid (ng/g) or Wet Weight (ng/L)

10.2 Modeling Information

EPA explored the potential to model DBP concentrations in human milk resulting from specific sources of maternal exposures with the aim of providing quantitative estimates of COU-specific milk exposures and risks. The Agency identified a pharmacokinetic model described in Kapraun et al. (2022) as the best available model to estimate transfer of lipophilic chemicals from mothers to infants during gestation and lactation; hereafter referred to as the Kapraun Model. The only chemical-specific parameter required by the Kapraun Model is the elimination half-life in the animal species of interest.

EPA considered the model input data available for DBP and concluded that uncertainties in establishing an appropriate half-life value precludes using the model to quantify lactational transfer and exposure

from TSCA COUs. Measurement of the parent phthalate (*i.e.*, DBP) in organs, tissues, and matrices is prone to error and contamination from sampling materials because of its rapid hydrolysis (Koch and Calafat, 2009). DBP is rapidly hydrolyzed to its primary monoester metabolite, MnBP, which is also a minor metabolite of benzyl butyl phthalate (BBP). This indicates that neither the parent compound nor the primary metabolite is a sensitive biomarker of exposure to DBP. As a result, measured half-life values for DBP and MnBP in plasma that were reported in Chang et al. (2013) and Fennell et al. (2004) were not considered. Furthermore, DBP's short 4-carbon side chain indicates that it is metabolized through only hydrolysis and degradation (Wang et al., 2019). Secondary oxidized metabolites are thus not readily detectable. These uncertainties in establishing an appropriate half-life value for DBP does not support using the model to quantify lactational transfer and exposure for TSCA COUs.

Instead, exposure estimates for workers, consumers, and the general population were compared against the hazard values designed to be protective of infants and expressed in terms of maternal exposure levels during gestation and the perinatal period.

10.3 Hazard Information

EPA considered multigenerational developmental and reproductive toxicity studies of rats that evaluated the effects of oral exposures to DBP. The critical effect is disruption to androgen action during the critical window of male reproductive development (*i.e.*, during gestation), leading to a spectrum of effects on the developing male reproductive system that is consistent with phthalate syndrome. These effects follow gestational or perinatal oral exposures to DBP and are attributable to antiandrogenic effects during gestation (see *Draft Human Health Hazard Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024f)). No studies were identified that evaluated only lactational exposure (*i.e.*, from birth to weaning) from quantified levels of DBP or its metabolites in milk. However, the hazard values are based on developmental and reproductive toxicity in the offspring following maternal exposure during gestation and the perinatal period. Because these values designed to be protective of infants are expressed in terms of maternal exposure and hazard values to assess direct exposures to infants are unavailable, EPA concluded that further characterization of infant exposure through human milk ingestion would not be informative.

10.4 Weight of Scientific Evidence Conclusions

EPA considered infant exposure to DBP through human milk because the available biomonitoring data demonstrate that DBP can be present in human milk and hazard data demonstrate that the developing male reproductive system may be particularly susceptible to the effects of DBP. Although EPA explored the potential to model milk concentrations and concluded that there is insufficient information (*e.g.*, sensitive and specific half-life data) available to support modeling of the milk pathway, the Agency also concluded that modeling is not needed to adequately evaluate risks associated with exposure through milk. This is because the POD used in this draft assessment is based on male reproductive effects resulting from maternal exposures throughout sensitive phases of development in multigenerational studies. EPA therefore has confidence that the risk estimates calculated based on maternal exposures are protective of a nursing infant.

11 URINARY BIOMONITORING

Reverse dosimetry is an approach, as shown in Figure 11-1, of estimating an external exposure or intake dose to a chemical using biomonitoring data (U.S. EPA, 2019b). In the case of phthalates, the U.S. Centers for Disease Control and Prevention's (CDC) National Health and Nutrition Examination Survey (NHANES) dataset provides a relatively recent (data available from 2017–2018) and robust source of urinary biomonitoring data that is considered a national, statistically representative sample of the non-institutionalized, U.S. civilian population. Phthalates have elimination half-lives on the order of several hours and are quickly excreted from the body in urine and to some extent feces (ATSDR, 2022; EC/HC, 2015). Therefore, the presence of phthalate metabolites in NHANES urinary biomonitoring data indicates recent phthalate exposure.

Reverse dosimetry is a powerful tool for estimating exposure, but reverse dosimetry modeling does not distinguish between routes or pathways of exposure and does not allow for source apportionment (*i.e.*, exposure from TSCA COUs cannot be isolated). Instead, reverse dosimetry provides an estimate of the total dose (or aggregate exposure) responsible for the measured biomarker. Therefore, intake doses estimated using reverse dosimetry are not directly comparable to the exposure estimates from the various environmental media presented in this document. However, the total intake dose estimated from reverse dosimetry can help contextualize the exposure estimates from TSCA COUs as being potentially underestimated or overestimated.

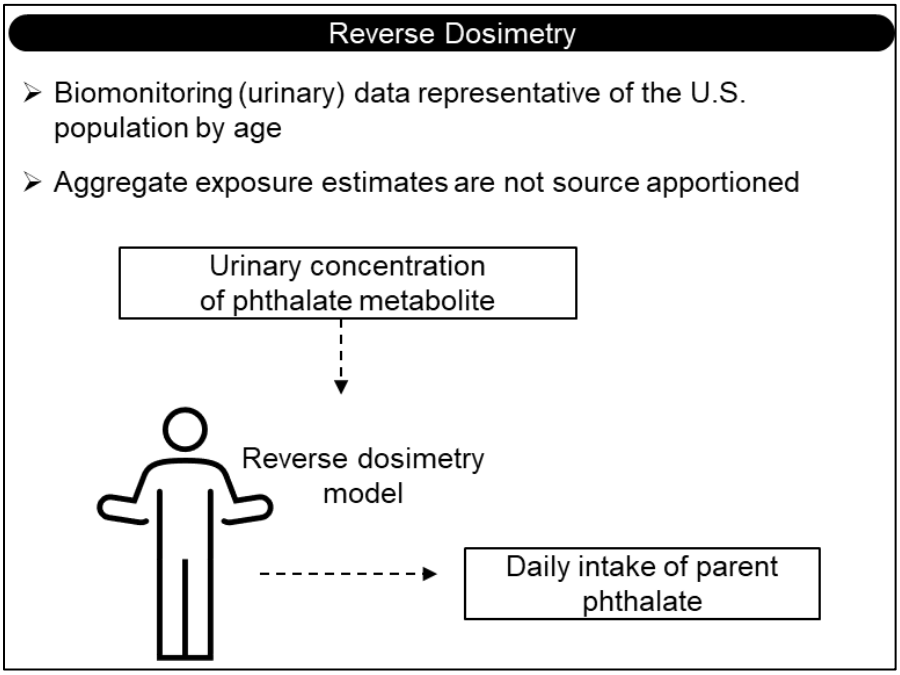


Figure 11-1. Reverse Dosimetry Approach for Estimating Daily Intake

11.1 Approach for Analyzing Biomonitoring Data

EPA analyzed urinary biomonitoring data from NHANES, which reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. Specifically, EPA analyzed data for two metabolites of DBP; mono-3-hydroxybutyl phthalate (MHBP) (measured in the 2013–2018 NHANES cycles) and mono-n-butyl phthalate (MnBP) (measured in the 1999–2018 NHANES cycles). Although MHBP was measured in the 2013 to 2018 NHANES cycles, the data for the 2013 to 2014 NHANES cycle was determined to be inaccurate due to procedural error and was only released as surplus data, which is not readily publicly available. As a result, the present analysis only includes urinary MHBP

data from the 2015 to 2018 NHANES cycles. Sampling details can be found in Appendix G.

Urinary concentrations of DBP metabolites were quantified for different life stages and included women of reproductive age (16–49 years), adults (16+ years), adolescents (11 to <16 years), children (6 to <11 years), and toddlers (3 to <6 years), when data were available. Urinary concentrations of DBP metabolites were analyzed for all available NHANES survey years to examine the temporal trend of DBP exposure. However, intake doses using reverse dosimetry were calculated for the NHANES cycle (2017–2018) as being most representative of current exposures because it was the most recently available data.

NHANES uses a multi-stage, stratified, clustered sampling design that intentionally oversamples certain demographic groups; to account for this, all data was analyzed using the survey weights provided by NHANES and analyzed using weighted procedures in SAS and SUDAAN statistical software. Median and 95th percentile concentrations were calculated in SAS and reported for life stages of interest. Median and 95th percentile concentrations are provided in Table_Apx G-2. Statistical analyses of DBP metabolite trends over time were performed with PROC DESCRIPT using SAS-callable SUDAAN.

11.1.1 Temporal Trend of MnBP

Figure 11-2 through Figure 11-7 show urinary MnBP concentrations plotted over time for the various populations to visualize the temporal exposure trends. All data used for the temporal exposure trends are provided in Table_Apx G-2. Overall, MnBP urinary concentrations have decreased over time for all life stages.

From 1999 to 2018, 50th and 95th percentile urinary MnBP concentrations significantly decreased over time among all children under 16 ($p < 0.001$ for both percentile exposures) (Figure 11-4), as well as for children aged 3 to less than 6 years ($p < 0.001$) (Figure 11-5), 6 to less than 11 years ($p < 0.001$) (Figure 11-6), and 11 to less than 16 years ($p < 0.001$) (Figure 11-7).

From 1999 to 2018, median and 95th percentile urinary MnBP concentrations significantly decreased among all adults ($p < 0.001$ for both percentile exposures), female adults ($p < 0.001$ for 50th and 95th percentile), male adults ($p < 0.001$ for 50th and 95th percentile) (Figure 11-2), and women of reproductive age ($p < 0.001$ for 50th and 95th percentile) (Figure 11-3).

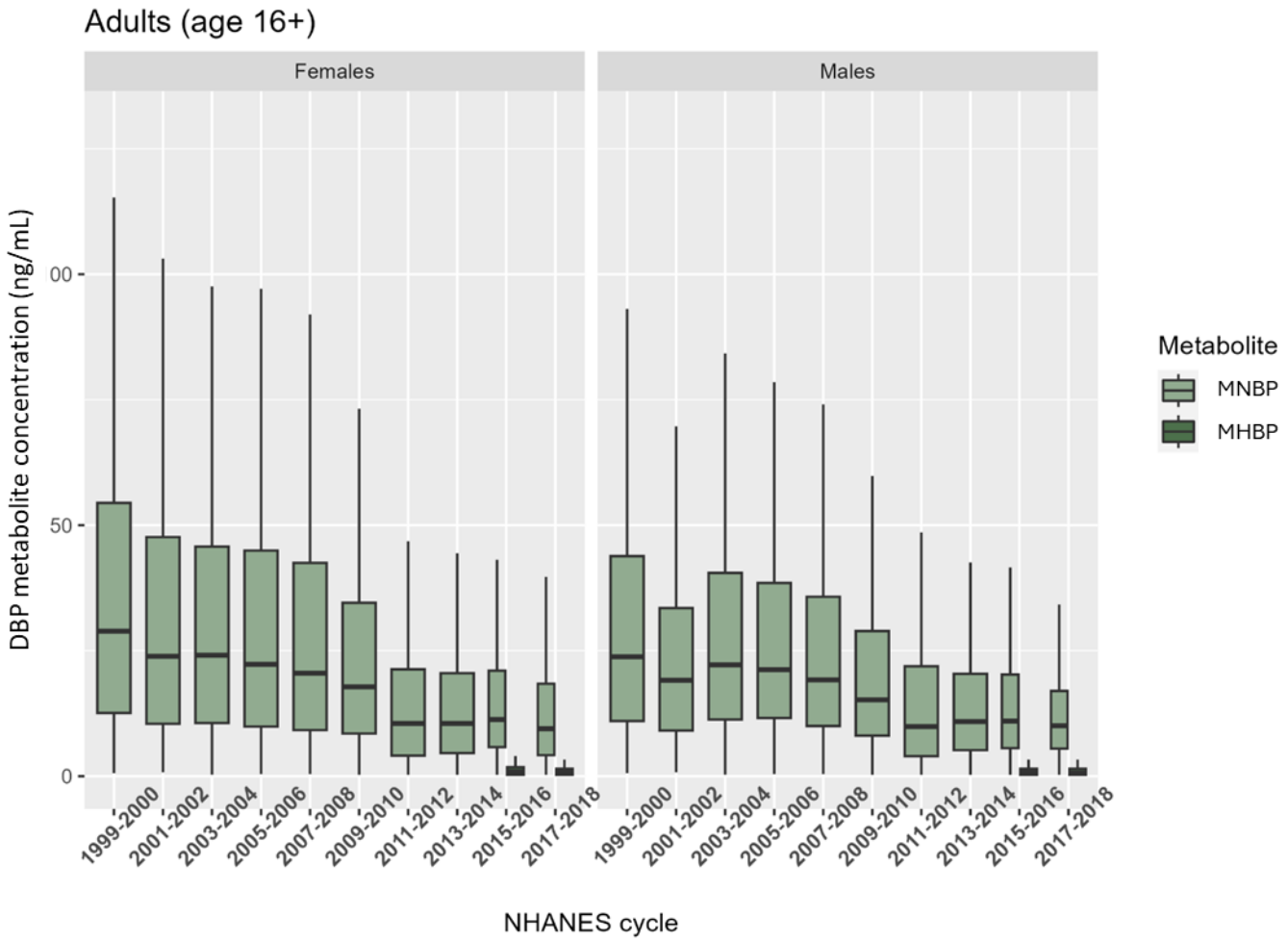


Figure 11-2. Urinary DBP Metabolite Concentrations for Adults (16+ Years)

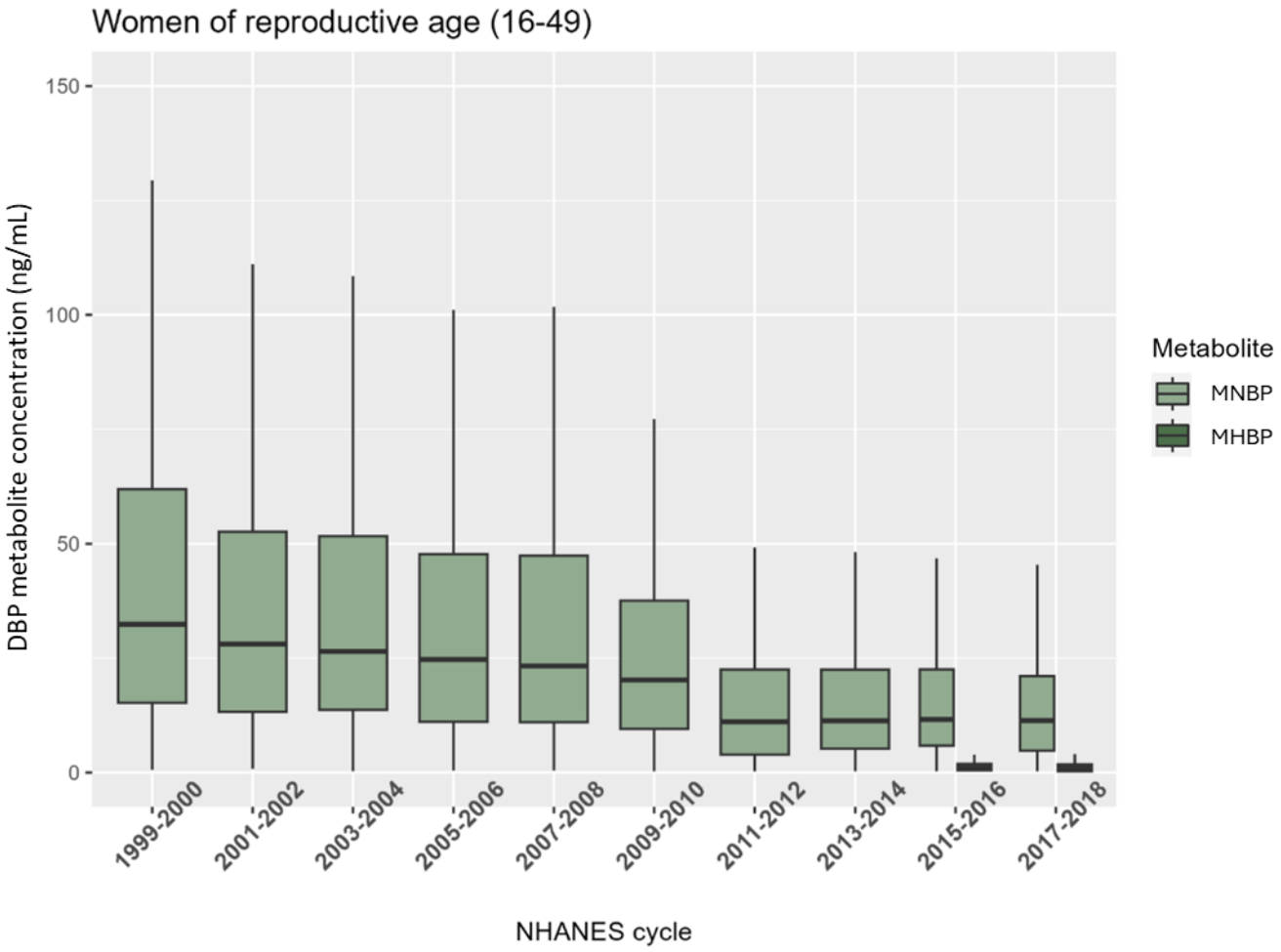


Figure 11-3. Urinary DBP Metabolite Concentrations for Women of Reproductive Age (16–49 Years)

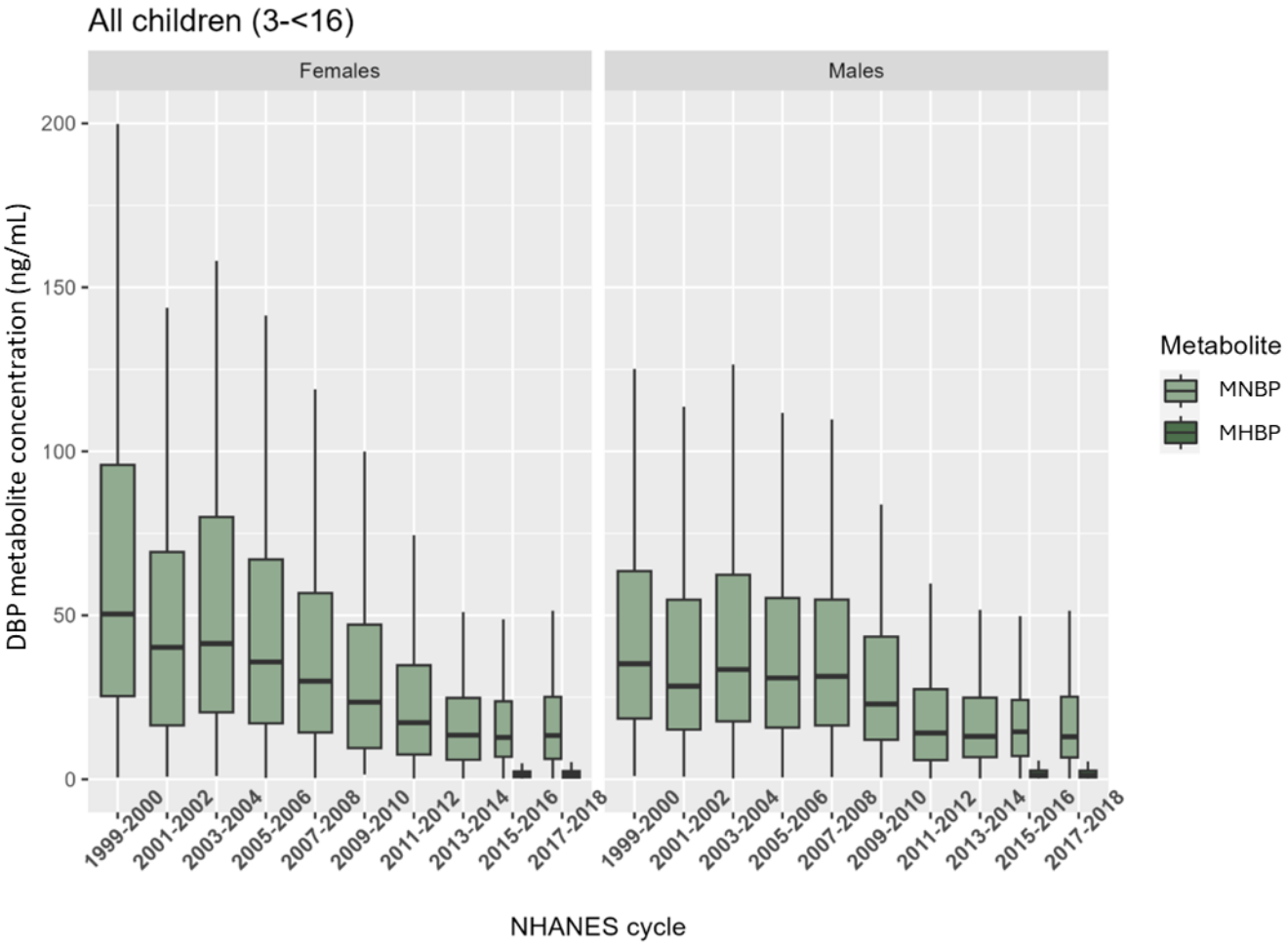


Figure 11-4. Urinary DBP Metabolite Concentrations for All Children (3 to <16 Years) by Sex

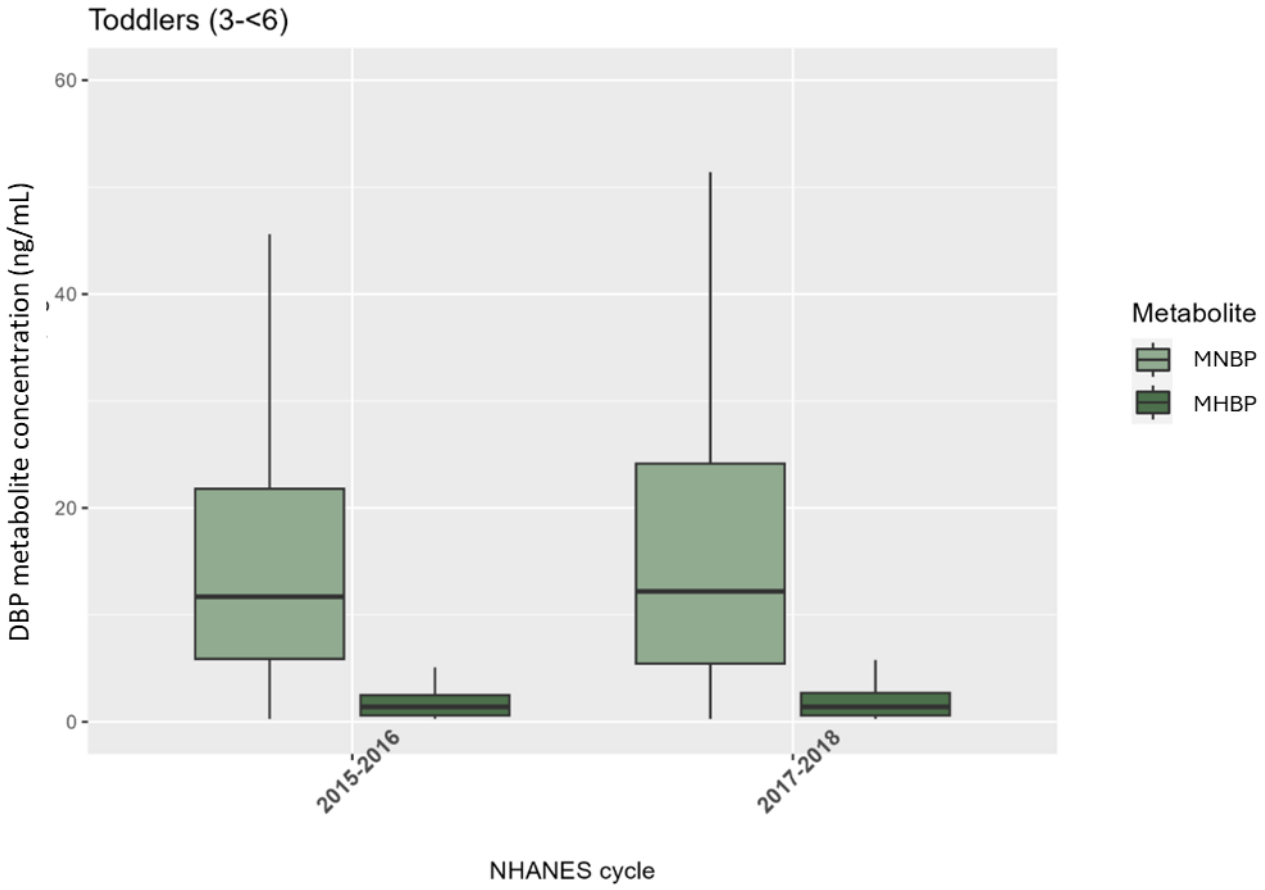


Figure 11-5. Urinary DBP Metabolite Concentrations for Toddlers (3 to <6 Years)

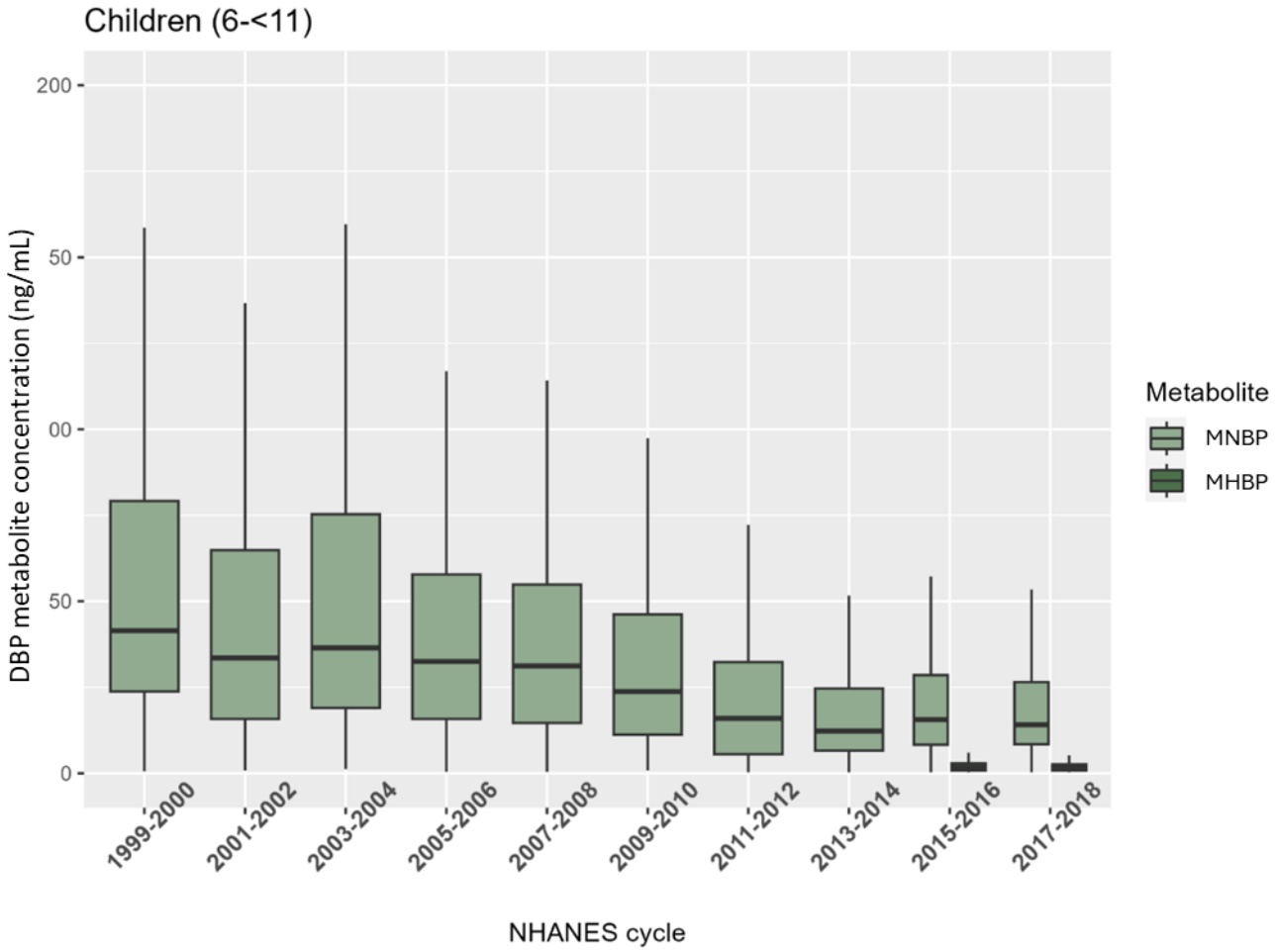


Figure 11-6. Urinary DBP Metabolite Concentrations for Children (6 to <11 Years)

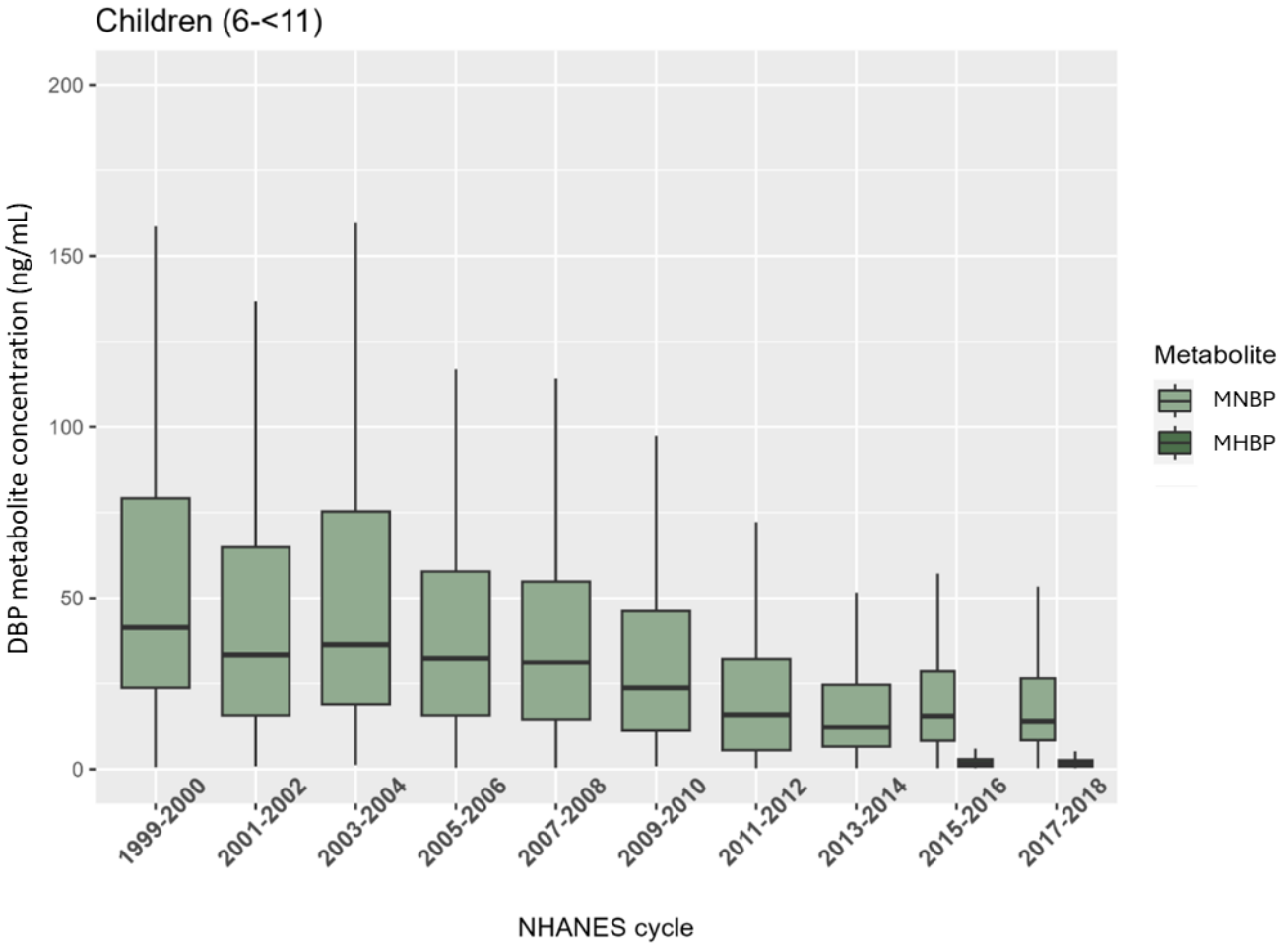


Figure 11-7. Urinary DBP Metabolite Concentrations for Adolescents (11 to <16 Years)

11.1.2 Changes in MHBP Concentrations

As mentioned in Section 11.1, only data from the 2015 to 2018 NHANES cycles were analyzed for MHBP resulting in the two data points shown for MHBP concentrations in Figure 11-2 through Figure 11-7. Therefore, a temporal trend analysis was not conducted for MHBP. However, a comparison of the metabolite concentrations between the 2015 to 2016 and 2017 to 2018 NHANES cycles show that while 95th percentile MHBP concentrations tended to decrease between the two cycles for children and adults, they increased among women of reproductive age. Meanwhile, 50th percentile MHBP concentrations tended to increase between the two cycles among children under 16 years, decrease for adults, and have no significant changes for women of reproductive age.

11.1.3 Daily Intake of DBP from NHANES

Using DBP metabolite concentrations measured in the most recently available sampling cycle (2017–2018), EPA estimated the daily intake of DBP through reverse dosimetry. Reverse dosimetry approaches that incorporate basic pharmacokinetic information are available for phthalates (Koch et al., 2007; Koch et al., 2003; David, 2000) and have been used in previous phthalate risk assessments conducted by U.S. Consumer Product Safety Commission (CPSC) (2014) and Health Canada (Health Canada, 2020) to estimate daily intake values for exposure assessment. For phthalates, reverse dosimetry can be used to estimate a daily intake (DI) value for a parent phthalate diester based on phthalate monoester metabolites measured in human urine using Equation 11-1 (Koch et al., 2007) below. For DBP, the phthalate monoester metabolites are MHBP and MnBP.

Equation 11-1. Calculating the Daily Intake Value from Urinary Biomonitoring Data

$$Phthalate\ DI = \frac{(\mathrm{UE}_{Sum} \times \mathrm{CE})}{\mathrm{Fue}_{sum}} \times MW_{Parent}$$

Where:

$Phthalate\ DI$	=	Daily intake (µg/kg-day) value for the parent phthalate diester
UE_{sum}	=	Sum molar concentration of urinary metabolites associated with the parent phthalate diester (µmol/g)
CE	=	Creatinine excretion rate normalized by body weight (mg/kg-day). CE can be estimated from the urinary creatinine values reported in biomonitoring studies (*i.e.*, NHANES) using the equations of Mage et al. (2008) based on age, gender, height, and race, as was done by Health Canada (Health Canada, 2020) and U.S. CPSC (2014).
Fue_{sum}	=	Summed molar fraction of urinary metabolites. The molar fraction describes the molar ratio between the amount of metabolite excreted in urine and the amount of parent compound taken up. F_{ue} values used for daily intake value calculations are shown in Table 11-1.
MW_{parent}	=	Molecular weight of the parent phthalate diester (g/mol)

Table 11-1. F_{ue} Values Used for the Calculation of Daily Intake Values by DBP

Metabolite	F_{ue} [a]	Reference	Study Population
MnBP	0.69	Anderson et al. (2011)	n = 10 men (20–42 years of age) and 10 women (18–77 years of age)

[a] F_{ue} values are presented on a molar basis and were estimated by study authors based on metabolite excretion over a 24-hour period.

Daily intake values were calculated for each participant from NHANES. A creatinine excretion rate for each participant was calculated using equations provided by Mage et al. (2008). The applied equation is dependent on the participant's age, height, race, and sex to accommodate variances in urinary excretion rates. Creatinine excretion rate equations were only reported for people who are non-Hispanic Black and non-Hispanic White, so the creatinine excretion rate for participants of other races were calculated using the equation for non-Hispanic White adults or children, in accordance with the approach used by U.S. CPSC (2015). Daily intake values for DBP are reported in Table 11-2.

Table 11-2. Daily Intake Values for DBP Based on Urinary Biomonitoring from the 2017–2018 NHANES Cycle

Demographic	50th Percentile Daily Intake Value (Median [95% CI]) (µg/kg-day)	95th Percentile Daily Intake Value (Median [95% CI]) (µg/kg-day)
All	0.33 (0.3–0.36)	1.16 (0.96–1.35)
Females	0.31 (0.27–0.35)	1.02 (0.93–1.11)
Males	0.34 (0.31–0.37)	1.33 (0.93–1.72)
White non-Hispanic	0.33 (0.29–0.38)	0.97 (0.7–1.24)
Black non-Hispanic	0.32 (0.28–0.37)	1.18 (0.84–1.52)
Mexican-American	0.29 (0.24–0.33)	0.91 (0.68–1.13)
Other	0.38 (0.31–0.44)	1.8 (–0.29–3.88)
Above poverty level	0.38 (0.33–0.43)	1.26 (0.91–1.62)
Below poverty level	0.31 (0.27–0.34)	1.04 (0.84–1.24)
Toddlers (3 to <6 years)	0.55 (0.5–0.6)	1.54 (1.07–2)
Children (6 to <11 years)	0.36 (0.31–0.41)	1.37 (0.88–1.86)
Adolescents (12 to <16 years)	0.28 (0.21–0.34)	0.62 (0.37–0.88)
Adults (16+ years)	0.21 (0.17–0.25)	0.61 (0.39–0.84)
Male toddlers (3 to <6 years)	0.56 (0.49–0.63)	2.02 (1.31–2.74)
Male children (6 to <11 years)	0.38 (0.32–0.44)	1.41 (–0.01 to 2.83)
Male adolescents (12 to <16 years)	0.33 (0.26–0.4)	0.62 (–1.03 to 2.27)
Male adults (16+ years)	0.21 (0.15–0.28)	0.59 (0.35–0.83)
Female toddlers (3 to <6 years)	0.51 (0.44–0.57)	1.44 (1.04–1.84)
Female children (6 to <11 years)	0.34 (0.28–0.41)	0.95 (0.62–1.29)
Female adolescents (12 to <16 years)	0.26 (0.17–0.34)	0.61 (0.29–0.94)
Women of reproductive age (16–49 years)	0.21 (0.16–0.26)	0.61[a]
Female adults (16+ years)	0.21 (0.16–0.26)	0.61[a]
All	0.33 (0.3–0.36)	1.16 (0.96–1.35)

[a] 95% confidence intervals (CI) could not be calculated due to small sample size or a standard error of zero

The calculated daily intake values in this analysis are similar to those reported by the U.S. CPSC (2014) and Health Canada (Health Canada, 2020). The daily intake values in the present analysis are calculated with all available NHANES data between 1999 and 2018, while the CPSC report only contains estimates for MnBP calculated with data from the 2005–2006 NHANES cycle and the Health Canada analysis used data from the 2007–2011 cycles of the Canadian Health Measures Survey.

Median and 95th percentile daily intake values in the U.S. CPSC (2014) report were estimated for men and women of reproductive age (15–45 years). U.S. CPSC reports a median daily intake value for adults aged 15 to 45 year as 0.66 µg/kg-day and a 95th percentile daily intake value of 2.6 µg/kg-day.

Health Canada assessment reports median daily intake values for male children and female children aged 6 to 11 as 1.3 µg/kg-day (Health Canada, 2020). Among 12 to 19 year-old males, the median daily intake value was 1.4 µg-kg/day and the 95th percentile was 3.2 µg-kg/day, and among 12 to 19 year-old females, the median daily intake value was 0.71 µg-kg/day and the 95th percentile was 1.8 µg-kg/day The reported median and 95th percentile daily intake values for adults (ages 20–49 years) were 0.58 and 1.8 µg/kg-day for males and 0.55 and 0.6 µg/kg-day for females.

As described previously, reverse dosimetry modeling does not distinguish between routes or pathways of exposure and does not allow for source apportionment (*i.e.*, exposure from TSCA COUs cannot be isolated). Therefore, general population exposure estimates from exposure to ambient air, surface water, and soil are not directly comparable. However, in contrasting the general population exposures estimated for a screening level analysis with the NHANES biomonitoring data, many of the acute dose rates or average daily doses from a single exposure scenario exceed the total daily intake values estimated using NHANES. Taken together with results from U.S. CPSC (2014) stating that DBP exposure comes primarily from personal care products for women and diet and indoor exposures for infants, toddlers, and children, and that the outdoor environment did not contribute to DBP exposures, the exposures to the general population ambient air, surface water, and drinking water quantified in this assessment are likely overestimates, as estimates from individual pathways exceed the total intake values measured even at the 95th percentile of the U.S. population for all ages. This supports the use of exposure values in this assessment for a screening level analysis for the general population.

11.2 Limitations and Uncertainties of Reverse Dosimetry Approach

Controlled human exposure studies have been conducted and provide estimates of the urinary molar excretion factor (*i.e.*, the F_{ue}) to support use of a reverse dosimetry approach. These studies most frequently involve oral administration of an isotope-labelled (*e.g.*, deuterium or carbon-13) phthalate diester to a healthy human volunteer and then urinary excretion of monoester metabolites is monitored over 24 to 48 hours. F_{ue} values estimated from these studies have been used by both U.S. CPSC (2014) and Health Canada (Health Canada, 2020) to estimate phthalate daily intake values using urinary biomonitoring data.

Use of reverse dosimetry and urinary biomonitoring data to estimate daily intake of phthalates is consistent with approaches employed by both U.S. CPSC (2014) and Health Canada (Health Canada, 2020). However, there are challenges and sources of uncertainty associated with the use of reverse dosimetry approaches. The U.S. CPSC considered several sources of uncertainty associated with use of human urinary biomonitoring data to estimate daily intake values and conducted a semi-quantitative evaluation of uncertainties to determine the overall effect on daily intake estimates (see Section 4.1.3 of (CPSC, 2014)). Identified sources of uncertainty include (1) analytical variability in urinary metabolite measurements; (2) human variability in phthalate metabolism and its effect on metabolite conversion factors (*i.e.*, the F_{ue}); (3) temporal variability in urinary phthalate metabolite levels; (4) variability in urinary phthalate metabolite levels due to fasting prior to sample collection; (5) variability due to fast elimination kinetics and spot samples; and (6) creatinine correction models for estimating daily intake values.

In addition to some of the limitations and uncertainties discussed above and outlined by U.S. CPSC (2014), the short half-lives of phthalates can be a challenge when using a reverse dosimetry approach. Phthalates have elimination half-lives on the order of several hours and are quickly excreted from the body in urine and to some extent feces (ATSDR, 2022; EC/HC, 2015). Therefore, spot urine samples, as collected through NHANES and many other biomonitoring studies, are representative of relatively recent exposures. Spot urine samples were used by Health Canada (Health Canada, 2020) and U.S.

CPSC (2014) to estimate daily intake values. However, due to the short half-lives of phthalates, a single spot sample may not be representative of average urinary concentrations that are collected over a longer term or calculated using pooled samples (Shin et al., 2019; Aylward et al., 2016). Multiple spot samples provide a better characterization of exposure, with multiple 24-hour samples potentially leading to better characterization, but are less feasible to collect for large studies (Shin et al., 2019). Due to rapid elimination kinetics, the U.S. CPSC concluded that spot urine samples collected at a short time (2–4 hours) since last exposure may overestimate human exposure, while samples collected at a longer time (<14 hours) since last exposure may underestimate exposure (see Section 4.1.3 of U.S. CPSC (2014) (U.S. CPSC, 2014)for further discussion).

11.3 Weight of Scientific Evidence Conclusions

For the urinary biomonitoring data, despite the uncertainties discussed in Section 11.2, overall, the U.S. CPSC (2014) concluded that factors that might lead to an overestimation of daily intake seem to be well balanced by factors that might lead to an underestimation of daily intake. Therefore, reverse dosimetry approaches "provide a reliable and robust measure of estimating the overall phthalate exposure." Given a similar approach and estimated daily intake values, *EPA has robust confidence in the estimated daily intake values calculated using reverse dosimetry on NHANES biomonitoring data.* Again, reverse dosimetry modeling does not distinguish between routes or pathways of exposure and does not allow for source apportionment (*i.e.*, exposure from TSCA COUs cannot be isolated), but EPA has robust confidence in the use of its total daily intake value calculated using NHANES to contextualize the exposure estimates from TSCA COUs as being overestimated as described in Section 11.1.3.

12 ENVIRONMENTAL BIOMONITORING AND TROPHIC TRANSFER

EPA assessed the environmental concentrations of DBP resulting from industrial and commercial release estimates. Because DBP fate and exposure from groundwater, biosolids, and landfills were not quantified, the Agency performed a qualitative assessment for all exposure scenarios (U.S. EPA, 2024g). The assessments described in this TSD include the potential DBP dietary exposures to aquatic and terrestrial organisms in the environment. EPA described the potential exposures of DBP to aquatic organisms and aquatic-dependent terrestrial species through a qualitative description of the biomonitoring data of studies of DBP in organism body tissue.

12.1 Aquatic Environmental Monitoring

Studies on DBP concentrations in aquatic species within the pool of reasonably available information were coupled with larger investigations on dialkyl phthalate esters. Measured DBP concentrations (wet, dry, or lipid equivalent) stemmed from studies examining phthalate ester levels in aquatic ecosystems. Multiple aquatic species had DBP concentrations quantified and reported, from a total of 17 studies. These DBP concentrations in aquatic organisms were evaluated to contextualize the qualitative evaluation of trophic transfer and were not ultimately used in a quantitative analysis.

Wet Weight Summaries

Measured DBP concentrations stemmed from studies examining phthalate ester levels in aquatic ecosystems. Multiple aquatic species had DBP wet weight (ww) concentrations reported and/or calculated from a total of nine studies. Upon examining the highest geometric mean and/or average DBP wet weight concentration at each trophic level, there is no discernable trend for DBP as it transfers up trophic levels. Because DBP is expected to partition to lipid-containing tissues, only whole body, liver, and brain tissue samples are reported in this TSD. Samples from muscle and soft tissue may provide an underestimate of DBP concentrations.

DBP wet weight concentrations were reported for two primary producers from aquatic ecosystems (Chi, 2009; McConnell, 2007). In Vancouver, British Columbia, Canada, the green algae (*Prasiola meridionalis)* from the urban False Creek Harbor had a geometric mean whole body DBP concentration at 0.02 mg/kg ww (McConnell, 2007). This was lower than the average DBP concentration found in the aquatic plant *Potamogeton crispus* from Northern China's Haihe River in the urban portion of Tianjin that was measured in the plant's above ground tissue at approximately 0.078 mg/kg ww (Chi, 2009).

DBP wet weight concentrations have been reported for 11 species of primary consumers (*e.g.*, crustaceans, mollusks, invertebrates, and herbivorous finfish) (Hu et al., 2016; McConnell, 2007; Giam et al., 1978). The hepatopancreas of the dungeness crab (*Cancer magister*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a geometric mean DBP concentration at 0.015 mg/kg ww (McConnell, 2007). For five mollusk species, geometric mean DBP concentrations ranged from 0.0023 to 0.034 mg/kg ww in the whole bodies of the softshell clam (*Mya arenaria*) and the blue mussel (*Mytilus edulis*), which were both measured from the urban False Creek Harbor in Vancouver, British Columbia, Canada, respectively (McConnell, 2007). The great blue spotted mudskipper (*Boleophthalmus pectinirostris*), an herbivorous finfish, from the coastal city Ningbo in the Yangtze River Delta in China had an average DBP concentration at approximately 0.022 mg/kg ww in homogenized organs (Hu et al., 2016). Thus, geometric mean/average DBP concentrations ranged from 0.0023 to 0.034 mg/kg ww for primary consumers (McConnell, 2007).

Omnivorous finfish are secondary and tertiary consumers that had DBP wet weight concentrations

reported and/or calculated for 11 species (Lucas and Polidoro, 2019; Hu et al., 2016; Jarosová et al., 2012; McConnell, 2007; Camanzo et al., 1987; De Vault, 1985; Giam et al., 1978; U.S. EPA, 1974). Homogenized organs of the flathead grey mullet (*Mugil cephalus*) from the coastal city Ningbo in the Yangtze River Delta had the lowest average DBP concentration at approximately 0.0064 mg/kg ww (Hu et al., 2016). Carp from tributaries/harbors of five Wisconsin and one Ohio river had the highest geometric mean, whole body DBP concentration at 8.36 mg/kg ww (De Vault, 1985). These samples were collected as part of a contaminant monitoring program in the Great Lakes region and were collected from areas with histories of known chemical contamination.

Similar to omnivorous finfish, piscivorous finfish are secondary and tertiary consumers. DBP wet weight concentrations were reported for 40 piscivorous species (Lucas and Polidoro, 2019; Hu et al., 2016; McConnell, 2007; Peijnenburg and Struijs, 2006; Camanzo et al., 1987; De Vault, 1985; Giam et al., 1978; U.S. EPA, 1974). The herring (*Clupea pallasii*) from the coastal city Wenling in the Yangtze River Delta had the lowest average DBP concentration in homogenized organs at approximately 0.0024 mg/kg ww (Hu et al., 2016). The striped bonito (*Sarda orientalis*) from the coastal city Wenling in the Yangtze River Delta had the highest average DBP concentration in homogenized organs at approximately 0.079 mg/kg ww (Hu et al., 2016). Additionally, bream and roach finfish, a piscivore and an omnivore, from a mix of contaminated and non-contaminated sites throughout the Netherlands were homogenized and had a geometric mean DBP concentration at 0.001 mg/kg ww (Peijnenburg and Struijs, 2006).

Dry Weight Summaries

Multiple aquatic species had DBP dry weight concentrations reported from a total of six studies. Upon examining the highest geometric mean and/or average DBP dry weight concentration at each trophic level, there is no discernable trend for DBP as it transfers up trophic levels due to only two levels being available for comparison. Because DBP is expected to partition to lipid-containing tissues, only whole body, liver, and brain tissue samples are reported here. Samples from muscle and soft tissue can provide an underestimate of DBP concentrations.

DBP dry weight concentrations were reported for two primary producers from aquatic ecosystems (Saliu et al., 2019; Chi, 2009). The aquatic plant *Potamogeton crispus* from Northern China's Haihe River in the urban portion of Tianjin had the highest average DBP concentration in its roots at 1.28 mg/kg dw (Chi, 2009). Whole-body plankton had the lowest mean DBP concentrations outside the Faafu Atoll, islands included in the Republic of Maldives, at 0.0069 mg/kg dw (Saliu et al., 2019).

Omnivorous finfish are secondary and tertiary consumers that had DBP dry weight concentrations reported for six species (Valton et al., 2014; Adeniyi et al., 2011; Huang et al., 2008). In the mouth of Nigeria's Ogun River, which flows through agriculture, urbanized, and industrial areas, the highest mean DBP concentration was measured in the whole body of *Synodontis* sp. at approximately 1.72 mg/kg dw (Adeniyi et al., 2011). The lowest mean DBP concentration was also measured in the mouth of Nigeria's Ogun River in the whole body of *Tilapia* sp. at approximately 0.69 mg/kg dw (Adeniyi et al., 2011).

Lipid Equivalent Summaries

Measured DBP concentrations stemmed from studies examining phthalate ester levels in aquatic ecosystems. Multiple aquatic species had DBP equivalent lipid concentrations reported and/or calculated from a total of four studies. If a study provided lipid content and reported concentrations in wet weights, equivalent lipid concentrations were calculated by dividing a species' wet weight concentration by its lipid content. Upon examining the highest geometric mean and/or average DBP equivalent lipid

concentration at each trophic level, DBP generally decreases in concentration as it transfers up trophic levels.

DBP equivalent lipid concentrations were reported for only one primary producer from aquatic ecosystems (McConnell, 2007). In Vancouver, British Columbia, Canada, the green algae (*Prasiola meridionalis*) from the urban False Creek Harbor had a geometric mean whole body DBP concentration at 4.9 mg/kg equivalent lipid (McConnell, 2007).

DBP concentrations were reported for three species of primary consumers (*e.g.*, crustaceans and mollusks) (McConnell, 2007). The dungeness crab (*Cancer magister*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a higher geometric mean DBP concentration in its muscle than its hepatopancreas at 0.56 and 0.25 mg/kg equivalent lipid, respectively (McConnell, 2007). For two mollusk species, geometric mean DBP concentrations ranged from 0.65 to 0.71 mg/kg equivalent lipid in the whole bodies of softshell clam (*Mya arenaria*) and blue mussel (*Mytilus edulis*), which were both from the urban False Creek Harbor in Vancouver, British Columbia, Canada (McConnell, 2007). As a collective, primary consumers had geometric mean DBP concentrations ranging from 0.25 to 0.71 mg/kg equivalent lipid (McConnell, 2007).

Omnivorous finfish are secondary and tertiary consumers that had DBP equivalent lipid concentrations reported and/or calculated for nine species (McConnell, 2007; Camanzo et al., 1987; De Vault, 1985). Carp from tributaries/harbors of five Wisconsin and one Ohio river had the highest geometric mean, whole body DBP concentration at approximately 22.56 mg/kg equivalent lipid (De Vault, 1985). The shiner perch (*Cymatogaster aggregata*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada, had the lowest geometric mean DBP concentration in its whole body at 0.73 mg/kg equivalent lipid (McConnell, 2007).

Similar to omnivorous finfish, piscivorous finfish are secondary and tertiary consumers. DBP equivalent lipid concentrations were reported for 13 piscivorous species (McConnell, 2007; Peijnenburg and Struijs, 2006; Camanzo et al., 1987; De Vault, 1985). The white-spotted greenling (*Hexogrammos stelleri*) had the lowest geometric mean DBP concentration in its muscle at 0.12 mg/kg equivalent lipid while the spiny dogfish (*Squalus acanthias*) had the highest geometric mean DBP concentration in its muscle at 0.3 mg/kg lipid equivalent, which were both from the urban False Creek Harbor in Vancouver, British Columbia, Canada (McConnell, 2007). Additionally, bream and roach finfish, a piscivore and an omnivore, from a mix of contaminated and non-contaminated sites throughout the Netherlands were homogenized and had a geometric mean DBP concentration at 0.2 mg/kg equivalent lipid based on a median lipid content of 0.5 percent (Peijnenburg and Struijs, 2006). It should be noted that the heads and tails of bream and roach finfish were removed before homogenization.

Unknown Unit Summaries

Measured DBP concentrations stemmed from studies examining phthalate ester levels in aquatic ecosystems. Two studies had DBP concentrations reported and/or calculated for multiple aquatic species, but did not specify their units as either wet, dry, or lipid equivalent concentrations. Upon examining the highest geometric mean/average DBP concentration at each trophic level, there is no discernable trend for DBP as it transfers up trophic levels due to only two levels being available for comparison.

Omnivorous finfish are secondary and tertiary consumers that had DBP concentrations reported and/or calculated for three species (Adeogun et al., 2015). The redbelly tilapia (*Tilapia zillii*) from the manmade Lake Eleyele in Ibadan, Nigeria, had the highest geometric mean DBP concentration in its

muscle, gill, liver, and kidney at approximately 0.35 mg/kg (Adeogun et al., 2015). Meanwhile, the *Morymyrus rume* from the manmade Lake Asejire in Ibadan, Nigeria, had the lowest geometric mean DBP concentration in its muscle, gill, liver, and kidney at approximately 0.19 mg/kg (Adeogun et al., 2015).

Similar to omnivorous finfish, piscivorous finfish are secondary and tertiary consumers that had DBP concentrations reported and/or calculated for two piscivorous species (Adeogun et al., 2015). Geometric mean DBP concentrations ranged from approximately 0.23 to 0.26 mg/kg in the muscle, gill, liver, and kidney of the obscure snakehead (*Parachanna obscura*) and the African pike characin *(Hepsetus odoe*), which were both from the manmade Lake Eleyele in Ibadan, Nigeria (Adeogun et al., 2015).

12.2 Trophic Transfer

EPA did not conduct a quantitative analysis of DBP trophic transfer. Due to its physical and chemical properties, environmental fate, and exposure parameters, DBP is not expected to persist in surface water, groundwater, or air. DBP has a water solubility of 11.2 mg/L, a log K_{OC} value of 3.69, an estimated BCF value of 159.4 L/kg, monitored fish BAF values between 110 and 1,247 L/kg, monitored aquatic invertebrate BAF values between 500 and 6,600 L/kg, and a terrestrial biota-sediment accumulation factor (BSAF) between 0.35 and 11.8 kg/kg. DBP is expected to have low bioaccumulation potential, no apparent biomagnification potential, and thus low potential for uptake overall. For further information on the sources of these values, please see the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g). A study in 18 marine species found that the food-web magnification factor for DBP is 0.70, indicating biodilution as trophic level increases (Mackintosh et al., 2004). DBP is (1) expected to degrade rapidly via direct and indirect photolysis; (2) have environmental biodegradation half-life in aerobic environments on the order of days to weeks; (3) is not subject to long range transport; (4) transforms in the environment via biotic and abiotic processes to form monobutyl phthalate, butanol, and phthalic acid; (5) shows strong affinity and sorption potential for organic carbon in soil and sediment; and (6) will be removed at rates between 65 and 98 percent in conventional wastewater treatment systems. DBP may persist in sediment, soil, biosolids, or landfills after release to these environments, but bioavailability is expected to be limited. The estimated BCF suggests DBP does not meet the criteria to be considered bioaccumulative (estimated BCF/BAF > 1,000 L/kg) and bioaccumulation and bioconcentration in aquatic and terrestrial organisms are not expected (U.S. EPA, 2012). Despite monitored BCF values exceeding 1,000 L/kg in the common carp (*Cyprinus carpio*), a bottom-feeding omnivorous fish, from a study in Asan Lake, South Korea in Lee et al. (2019) (although these samples were desiccated before analysis, suggesting that they overestimate body burden in the live fish, and Asan Lake is one of the largest artificial lakes in Korea and is mainly used for agricultural and industrial purposes, meaning it is likely affected by pollution coming from an industrial complex and two nearby cities), and) as well as in several aquatic invertebrates (Mayer Jr et al., 1973), the available evidence from body burdens in higher trophic level piscivorous fish and the food-web magnification factor study conducted by Mackintosh et al. (2004) provide evidence that trophic transfer of DBP is not a likely source of significant DBP exposure. This conclusion is consistent with the observations made for other phthalates with measured BCF/BAFs such as di-isodecyl phthalate (DIDP) (U.S. EPA, 2024h), di-isononyl phthalate (DINP) (U.S. EPA, 2024i), dicyclohexyl phthalate (DCHP) (U.S. EPA, 2024d), and di-ethylhexyl phthalate (DEHP) (U.S. EPA, 2024e).

12.3 Weight of Scientific Evidence Conclusions

Based on the reasonably available data, EPA has robust confidence that that DBP is found in relatively low concentrations (or not at all) in aquatic organism tissues—especially at higher trophic levels. Furthermore, DBP has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms, and thus low potential for trophic transfer through food webs. EPA therefore does not expect

risk from trophic transfer in wildlife at environmentally relevant concentrations of DBP and has proceeded with a qualitative assessment of trophic transfer in the environmental risk characterization (see Section 5.3 of the *Draft Risk Evaluation for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025d).

13 CONCLUSION OF ENVIRONMENTAL MEDIA CONCENTRATION, GENERAL POPULATION EXPOSURE, AND RISK SCREEN

13.1 Environmental Exposure Conclusions

DBP is expected to be released to the environment via air, water, and biosolids to landfills as detailed within the environmental release assessment presented in the *Draft Environmental Release and Occupational Exposure Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2025b). Environmental media concentrations were quantified in ambient air, soil from ambient air deposition, biosolids, surface water, and sediment. Further details on the environmental partitioning and media assessment can be found in the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g).

For the land pathway, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered. However, based on high-quality physical and chemical property data, EPA determined that DBP has low persistence potential and mobility in soils. Therefore, groundwater concentrations resulting from releases to the landfill or to agricultural lands via biosolids applications were not quantified but are discussed qualitatively. Modeled soil DBP concentrations from air deposition to soil (Section 8) and modeled DBP concentrations in biosolids-amended soils from OESs (Table 3-2) with the resulting highest concentrations to soil are assessed quantitatively with hazard thresholds (U.S. EPA, 2024c) for relevant soil-dwelling organisms and plants within the DBP environmental risk characterization section (U.S. EPA, 2025d).

For the water pathway, relevant flow data from the associated receiving waterbody were collected for facilities reporting to TRI. Quantified release estimates to surface water were evaluated with PSC modeling. For each COU with surface water releases, the highest estimated release to surface water was modeled. Releases were evaluated for resulting environmental media concentrations at the point of release (*i.e.*, in the immediate receiving waterbody receiving the effluent). Due to uncertainty about the prevalence of wastewater treatment from DBP-releasing facilities, all releases are assumed initially to be released to surface water without treatment. The resulting surface water, pore water, and benthic sediment concentrations are presented within Table 4-3 and will be utilized within the environmental risk characterization for DBP for quantitative risk characterization.

Based on the conclusions on the physical and chemical and fate properties of DBP presented in the *Draft Physical Chemistry, Fate, and Transport Assessment for Dibutyl Phthalate (DBP)* (U.S. EPA, 2024g), EPA conducted a qualitative assessment trophic transfer in biota. Multiple aquatic species had DBP concentrations quantified and reported from a total of 17 studies. Because DBP does not biomagnify and is characterized as demonstrating trophic dilution, EPA did not conduct a quantitative modeling analysis of the trophic transfer of DBP through food webs. The Agency has robust confidence that DBP has limited bioaccumulation and bioconcentration potential based on physical chemical and fate properties, biotransformation, and empirical bioaccumulation metrics0. Additionally, due to the physical chemical properties, environmental fate, and exposure parameters of DBP, it is not expected to persist in surface water, groundwater, or air.

13.2 Weight of Scientific Evidence Conclusions for Environmental Exposure

The weight of scientific evidence supporting the exposure estimate is decided based on the strengths, limitations, and uncertainties associated with the exposure estimates, which are discussed in detail for

biosolids (Section 3.1.1), landfills (Section 3.2.1), surface water (Section 4.4), ambient air (Section 8.3.1), and environmental biomonitoring and trophic transfer (Section 12.3). EPA summarized its weight of scientific evidence using confidence descriptors: robust, moderate, slight, or indeterminate confidence descriptors. The Agency used general considerations (*i.e.*, relevance, data quality, representativeness, consistency, variability, uncertainties) as well as chemical-specific considerations for its weight of scientific evidence conclusions.

For its quantitative assessment, EPA modeled exposure due to various exposure scenarios resulting from different pathways of exposure. Exposure estimates used high-end inputs for the purpose of a screening level analysis as demonstrated within the land pathway for modeled concentrations of DBP in biosolids-amended soils at relevant COUs and air to soil deposition of DBP (Section 3.1). Within the water pathway, the release resulting in the highest environmental concentrations are presented within Section 4.1. When available, monitoring data were compared to modeled estimates to evaluate overlap, magnitude, and trends. Differences in magnitude between modeled and measured concentrations (Section 4.2) may be due to measured concentrations not being geographically or temporally close to known releasers of DBP. The high-end modeled concentrations in the surface water for TRI-reported releases and the modeled concentrations for generic release scenarios using a P75 or P90 flow (these flow rates are considered more likely than the P50 to receive high-end industrial and commercial releases) are the same order of magnitude as the high-end monitored concentrations found in surface water. This confirms EPA's expectation that a screening approach with high-end modeled releases is appropriate. The Agency has robust confidence that DBP has limited bioaccumulation and bioconcentration potential based on physical chemical and fate properties, biotransformation, and empirical metrics of bioaccumulation metrics.

13.3 General Population Screening Conclusions

The general population can be exposed to DBP from various exposure pathways. As shown in Table 2-1, exposures to the general population via surface water, drinking water, fish ingestion, and ambient air were quantified using a conservative, high-end scenario screening approach while exposures via the land pathway (*i.e.*, biosolids and landfills) were qualitatively assessed. Based on the high-end estimates of environmental media concentrations summarized in Table 13-1, general population exposures were estimated for the lifestage that would be most exposed based on intake rate and body weight.

The maximum fugitive release value used in this assessment was reported to the 2017 NEI dataset and is associated with the Application of paints, coatings, adhesives and sealants (from institutional furniture manufacturing) OES. The maximum stack release value used in this assessment was reported to the TRI dataset and is associated with the Waste handling, treatment, and disposal (from paint and coating manufacturing) OES.

Table 13-1. Summary of High-End DBP Concentrations in Various Environmental Media from Environmental Releases

OES[a]	Release Media	Environmental Media	DBP Concentration
Manufacturing (P50)	Water	Surface water (30Q5 flow)	616 µg/L
		Surface water (harmonic mean flow)	885 µg/L
Waste handling, treatment, disposal	Water	Surface water (30Q5 flow)	14.5 µg/L
		Surface water (harmonic mean)	14.5 µg/L
Highest monitored surface water (NWQMC, 2021)	Water	Surface water (30Q5 flow)	26.8 µg/L
		Surface water (harmonic mean)	26.8 µg/L
Waste handling, treatment, disposal (Stack)	Ambient air	Daily-averaged total (fugitive and stack, 100m)	17.26 µg/m^3
Application of paints, coatings, adhesives, and sealants (Fugitive)		Annual-averaged total (fugitive and stack, 100m)	11.82 µg/m^3
[a] Table 1-1 provides the crosswalk of OESs to COUs			

Table 13-2 summarizes the conclusions for the exposure pathways and lifestages that were assessed for the general population. EPA conducted a quantitative evaluation for the following: incidental dermal and incidental ingestion from swimming in surface water, drinking water ingestion, fish ingestion, and ambient air. Biosolids and landfills were assessed qualitatively in Sections 3.1 and 3.2, respectively. Results indicate that no pathways were of concern for DBP for the highest exposed populations except for one—fish ingestion for Tribal populations. Because screening risk estimates resulted in risk values below the benchmark for fish ingestion for tribal populations using water solubility as the water concentration, EPA refined its evaluation by using the three OESs that resulted in the highest modeled surface water concentrations based on releases to water combined with the flow rate of the receiving water body (Section 4.1). This refined analysis resulted in screening level risk estimates below the benchmark for the PVC plastic compounding OES based on current 95th percentile ingestion rate and heritage ingestion rate (see Section 7.3). Therefore, ingestion of fish potentially contaminated with DBP can be a pathway of concern for tribal populations.

Table 13-2. Risk Screen for High-End Exposure Scenarios for Highest Exposed Populations

OES(s)	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Pathway of Concern[b]
All	Biosolids (Section 3.1)	No specific exposure scenarios were assessed for qualitative assessments			No
All	Landfills (Section 3.2)	No specific exposure scenarios were assessed for qualitative assessments			No
Manufacturing	Surface water	Dermal	Dermal exposure to DBP in surface water during swimming (Section 5.1.1)	All	No
		Oral	Incidental ingestion of DBP in surface water during swimming (Section 5.1.2)	All	No
Manufacturing Waste handling, treatment, disposal	Drinking water	Oral	Ingestion of drinking water (Section 6.1.1)	All	No
Manufacturing Waste handling, treatment, disposal	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	Adults and young toddlers (1–2 years)	No
			Ingestion of fish for subsistence fishers (Section 7.2)	Adults (16 to <70 years)	No
			Ingestion of fish for tribal populations (Section 7.3)	Adults (16 to <70 years)	No
Waste handling, treatment, disposal (stack)	Ambient air	Inhalation	Inhalation of DBP in ambient air resulting from industrial releases (Section 9)	All	No
Application of paints, coatings, adhesives, and sealants (fugitive)		Oral	Ingestion of soil from air to soil deposition resulting from industrial releases (Section 9)	Infants and children (6 month to 12 years)	No

[a] Table 1-1 provides a crosswalk of COUs to OES
[b] Using the MOE approach as a risk screening tool, an exposure pathway was determined to not be a pathway of concern if the MOE was equal to or exceeded the benchmark MOE of 30.

13.4 Weight of Scientific Evidence Conclusions for General Population Exposure

The weight of scientific evidence supporting the exposure estimate is decided based on the strengths, limitations, and uncertainties associated with the exposure estimates, which are discussed in detail for biosolids (Section 3.1.1), landfills (Section 3.2.1), surface water (Section 4.3.1 and 4.4), drinking water (Section 6.4), fish ingestion (Section 7.4.1), ambient air (Sections 8.3.1 and 8.4), human milk (Section 10.4), and urinary biomonitoring (Section 11.2 and 11.3).

EPA summarized its weight of scientific evidence using confidence descriptors: robust, moderate, slight, or indeterminate confidence descriptors. The Agency used general considerations (*i.e.*, relevance, data quality, representativeness, consistency, variability, uncertainties) as well as chemical-specific considerations for its weight of scientific evidence conclusions.

EPA determined robust confidence in its qualitative assessment and conclusions pertaining to exposures from biosolids (Section 3.1.1) and landfills (Section 3.2.1). For its quantitative assessment, the Agency modeled exposure due to various exposure scenarios resulting from different pathways of exposure. Exposure estimates used high-end inputs for the purpose of a screening level analysis. When available, monitoring data were compared to modeled estimates to evaluate overlap, magnitude, and trends to inform confidence in the quantitative exposure assessment of surface water (Sections 4 and 5), drinking water (Section 6), fish ingestion (Section 7), ambient air (Sections 8 and 9), and human milk (Section 10). EPA has robust confidence that the screening level analysis was appropriately conservative to determine that no environmental pathway has the potential for non-cancer risks to the general population. Despite slight to moderate confidence in the estimated absolute values themselves, confidence in exposure estimates capturing high-end exposure scenarios was robust given the many conservative assumptions. Additionally, EPA conducted reverse dosimetry to calculate daily intake values for DBP using biomonitoring data from NHANES. Notably, many of the acute dose rates or average daily doses from a single exposure scenario exceed the total daily intake values estimated even at the 95th percentile of the U.S. population for all ages using NHANES. Furthermore, risk estimates for high-end exposure scenarios were still consistently above the benchmarks adding to confidence that non-cancer risks are not expected.

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APPENDICES

Appendix A EXPOSURE FACTORS

Table_Apx A-1. Body Weight by Age Group

Age Group[a]	Mean Body Weight (kg)[b]
Infant (<1 year)	7.83
Young toddler (1 to <2 years)	11.4
Toddler (2 to <3 years)	13.8
Small child (3 to <6 years)	18.6
Child (6 to <11 years)	31.8
Teen (11 to <16 years)	56.8
Adults (16+ years)	80.0

[a] Age group weighted average
[b] See Table 8-1 of U.S. EPA (2011a)

Table_Apx A-2. Fish Ingestion Rates by Age Group

Age Group	Fish Ingestion Rate (g/kg-day)[a]	
	50th Percentile	90th Percentile
Infant (<1 year) [b]	N/A	N/A
Young toddler (1 to <2 years) [b]	0.053	0.412
Toddler (2 to <3 years) [b]	0.043	0.341
Small child (3 to <6 years) [b]	0.038	0.312
Child (6 to <11 years) [b]	0.035	0.242
Teen (11 to <16 years) [b]	0.019	0.146
Adult (16+ years) [c]	0.063	0.277
Subsistence fisher (adult) [d]	1.78	

[a] Age group weighted average, using body weight from Table_Apx A-1
[b] See Table 20a of U.S. EPA (2014)
[c] See Table 9a of U.S. EPA (2014)
[d] U.S. EPA (2000b)

2772 **Table_Apx A-3. Recommended Default Values for Common Exposure Factors**

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source/Notes
		Occupational	Residential	
ED	Exposure duration (hours/day)	8	24	
EF	Exposure frequency (days/year)	250	365	
EY	Exposure years (years)	40	Varies for Adult (chronic non-cancer) 78 (Lifetime) 1 Infant (birth to <1 year) 5 Toddler (1–5 years) 5 Child (6–10 years) 5 Youth (11–15 years) 5 Youth (16–20 years)	Number of years in age group Note: These age bins may vary for different measurements and sources
AT	Averaging time non-cancer	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	See pg. 6–23 of Risk assessment guidance for superfund, volume I: Human health evaluation manual (Part A). (U.S. EPA, 1989)
	Averaging time cancer	78 years (28,470 days)	78 years (28,470 days)	See Table 18-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)
BW	Body weight (kg)	80	80 Adult 7.83 Infant (birth to <1 year) 16.2 Toddler (1–5 years) 31.8 Child (6–10 years) 56.8 Youth (11–15 years) 71.6 Youth (16–20 years) 65.9 Adolescent woman of childbearing age (16 to <21) – apply to all developmental exposure scenarios	See Table 8-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a) (Refer to Figure 31 for age-specific BW) Note: These age bins may vary for different measurements and sources See Table 8-5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)
$IR_{dw\text{-}acute}$	Drinking water ingestion rate (L/day) – acute	3.219 Adult	3.219 Adult 1.106 Infant (birth to <1 year) 0.813 Toddler (1–5 years) 1.258 Child (6–10 years) 1.761 Youth (11–15 years) 2.214 Youth (16–20 years)	See Tables 3-15 and 3-33; weighted average of 90th percentile consumer-only ingestion of drinking water (birth to <6 years) (U.S. EPA, 2011a)
$IR_{dw\text{-}chronic}$	Drinking water ingestion rate (L/day) – chronic	0.880 Adult	0.880 Adult 0.220 Infant (birth to <1 year)	Chapter 3 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 3-9 per capita

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source/Notes
		Occupational	Residential	
			0.195 Toddler (1–5 years) 0.294 Child (6–10 years) 0.315 Youth (11–15 years) 0.436 Youth (16–20 years)	mean values; weighted averages for adults (21–49 and 50+ years), for toddlers (years 1–2, 2–3, and 3 to <6).
IR_{inc}	Incidental water ingestion rate (L/h)		0.025 Adult 0.05 Child (6 to < 16 years)	Evaluation of Swimmer Exposures Using the SWIMODEL Algorithms and Assumptions (U.S. EPA, 2015a)
IR_{fish}	Fish ingestion rate (g/day)		22 Adult	Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (U.S. EPA, 2014) This represents the 90th percentile consumption rate of fish and shellfish from inland and nearshore waters for the U.S. adult population 21 years of age and older, based on NHANES data from 2003–2010
IR_{soil}	Soil ingestion rate (mg/day)	50 Indoor workers 100 Outdoor workers	100 Infant (<6 months) 200 Infant to Youth (6 months to <12 years) 100 Youth to Adult (12+ years) 1,000 Soil Pica Infant to Youth (1 to <12 years) 50,000 Geophagy (all ages)	U.S. EPA Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (1991) Chapter 5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 5-1, Upper percentile daily soil and dust ingestion
SA_{water}	Skin surface area exposed (cm^2) used for incidental water dermal contact		19,500 Adult 7,600 Child (3 to < 6 years) 10,800 Child (6 to < 11 years) 15,900 Youth (11 to < 16 years)	Chapter 7 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 7-1; recommended mean values for total body surface area, for children (sexes combined) and adults by sex
K_p	Permeability constant (cm/h) used for incidental water dermal contact		0.001 Or calculated using K_p equation with chemical specific K_{OW} and MW (see exposure formulas)	*EPA Dermal Exposure Assessment: Principles and Applications* (U.S. EPA, 1992), Table 5-7, "Predicted K_p Estimates for Common Pollutants"

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source/Notes
		Occupational	Residential	
SA_{soil}	Skin surface area exposed (cm^2) used for soil dermal contact	3,300 Adult	5,800 Adult 2,700 Child	*EPA Risk Assessment Guidance for Superfund RAGS Part E for Dermal Exposure* (U.S. EPA, 2004)
AF_{soil}	Adherence factor (mg/cm^2) used for soil dermal contact	0.2 Adult	0.07 Adult 0.2 Child	*EPA Risk Assessment Guidance for Superfund RAGS Part E for Dermal Exposure* (U.S. EPA, 2004)

Table_Apx A-4. Mean and Upper Milk Ingestion Rates by Age

Age Group	Milk Ingestion (mL/kg day)	
	Mean	Upper (95th percentile)
Birth to <1 month	150	220
1 to <3 month	140	190
3 to <6 month	110	150
6 to <12 month	83	130
Birth to <1 year	104.8	152.5

A.1 Surface Water Exposure Activity Parameters

Table_Apx A-5. Incidental Dermal (Swimming) Modeling Parameters

Input	Description (Units)	Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)	Notes	Reference
BW	Body weight (kg)	80	56.8	31.8	Mean body weight. Chapter 8 of the *Exposure Factors Handbook*, Table 8-1	U.S. EPA (2021)
SA	Skin surface area exposed (cm^2)	19,500	15,900	10,800	U.S. EPA Swimmer Exposure Assessment Model (SWIMODEL)	U.S. EPA (2015a)
ET	Exposure time (h/day)	3	2	1	High-end default short-term duration from U.S. EPA Swimmer Exposure Assessment Model (SWIMODEL)	U.S. EPA (2015a)
ED	Exposure duration (years for ADD)	57	5	5	Number of years in age group	U.S. EPA (2021)
AT	Averaging time (years for ADD)	57	5	5	Number of years in age group	U.S. EPA (2021)
K_p	Permeability coefficient (cm/h)	0.0071 cm/h			CEM estimate aqueous K_p	(U.S. EPA; ICF Consulting, 2022)

Table_Apx A-6. Incidental Oral Ingestion (Swimming) Modeling Parameters

Input	Description (Units)	Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)	Notes	Reference
IR_{inc}	Ingestion rate (L/h)	0.092	0.152	0.096	Upper percentile ingestion while swimming. Chapter 3 of the *Exposure Factors Handbook*, Table 3-7.	U.S. EPA (2019a)
BW	Body weight (kg)	80	56.8	31.8	Mean body weight. Chapter 8 of the *Exposure Factors Handbook*, Table 8-1.	U.S. EPA (2021)
ET	Exposure time (hr/day)	3	2	1	High-end, default, short-term duration from U.S. EPA Swimmer Exposure Assessment Model (SWIMODEL); based on competitive swimmers in the age class	U.S. EPA (2015a)
$IR_{inc\text{-}daily}$	Incidental daily ingestion rate (L/day)	0.276	0.304	0.096	Calculation: ingestion rate × exposure time	
IR/BW	Weighted incidental daily ingestion rate (L/kg-day)	0.0035	0.0054	0.0030	Calculation: ingestion rate/body weight	
ED	Exposure duration (years for ADD)	57	5	5	Number of years in age group	U.S. EPA (2021)
AT	Averaging time (years for ADD)	57	5	5	Number of years in age group	U.S. EPA (2021)
CF1	Conversion factor (mg/µg)	1.00E−03				
CF2	Conversion factor (days/year)	365				

Appendix B ESTIMATING HYDROLOGICAL FLOW DATA FOR SURFACE WATER MODELING

EPA's ECHO database was accessed via the Application Programming Interface (API) and queried for facilities regulated under the Clean Water Act. All available NPDES permit IDs were retrieved from the facilities returned by the query. An additional query of the DMR REST service was conducted via the ECHO API to return the National Hydrography Dataset Plus (NHDPlus) reach code associated with the receiving waterbody for each available facility. Modeled flow metrics were then extracted for the retrieved reach codes from the NHDPlus V2.1 Flowline Network's Enhanced Runoff Method (EROM) Flow database. The EROM database provides modeled monthly average flows for each month of the year. While the EROM flow database represents averages across a 30-year time period, the lowest of the monthly average flows was selected as a substitute for the 30Q5 (the lowest 30-day average flow that occurs on average once every 5 years) flow used in modeling, as both approximate the lowest observed monthly flow at a given location. The substitute 30Q5 flow was then plugged into the regression equation used by the EPA surface water model, E-FAST, to convert between these flow metrics and solved for the 7Q10 (the lowest 7-day average flow that occurs on average once every 10 years) using Equation_Apx B-1. In previous assessments, the EPA has selected the 7Q10 flow as a representative low flow scenario to assess ecological impacts from effluent discharges into streams, while the harmonic mean represents a more average flow for assessing chronic drinking water exposure.

Equation_Apx B-1. Calculating the 7Q10 Flow

$$7Q10 = \frac{\left(0.409\frac{cfs}{MLD} \times \frac{30Q5}{1.782}\right)^{1.0352}}{0.409\frac{cfs}{MLD}}$$

Where:

$7Q10$ = Modeled 7Q10 flow, in million liters per day (MLD)
$30Q5$ = Lowest monthly average flow from NHD, in MLD

Further, the harmonic mean (HM) flow was calculated using Equation_Apx B-2, derived from the relevant E-FAST regression (U.S. EPA, 2007).

Equation_Apx B-2. Calculating the Harmonic Mean Flow

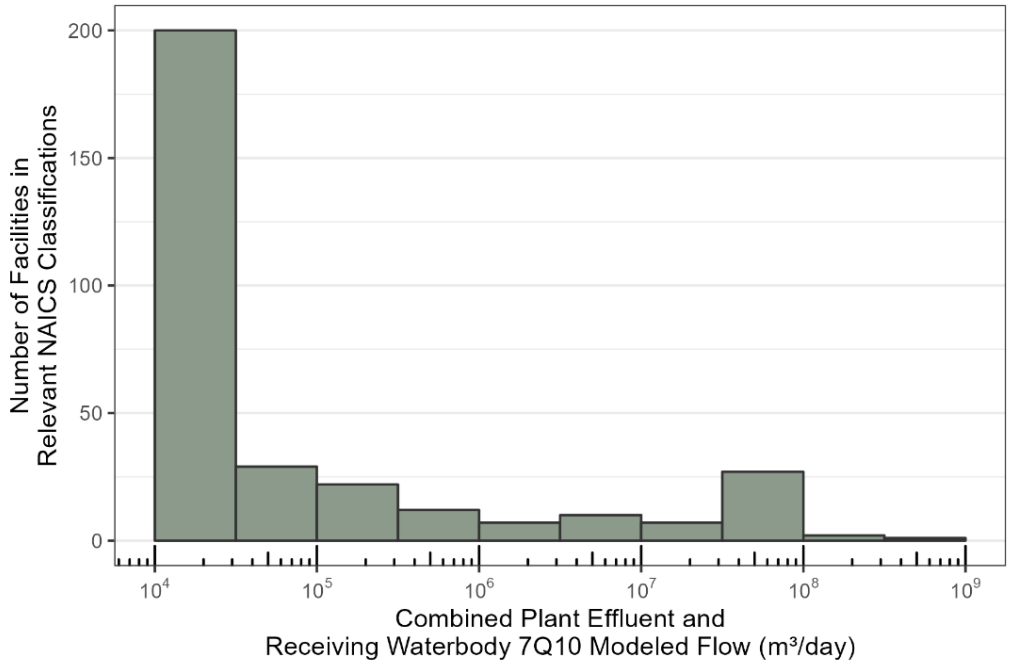
$$HM = 1.194 \times \frac{\left(0.409\frac{cfs}{MLD} \times AM\right)^{0.473} \times \left(0.409\frac{cfs}{MLD} \times 7Q10\right)^{0.552}}{0.409\frac{cfs}{MLD}}$$

Where:

HM = Modeled harmonic mean flow, in MLD
AM = Annual average flow from NHD, in MLD
$7Q10$ = Modeled 7Q10 flow from the previous equation, in MLD

In addition to the hydrologic flow data retrieved from the NHDPlus database, information about the facility effluent rate was collected, as available, from the ECHO API. The receiving waterbody flow was then calculated as the sum of the hydrologic flow estimated from regression, and the facility effluent

flow. From the distribution of resulting receiving waterbody flow rates across the pooled flow data of all relevant NAICS codes, the median (P50) flow rate was applied as a conservative low flow condition across the modeled releases (Figure_Apx B-1). Additional refined analyses were conducted for the scenarios resulting in the greatest environmental concentrations by applying the 75th and 90th percentile (P75 and P90, respectively) flow metrics from the distribution, which were expected to be more representative of the flow conditions associated with high-end releases.



Figure_Apx B-1. Distribution of Receiving Waterbody 7Q10 Modeled Flow for Facilities with Relevant NAICS Classifications

For each COU with surface water releases, the highest estimated release of DBP to surface water was used to estimate the corresponding DBP concentrations in the receiving water body. The total days of release associated with the highest COU release was applied as continuous days of release per year (*e.g.*, a scenario with 250 days of release per year was modeled as 250 consecutive days of release, followed by 115 days of no release, per year). Raw daily concentration estimates from PSC were manually evaluated for the highest resulting concentrations in an averaging window equal to the total days of release (for example, a scenario with 250 days of release was evaluated for the highest 250-day average concentration). The frollmean function in the data.table package in R was used to calculate the rolling averages. The function takes in the concentration values to be averaged (extracted from the PSC Daily Output File) and the number of values to include in the averaging window which was total days of release (extracted from the PSC Summary Output File). The function outputs a list of averages from consecutive averaging windows (for example, the first average will be for values 1- total days of release and the second average will be for values 2- total days of release +1).

Appendix C SURFACE WATER RISK SCREENING RESULTS

C.1 Incidental Dermal Exposures (Swimming)

Based on the estimated dermal doses in [ADD], EPA screened for risk to adults (21+ years), youth (11–15 years), and children (6–10 years). Table_Apx C-1 summarizes the acute MOEs based on the dermal doses. Using the total acute dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 (U.S. EPA, 2024f). *Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects for dermal absorption through swimming is not expected.*

Table_Apx C-1. Risk Screen for Modeled Incidental Dermal (Swimming) Doses for Adults, Youths, and Children from Modeling and Monitoring Results

Scenario	Water Column Concentrations		Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	Acute MOE	Acute MOE	Acute MOE
Manufacturing (P50)	885	616	203	265	437
Highest monitored surface water (NWQMC, 2021)	26.8	26.8	6,697	8,748	14,420

C.2 Incidental Ingestion

Based on the estimated incidental ingestion doses in Table 5-2, EPA screened for risk to adults (21+ years), youth (11–15 years), and children (6–10 years). Table_Apx C-2 summarizes the acute MOEs based on the incidental ingestion doses. Using the total acute dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 (U.S. EPA, 2024f). *Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects for incidental ingestion through swimming is not expected.*

Table_Apx C-2. Risk Screen for Modeled Incidental Ingestion Doses for Adults, Youths, and Children from Modeling and Monitoring Results

Scenario	Water Column Concentrations		Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	Acute MOE	Acute MOE	Acute MOE
Manufacturing (P50)	885	616	688	443	786
Highest monitored surface water (NWQMC, 2021)	26.8	26.8	22,713	14,641	25,956

Appendix D GENERAL POPULATION DRINKING WATER RISK SCREENING RESULTS

Based on the estimated drinking water doses in Table 6-1, EPA screened for risk to adults (21+ years), infants (birth to <1 year), and toddlers (1–5 years). Table_Apx D-1 summarizes the acute and chronic MOEs based on the drinking water doses. Using the total acute and chronic dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 (U.S. EPA, 2024f) except for the Manufacturing OES, which is based on a high-end release estimate to multiple environmental media, paired with a very low flow assumptions. This protective screening scenario, with the entirety of the estimated environmental release assumed to be released directly to surface water, results in an MOE less than the benchmark in only the most extreme hypothetical exposure scenario with an unlikely confluence of factors. *Based on the conservative modeling parameters for drinking water concentration and exposure factors parameters, risk for non-cancer health effects for drinking water ingestion is not expected.*

This assessment assumes that concentrations at the point of intake for the drinking water system are equal to the concentrations in the receiving waterbody at the point of release, where treated effluent is being discharged from a facility. In reality, some distance between the point of release and a drinking water intake would be expected, providing space and time for additional reductions in water column concentrations via degradation, partitioning, and dilution. Some form of additional treatment would typically be expected for surface water at a drinking water treatment plant, including coagulation, flocculation, and sedimentation, and/or filtration. This treatment would likely result in even greater reductions in DBP concentrations prior to releasing finished drinking water to customers.

Table_Apx D-1. Risk Screen for Modeled Drinking Water Exposure for Adults, Infants, and Toddlers from Modeling and Monitoring Results

Scenario	Water Column Concentrations		Adult (21+ years)		Infant (Birth to <1 year)		Toddler (1–5 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	Acute MOE	Chronic MOE	Acute MOE	Chronic MOE	Acute MOE	Chronic MOE
Manufacturing (P50 flow)	616	885	59	110,000	17	44,000	47	100,000
Manufacturing (P75 flow)	24.4	46.6	1,120	2,900,000	319	1,100,000	898	2,600,000
Manufacturing (P90 flow)	1.7	3.0	17,000	41,000,000	4,958	16,000,000	14,000	37,000,000
Waste Handling, Treatment, and Disposal (TRI Reported Release)	14.5	14.5	3,599	4,800,000	1,026	1,900,000	2,884	4,400,000
High from Monitoring *Without Wastewater Treatment* (NWQMC, 2021)	26.8	26.8	1,947	2,601,209	555	1,018,360	1,561	2,376,062

Appendix E FISH INGESTION RISK SCREENING RESULTS

E.1 General Population

Using conservative exposure estimates based on the water solubility limit as the surface water concentration, acute and chronic non-cancer risk estimates for the general population were below the benchmark of 30 for both fish species (Table_Apx E-1). In comparison, the risk estimates using the highest monitored surface water concentration (NWQMC, 2021) (Section 4.2.1) exceed the benchmark by two to three orders of magnitude. EPA then refined its analysis by modeling surface water concentrations based on the high-end harmonic mean release for the Manufacturing OES. The acute, non-cancer risk estimate using modeled surface water concentration for the PVC plastics compounding OES exceeded the benchmark of 30. These results indicate that fish ingestion is not a pathway of concern for DBP for the general population.

Table_Apx E-1. Risk Estimates for Fish Ingestion Exposure for General Population

	Acute Non-Cancer MOE UFs = 30		Adult, Chronic and Non-Cancer MOE UFs = 30
	Adult	Young Toddler	
Water solubility limit (11.2 mg/L)	2 (tilapia) 2.2 (common carp)	1 (tilapia) 1.4 (common carp)	7 (tilapia) 9 (common carp)
PVC plastics compounding (HE, 1.78E–02 mg/L)	1,037 (tilapia) 1,354 (common carp)	698 (tilapia) 912 (common carp)	4,567 (tilapia) 5,964 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	756 (tilapia) 988 (common carp)	510 (tilapia) 665 (common carp)	3,332 (tilapia) 4,351 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	2,251 (tilapia) 2,939 (common carp)	1,516 (tilapia) 1,980 (common carp)	9,915 (tilapia) 12,946 (common carp)

HE = high-end; MOE = margin of exposure; PVC = polyvinyl chloride; UF = uncertainty factor

E.2 Subsistence Fishers

Acute and chronic non-cancer risk estimates for subsistence fishers were below the benchmark using the water solubility limit as the surface water concentration for both fish species (Table_Apx E-2). In comparison, the risk estimates using the highest monitored surface water concentration (NWQMC, 2021) (Section 4.2.1) exceed the benchmark by one order of magnitude. EPA then refined its analysis by modeling surface water concentrations based on the high-end harmonic release for the Manufacturing OES. The acute and chronic non-cancer risk estimates exceeded the benchmark of 30 for both fish species. These results indicate that fish ingestion is not a pathway of concern for DBP for subsistence fishers.

Table_Apx E-2. Risk Estimates for Fish Ingestion Exposure for Subsistence Fishers

	Acute and Chronic Non-Cancer MOE UFs = 30
Water solubility limit (11.2 mg/L)	0.3 (tilapia) 0.3 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	198 (tilapia) 154 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	351 (tilapia) 458 (common carp)
HE = high-end; MOE = margin of exposure; UF = uncertainty factor Note: The acute and chronic MOEs are identical because the exposure estimates and the POD do not change between acute and chronic.	

E.3 Tribal Populations

Acute and chronic non-cancer risk estimates were below the benchmark using the water solubility limit as the surface water concentration (Table_Apx E-2). EPA then refined its analysis by using the three OESs that reported releases and resulted in the highest modeled surface water concentrations. The Agency also included the highest monitored surface water concentrations from the WQP (NWQMC, 2021) (Section 4.2.1). The highest modeled surface water concentration based on the PVC plastics compounding OES resulted in some non-cancer risk estimates to be below the benchmark. Risk estimates for other OESs are two to three orders of magnitude above the benchmark. Non-cancer risk estimates are below the benchmark for the PVC plastics compounding OES. These results indicate that fish ingestion can be a pathway of concern for DBP for Tribal populations.

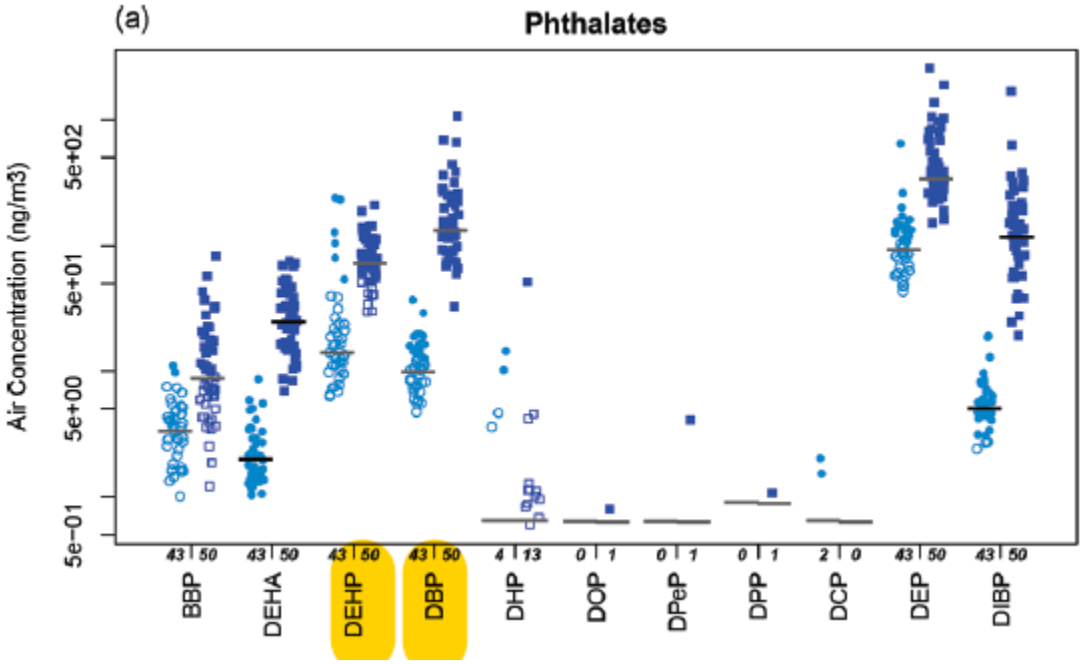
Table_Apx E-3. Risk Estimates for Fish Ingestion Exposure for Tribal Populations

	Acute and Chronic Non-Cancer MOE UFs = 30		
	Current IR, Mean	Current IR, 95th Percentile	Heritage IR
Water solubility limit (11.2 mg/L)	0.2 (tilapia) 0.2 (common carp)	0.0 (tilapia) 0.1 (common carp)	0.0 (tilapia) 0.0 (common carp)
Manufacturing OES, P75, HE (generic scenario) (2.24E–02 mg/L)	78 (tilapia) 102 (common carp)	19 (tilapia) 25 (common carp)	10 (tilapia) 13 (common carp)
Manufacturing OES, P90, HE (generic scenario) (1.7E–03 mg/L)	1,116 (tilapia) 1,457 (common carp)	276 (tilapia) 361 (common carp)	146 (tilapia) 191 (common carp)
Waste Handling, Treatment, Disposal-POTW (TRI reported release) (1.45E–02 mg/L)	231 (tilapia) 171 (common carp)	57 (tilapia) 42 (common carp)	30 (tilapia) 22 (common carp)
Monitored surface water concentration (8.2E–03 mg/L) (NWQMC, 2021)	231 (tilapia) 302 (common carp)	57 (tilapia) 75 (common carp)	30 (tilapia) 40 (common carp)
CT = central tendency; HE = high end; IR = ingestion rate; OES = occupational exposure scenario Note: The acute and chronic MOEs are identical because the exposure estimates and the point of departure (POD) do not change between acute and chronic.			

Appendix F AMBIENT AIR MONITORING STUDY SUMMARY

China Study (Zhu et al., 2016)

Chinese study saying cancer risks 3.51×10^{-8} to 9.75×10^{-11} well below 1×10−6.



Although the phthalates DEHP, DEHA, and DIBP are typically considered indoor contaminants from plastics and consumer goods, the concentration difference between outdoor air in urban/industrial and rural communities suggests some industrial or transportation sources as well.

New York City Study (Bove et al., 1978)

Airborne di-Butyl and di-(2-Ethylhexyl)-phthalate at three New York City Air Sampling Stations

Di-butyl phthalate concentrations in New York City air were 3.73, 5.69, and 3.28 ng/m^3, while di(2-ethylhexyl)-phthalate concentrations were 10.20, 16.79, and 14.20 ng/m^3.

Appendix G URINARY BIOMONITORING METHODS AND RESULTS

EPA analyzed urinary biomonitoring data from the CDC's NHANES, which reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. Two metabolites of DBP, mono-n-butyl phthalate (MnBP) and mono-3-hydroxybutyl phthalate (MHBP), have been reported in the NHANES data.

MnBP has been reported in NHANES beginning with the 1999 cycle and measured in 26,740 members of the general public, including 7,331 children under 16 year and 19,409 adults aged 16 years and older. Although MHBP was measured in the 2013 to 2018 NHANES cycles, the data for the 2013 to 2014 NHANES cycle was determined to be inaccurate due to procedural error and only released as surplus data, which is not readily publicly available (https://wwwn.cdc.gov/Nchs/Nhanes/2013-2014/SSPHTE_H.htm). As a result, the present analysis only includes urinary MHBP data from the 2015 to 2018 NHANES cycles. The present analysis of MHBP includes data from the 2015 to 2018 NHANES cycles and has been measured in 5,737 participants, including 1,961 children under 16 years and 3,776 adults aged 16 years and older.

Urinary MnBP and MHBP concentrations were quantified using high performance liquid chromatography-electrospray ionization-tandem mass spectrometry. Limits of detection (LOD) for each cycle on NHANES are provided in Table_Apx G-1. Values below the LOD were replaced by the lower LOD divided by the square root of two (NCHS, 2021).

Table_Apx G-1. Limit of Detection of Urinary DBP Metabolites by NHANES Cycle

NHANES Cycle	MnBP	MHBP
1999–2000	0.94	–
2001–2002	0.94	–
2003–2004	0.4	–
2005–2006	0.6	–
2007–2008	0.6	–
2009–2010	0.4	–
2011–2012	0.2	–
2013–2014	0.4	–
2015–2016	0.4	0.4
2017–2018	0.4	0.4

2976 **Table_Apx G-2. Summary of Urinary DBP Metabolite Concentrations (ng/mL) from all NHANES Cycles Between 1999–2018**

NHANES Cycle	Metabolite	Age Group	Subset	Sample Size	Detection Frequency	50th Percentile (95% CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95% CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2017–2018	MHBP	Adults	All adults	1,896	1,896 (70.94%)	0.7 (0.6–0.7)	3.2 (2.7–3.9)	0.8 (0.73–0.9)	3.87 (3.28–4.4)
2017–2018	MHBP	Adults	At or above poverty level	467	467 (75.16%)	0.5 (0.5–0.7)	2.8 (2.4–4.1)	0.78 (0.7–0.85)	3.5 (2.74–4)
2017–2018	MHBP	Adults	Below poverty level	337	337 (72.7%)	0.8 (0.28–1.3)	4.9 (2.7–11.8)	1.04 (0.9–1.23)	5.16 (4.22–6.83)
2017–2018	MHBP	Adults	Black non-Hispanic	438	438 (75.34%)	0.9 (0.7–1.4)	4.6 (2.6–6.6)	0.71 (0.6–0.84)	3.84 (2.79–5.71)
2017–2018	MHBP	Adults	Females	952	952 (69.01%)	0.9 (0.6–1.1)	4.4 (3.3–7)	1.13 (0.98–1.33)	4.51 (3.73–5.26)
2017–2018	MHBP	Adults	Males	944	944 (72.88%)	0.7 (0.6–0.7)	3.1 (2.7–4.1)	0.67 (0.62–0.74)	3.33 (2.76–4)
2017–2018	MHBP	Adults	Mexican American	278	278 (66.55%)	0.4 (0.28–0.7)	2.7 (1.6–4.9)	0.85 (0.65–0.96)	3.51 (2.86–4.05)
2017–2018	MHBP	Adults	Other	532	532 (67.48%)	0.5 (0.28–0.8)	3.1 (1.9–4.1)	0.9 (0.74–1.05)	4.67 (3.82–6.09)
2017–2018	MHBP	Adults	Unknown income	840	840 (67.14%)	0.6 (0.4–1)	3.3 (1.4–4.4)	0.79 (0.63–0.99)	4.71 (3.08–6.78)
2017–2018	MHBP	Adults	White non-Hispanic	648	648 (72.69%)	0.5 (0.4–0.7)	3.2 (2.1–4.3)	0.79 (0.7–0.92)	3.75 (2.92–4.4)
2017–2018	MHBP	Children	Adolescents (11 to <16 years)	213	213 (81.69%)	4.2 (3.3–5.9)	32 (24–45.5)	0.98 (0.78–1.16)	2.45 (2.13–3.47)
2017–2018	MHBP	Children	Adolescents (11 to <16 years)	213	213 (81.69%)	4.2 (3.3–5.9)	32 (24–45.5)	0.98 (0.78–1.16)	2.78 (2.13–3.63)
2017–2018	MHBP	Children	All children	866	866 (84.18%)	1.3 (1.1–1.4)	4.9 (4.4–5.8)	1.15 (0.93–1.49)	4.4 (3.47–5.37)
2017–2018	MHBP	Children	At or above poverty level	231	231 (88.31%)	1.3 (1.1–1.4)	4.7 (3.7–5.8)	1.11 (0.79–1.55)	3.89 (2.94–4.88)
2017–2018	MHBP	Children	Below poverty level	234	234 (85.9%)	1.4 (1.2–2)	5.9 (4.8–7)	1.45 (1.16–1.62)	5.23 (3.79–7.02)
2017–2018	MHBP	Children	Black non-Hispanic	207	207 (87.44%)	1.5 (1–2.1)	5.2 (3.7–7.7)	1.06 (0.84–1.18)	3.99 (2.59–7.02)
2017–2018	MHBP	Children	Children (6 to <11 years)	274	274 (89.05%)	5.8 (4.2–9)	38.4 (29.7–103.7)	1.83 (1.44–2.18)	4.91 (4.5–5.56)
2017–2018	MHBP	Children	Children (6 to <11 years)	274	274 (89.05%)	5.8 (4.2–9)	38.4 (29.7–103.7)	1.83 (1.44–2.18)	5.71 (4.4–7.78)
2017–2018	MHBP	Children	Females	447	447 (82.77%)	1.2 (0.7–1.5)	4.9 (4–6.2)	1.33 (0.98–1.89)	4.41 (3.73–6.21)
2017–2018	MHBP	Children	Males	419	419 (85.68%)	1.2 (1–1.3)	4.9 (3.9–6.6)	0.97 (0.82–1.22)	4.4 (2.87–6.67)
2017–2018	MHBP	Children	Mexican American	139	139 (80.58%)	1 (0.5–1.3)	3.3 (2.5–5.9)	1.04 (0.91–1.22)	3.3 (2.18–6.78)
2017–2018	MHBP	Children	Other	262	262 (83.97%)	1.2 (0.9–1.7)	6.3 (4.9–23.3)	1.45 (1–1.85)	6.51 (3.61–138)
2017–2018	MHBP	Children	Toddlers (3 to <6 years)	379	379 (82.06%)	5.7 (4.4–8.1)	25 (13.7–34.9)	0.71 (0.38–0.79)	1.51 (1.09–2.35)
2017–2018	MHBP	Children	Toddlers (3 to <6 years)	379	379 (82.06%)	5.7 (4.4–8.1)	25 (13.7–34.9)	0.71 (0.38–0.79)	1.86 (1.42–2.65)
2017–2018	MHBP	Children	Unknown income	316	316 (80.7%)	1.1 (0.5–1.4)	5.9 (2.4–23.3)	1.05 (0.82–1.35)	7.78 (1.84–18.49)
2017–2018	MHBP	Children	White non-Hispanic	258	258 (83.72%)	1.2 (1.1–1.5)	4 (2.9–5.2)	1.15 (0.78–1.78)	3.83 (2.87–5.37)
2017–2018	MnBP	Adults	All adults	1,896	1,896 (99.26%)	9.4 (7.7–10.6)	35 (30.5–42.1)	8.63 (7.92–9.26)	34.4 (29.74–38.02)
2017–2018	MnBP	Adults	At or above poverty level	467	467 (99.14%)	9 (6.7–11)	34.2 (26.6–42.1)	8.5 (7.5–9.36)	30.63 (26.76–34.4)
2017–2018	MnBP	Adults	Below poverty level	337	337 (99.41%)	9.8 (5.6–13.4)	54.9 (31.2–84.3)	10.75 (9.41–12.73)	44.48 (39.52–56.27)
2017–2018	MnBP	Adults	Black non-Hispanic	438	438 (99.54%)	14.2 (10.9–18.4)	56.6 (34.8–71.5)	8.83 (8.15–9.52)	41 (30.96–57.26)

NHANES Cycle	Metabolite	Age Group	Subset	Sample Size	Detection Frequency	50th Percentile (95% CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95% CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2017–2018	MnBP	Adults	Females	952	952 (99.16%)	11.5 (8.2–14)	43.4 (33–54.6)	11.67 (10–12.69)	38 (33.18–42.05)
2017–2018	MnBP	Adults	Males	944	944 (99.36%)	9 (7.5–10.6)	35 (30.2–43.6)	7.41 (6.69–8.11)	29 (26.5–34.17)
2017–2018	MnBP	Adults	Mexican American	278	278 (100%)	8.3 (5.6–11.7)	31 (18.7–36.3)	9.2 (7.44–10.66)	30 (26.25–38.89)
2017–2018	MnBP	Adults	Other	532	532 (98.87%)	7.8 (5.8–10.7)	35.8 (30.7–51.7)	9.64 (8.09–11.23)	46.5 (37.77–67.67)
2017–2018	MnBP	Adults	Unknown income	840	840 (99.4%)	9.2 (6–11)	36.2 (22.8–69.4)	7.93 (6.84–11.09)	39.38 (29.43–83.68)
2017–2018	MnBP	Adults	White non-Hispanic	648	648 (99.07%)	8.2 (6.1–10.9)	32.9 (24.3–47.4)	8.32 (7.47–9.02)	32.27 (28.08–36.5)
2015–2016	MHBP	Adults	All adults	1,880	1,880 (72.71%)	0.7 (0.5–0.8)	3.8 (2.8–4.8)	0.89 (0.8–0.97)	4.11 (3.64–4.67)
2015–2016	MHBP	Adults	At or above poverty level	461	461 (74.4%)	0.7 (0.5–0.8)	3.7 (2.6–4)	0.87 (0.8–0.93)	3.6 (3.06–4)
2015–2016	MHBP	Adults	Below poverty level	399	399 (76.94%)	0.9 (0.7–1.2)	4.6 (2–11.9)	1.08 (0.97–1.26)	5.97 (4.86–6.93)
2015–2016	MHBP	Adults	Black non-Hispanic	427	427 (74.24%)	1 (0.8–1.2)	3.6 (2–5.3)	0.72 (0.67–0.85)	5.26 (4.15–6.8)
2015–2016	MHBP	Adults	Females	984	984 (74.59%)	0.8 (0.7–1.1)	4.7 (3.5–6.6)	1.27 (1.1–1.38)	4.77 (4.29–5.26)
2015–2016	MHBP	Adults	Males	896	896 (70.65%)	0.6 (0.5–0.8)	3.8 (2.7–4.9)	0.73 (0.65–0.8)	3.37 (2.89–3.85)
2015–2016	MHBP	Adults	Mexican American	342	342 (70.76%)	0.6 (0.28–0.7)	3.7 (2.3–6.8)	1.03 (0.93–1.08)	5 (4–6.15)
2015–2016	MHBP	Adults	Other	540	540 (72.59%)	0.6 (0.5–0.8)	3.3 (2.6–4.8)	0.8 (0.73–0.96)	4.19 (3.5–4.73)
2015–2016	MHBP	Adults	Unknown income	833	833 (68.91%)	0.7 (0.28–1.6)	5.3 (1.2–7.5)	0.88 (0.69–1.14)	5.19 (3.23–6.14)
2015–2016	MHBP	Adults	White non-Hispanic	571	571 (72.85%)	0.7 (0.5–0.8)	3.9 (2.9–5.9)	0.9 (0.8–1)	3.75 (3.09–4.34)
2015–2016	MHBP	Children	Adolescents (11 to <16 years)	284	284 (85.21%)	7.3 (5.4–10.3)	61.8 (38.7–80.6)	1.1 (0.79–1.4)	3.38 (2.88–3.84)
2015–2016	MHBP	Children	Adolescents (11 to <16 years)	284	284 (85.21%)	7.3 (5.4–10.3)	61.8 (38.7–80.6)	1.1 (0.79–1.4)	3.81 (3.04–4)
2015–2016	MHBP	Children	All children	1,095	1,095 (87.67%)	1.2 (1.1–1.4)	5.5 (4.7–6.1)	1.36 (1.24–1.54)	5 (4.29–6.09)
2015–2016	MHBP	Children	At or above poverty level	282	282 (89.01%)	1.2 (1.1–1.4)	5.4 (3.6–7.2)	1.33 (1.16–1.46)	4.41 (3.81–5.65)
2015–2016	MHBP	Children	Below poverty level	329	329 (85.71%)	1.4 (1.2–1.8)	8.3 (4–12.5)	1.44 (1.24–1.72)	8.33 (4.76–11.24)
2015–2016	MHBP	Children	Black non-Hispanic	271	271 (86.72%)	1.3 (1–1.9)	5.9 (4.6–11.8)	1.2 (0.88–1.53)	9.09 (4.76–11.24)
2015–2016	MHBP	Children	Children (6 to <11 years)	346	346 (90.75%)	10.4 (8.1–13.3)	81.3 (64.8–173.9)	2 (1.67–2.35)	4.93 (4.4–6)
2015–2016	MHBP	Children	Children (6 to <11 years)	346	346 (90.75%)	10.4 (8.1–13.3)	81.3 (64.8–173.9)	2 (1.67–2.35)	8.18 (6.07–10.98)
2015–2016	MHBP	Children	Females	517	517 (87.81%)	1.2 (1–1.4)	5.6 (5–7.1)	1.43 (1.29–1.61)	6.06 (4.67–8.18)
2015–2016	MHBP	Children	Males	578	578 (87.54%)	1.3 (1.1–1.5)	5.7 (3.7–7.7)	1.29 (1.03–1.58)	4.41 (3.81–5.65)
2015–2016	MHBP	Children	Mexican American	253	253 (85.77%)	1.2 (1–1.5)	5.3 (4.2–11.3)	1.34 (1.14–1.61)	5.65 (4.23–8.33)
2015–2016	MHBP	Children	Other	280	280 (88.57%)	1.2 (1–1.6)	4.7 (3.6–5.4)	1.34 (1.04–1.72)	4.35 (3.26–5.25)
2015–2016	MHBP	Children	Toddlers (3 to <6 years)	465	465 (86.88%)	6.8 (4.2–13.8)	55.3 (20.8–77.8)	0.49 (0.35–0.69)	1.53 (1.27–2.43)
2015–2016	MHBP	Children	Toddlers (3 to <6 years)	465	465 (86.88%)	6.8 (4.2–13.8)	55.3 (20.8–77.8)	0.49 (0.35–0.69)	2.06 (0.98–5.65)
2015–2016	MHBP	Children	Unknown income	388	388 (87.89%)	1.6 (0.8–2.4)	4.6 (2.3–19.8)	1.82 (1.11–2.12)	4.71 (3.5–15.59)

NHANES Cycle	Metabolite	Age Group	Subset	Sample Size	Detection Frequency	50th Percentile (95% CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95% CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2015–2016	MHBP	Children	White non-Hispanic	291	291 (89.35%)	1.3 (1–1.8)	5.6 (4.2–7.7)	1.39 (1.23–1.67)	4.62 (4–6.22)
2015–2016	MnBP	Adults	All adults	1,880	1,880 (99.04%)	9.5 (7.9–10.9)	44.9 (32.7–53.8)	9.94 (8.95–10.63)	36.02 (34.44–38.2)
2015–2016	MnBP	Adults	At or above poverty level	461	461 (99.57%)	9.2 (7.6–10.3)	39 (32.5–44.9)	9.24 (8.64–10.11)	32.89 (28.94–36.06)
2015–2016	MnBP	Adults	Below poverty level	399	399 (99%)	12.4 (9.1–15.8)	55.4 (24.8–157.6)	12.5 (10.97–14.39)	56.3 (41.41–76.07)
2015–2016	MnBP	Adults	Black non-Hispanic	427	427 (99.06%)	13.5 (9.6–19.2)	46.6 (27.4–114.6)	10.4 (9.38–11.3)	47.37 (40.2–74.42)
2015–2016	MnBP	Adults	Females	984	984 (98.88%)	10.5 (9.1–12)	44.5 (37.9–65.1)	13.52 (11.88–15.23)	43.85 (37.64–46.84)
2015–2016	MnBP	Adults	Males	896	896 (99.22%)	9.6 (7.7–10.9)	44.9 (31.6–55.1)	8.4 (7.89–8.93)	31.14 (26.62–34.95)
2015–2016	MnBP	Adults	Mexican American	342	342 (98.54%)	9.6 (6.7–11.6)	55.1 (35.3–111.7)	10.82 (10.05–12.15)	48.61 (36.92–67.65)
2015–2016	MnBP	Adults	Other	540	540 (99.26%)	11.7 (7.5–15.7)	37.5 (29.9–45.1)	10.13 (9.32–10.97)	37.04 (33.52–45.23)
2015–2016	MnBP	Adults	Unknown income	833	833 (98.68%)	11.7 (6.2–20.4)	55.6 (14.1–68)	11.6 (8.6–14.92)	46.55 (28.92–72.21)
2015–2016	MnBP	Adults	White non-Hispanic	571	571 (99.12%)	8.4 (6.8–10)	44.9 (22.8–55.6)	9.24 (8.57–10.6)	34.52 (29.71–36.25)
2013–2014	MnBP	Adults	All adults	2,040	2,040 (98.28%)	10.2 (9.4–11.3)	44.6 (37–50.5)	8.93 (8.25–9.54)	34.63 (29.89–42.93)
2013–2014	MnBP	Adults	At or above poverty level	484	484 (98.14%)	9.6 (8.5–11.4)	40 (32–50.5)	8.77 (8.09–9.37)	33.86 (28.33–45.24)
2013–2014	MnBP	Adults	Below poverty level	454	454 (98.9%)	11.8 (9.1–17.3)	49.5 (38.9–72.6)	10.65 (9.53–12.1)	42.22 (29.94–52.86)
2013–2014	MnBP	Adults	Black non-Hispanic	442	442 (98.64%)	12.3 (10.2–16.8)	66.7 (44.7–74.1)	8.9 (8–9.78)	32.89 (28.36–38.72)
2013–2014	MnBP	Adults	Females	1,076	1,076 (97.86%)	10.9 (9.1–12.6)	53.2 (42.6–75)	11.18 (10.27–12.26)	46 (34.37–64.21)
2013–2014	MnBP	Adults	Males	964	964 (98.76%)	10.1 (9.3–11.4)	42.6 (33.6–50.5)	7.67 (6.97–8.38)	28.76 (22.69–35.76)
2013–2014	MnBP	Adults	Mexican American	282	282 (98.23%)	8.6 (5.8–11.8)	53.5 (20.7–78.7)	9.71 (7.85–11.34)	36.71 (27.96–45.78)
2013–2014	MnBP	Adults	Other	496	496 (98.99%)	10.6 (9–14)	49.7 (37–77.8)	10 (9.21–11.16)	38.04 (31.25–45.24)
2013–2014	MnBP	Adults	Unknown income	921	921 (97.94%)	9.2 (5.6–15.3)	29.3 (26.6–74.2)	7.69 (6.48–9.75)	26.95 (19.52–36.32)
2013–2014	MnBP	Adults	White non-Hispanic	820	820 (97.68%)	9.6 (8.7–11.5)	32 (26–50.2)	8.68 (7.67–9.54)	33.1 (24.03–55.5)
2011–2012	MnBP	Adults	All adults	1,894	1,894 (93.66%)	9.2 (8.2–10.6)	46.9 (37.3–61.3)	8.93 (8.13–9.8)	42.27 (32.22–54.75)
2011–2012	MnBP	Adults	At or above poverty level	449	449 (93.32%)	9.2 (8–11.1)	46.3 (35.3–61.3)	8.73 (7.96–9.51)	38.89 (29.71–51.79)
2011–2012	MnBP	Adults	Below poverty level	441	441 (95.01%)	10 (6.3–15.8)	58.6 (43.1–99.7)	9.67 (8.29–11.28)	50.88 (36.74–66.42)
2011–2012	MnBP	Adults	Black non-Hispanic	499	499 (95.79%)	14.1 (10.7–17.3)	63.3 (47.5–96.2)	11 (9.55–11.92)	43.5 (34.42–55.77)
2011–2012	MnBP	Adults	Females	933	933 (93.46%)	9.4 (7–11.8)	58.5 (41.7–129.3)	11.31 (9.77–13.33)	47.44 (42.09–54.75)
2011–2012	MnBP	Adults	Males	961	961 (93.86%)	9.2 (8.2–10.7)	46.7 (36.4–61.3)	8.06 (7.54–8.85)	34.58 (24.13–55.19)
2011–2012	MnBP	Adults	Mexican American	186	186 (96.24%)	8.8 (6.8–12.5)	35.8 (23.5–46.4)	10.24 (8.62–12.21)	41.18 (32.47–55.6)
2011–2012	MnBP	Adults	Other	545	545 (92.48%)	9.5 (8.2–11.6)	52.2 (38.5–68.5)	10.88 (9.8–11.69)	50 (46.16–73.28)
2011–2012	MnBP	Adults	Unknown income	821	821 (92.94%)	10 (5.7–13.3)	37 (17.1–64.3)	9.86 (6.43–12.72)	54.64 (22.86–2863.14)
2011–2012	MnBP	Adults	White non-Hispanic	664	664 (92.32%)	8.6 (7.9–10.1)	44.3 (26.7–76.3)	8.03 (7.43–9.02)	34.62 (27.94–54.75)
2009–2010	MnBP	Adults	All adults	2,127	2,127 (99.44%)	14.59 (12.94–16.33)	70.32 (61.73–82.47)	13.82 (13.04–14.87)	56.11 (49.62–65.82)
2009–2010	MnBP	Adults	At or above poverty level	550	550 (99.45%)	13.91 (12.25–16.11)	65.27 (54.59–70.34)	13.42 (12.6–14.33)	49.83 (45.17–55.02)

NHANES Cycle	Metabolite	Age Group	Subset	Sample Size	Detection Frequency	50th Percentile (95% CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95% CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2009–2010	MnBP	Adults	Below poverty level	469	469 (99.36%)	15.04 (12.11–16.48)	133.91 (71.74–161.63)	16.09 (13.55–18.89)	79.91 (63.41–107.08)
2009–2010	MnBP	Adults	Black non-Hispanic	400	400 (99.75%)	19.61 (16.86–27.12)	105.11 (65.27–193.05)	14.81 (12.97–18.14)	52.32 (43.98–73.54)
2009–2010	MnBP	Adults	Females	1,040	1,040 (99.33%)	19.38 (14.12–22.7)	83.85 (60.63–123.12)	17.69 (15.34–18.89)	70.96 (53.78–89.24)
2009–2010	MnBP	Adults	Males	1087	1087 (99.54%)	14.29 (12.65–16.33)	70.34 (61.41–82.63)	12.81 (11.76–13.57)	45.2 (39.66–53.78)
2009–2010	MnBP	Adults	Mexican American	393	393 (99.49%)	15.77 (11.4–21.88)	55.77 (43.56–82.63)	14.13 (13.28–15.57)	87.68 (59.71–99.03)
2009–2010	MnBP	Adults	Other	336	336 (99.7%)	13.5 (11.63–17.39)	160.59 (52.99–418.4)	15.08 (11.96–20.14)	81.52 (48.38–362.56)
2009–2010	MnBP	Adults	Unknown income	905	905 (99.34%)	17.045 (12.67–31.19)	322.68 (40.3–322.68)	17.21 (13.39–20.04)	70.96 (28.63–1933.78)
2009–2010	MnBP	Adults	White non-Hispanic	998	998 (99.2%)	13.46 (10.85–16.85)	69.53 (54.75–81.95)	13.46 (12.79–14.45)	50.85 (44.79–57.8)
2007–2008	MnBP	Adults	All adults	2,021	2,021 (99.16%)	18.8 (16–20.9)	80.8 (63.8–99.4)	17.47 (15.94–19.16)	77.12 (61.63–90)
2007–2008	MnBP	Adults	At or above poverty level	505	505 (99.41%)	19.1 (16–22.5)	79.5 (55.6–95.7)	16.82 (15.24–18.68)	72.26 (59.5–84.47)
2007–2008	MnBP	Adults	Below poverty level	392	392 (99.23%)	19.3 (15.4–24.1)	110.2 (63.8–156.9)	22.41 (18.75–26.15)	102.06 (77.12–159.63)
2007–2008	MnBP	Adults	Black non-Hispanic	434	434 (99.54%)	21.4 (17.8–26.8)	110.2 (57.4–338.3)	17.31 (14.79–20)	78.11 (51.6–125.23)
2007–2008	MnBP	Adults	Females	1,030	1,030 (99.03%)	23 (18.9–28.9)	114.2 (83.7–161.7)	24.54 (21.12–27.52)	100.64 (80–144.88)
2007–2008	MnBP	Adults	Males	991	991 (99.29%)	18.9 (15.9–21.3)	79.1 (61.6–99.4)	14.69 (13.33–16.27)	55.2 (45.93–65.22)
2007–2008	MnBP	Adults	Mexican American	371	371 (99.73%)	19.6 (14.7–27.6)	92.2 (61.8–141.1)	19.8 (15.19–25.48)	100.32 (59.5–193.03)
2007–2008	MnBP	Adults	Other	294	294 (99.66%)	19.2 (12.6–31.7)	61.2 (50–168.5)	19.03 (14.21–24.44)	89.5 (55.04–103.41)
2007–2008	MnBP	Adults	Unknown income	948	948 (98.84%)	14.8 (11–40.8)	63.4 (33.3–84.1)	16.79 (14.67–26.25)	73.33 (51.87–158.45)
2007–2008	MnBP	Adults	White non-Hispanic	922	922 (98.59%)	18.8 (15–21.5)	73.5 (53.4–94.5)	16.8 (15.41–18.77)	71.83 (57.43–84.17)
2005–2006	MnBP	Adults	All adults	1,831	1,831 (99.67%)	21.2 (19–24)	86 (66.2–118.1)	18.07 (16.41–19.71)	73.38 (62.58–94.78)
2005–2006	MnBP	Adults	At or above poverty level	436	436 (99.08%)	20.9 (18.4–24)	78.9 (63.8–104.9)	17.73 (15.91–19.62)	66.69 (53.73–84.64)
2005–2006	MnBP	Adults	Below poverty level	340	340 (99.71%)	25.4 (18–35.3)	124.4 (101.2–222.8)	20.48 (18.25–23.09)	99.24 (76.72–115.98)
2005–2006	MnBP	Adults	Black non-Hispanic	464	464 (100%)	24.9 (21.6–27.2)	111.7 (84.3–139)	17.3 (15.07–19.76)	70.56 (51.28–100.56)
2005–2006	MnBP	Adults	Females	935	935 (99.57%)	22.8 (19.7–26.6)	113.2 (97.1–132.6)	25.38 (20.53–30.36)	111.55 (78.54–139.17)
2005–2006	MnBP	Adults	Males	896	896 (99.78%)	20.7 (18.5–23.9)	86 (63.8–118.7)	15.42 (14.22–16.41)	51.02 (46.1–65.61)
2005–2006	MnBP	Adults	Mexican American	390	390 (99.49%)	22.6 (15.8–27.6)	105.8 (74.3–127.5)	18.07 (15.13–21.23)	99.46 (69.86–161.41)
2005–2006	MnBP	Adults	Other	131	131 (100%)	28 (22–54.2)	176.2 (51.9–1063.6)	21.89 (15.63–29.61)	73.38 (47.75–178.24)
2005–2006	MnBP	Adults	Unknown income	955	955 (99.9%)	18.8 (8.6–38.7)	98.8 (38.7–170.5)	19.35 (13.48–29.16)	108.6 (50.5–177.4)
2005–2006	MnBP	Adults	White non-Hispanic	846	846 (99.53%)	18.8 (17.6–20.7)	72.6 (55.4–112.8)	17.9 (16.22–19.53)	67.35 (56.44–95.7)
2003–2004	MnBP	Adults	All adults	1,889	1,889 (99.42%)	20.7 (16.9–24.3)	80.7 (64.2–109.1)	17.84 (16.25–19.62)	83.64 (68.28–110)
2003–2004	MnBP	Adults	At or above poverty level	474	474 (99.58%)	19.6 (16–24)	70.2 (60.6–97.9)	17 (15.53–18.47)	78.1 (62.31–100.95)
2003–2004	MnBP	Adults	Below poverty level	393	393 (99.24%)	23.9 (17.9–31.4)	105.9 (67.5–172.1)	22.5 (20.35–24.2)	129.78 (98.84–141.7)
2003–2004	MnBP	Adults	Black non-Hispanic	423	423 (99.76%)	30.3 (26.5–32.6)	118.9 (88.9–135)	20.93 (18.47–24.37)	87.43 (70.11–100.27)
2003–2004	MnBP	Adults	Females	980	980 (99.69%)	25.2 (22.7–31)	127.4 (101.7–163.7)	25.27 (22.44–29.69)	121.21 (83.64–143.14)

NHANES Cycle	Metabolite	Age Group	Subset	Sample Size	Detection Frequency	50th Percentile (95% CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95% CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2003–2004	MnBP	Adults	Males	909	909 (99.12%)	20.6 (16.6–24.3)	75.8 (62.9–104.2)	14.84 (13.61–16.03)	59.43 (50.31–81.5)
2003–2004	MnBP	Adults	Mexican American	423	423 (99.29%)	21.1 (16.6–32.7)	73 (60.9–107.7)	20.13 (16.63–24.61)	109.13 (80.75–149.83)
2003–2004	MnBP	Adults	Other	142	142 (100%)	23 (13.4–38.1)	172.1 (36–3191.3)	20.39 (16.67–27.36)	123.33 (83.8–415.06)
2003–2004	MnBP	Adults	Unknown income	904	904 (99.34%)	26.8 (15.7–52.7)	99.1 (34.5–124.1)	22.15 (13.19–29.14)	86.81 (41.96–155)
2003–2004	MnBP	Adults	White non-Hispanic	901	901 (99.22%)	18.8 (14.5–22.8)	66.7 (52.7–94)	16.82 (15.27–18.63)	73.35 (58.09–99.23)
2001–2002	MnBP	Adults	All adults	2,004	2,004 (98.1%)	19.3 (16.3–21.5)	91.7 (64.7–117.4)	16.46 (15.29–17.53)	84.3 (72.35–103.08)
2001–2002	MnBP	Adults	At or above poverty level	463	463 (96.98%)	18.6 (15.2–21.2)	79.6 (57.1–103.4)	15.71 (14.56–16.62)	76.21 (62.32–91.88)
2001–2002	MnBP	Adults	Below poverty level	361	361 (98.89%)	23.1 (16.1–29.4)	101.2 (59.1–143.1)	20.3 (17.58–24.02)	130.51 (72.31–220)
2001–2002	MnBP	Adults	Black non-Hispanic	414	414 (99.52%)	26.7 (20.8–31.3)	93.9 (67.3–143.6)	19.02 (14.92–23)	84.3 (67.38–103.57)
2001–2002	MnBP	Adults	Females	1,019	1,019 (98.14%)	22.4 (18.6–29.2)	105.5 (86.8–122)	23.62 (21.18–26.6)	110.63 (90.71–138.18)
2001–2002	MnBP	Adults	Males	985	985 (98.07%)	19.3 (15.8–21.4)	87.5 (60.5–117.4)	13.68 (12.92–14.86)	60 (50.32–78.39)
2001–2002	MnBP	Adults	Mexican American	445	445 (98.43%)	18.4 (15.1–23.1)	88 (47.8–313.5)	18.2 (15.88–19.92)	84.47 (62.02–128.76)
2001–2002	MnBP	Adults	Other	162	162 (96.91%)	19.8 (14.7–24.6)	83.7 (47.8–111.9)	16.07 (12.61–19.43)	59.02 (48.83–74.17)
2001–2002	MnBP	Adults	Unknown income	1,052	1,052 (98.29%)	21.8 (14.5–41.2)	180.3 (40.6–322.1)	15.59 (9.55–23.78)	103.57 (50.32–135.85)
2001–2002	MnBP	Adults	White non-Hispanic	983	983 (97.56%)	18.2 (14.3–21.2)	92.7 (55.8–129.6)	15.88 (14.38–17.31)	91.03 (70–115.26)
1999–2000	MnBP	Adults	All adults	1,827	1,827 (98.69%)	23.1 (20.9–24.7)	111.1 (92.3–125.6)	20.81 (18.93–23.19)	93.17 (75.98–114.08)
1999–2000	MnBP	Adults	At or above poverty level	412	412 (99.27%)	22.8 (20.6–25.3)	98.6 (85.2–114.1)	19.82 (17.34–22.59)	93.02 (67.12–116.99)
1999–2000	MnBP	Adults	Below poverty level	377	377 (99.2%)	23.4 (14.5–33.5)	162.7 (60.6–224.6)	25.15 (20.13–30.67)	105.44 (74.57–139.12)
1999–2000	MnBP	Adults	Black non-Hispanic	363	363 (99.17%)	30.9 (24–38.9)	114.1 (85.4–143.4)	24.9 (19.69–29.39)	93.15 (73.11–113.04)
1999–2000	MnBP	Adults	Females	964	964 (98.65%)	32.6 (27.6–41.2)	155.9 (98.9–412.1)	30.48 (27.74–34.29)	134.09 (99.53–196.13)
1999–2000	MnBP	Adults	Males	863	863 (98.73%)	22.7 (20.5–24.1)	108 (91.1–120.8)	16.97 (15.53–18.74)	64.7 (57.33–71.51)
1999–2000	MnBP	Adults	Mexican American	550	550 (98.91%)	23.5 (18.4–24.9)	104.8 (63.8–117)	19.26 (17.86–21.69)	94.15 (73.87–117.78)
1999–2000	MnBP	Adults	Other	176	176 (99.43%)	29.3 (19.6–33.5)	162.7 (82.3–224.6)	24.44 (18.93–30.46)	107.55 (71.51–196.13)
1999–2000	MnBP	Adults	Unknown income	798	798 (97.99%)	19.2 (8–33.4)	93.3 (50.6–140.4)	22.04 (18.08–30.09)	83.15 (62.62–130.62)
1999–2000	MnBP	Adults	White non-Hispanic	738	738 (98.1%)	20.7 (16.7–23.2)	96.2 (78.8–119.8)	20.11 (17.61–23.16)	92.27 (63.62–136.9)

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2978 **Table_Apx G-3. Regression Coefficients and P-values for Statistical Analyses of DBP Metabolite Concentrations**

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	P-value, 50th Percentile	Regression Coefficient, 95th Percentile	P-value, 95th Percentile
2015–2018	MHBP	Adults	All adults	Age	Sex race income	–	<0.001	–	<0.001
2015–2018	MHBP	Adults	All adults	Income	Age sex race	–	0.0036	–	<0.001
2015–2018	MHBP	Adults	All adults	Race	Age sex income	–	<0.001	–	<0.001
2015–2018	MHBP	Adults	All adults	Sex	Age race income	–	<0.001	–	<0.001
2015–2018	MHBP	Adults	All adults	Years	Age sex race income	−0.0601	<0.001	−0.3351	<0.001
2015–2018	MHBP	Adults	All adults	Years	Age sex race income	−0.0601	<0.001	−0.3351	<0.001
2015–2018	MHBP	Adults	At or above poverty level	Years	Age sex race	0.02505	0.2319	0.05601	0.0758
2015–2018	MHBP	Adults	At or above poverty level	Years	Age sex race	0.02505	0.2319	0.05601	0.0758
2015–2018	MHBP	Adults	Below poverty level	Years	Age sex race	0.05588	0.1268	0.06424	0.0794
2015–2018	MHBP	Adults	Below poverty level	Years	Age sex race	0.05588	0.1268	0.06424	0.0794
2015–2018	MHBP	Adults	Black non-Hispanic	Years	Age sex income	0.03770	0.3541	−0.0619	0.1399
2015–2018	MHBP	Adults	Black non-Hispanic	Years	Age sex income	0.03770	0.3541	−0.0619	0.1399
2015–2018	MHBP	Adults	Females	Years	Age race income	−0.1028	<0.001	−0.3133	<0.001
2015–2018	MHBP	Adults	Females	Years	Age race income	−0.1028	<0.001	−0.3133	<0.001
2015–2018	MHBP	Adults	Males	Years	Age race income	−0.0057	0.7635	−0.108	<0.001
2015–2018	MHBP	Adults	Males	Years	Age race income	−0.0057	0.7635	−0.108	<0.001
2015–2018	MHBP	Adults	Mexican-American	Years	Age sex income	−0.0629	0.3873	0.67195	<0.001
2015–2018	MHBP	Adults	Mexican-American	Years	Age sex income	−0.0629	0.3873	0.67195	<0.001
2015–2018	MHBP	Adults	Other	Years	Age sex income	−0.0766	0.0866	−0.8002	<0.001
2015–2018	MHBP	Adults	Other	Years	Age sex income	−0.0766	0.0866	−0.8002	<0.001
2015–2018	MHBP	Adults	Unknown income	Years	Age sex race	−1.5314	<0.001	−4.2629	<0.001
2015–2018	MHBP	Adults	Unknown income	Years	Age sex race	−1.5314	<0.001	−4.2629	<0.001
2015–2018	MHBP	Adults	White non-Hispanic	Years	Age sex income	−0.1358	<0.001	0.26398	<0.001
2015–2018	MHBP	Adults	White non-Hispanic	Years	Age sex income	−0.1358	<0.001	0.26398	<0.001
2015–2018	MHBP	Children	All children (<16 years)	Age	Sex race income	–	<0.001	–	<0.001
2015–2018	MHBP	Children	All children (<16 years)	Income	Age sex race	–	0.0877	–	<0.001
2015–2018	MHBP	Children	All children (<16 years)	Race	Age sex income	–	0.0131	–	<0.001
2015–2018	MHBP	Children	All children (<16 years)	Sex	Age race income	–	0.9056	–	<0.001
2015–2018	MHBP	Children	Adolescents (11 to <16 years)	Years	Sex race income	0.22160	<0.001	−0.3986	<0.001
2015–2018	MHBP	Children	Adolescents (11 to <16 years)	Years	Sex race income	0.22160	<0.001	−0.3986	<0.001
2015–2018	MHBP	Children	Toddlers (3 to <6 years)	Years	Sex race income	0.22821	0.0773	0.19641	0.0885
2015–2018	MHBP	Children	Toddlers (3 to <6 years)	Years	Sex race income	0.22821	0.0773	0.19641	0.0885
2015–2018	MHBP	Children	Children (6 to <10 years)	Years	Sex race income	−0.1095	0.0533	−0.8971	<0.001

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	P-value, 50th Percentile	Regression Coefficient, 95th Percentile	P-value, 95th Percentile
2015–2018	MHBP	Children	Children (6 to <10 years	Years	Sex race income	−0.1095	0.0533	−0.8971	<0.001
2015–2018	MHBP	Children	All children (<16 years	Years	Age sex race income	0.13948	<0.001	−0.6881	<0.001
2015–2018	MHBP	Children	All children (<16 years	Years	Age sex race income	0.13948	<0.001	−0.6881	<0.001
2015–2018	MHBP	Children	At or above poverty level	Years	Age sex race	−0.127	0.0043	−0.2311	<0.001
2015–2018	MHBP	Children	At or above poverty level	Years	Age sex race	−0.127	0.0043	−0.2311	<0.001
2015–2018	MHBP	Children	Below poverty level	Years	Age sex race	0.33899	<0.001	−1.0209	<0.001
2015–2018	MHBP	Children	Below poverty level	Years	Age sex race	0.33899	<0.001	−1.0209	<0.001
2015–2018	MHBP	Children	Black non-Hispanic	Years	Age sex income	0.21667	0.0049	−0.8785	<0.001
2015–2018	MHBP	Children	Black non-Hispanic	Years	Age sex income	0.21667	0.0049	−0.8785	<0.001
2015–2018	MHBP	Children	Females	Years	Age race income	0.11178	0.0274	−0.0377	0.5194
2015–2018	MHBP	Children	Females	Years	Age race income	0.11178	0.0274	−0.0377	0.5194
2015–2018	MHBP	Children	Males	Years	Age race income	0.07433	0.1299	−0.9418	<0.001
2015–2018	MHBP	Children	Males	Years	Age race income	0.07433	0.1299	−0.9418	<0.001
2015–2018	MHBP	Children	Mexican-American	Years	Age sex income	−0.4431	<0.001	−0.5245	<0.001
2015–2018	MHBP	Children	Mexican-American	Years	Age sex income	−0.4431	<0.001	−0.5245	<0.001
2015–2018	MHBP	Children	Other	Years	Age sex income	0.06189	0.549	−0.1149	0.4289
2015–2018	MHBP	Children	Other	Years	Age sex income	0.06189	0.549	−0.1149	0.4289
2015–2018	MHBP	Children	Unknown income	Years	Age sex race	–	0.0123	–	<0.001
2015–2018	MHBP	Children	Unknown income	Years	Age sex race	–	0.0123	–	<0.001
2015–2018	MHBP	Children	White non-Hispanic	Years	Age sex income	0.11139	0.0311	0.43391	<0.001
2015–2018	MHBP	Children	White non-Hispanic	Years	Age sex income	0.11139	0.0311	0.43391	<0.001
2015–2018	MHBP	Women	All women of reproductive age	Age	Sex race income	–	<0.001	–	<0.001
2015–2018	MHBP	Women	All women of reproductive age	Income	Age sex race	–	0.1377	–	0.2221
2015–2018	MHBP	Women	All women of reproductive age	Race	Age sex income	–	0.1005	–	<0.001
2015–2018	MHBP	Women	All women of reproductive age	Sex	Age race income	–	<0.001	–	<0.001
2015–2018	MHBP	Women	All women of reproductive age	Years	Age sex race income	−0.0308	0.5852	1.42648	<0.001
2015–2018	MHBP	Women	At or above poverty level	Years	Age sex race	0.01807	0.8223	0.11482	0.7696
2015–2018	MHBP	Women	Below poverty level	Years	Age sex race	−0.1646	0.1681	−0.6382	0.1531
2015–2018	MHBP	Women	Black non-Hispanic	Years	Age sex income	−0.0315	0.8479	0.77272	0.0866
2015–2018	MHBP	Women	Females	Years	Age race income	−0.0308	0.5852	1.42648	<0.001
2015–2018	MHBP	Women	Mexican-American	Years	Age sex income	0.10197	0.3969	2.08916	<0.001
2015–2018	MHBP	Women	Other	Years	Age sex income	−0.0185	0.848	0.74702	0.0093
2015–2018	MHBP	Women	Unknown income	Years	Age sex race	0.29205	0.0681	2.21315	<0.001
2015–2018	MHBP	Women	White non-Hispanic	Years	Age sex income	−0.0244	0.8612	2.05854	0.0229

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	P-value, 50th Percentile	Regression Coefficient, 95th Percentile	P-value, 95th Percentile
1999–2018	MnBP	Adults	All adults	Age	Sex race income	–	<0.001	–	<0.001
1999–2018	MnBP	Adults	All adults	Income	Age sex race	–	0.1101	–	<0.001
1999–2018	MnBP	Adults	All adults	Race	Age sex income	–	<0.001	–	<0.001
1999–2018	MnBP	Adults	All adults	Sex	Age race income	–	<0.001	–	<0.001
1999–2018	MnBP	Adults	All adults	Years	Age sex race income	−0.5043	<0.001	−1.5193	<0.001
1999–2018	MnBP	Adults	At or above poverty level	Years	Age sex race	−0.7337	<0.001	−1.9643	<0.001
1999–2018	MnBP	Adults	Below poverty level	Years	Age sex race	−0.8590	<0.001	−2.304	<0.001
1999–2018	MnBP	Adults	Black non-Hispanic	Years	Age sex income	−0.3549	<0.001	−1.8314	<0.001
1999–2018	MnBP	Adults	Females	Years	Age race income	−0.3713	<0.001	−1.8329	<0.001
1999–2018	MnBP	Adults	Males	Years	Age race income	−0.5328	<0.001	−1.1366	<0.001
1999–2018	MnBP	Adults	Mexican-American	Years	Age sex income	−0.7860	<0.001	−2.2968	<0.001
1999–2018	MnBP	Adults	Other	Years	Age sex income	−0.6674	<0.001	−1.224	<0.001
1999–2018	MnBP	Adults	Unknown income	Years	Age sex race	−0.04	0.2986	−0.5050	<0.001
1999–2018	MnBP	Adults	White non-Hispanic	Years	Age sex income	−0.6614	<0.001	−1.8375	<0.001
1999–2018	MnBP	Children	All children (<16 years	Age	Sex race income	–	0.386	–	0.0073
1999–2018	MnBP	Children	All children (<16 years	Income	Age sex race	–	0.2985	–	0.5367
1999–2018	MnBP	Children	All children (<16 years	Race	Age sex income	–	<0.001	–	<0.001
1999–2018	MnBP	Children	All children (<16 years	Sex	Age race income	–	0.0012	–	<0.001
1999–2018	MnBP	Children	Adolescents (11 to <16 years	Years	Sex race income	−0.7676	<0.001	−1.5696	<0.001
1999–2018	MnBP	Children	Toddlers (3 to <6 years	Years	Sex race income	−1.4556	<0.001	−2.027	<0.001
1999–2018	MnBP	Children	Children (6 to <10 years	Years	Sex race income	−0.6346	<0.001	−0.8292	<0.001
1999–2018	MnBP	Children	All children (<16 years	Years	Age sex race income	−0.7062	<0.001	−1.0890	<0.001
1999–2018	MnBP	Children	At or above poverty level	Years	Age sex race	−1.3871	<0.001	−2.6951	<0.001
1999–2018	MnBP	Children	Below poverty level	Years	Age sex race	−0.7066	<0.001	−1.7833	<0.001
1999–2018	MnBP	Children	Black non-Hispanic	Years	Age sex income	−1.7075	<0.001	−4.8491	<0.001
1999–2018	MnBP	Children	Females	Years	Age race income	−0.9803	<0.001	−0.3950	<0.001
1999–2018	MnBP	Children	Males	Years	Age race income	−0.6468	<0.001	−1.7490	<0.001
1999–2018	MnBP	Children	Mexican-American	Years	Age sex income	−0.7349	<0.001	−0.3946	<0.001
1999–2018	MnBP	Children	Other	Years	Age sex income	−0.975	<0.001	−0.7710	<0.001
1999–2018	MnBP	Children	Unknown income	Years	Age sex race	−0.5003	<0.001	0.70492	<0.001
1999–2018	MnBP	Children	White non-Hispanic	Years	Age sex income	−0.4363	<0.001	−1.1186	<0.001
1999–2018	MnBP	Women	All women of reproductive age	Age	Sex race income	–	<0.001	–	<0.001
1999–2018	MnBP	Women	All women of reproductive age	Income	Age sex race	–	0.3669	–	<0.001
1999–2018	MnBP	Women	All women of reproductive age	Race	Age sex income	–	0.0068	–	<0.001

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	P-value, 50th Percentile	Regression Coefficient, 95th Percentile	P-value, 95th Percentile
1999–2018	MnBP	Women	All women of reproductive age	Sex	Age race income	–	<0.001	–	<0.001
1999–2018	MnBP	Women	All women of reproductive age	Years	Age sex race income	−1.1953	<0.001	−1.1005	<0.001
1999–2018	MnBP	Women	At or above poverty level	Years	Age sex race	−1.0600	<0.001	−3.9577	<0.001
1999–2018	MnBP	Women	Below poverty level	Years	Age sex race	−1.4453	<0.001	−3.7430	<0.001
1999–2018	MnBP	Women	Black non-Hispanic	Years	Age sex income	−1.6397	<0.001	−3.9001	<0.001
1999–2018	MnBP	Women	Females	Years	Age race income	−1.1953	<0.001	−1.1005	<0.001
1999–2018	MnBP	Women	Mexican-American	Years	Age sex income	−1.1381	<0.001	0.91770	<0.001
1999–2018	MnBP	Women	Other	Years	Age sex income	−1.4323	<0.001	−4.7382	<0.001
1999–2018	MnBP	Women	Unknown income	Years	Age sex race	−1.1137	<0.001	−0.2231	0.1547
1999–2018	MnBP	Women	White non-Hispanic	Years	Age sex income	−0.9298	<0.001	−2.7311	<0.001