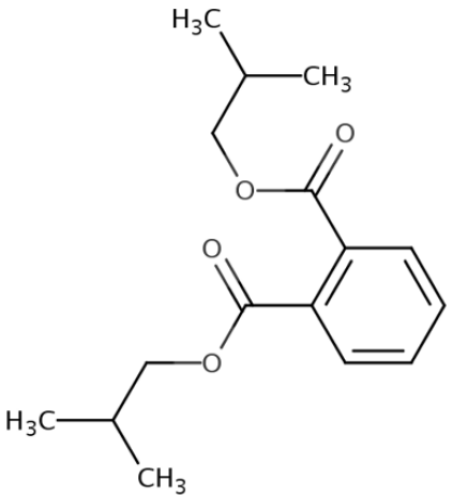
United States
Environmental Protection Agency

EPA-740-D-25-010
July 2025
Office of Chemical Safety and
Pollution Prevention

Draft Environmental Media and General Population Exposure and Environmental Exposure for Diisobutyl Phthalate (DIBP)

Technical Support Document for the Draft Risk Evaluation

CASRN 84-69-5



July 2025

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDIX TABLES

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
BSAF	biota-sediment accumulation factors
CDC	Centers for Disease Control and Prevention (U.S.)
CEM	Consumer Exposure Model
COU	Condition of use
CPSC	Consumer Product Safety Commission (U.S.)
DAD	Dermal absorbed dose
DI	Daily intake

DIBP	Diisobutyl phthalate
dw	Dry weight
ECHO	EPA Enforcement and Compliance History Online Database
EPA	Environmental Protection Agency (U.S.)
FOD	Frequency of detection
F_{ue}	Fractional urinary excretion
IIOAC	Integrated indoor-outdoor air calculator
EPA	Environmental Protection Agency (U.S.)
HEC	Human equivalent concentration
HED	Human equivalent dose
HM	Harmonic mean
K_{OA}	Octanol:air coefficient
K_{OC}	Organic carbon:water partition coefficent
K_p	Dermal permeability coefficient
LADD	Lifetime average daily dose
MCNP	Mono-(carboxynonyl) phthalate
MOE	Margin of exposure
NAICS	North American Industry Classification System
NEI	National Emissions Inventory
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
PSC	Point Source Calculator tool
PESS	Potentially exposed or susceptible subpopulation(s)
POD	Point of departure
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
UF	Uncertainty factor
U.S.	United States
ww	Wet weight
WWTP	Wastewater treatment plant

SUMMARY

DIBP– Environmental Media Concentration and General Population Exposure Assessment Summary:
Key Points

EPA (or the Agency) evaluated the reasonably available information for various environmental media concentrations and estimated exposure using conservative exposure scenarios as a screening level approach. The conservative high-end exposure was assumed to result from the highest DIBP 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 assessed environmental concentrations of DIBP in air, water, and land (soil, biosolids, and groundwater) for use in environmental exposure and general population exposure assessment.
  - 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 DIBP will have low persistence potential and mobility in soils. Therefore, groundwater concentrations resulting from releases of DIBP to landfills or to agricultural lands via biosolids applications were not quantified but are discussed qualitatively.
  - For the water pathway, DIBP in water releases is expected to predominantly partition into sediment and suspended particles in the water column. 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 modeled concentration to estimate risk is protective.
  - For the ambient air pathway, the modeled DIBP 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 modeled concentration to estimate risk is protective.
- Screening level risk estimates using high-end modeled water concentrations for DIBP exceeded the benchmark (and therefore refinement was not necessary) for incidental dermal contact, incidental ingestion from swimming, ingestion of drinking water, and ingestion of fish. The same is true using high-end modeled air concentrations for inhalation of ambient air ingestion of soil from air to soil deposition.
- EPA concluded that there are no exposure pathways of concern for the general population for DIBP.
- DIBP is found in relatively low concentrations in aquatic or terrestrial organisms and has low bioaccumulation and biomagnification potential. Therefore, DIBP 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 Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025i). DIBP is a common chemical name for a category of chemical substances under one CASRN (84-61-7): bis(2-methylpropyl) benzene-1,2-dicarboxylate (IUPAC), di-isobutyl phthalate, 1,2-benzenedicarboxylic acid, among others. DIBP is commonly used as a plasticizer in the production of plastics and other polymers for use in consumer, commercial, and industrial applications.

This document describes the use of reasonably available information to estimate environmental concentration of DIBP in different environmental media and the use of the estimated concentrations to evaluate exposure to the general population from releases associated with Toxic Substances Control Act (TSCA) conditions of use (COUs). EPA evaluated the reasonably available information for releases of DIBP from facilities that use, manufacture, or process DIBP under industrial and/or commercial COUs as detailed in the *Draft Environmental Release and Occupational Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025e). 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	Repackaging into large and small containers
Processing	Incorporation into article	Plasticizer in plastic product manufacturing	Plastics converting
		Plasticizer in transportation equipment manufacturing	Plastics converting
	Incorporation in formulation, mixture, or reaction product	Plasticizer in adhesive manufacturing	Incorporation into adhesives and sealants
		Plasticizer in plastic product manufacturing	Plastic compounding
		Solvents (which become part of product formulations or mixture) in plastic material and resin manufacturing	Plastic compounding
		Paints and coatings	Incorporation into paints and coatings
		Processing aids, not otherwise listed	Plastic compounding
		Repackaging (*e.g.*, laboratory chemicals)	Repackaging into large and small containers
		Plastic and rubber products not covered elsewhere	Rubber manufacturing
		Catalyst (*e.g.*, catalyst component for polyolefins production)	Use as a catalyst
	Processing – as a	Intermediate in plastic	Use as a catalyst

Life Cycle Stage	Category	Subcategory	OES
	reactant	manufacturing	
	Recycling	Recycling	Recycling
Distribution in Commerce	Distribution in commerce	Distribution in commerce	Distribution in commerce
Industrial Uses	Foam	Pipeline pigs	N/A[a]
	Paints and coatings	Paints and coatings	Application of paints and coatings
	Plastic and rubber products not covered elsewhere	Plastic and rubber products not covered elsewhere	Fabrication of final product from articles
	Adhesives and sealants	Two component glues and adhesives	Application of adhesives and sealants
		Transportation equipment manufacturing	Application of adhesives and sealants
		Two component glues and adhesives	Application of adhesives and sealants
Commercial Uses	Adhesives and sealants	Two component glues and adhesives	Application of adhesives and sealants
	Paints and coatings	Paints and coatings	Application of paints and coatings
	Other articles with routine direct contact during normal use including rubber articles; plastic articles (hard)	Other articles with routine direct contact during normal use including rubber articles; plastic articles (hard)	Fabrication of final product from articles
	Laboratory chemicals	Laboratory chemicals	Use of laboratory chemicals
	Toys, playground, and sporting equipment	Toys, playground, and sporting equipment	Fabrication of final product from articles
Disposal	Disposal	Disposal	Waste handling, treatment, and disposal

[a] As discussed in the *Draft Risk Evaluation for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025i), EPA may assess this COU and include the assessment results in the final version of this document.

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

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
Manufacturing	Fugitive air
	Stack air
	Wastewater to onsite treatment or discharge to POTW
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
Repackaging into large and small containers	Fugitive air
	Wastewater to onsite treatment, discharge to POTW, or landfill
Plastics converting	Fugitive or stack air
	Stack air
	Fugitive air, onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Fugitive air, onsite wastewater treatment or discharge to POTW, direct to surface water
	Incineration or landfill
Incorporation into adhesives and sealants	Fugitive air
	Stack air
	Wastewater to onsite treatment or discharge to POTW
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
Plastic compounding	Fugitive or stack air
	Stack air
	Fugitive air, onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Incineration or landfill
Incorporation into paints and coatings	Fugitive air
	Stack air
	Wastewater to onsite treatment or discharge to POTW
	Onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
Use as a catalyst	Fugitive air
	Stack air
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
Application of paints and coatings [with engineering controls]	Fugitive air
	Stack air
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
	Incineration or landfill
Application of paints and coatings [without engineering controls]	Fugitive air
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
	Air, onsite wastewater treatment or discharge to POTW, incineration, or landfill
	Incineration or landfill
Application of adhesives and sealants	Fugitive or stack air
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
Use of laboratory chemicals – liquid	Fugitive or stack air
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
Use of laboratory chemicals – solid	Stack air
	Unknown media (fugitive air, onsite wastewater treatment or discharge to POTW, incineration, or landfill)
	Onsite wastewater treatment or discharge to POTW, incineration, or landfill
Rubber manufacturing	Fugitive or stack air
	Fugitive air, onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Stack air
	Onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Onsite wastewater treatment or discharge to POTW, direct to surface water
	Incineration or landfill
Recycling and disposal	Stack air
	Fugitive air, onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Onsite wastewater treatment or discharge to POTW, direct to surface water, incineration, or landfill
	Onsite wastewater treatment or discharge to POTW

[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

Releases from all OESs were considered, but EPA focused on estimating high-end concentrations of

DIBP from the largest estimated releases by media for its screening level assessment of environmental and general population exposures. This means that EPA considered the concentration of DIBP in a given environmental media resulting from the OES that had the highest release compared to the other OESs. The OES resulting in the highest environmental concentration of DIBP varied by environmental media as shown in Table 1-3. 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 *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h). Briefly, based on DIBP's fate parameters (*e.g.*, Henry's Law constant, log K_{OC}, water solubility, fugacity modeling), EPA anticipates DIBP to be predominantly in water, soil, and sediment. However, because DIBP is released to the ambient air from industrial facilities and processes, inhalation of ambient air is a possible exposure pathway. EPA quantitatively assessed concentrations of DIBP in surface water, sediment, and ambient air. Soil concentrations of DIBP from land application of biosolids were not quantitatively assessed as DIBP is expected to have limited persistence potential and mobility in soils receiving biosolids.

Environmental exposures using the predicted concentrations of DIBP are presented in Section 12. As DIBP 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, 2025h). Additionally, EPA discusses the potential DIBP dietary exposures to aquatic and terrestrial organisms in the environment in Section 12. EPA did not conduct a quantitative analysis of DIBP trophic transfer, as DIBP 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 DIBP, please see the *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h).

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. The Agency assumed that if there is no risk for an individual identified as having the potential for the highest exposure associated with a COU for a given pathway of exposure, then that pathway was determined not to be a potential pathway of exposure for the general population and was not pursued further. 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, refinement of exposure estimates, and exposure estimates for additional subpopulations and COUs/OESs.

Table 1-3 summarizes the exposure pathways assessed for the general population. For DIBP, 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 DIBP is not expected to be persistent or mobile in soils. No monitoring data for DIBP in biosolids or landfills were available. 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, fish ingestion, and ambient air are not pathways of concern for DIBP for highly exposed populations based on the OES leading to high-end concentrations of DIBP 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)	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
Plastic compounding	Surface water	Dermal	Dermal exposure to DIBP in surface water during swimming (Section 5.1.1)	No
		Oral	Incidental ingestion of DIBP in surface water during swimming (Section 5.1.2)	No
Plastic compounding	Drinking water	Oral	Ingestion of drinking water (Section 6.1.1)	No
All	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	No
All			Ingestion of fish for subsistence fishers (Section 7.2)	No
Plastic compounding			Ingestion of fish for tribal populations (Section 7.3)	No
Plastic compounding (fugitive and stack)	Ambient air	Inhalation	Inhalation of DIBP in ambient air resulting from industrial releases (Section 9)	No
		Oral	Ingestion from air to soil deposition resulting from industrial releases (Section 9)	No

[a] Table 1-1 provides a crosswalk of industrial and commercial COUs to OESs.
[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.

2 SCREENING LEVEL ASSESSMENT OVERVIEW

Screening level assessments are useful when there is little facility location- or scenario-specific information reasonably available. EPA began its DIBP exposure assessment using a screening level approach because of limited reasonably available environmental monitoring data and absence of location data for DIBP releases. A screening level analysis relies on conservative assumptions, including default input parameters for modeling exposure, to assess exposures that would be expected to be on the high end of the expected exposure distribution. 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 DIBP per body weight were considered to be those at the upper end of the exposure. Taken together, these exposure estimates are conservative because they were determined using the highest environmental media concentrations and greatest intake rate of DIBP 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 using high-end exposure estimates to determine if exposure pathways were pathways of concern for potential 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. 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 OES/COUs.

2.1 Estimating High-End Exposure

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

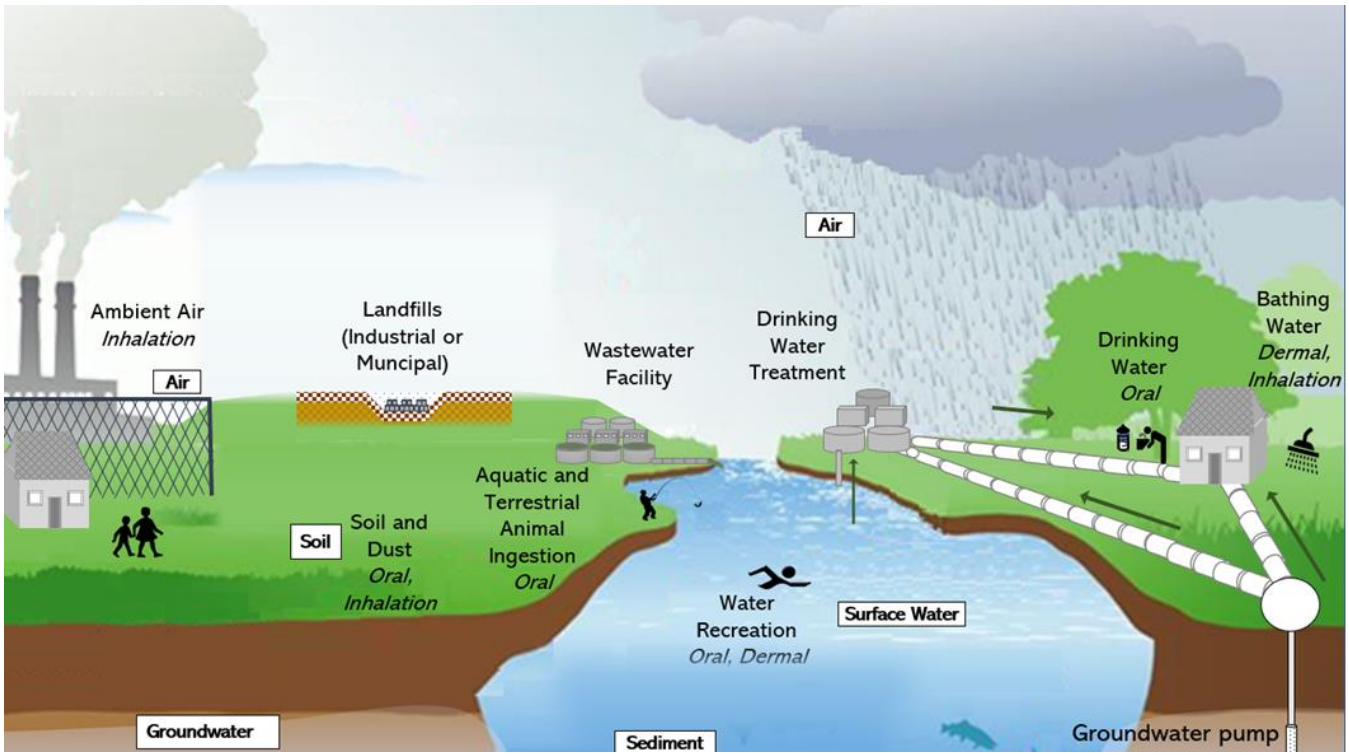


Figure 2-1. Potential Human Exposure Pathways for the General Population
The diagram presents the media (white text boxes) and routes of exposure (italics for oral, inhalation, or dermal) for the general population. Sources of drinking water from surface or water pipes are depicted with grey arrows.

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 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 DIBP 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 DIBP environmental releases from biosolids and landfills (and therefore resulting soil concentrations) were not quantified, exposure from soil or groundwater resulting from DIBP release to the environment via biosolids or landfills was not quantitatively assessed. However, the scenarios were assessed qualitatively for exposures potentially resulting from biosolids and landfills.

Table 2-1. Exposure Scenarios Assessed in Risk Screening for DIBP

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
Plastic compounding	Surface water	Dermal	Dermal exposure to DIBP in surface water during swimming	Adult, youth, and children	Quantitative, Section 5.1.1
		Oral	Incidental ingestion of DIBP in surface water during swimming	Adult, youth, and children	Quantitative, Section 5.1.2
Plastic compounding	Drinking water	Oral	Ingestion of drinking water	Adult, youth, and children	Quantitative, Section 6
All	Fish ingestion	Oral	Ingestion of fish for general population	Adult and children	Quantitative, Section 7.1
Plastic compounding			Ingestion of fish for subsistence fishers	Adult	Quantitative, Section 7.2
			Ingestion of fish for tribal populations	Adult	Quantitative, Section 7.3
Plastic compounding (fugitive and stack)	Ambient air	Inhalation	Inhalation of DIBP in ambient air resulting from industrial releases	All	Quantitative, Section 9
		Oral	Ingestion of soil from air to soil deposition resulting from industrial releases	Infants and Children	Quantitative, Section 9

As part of the general population exposure assessment, EPA utilized previously peer reviewed methodologies to conduct screening level analyses of general population exposures to DEHP associated with TSCA COUs via the ambient air, ambient water, ambient land, and fish ingestion pathways/routes For other exposure pathways, EPA's screening method assessing high-end exposure scenarios used release data that reflect exposures expected to occur in proximity to releasing facilities, which would include fenceline populations.

Modeled surface water concentrations (Section 4.1) were used to estimate incidental dermal exposures (Section 5.1.1) and incidental oral exposures (Section 5.1.2) during swimming, oral drinking water exposures (6.1.1), and fish ingestion exposure (Section 7). 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 Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025g). 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

Exposure Scenario	Target Organ System	Species	Duration	POD (mg/kg-day)	Effect	HED (mg/kg-day)	HEC (mg/m^3) [ppm]	Benchmark MOE	Reference
Acute, intermediate, chronic	Developmental toxicity	Rat	4 days during gestation (GDs 14–18)	$BMDL_5$ = 24	↓ *ex vivo* fetal testicular testosterone production	5.7	30.9 [2.71]	UF_A= 3 [a] UF_H=10 *Total UF=30*	Gray et al. (2021)

HEC = human equivalent concentration; HED = human equivalent dose; MOE = margin of exposure; NOAEL = no-observed-adverse-effect level; POD = point of departure; UF = uncertainty factor

[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 UF_A was reduced from 10 to 3.

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

Phthalates may be present in land pathways for several potential reasons. These include the amendment of soils with biosolids containing phthalates, contamination of soils and groundwater from leaking landfills, and from potential air deposition. EPA searched databases, peer-reviewed literature, and gray literature for environmental monitoring data identified during systematic review to determine the potential concentrations of DIBP in these land pathways (*i.e.*, biosolids, wastewater sludge, agricultural soils, landfills, and landfill leachate).

Broadly, databases did not yield any relevant monitoring information for DIBP. Academic and monitoring studies have identified DIBP in various relevant compartments including leachate, activated sludge, and biosolids. These studies are discussed in the subsequent and corresponding sections. However, EPA does not have any facility-specific DIBP release information as facilities do not report releases of DIBP to land from TSCA COUs in the United States. As a result, this assessment is qualitative and uses fate and physical-chemical characteristics of DIBP to estimate exposure. Experimental and field data were used to support this assessment.

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. DIBP may be introduced to biosolids by the absorption or adsorption of DIBP to particulate or organic material during wastewater treatment. Wastewater treatment is expected to remove 90 percent or more of DIBP during wastewater treatment through sorption to biosolids (Berardi et al., 2019; Tran et al., 2014; Shao and Ma, 2009; Fauser et al., 2003; Marttinen et al., 2003). The STPWIN™ model in EPI Suite™ predicts 94 percent removal of DIBP removal in wastewater treatment with 93.21 percent of removal (out of 94 percent overall removal) resulting from sorption to activated sludge and solids (U.S. EPA, 2017a).

Although DIBP is largely removed through sorption, some small fraction may be metabolized by the microbial community in activated sludge to form several metabolites that may remain in the sludge or stabilized biosolids. The known metabolites of DIBP identified in activated sludge and stabilized biosolids include 2-isobutyl phthalate (*i.e.*, monoester variant of DIBP), 2-ethylhexanol, 2-ethylhexanal, and 2-ethylhexonoic acid (Beauchesne et al., 2008).

DIBP has been identified in several U.S.-based and international surveys of wastewater sludge, composted, and stabilized biosolids. A 2012 survey of North American wastewater plants (Canada and United States) identified DIBP in sludge at concentrations ranging from 0.1 to 76.7 ng/g dry weight (dw) (Ikonomou et al., 2012). Beyond North America, DIBP has been identified in sludge at various concentrations in wastewater plants located in China (Zhu et al., 2019; Meng et al., 2014)

There are currently no U.S.-based studies reporting DIBP concentration in soil after land application. DIBP containing sludge and biosolids have not been reported for uses in surface land disposal or agricultural application. If DIBP containing sludge were be used for agricultural or fertilizing applications, they are likely to be persistent in the top layers of incorporated soil (NCBI, 2020; Net et al., 2015). No anaerobic or aerobic degradation studies were identified during systematic review. However, similar phthalates including its primary isomer, dibutyl phthalate (DBP), reported half-lives in soil

ranging from hours to several hundred days (Net et al., 2015).

Other sources of DIBP in biosolids-amended soils may include atmospheric deposition to soil. While long-range transport and deposition of DIBP in the atmosphere has not been directly monitored, Net et al. (2015) noted possible atmospheric deposition of DIBP and other phthalates in agricultural settings. A 2008 study noted concentrations up to 3,976 ng/L of DIBP in precipitation samples (Peters et al., 2008) while a 2010 study on atmospheric deposition of phthalates notes bulk wet and dry deposition of DIBP and other phthalates in the atmosphere around agricultural sites (Zeng et al., 2010). Once in the topsoil, DIBP is unlikely to be substantially taken up into plant tissues.

DIBP 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 DIBP to sorb strongly to organic media and soil, potential leaching is limited. Any leaching that does occur in the uppermost soil layers will sorb to soil lower in the column and show minimal potential to interact with groundwater systems. DIBP is not readily taken up by agricultural crop or cover crops planted in soils fertilized with biosolids. A study evaluating the potential for DBP (a DIBP isomer) to be taken up by crops demonstrated the largest concentration of DBP was on the surface of crop leaves resulting from localized volatilization and subsequent deposition of DBP from soil and particulate onto the plants 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). DIBP might be present on the surface of any plants growing in the vicinity resulting from localized atmospheric deposition of DIBP transported up by the wind or volatizing out of the top layer of soil. Although possible, no studies identified thus far in systematic review have reported that DIBP is susceptible to longer range atmospheric transport resulting in land application of DIBP-containing biosolids beyond the immediate region of initial application.

Concentrations of DIBP in soil following agricultural application of municipal biosolids were not identified from the Toxics Release Inventory (TRI) or National Emissions Inventory (NEI) release data nor were any monitoring studies identified during systematic review. As such, DIBP concentrations in soil were estimated using high-quality monitoring data, with concentrations identified in sludge concentrations ranging from 0.1 to 76.7 ng/g dry weight (dw) (Ikonomou et al., 2012). Using the EPA recommended application rate and volume and application limitation in accordance with 40 CFR part 503, *Standards for the Use of Disposal of Sewage Sludge*. 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 Land 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	5–00
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 (0.1 ng/g) and maximum (77 ng/g) concentrations of DIBP in biosolids were selected from the observed concentration in biosolids during the 2008 EPA National Sewage Survey (U.S. EPA, 2009). The 2008 survey of wastewater was determined to have a high confidence level during systematic review. DIBP concentrations in sludge selected from the wastewater sludge monitoring study was not used to quantify exposures estimates in the draft DIBP risk evaluation. The information instead provides general insight on the concentrations that may result if biosolids containing DIBP is applied to agricultural land at the recommended application rates at the observed concentrations.

Table 3-2. Estimated DIBP 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) [c d]
Corn	0.1	5,080	1	1.3E−04	5.0E−07
Corn	0.1	10,161	1	2.5E−04	1.0E−06
Corn	77	5,080	1	9.6E−02	3.9E−04
Corn	77	10,161	1	1.9E−01	7.7E−04
Hay	0.1	2,032	1	5.0E−05	2.0E−07
Hay	0.1	5,080	3	3.8E−04	1.5E−06
Hay	77	2,032	1	3.9E−02	1.5E−04
Hay	77	5,080	3	2.9E−01	1.2E−03
Small grains	0.1	2,032	1	5.0E−05	2.0E−07
Small grains	0.1	5,080	3	3.8E−04	1.5E−06
Small grains	77	2,032	1	3.9E−02	1.5E−04
Small grains	77	5,080	3	2.9E−01	1.2E−03
Soybeans	0.1	5,080	1	1.3E−04	5.0E−07
Soybeans	0.1	20,321	1	5.0E−04	2.0E−06
Soybeans	77	5,080	1	9.6E−02	3.9E−04

Crop	Sludge Concentration (mg/kg) [a]	Application Rate (kg/acre) [b]	Frequency ($year^{-1}$) [b]	Surface Concentration (mg/m^2)	Topsoil Concentration (mg/kg) [c d]
Soybeans	77	20,321	1	3.9E−01	1.5E−03

[a] Source: Targeted National Sewage Sludge Survey Sampling and Analysis Technical Report (Data Quality: High Confidence) (U.S. EPA, 2009)

[b] Source: 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/yd^3 (1,500 kg/m^3) was selected from NRCS Soil Quality Indicators (USDA NRCS, 2008)

Using the generic application scenarios and biosolids concentrations collected from national surveys, the typical concentration of DIBP in biosolids may vary significantly depending largely on the source material and method of application. The surface loading rate for spray or near surface injection applications range from 5×10^{-5} to 0.4 mg/m^2, while mixing applications (assuming a 7-inch [18-cm] tilling depth) may range from 2×10^{-7} to 0.002 mg/m^3 depending on the application rate, frequency, and applied biosolids concentration.

Once in the soil, DIBP is expected to have a high affinity to soil (log K_{OC} = 5.5) and organic media (log K_{OW} = 4.34) which would limit mobility from biosolids or biosolid amended soils. Similarly, high sorption to particulates and organics would likely lead to high retardation, which would limit infiltration to and mobility within surrounding groundwater systems. DIBP is slightly soluble in water (6.2 mg/L) and has limited potential to leach from biosolids and infiltrate into deeper soil strata. However, it is not expected to migrate as far as groundwater given the minimum depth to groundwater required for biosolids agricultural applications stated in 40 CFR part 503. Because DIBP does have high hydrophobicity and a high affinity for soil sorption, it is unlikely that DIBP will migrate from potential biosolids-amended soils via groundwater infiltration. DIBP has been detected in surface runoff originating from landfills containing DIBP (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 reasonably available related to the uptake and bioavailability of DIBP in land applied soils. DIBP'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 DIBP. Based on the solubility (6.2 mg/L) and hydrophobicity (log K_{OW} = 4.34; log K_{OC} = 5.5), DIBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of DIBP into crops planted in DIBP containing soils found that DIBP was not found in any of the plant tissues (*i.e.*, roots, shoots, leaves) resulting from the uptake via soil or water. DBP, a DIBP 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). The bioaccumulation factor (BAF) and bioconcentration factor (BCF) were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 1.48 to 1.66 (upper-lower trophic levels) and log BAF ranging from 1.48 to 1.66 (upper-lower trophic levels) (U.S. EPA, 2017a).

There is limited measured data on concentrations of DIBP 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 DIBP 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 DIBP in biosolids. There is currently no direct evidence that biosolids containing DIBP are being consistently applied to 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. Because there is limited data that any biosolids containing DIBP are being consistently applied to biosolids, there is similarly very limited direct evidence that DIBP is present in agricultural products or subsequently that the general populus may be regularly exposed to DIBP resulting from the applications of biosolids to agricultural fields.

The limited research that does exist suggests that DBP present in biosolid amended soils will likely not be absorbed by any plants or crops growing in the soil. Although field and experimental data are limited, soil dwelling organisms may be exposed to DBP through soils that have been amended with DBP containing biosolids applied as fertilizers but are not expected to readily accumulate DBP through ingestion or absorption.

There is robust confidence that DIBP in soils will not be mobile and will have low persistence potential due to the high confidence in the biodegradation rates and physical and chemical properties. The existing literature suggests that DIBP present in biosolid amended soils will likely not be absorbed by any plants or crops growing in the soil. Although experimental data are limited, soil dwelling organisms may be exposed to DIBP in biosolid amended soils but will not bioaccumulate the chemical.

3.2 Landfills

Landfills are a potential source of chemicals in the environment. DIBP may be deposited into landfills through various waste streams including consumer waste, residential waste, industrial waste, and municipal waste including dewatered wastewater biosolids. This qualitative assessment reviewed reasonably available information using EPA's systematic review process with overall data quality ratings of high as well as transport and fate properties to understand potential exposures from landfills.

No studies were identified through systematic review that could provide the concentration of DIBP in refuse or waste in the United States. No TRI data was reported on releases of DIBP into landfills or from recycling facilities. One 1997 German study did, however, examine the presence of five other phthalates (*i.e.* DMP, DEP, DBP, BBP, DEHP) in residential waste. The five phthalates were shown consistently in residential mixed refuse with the highest concentrations of phthalates typically in "compound materials", "8–40 mm" fragmented plastics, and "Other plastics" with an estimated total phthalate composition ranging from 190.2 to 1,599.3 mg/kg dw residential mixed refuse. Although the five phthalates are often used in similar applications, concentrations of DIBP cannot be asserted from the presented study for a quantified analysis and instead only demonstrates the general presence of phthalates in mixed residential waste.

No studies were identified that reported the concentration of DIBP in landfills or in the surrounding land. There is limited information regarding DIBP in dewatered biosolids, which may be sent to landfills for disposal. As previously noted, DIBP has been identified in wastewater sludge in the United States and Canada (Ikonomou et al., 2012), as well as at various facilities across China (Zhu et al., 2019; Meng

et al., 2014). A 2012 high-quality survey of North American wastewater plants (Canada and United States) identified DIBP in sludge at concentrations ranging from 0.1 to 76.7 dw (Ikonomou et al., 2012). Wastewater sludge and waste may be one source of DIBP relevant to landfills.

DIBP is capable of leaching from bioreactors simulating landfill conditions using residential waste. One 1997 study evaluating a variety of phthalates leaching from 50 kg of unaltered mixed refuse over 90 days reported an overall leaching potential of 1.1 g of total phthalates per ton of normal mixed refuse (Bauer and Herrmann, 1997). The 1997 study did not expressly evaluate for DIBP and is not being used to quantify concentrations of DIBP in landfill leachate but does demonstrate that phthalates with similar physical and chemical properties to DIBP may leach from refuse where they are present. No studies have directly evaluated the presence of DIBP in landfill or waste leachate. However, DIBP is expected to have a high affinity to particulate (log K_{OC} = 5.5) and organic media (log K_{OW} = 4.34) that 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 DIBP will migrate from landfills after groundwater infiltration. Nearby surface waters, however, may be susceptible to DIBP contamination via surface water runoff if it is not captured before interacting with surface water.

Although persistence in landfills has not been directly measured, DIBP can undergo abiotic degradation via carboxylic acid ester hydrolysis to form 2-isobutyl phthalate (major product) and 2-ethylhexyl phthalate (minor product) (U.S. EPA, 2024a). Hydrolysis is not expected to be a significant degradation pathway in landfills, with an estimated half-life for DIBP of 5.3 years under standard environmental conditions (at pH 7 and 20 °C) (U.S. EPA, 2017a).

To further understand potential transport and subsequent exposure from this setting, landfills are divided into two zones: (1) “upper-landfill” zone with normal environmental temperatures and pressures (*i.e.* 1 atm, 20–25 °C, aerobic conditions), where biotic processes are the predominant route of degradation for DIBP; 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 can still be present in the subsurface, conditions may still be favorable for aerobic biodegradation. 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 anaerobic biodegradation of DIBP. 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., 2013).

Temperature in lower landfills, however, often exceed 70 °C in very complex matrices. In such matrices, temperature, pressure, ionic strength, and chemical activity can 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.

In both the upper and lower layers of a landfill, DIBP is shielded from light and photolysis is not considered a significant abiotic degradation pathway. In the lower landfill, high temperatures (>60 °C) and low water content can partially or completely inhibit biological degradation (Huang et al., 2013). Aerobic and anaerobic degradation of DIBP has not been directly measured *in situ* in landfills or in landfill leachate. Aerobic degradation of DBP; however, has been measured experimentally in several high-quality studies. DIBP is readily degradable in aerobic soil conditions with a half-life ranging from 39 to 65 days (88–97% removal in 200 days) (Yuan et al., 2002). DIBP is less likely to be degraded

under anaerobic conditions such as those that would exist in lower landfills. While anaerobic biodegradation of DIBP has not been directly recorded, anaerobic biodegradation of DIBP in soil has been measured with a half-life exceeding 252 days (0–30% removal in 90 days) (NCBI, 2020). In landfills with high leachate production, DIBP may be more persistent with areas saturated with leachate, such as the lowest sections of the landfill adjacent the impermeable clay or geotextile liners, where temperatures often exceed the habitable zones for most microorganisms capable of degrading DIBP.

DIBP's sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms adjacent to landfills. DIBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of DIBP into crops planted in DIBP containing soils found that DIBP was not found in any of the plant tissues (*i.e.*, roots, shoots, leaves) resulting from the uptake via soil or water. DIBP 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 in one high-quality study (Müller and Kördel, 1993).

BAF and BCF were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 1.48 to 1.66 (upper-lower trophic levels) and log BAF ranging from 1.48 to 1.66 (upper-lower trophic levels) (U.S. EPA, 2017a).

3.2.1 Weight of Scientific Evidence Conclusions

Information on the presence of DIBP in landfills is limited. This scarcity of information may be attributed to a lack of focused efforts to measure the chemical in landfills of the United States.

There is uncertainty in the relevancy of the landfill leachate monitoring data to the COUs considered in this evaluation. Although 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. While the data demonstrates that DBP may exist in and leach from landfill refuse, there is uncertainty as to if the presented study accurately reflects the current state of DIBP in refuse and landfill 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 DIBP will be persistent in landfills. There is currently no direct evidence that the general populus or surrounding fauna have been directly exposed to DIBP through refuse or waste disposed of through landfills. Although possible, there has been no data to suggest that DIBP is present in environmental compartment adjacent to landfills as the direct result of landfill operations.

DIBP is unlikely to be present in landfill leachates and migrate through groundwater. While experimental data are limited, soil dwelling organisms may be exposed to DIBP in amended soils but will not bioaccumulate the chemical due to landfill disposal of biosolids or refuse. Similarly, the existing literature suggests that DIBP present in landfills will not be accumulated by living plants. EPA has high confidence in these conclusions based on the qualitative assessment of DIBP in landfills and review of readily available literature.

4 SURFACE WATER CONCENTRATION

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of DIBP in ambient surface water and aquatic sediments. Though the available monitoring data were limited, DIBP was detected in surface water, finished drinking water, and aquatic sediments. However, EPA cannot correlate monitoring levels with any releases associated with DIBP TSCA COUs. In addition, DIBP is not a listed priority pollutant in the Clean Water Act and is not reported in EPA's permit database as a monitored pollutant within the National Pollutant Discharge Elimination System (NPDES). That is, EPA does not have any facility-specific DIBP release data since facilities do not report releases of DIBP to surface waters from TSCA COUs to EPA programs. Therefore, EPA estimated the releases to surface water as described in *Draft Environmental Release and Occupational Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025e). Using these release estimates, EPA conducted modeling to assess the expected resulting environmental media concentrations from the 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 with EPA's Variable Volume Water Model (VVWM) in the Point Source Calculator tool (PSC) (U.S. EPA, 2019c) to estimate concentrations of DIBP within surface water and sediment resulting from TSCA COU releases. PSC considers model inputs of physical and chemical properties of DIBP (*e.g.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and estimates DIBP releases to water (U.S. EPA, 2025e), which are used to predict receiving water column concentrations. PSC was also used to estimate DIBP concentrations in settled 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. Physical and chemical properties of the chemical itself also influence partitioning and half-lives into environmental media. DIBP has a log K_{OC} of 3.14, 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 and chemical, and 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 Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h). A half-life based on anaerobic sediment was selected for the benthic half-life input as a more protective value than the aerobic sediment value, and in consideration of the potential for lower levels of oxygen in benthic sediments impacted by industrial releases. In addition to the values described in the *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h), 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[a]
Koc	1,380 mL/g
Water column half-life	14 days at 25 °C
Photolysis half-life	1.15 days at 30N
Hydrolysis half-life	1,934.5 days at 25 °C
Benthic Half-life	100 days at 25 °C
Molecular weight	278.35 g/mol
Vapor pressure (torr)	0.0000476
Solubility	6.2 mg/L at 25 °C
Henry's Law constant	0.000000183 atm m^3/mol at 25 °C
Heat of Henry	45,727 J/mol
Reference temperature	25 °C
[a] Selected values for these parameters are described in the *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h)	

A common setup for the model environment and media parameters were 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. Standard 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 differences in COU scenarios.

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/cm^3
Benthic f_{oc}	0.04
Benthic DOC	5.0 mg/L
Benthic biomass	0.006 g/m^2
Mass transfer coefficient	0.00000001 m/s

A required input for the PSC model is the hydrologic flow rate of the receiving water body. Since there were no reported data from available sources (*e.g.,* TRI and Discharge Monitoring Report [DMR]), EPA used modeling approaches to assess releases of DIBP to water for all OESs (U.S. EPA, 2025e). Without TRI and DMR data, EPA cannot identify the receiving water bodies and their location-specific hydrological flow data. Thus, EPA 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 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. This modeled distribution of hydrological flow data are specific to an industry sector rather than a facility but provides a reasonable estimate of the distribution of location-specific values. The complete methods for retrieving and processing flow data by NAICS code are detailed in Appendix B.

A number of hydrologic flow rates were estimated from the distribution to represent higher and lower flows and to therefore capture a range of corresponding surface water concentrations. The 30Q5 flows (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 (lowest 7-day average flow that occurs in a 10-year period) is used to estimate exceedances of concentrations of concerns 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.

Plastic compounding 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. Although the generic release scenario for Plastic compounding OES had releases estimated to multiple media types (Wastewater to onsite treatment, discharge to POTW (with or without pretreatment), direct to surface water, incineration, or landfill), it also had releases that were water specific and categorized as wastewater to onsite treatment, discharge to POTW (with or without pretreatment). Therefore, EPA was able to estimate surface water concentrations for Plastic compounding OES from releases known to discharge to water.

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 4-3 and Table 4-4 presents the surface water concentrations associated with the Plastic compounding 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. The total days of release value associated with the Plastic compounding OES was applied as continuous days of release per year as a conservative approach (*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). The highest water column concentration averaged over the number of release days (*i.e.,* 250)

was used to estimate general population and aquatic exposure. Appendix B describes the methods to calculate the rolling averages.

When considering the release quantity for the Plastic compounding OES discharging into an unknown water body, EPA considered the relationship between the magnitude of loading from facilities and the flow in the receiving waterbody. Because facility-specific data on releases and receiving waterbodies were not available to the Agency for DIBP, releases of the phthalates DEHP and DBP were examined. Across the facilities with known release data from various OES, but predominantly Plastic compounding, there appears to be a general trend as shown in Figure 4-1 of larger amounts of loading being released to larger receiving waterbodies. Due to the uncertainty associated with the receiving waterbody for the generic scenario, EPA models the Plastic compounding OESs with the P50, P75, and P90 flows from the distribution of receiving waterbodies developed as a generic distribution. Due to the assumed release for the Plastic compounding OES being orders of magnitude greater than any of the known releases reported to EPA programs, and the assumption of the continuation of this trend of larger releases tending to be released in larger waterbodies, the Agency has greater confidence in the higher percentile (*i.e.*, P75 and P90) flows from the generic distribution as being more representative of the expected receiving waterbody for this high-end release scenario.

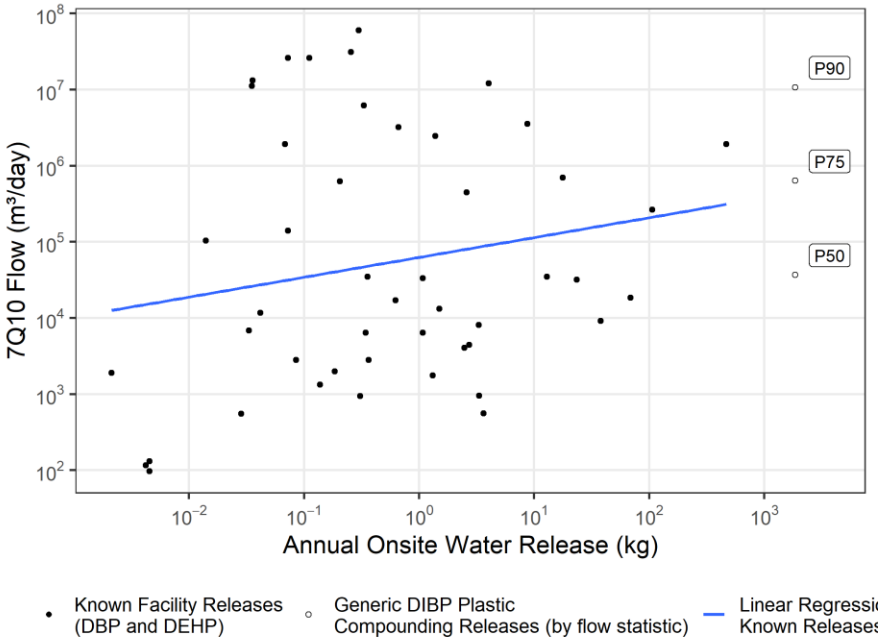


Figure 4-1. Plot of Known Phthalate Facility Releases of DEHP and DBP with High-End Generic Scenario Estimations for DIBP

The modeled releases were evaluated for resulting environmental media concentrations at the point of discharge (*i.e.*, in the immediate receiving waterbody receiving the effluent). Due to uncertainty about the prevalence of wastewater treatment from DIBP-releasing facilities, all modeled releases were assumed to be released to surface water without treatment. However, due to the partitioning of the compound to sediment, wastewater treatment is expected to be highly effective at removing DIBP from the water column prior to discharge, with treated effluent showing up to a 96.7 percent reduction in one study (Tran et al., 2014), and a typical removal efficiency of 68 percent found in an EPA study (U.S. EPA, 1982). Water column, pore water and benthic sediment concentration estimates assuming the 7Q10 low hydrologic flow are presented in Table 4-3. This analysis resulted in high estimated DIBP concentrations in the receiving waterbody and sediment because of a high-end release amount combined

with lower hydrologic flow and without consideration of wastewater treatment. These values are carried through to the ecological risk assessment for further evaluation as a conservative high-end approach to screen for ecological risk discussed in Section 12.

Table 4-3. Water and Benthic Sediment in the Receiving Waterbody, Applying a Median 7Q10 Flow

OES	Number of Operating Days Per Year	Daily Release (kg/day) [a]	Median 7Q10 Total Water Column Concentration (µg/L)	Median 7Q10 Benthic Pore Water Concentration (µg/L)	Median 7Q10 Benthic Sediment Concentration (µg/m³)
Plastic compounding	250	8.44	228	175	9,760

[a] Details on operating days and daily releases are provided in the *Draft Environmental Release and Occupational Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025e).

The OES with the highest total water column concentrations (Plastic compounding) was also run under the median harmonic mean and 30Q5 flow conditions (Table 4-4). These additional results were selected to screen for risks to human health. Two scenarios were run for this high-end release: one without any wastewater treatment applied to reduce DIBP concentrations (as in the modeling shown previously in this section), and another with a wastewater treatment removal efficiency of 68 percent applied (U.S. EPA, 1982), reducing the modeled concentrations in the receiving waterbody. The DIBP surface water concentration after application of the removal efficiency represents the likely human exposure to DIBP in drinking water, as drinking water treatment systems are anticipated to be effective in removing DIBP adhered to suspended solids.

Table 4-4. PSC Modeling Results for Total Water Column, Applying a Median Harmonic Mean Flow and a Median 30Q5 Flow

Scenario	Release Estimate (kg/day)	Median Harmonic Mean Flow (m³/d)	Median 30Q5 Flow (m³/d)	Removal Efficiency Applied (%)	Harmonic Mean Concentration (µg/L)	30Q5 Concentration (µg/L)
Plastic compounding *Without wastewater treatment*	8.44	69,800	48,600	0.00	121	174
Plastic compounding *With wastewater treatment*	8.44	69,800	48,600	68	38.7	55.7

4.2 Measured Concentrations

4.2.1 Measured Concentrations in Surface Water

EPA identified monitoring studies through systematic review to provide context to modeling results. The monitoring studies presented here were not used as part of the analysis for quantifying exposure

estimates. One study was identified from the United States that examined DIBP in surface water (Liu et al., 2013) (Table 4-5). 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 six sites located about 1 mile apart. DIBP was detected in 83 percent of these samples with concentrations ranging from non-detect to 3.3 µg/L. Fifty-four samples were also collected from the central lake area at six sites located near Lake Maurepas to the Causeway Bridge, with one site near the Manchac Pass. DIBP was detected in 67 percent of these samples with concentrations up to 0.69 µg/L.

For the central lake area, authors reported that concentrations of phthalates 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 a 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).

Four additional studies, three from France and one from South Korea, reported levels of DIBP 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 that mean DEHP levels were highest among seven phthalates, followed by DIBP at 743 ng/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. DIBP was detected once at a concentration of 1,650 ng/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. DIBP was detected in all samples with a mean concentration of 37.3 ng/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. It is likely affected by pollution coming from an industrial complex and two nearby cities. Forty-seven surface water samples were collected at 12 sampling locations. DIBP was detected in approximately 26 percent of samples at a mean concentration of 0.01 µg/L.

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

Reference	Sampling Location	DIBP Concentrations	Sampling Notes	Study Quality Rating
Liu et al. (2013)	United States	Bonnet Carré Spillway (6 locations; n = 42) FOD: 83%, <0.05–3.3 µg/L Central lake area (6 locations; n = 54) FOD: 67%, <0.05–0.69 µg/L	Freshwater samples from Lake Pontchartrain, LA, before, during, and after opening of the Bonnet Carré Spillway that occurred April/May 2008, March 2008–June 2009	Medium
Valton et al. (2014)	France	FOD and sample number NR Mean ± SD = 743 ± 470 ng/L	Freshwater samples from the outlet of the Orge River basin, date NR	Medium
Bach et al. (2020)	France	FOD = 0.88% (calculated) (n = 114), <150–1,650 ng/L LOQ = 150 ng/L	National screening study to examine phthalates in raw surface water (prior to treatment for use as drinking water), November 2015–July 2016	High
Schmidt et al. (2020)	France	FOD 100% (n = 22) Median, mean ± SD (range) = 39.7, 37.3 ± 21.6 (11.1–78.2) ng/L LOQ = 0.03 ng/L	Monthly Rhône River samples, May 2017–April 2018	High
Lee et al. (2019)	South Korea	FOD 25.5% (n = 47) Mean (range) = 0.01 (ND [a] to 0.07) µg/L	Freshwater samples from Asan Lake collected at 12 sampling locations, 2016–2017	High

FOD = frequency of detection; ND = non-detect; NR = not reported; SD = standard deviation; LOD = limit of detection; LOQ = limit of quantification
[a] A value of 0 was used for non-detects; LOD and LOQ were 0.01 and 0.02 µg/L, respectively.

4.2.2 Measured Concentrations in Sediment

EPA identified monitoring studies through systematic review to provide context to modeling results. The monitoring studies presented here were not used as part of the analysis for quantifying exposure estimates. EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of DIBP in sediment. Washington State Department of Ecology data were obtained through the Water Quality Portal (WQP) (NWQMC, 2021), which houses publicly available water quality data from the United States Geological Survey (USGS), EPA, and state, federal, tribal, and local agencies. All results obtained from the WQP were non-detects. No other references for the United States or Canada published after 2012 were available.

Table 4-6 presents DIBP levels in sediment from one study conducted in South Korea. 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. Forty-seven sediment samples were collected at 12 sampling locations. DIBP was detected in approximately 19 percent of samples at a mean concentration of 3 µg/kg dw.

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

Reference	Sampling Location	DIBP Concentrations	Sampling Notes	Study Quality Rating
Lee et al. (2019)	South Korea	FOD 19.1% (n = 47) Mean (range) = 3.0 (ND*–43.2) µg/kg dw *A value of zero was used for non-detect. LOD and LOQ were 1.32 and 3.97 µg/kg dw, respectively.	Sediment samples from Asan Lake collected at 12 sampling locations, 2016–2017	High
dw = dry weight; FOD = frequency of detection; ND = non-detect				

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 DIBP in surface water and sediment using modeled release amounts and estimated receiving waterbody flow rates from a distribution of known releasing facilities. PSC considers model inputs of physical and chemical properties of DIBP (*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and allows EPA to model predicted sediment concentrations in addition to water column concentrations. The use of physical and chemical properties of DIBP refined through the systematic review process and supplemented by EPA models increases confidence in the application of the PSC model. A standard EPA waterbody was used to represent a consistent and conservative receiving waterbody scenario. Uncertainty associated with location-specific model inputs (*e.g.*, flow parameters and meteorological data) is present as no facility locations were identified for DIBP releases and modeled values for DIBP release to surface water were used. EPA has moderate confidence in the estimated releases from facilities to surface water which were applied as inputs to the surface water modeling conducted in this assessment.

The modeled data represent estimated surface water (water column, benthic porewater, and sediment) concentrations near facilities that would be releasing DIBP to surface water. Because the release of DIBP to surface water is expected, but the specific locations and amounts of releases are unknown, the release scenarios were estimated using the data available to EPA. The reported measured concentrations represent sampled ambient water concentrations of DIBP. However, these monitored concentrations are not necessarily tied to TSCA COUs, and the origin of these concentrations are unknown and could represent aggregation of multiple sources. Furthermore, the measured data may not represent locations where the general population may be exposed, either incidentally or via drinking water. Measured DIBP 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 spatially or temporally associated with releases of DIBP. In addition, when modeling with PSC, EPA considered the generic scenario releases directly discharged to surface waters both with and without prior treatment. A generic removal efficiency is applied when treatment is considered. EPA recognizes that the untreated scenario is a conservative assumption that results in no removal of DIBP prior to release to surface water.

Concentrations of DIBP within the sediment were estimated using the high-end release estimates from generic scenarios and estimates of 7Q10 hydrologic flow data for the receiving water body that were

derived from NHD-modeled EROM flow data. 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. Surrogate flow data collected via 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. 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. However, there is uncertainty in how representative the median flow rates are as applied to the facilities and COUs represented in the DIBP release modeling. Additionally, a regression-based calculation was applied to estimate flow statistics from NHD-acquired flow data, which introduces some additional uncertainty. EPA assumes that the results presented in this section include a bias toward over-estimation of resulting environmental concentrations due to conservative assumptions that remain protective where there are uncertainties in release details.

4.4 Weight of Scientific Evidence Conclusions

Due to the lack of reported release data for facilities discharging DIBP to surface waters, releases were modeled, and the high-end estimate for each COU was applied for surface water modeling. Additionally, due to the lack of site-specific release information, a generic distribution of hydrologic flows was developed from facilities which had been classified under relevant NAICS codes, and which had NPDES permits. Due to the lower flow rates selected from the generated distributions, coupled with high-end release scenarios, EPA has slight to moderate confidence in the modeled concentrations as being representative of actual releases, with a slight bias toward over-estimation based on the many conservative assumptions including no wastewater treatment, all releases directly to surface water, and the pairing of low flow scenarios with high releases. Additionally, the Agency has robust confidence that no surface water release scenarios result in water concentrations which exceed the concentrations presented in this evaluation due to the conservative assumptions used. Other model inputs were derived from reasonably available literature collected and evaluated through EPA's systematic review process for TSCA risk evaluations. All monitoring and experimental data included in this analysis were from articles rated as medium- or high-quality from this process.

The high-end modeled concentrations in the surface water and sediment exceeded the highest values available from monitoring studies by more than an order of magnitude. This confirms EPA's expectation that modeled concentrations presented herein are biased toward overestimation and are appropriate to be used as a screening evaluation.

5 SURFACE WATER EXPOSURE TO GENERAL POPULATION

Concentrations of DIBP in surface water 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, 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 using the highest concentration of DIBP in surface water based on the highest releasing OES (Plastic compounding) as estimated in Section 4.1 for various lifestages (*e.g.*, adult, youth, children).

5.1 Modeling Approach

5.1.1 Dermal Exposure

The general population may swim in surface waters (streams and lakes) that could be affected by DIBP 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

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

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 dermal permeability coefficient (K_p) of 0.016 cm/hr (U.S. EPA, 2025b). EPA used the Consumer Exposure Model (CEM) Version 3.2 (U.S. EPA; ICF Consulting, 2022) to estimate the steady-state aqueous permeability coefficient of DIBP.

Table 5-1. shows a summary of the estimates of ADRs and ADDs due to dermal exposure while swimming for adults, youth, and children. Dermal doses were calculated with Equation 5-1 and Equation 5-2 using the highest surface water concentration from the Plastic compounding OES. Dose values are presented both with and without a wastewater treatment removal efficiency of 68 percent applied. As details of the releasing facilities and their treatment technologies are not readily available, this hypothetical treated concentration is included for reference, and exposure screening is primarily conducted with the high-end untreated release estimate. Dermal doses were also calculated using the highest values from ambient surface water monitoring data (Section 4.2.1) as the surface water concentration. Doses calculated using the surface water monitoring data are up to two orders of magnitude lower than corresponding doses modeled using the high-end Plastic compounding OES.

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)
Plastic compounding[b] *without wastewater treatment*	174	121	2.04E−03	8.49E−04	1.56E−03	6.50E−04	9.46E−04	3.95E−04
Plastic compounding[b] *with wastewater treatment*	55.7	38.7	6.51E−04	2.72E−04	4.99E−04	2.08E−04	3.03E−04	1.26E−04
High from monitored surface water[c]	3.30	3.30	3.86E−05	2.64E−05	2.96E−05	2.02E−05	1.79E−05	1.23E−05

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.
[c] Liu et al. (2013) reported the highest monitored surface water concentration from Louisiana, the only U.S. study, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations. It is important to note that monitored concentrations do not distinguish between sources and cannot be correlated to any TSCA COUs.

5.1.2 Oral Ingestion Exposure

The general population may swim in surfaces waters (streams and lakes) that could be affected by DIBP 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 Plastic compounding OES that resulted in the highest modeled surface water concentrations:

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 used 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)
Plastic compounding[a]	174	121	6.00E−04	2.50E−04	9.31E−04	3.89E−04	5.25E−04	2.19E−04

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)
without wastewater treatment								
Plastic compounding[a] *with wastewater treatment*	55.7	38.7	1.92E−04	8.02E−05	2.98E−04	1.24E−04	1.68E−04	7.01E−05
High from monitored surface water[b]	3.3	3.3	1.14E−05	2.27E−05	1.77E−05	3.52E−05	9.96E−06	1.99E−05

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.
[b] Liu et al. (2013) reported the highest monitored surface water concentration from Louisiana, the only U.S. study, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations. It is important to note that monitored concentrations do not distinguish between sources and cannot be correlated to any TSCA COUs.

5.2 Weight of Scientific Evidence Conclusions

No facility- or site-specific information was reasonably available when estimating release of DIBP to the environment. Environmental releases to water were estimated using generic scenarios (U.S. EPA, 2025e). Due to uncertainties inherent in this approach, conservative assumptions and methods were utilized to evaluate an upper-bounding limit to be applied as a protective screening assessment. As stated in Section 4.4, there is moderate confidence in the modeled concentrations as being representative of actual releases, with a bias toward over-estimation. Screening level risk estimates derived from the exposures modeled in this section are discussed in Appendix C and demonstrate no risk estimates to 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 for two routes of exposure (youth being exposed dermally as well as through incidental ingestion while swimming in surface water) were assessed as high-end potential exposures to DIBP in surface waters. EPA's *Exposure Factors Handbook* provided detailed information on the youth skin surface areas and events per day of the various scenarios (U.S. EPA, 2017b). Non-diluted surface water concentrations (*i.e.*, dilution was only considered for receiving water at the point of discharge as opposed to downstream dilution) were used when estimating dermal exposures to youth swimming in streams and lakes. DIBP concentrations will dilute when released to surface waters, but it is unclear what level of dilution will occur when members of the general population swim in waters with DIBP releases.

6 DRINKING WATER EXPOSURE TO GENERAL POPULATION

Drinking water in the United States typically comes from surface water (*e.g.,* lakes, rivers, reservoirs) and groundwater. The source water then flows to a treatment plant where it undergoes a series of water treatment steps before being distributed to homes and communities. Public drinking water systems often use a combination of treatment processes that include coagulation, flocculation, sedimentation, filtration, and disinfection to meet drinking water quality standards. The exact treatment processes used to meet drinking water quality standards differ between systems.

Very limited information is reasonably available on the removal of DIBP in drinking water treatment plants. As stated in the *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate* (U.S. EPA, 2025h), no data were identified by the EPA for DIBP in drinking water in the United States. Based on the low water solubility and log K_{OW}, DIBP 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 DIBP 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. As a screening analysis, the highest modeled facility release was included in the drinking water exposure analysis, alongside the highest monitored surface water concentration. A representative wastewater treatment efficiency of 68 percent was applied (U.S. EPA, 1982). This treatment is assumed to occur at the facility prior to effluent discharge to the receiving waterbody and prior to becoming influent at a downstream drinking water treatment plant. No further drinking water treatment is considered, which is expected to be a conservative scenario 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 (ppb or µg/L; 30Q5 conc for ADR, harmonic mean for ADD, LADD, LADC)
DWT	=	Removal during drinking water treatment (assume 0% for DIBP)
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 (ppb or µg/L; 30Q5 conc for ADR, harmonic mean for ADD, LADD, LADC)
DWT	=	Removal during drinking water treatment (assume 0% for DIBP)
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 effects 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 DIBP (see *Draft Non-Cancer Human Health Hazard Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025g)). Therefore, EPA is not evaluating DIBP for carcinogenic risk. Table 6-1 summarizes the drinking water doses for adults, infants, and toddlers for a scenario applying no wastewater treatment and another scenario applying wastewater treatment. Exposure estimates are low for all lifestages and scenarios, including for infants with the highest drinking water intake per body weight and assuming no wastewater treatment is applied.

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)
Plastic compounding[a] w*ithout wastewater treatment*	174	121	7.00E−03	7.98E−04	2.46E−02	2.04E−03	8.74E−03	8.74E−04
Plastic compounding[a] *with wastewater treatment*	55.7	38.7	2.24E−03	2.55E−04	7.86E−03	6.52E−04	2.80E−03	2.80E−04
Highest monitored surface water[b]	3.3	3.3	1.33E−04	3.80E−05	4.66E−04	9.71E−05	1.66E−04	4.16E−05

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.
[b] Liu et al. (2013) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored 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 DIBP in drinking water. No references for the United States or Canada published after 2012 were identified, and monitoring of DIBP is not currently federally required under the Safe Drinking Water Act. Table 6-2 presents DIBP 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. DIBP was detected in a single sample at approximately 1,300 ng/L. In a second study conducted in Romania in 2017 (Sulentic et al., 2018), phthalates were measured in municipal drinking water and consumed bottled water. Ten tap water samples and 16 bottled water samples that represented unique combinations of brand, type (still or gas), and storage conditions (room temperature or refrigerated) were collected and analyzed for four phthalates. Phthalates were generally not detected in the tap water samples except for two samples with DIBP (0.084 and 0.104 µg/L). Overall, the median level of DIBP in bottled water was 0.77 µg/L. Still water (1.36 µg/L) had a higher median concentration of DIBP than gas water (0.48 µg/L). Bottled water at room temperature (2.23 µg/L) had a higher median concentration of DIBP than bottled water that was refrigerated (0.41 µg/L).

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

Reference	Sampling Location	DIBP Concentrations	Sampling Notes
Bach et al. (2020)	France	FOD = 0.4% (n = 283) **Level by supply type (ng/L):** Surface water (n = 89): <150–1,296 Groundwater (n = 166): <150 Mixture of surface and groundwater (n = 28): <150 LOQ = 150 ng/L	National screening study to examine phthalates in treated tap water, November 2015–July 2016
Sulentic et al. (2018)	Romania	**Tap (n = 10) (µg/L):** FOD 20%*, median (IQR) = ND (ND, ND), <0.015–0.104 **Bottled (n = 16) (µg/L):** FOD = NR, median (IQR) = 0.77 (0.25, 2.50) LOD = 0.015 µg/L *Calculated	Tap water and bottled water samples collected as part of an exposure assessment in Romanian adolescents, 2017

FOD = frequency of detection; IQR = interquartile range; LOQ = limit of quantification; ND = non-detect; NR = not reported; LOD = level of detection

6.3 Evidence Integration for Drinking Water

Based on modeling of the estimated releases, EPA estimates little to no potential exposure to DIBP via drinking water, even under conservative 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. While recent monitoring data in the United States were not identified, available finished drinking water concentrations reported from France were less than 1.3 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 D, and no screening level risk estimates were below the benchmark MOE at the upper-bound of exposure.

6.4 Weight of Scientific Evidence Conclusions

EPA has moderate to high confidence in surface water as drinking water not being a pathway of concern for the general population due to the high-end screening approach with protective assumptions presenting an upper-bound of exposure in which risk does not exceed 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 two surface water concentrations in its assessment: (1) the water solubility of 6.20 mg/L (U.S. EPA, 2025h), and (2) the maximum modeled surface water concentration. The range of water solubility values measured at ambient temperature is 5.1 to 9.6 mg/L, and the selected value best represents DIBP's mean water solubility limit under normal environmental conditions. If refinements to the screening level analysis are needed, EPA used the maximum modeled surface water concentration associated with Plastic compounding (1.21×10^{-1} mg/L). This value corresponds to the harmonic mean based on the highest modeled 95th percentile release, 50th percentile flow metric distribution (P50), and no consideration of wastewater treatment. It represents the upper-bound of DIBP concentration in receiving waters that is OES-specific.

Another important parameter in estimating human exposure to a chemical through fish ingestion is the bioaccumulation factor (BAF). BAF is preferred over the bioconcentration factor (BCF) because it considers the animal's uptake of a chemical from both diet and the water column. However, for DIBP, the estimated BAF and BCF values using the Arnot-Gobas method for upper trophic organisms are both 30.2 L/kg (see *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h)). Table 7-1 compares the fish tissue concentration calculated using the different surface water concentrations and BAF/BCF with the measured fish tissue concentrations obtained from literature. The measured concentrations identified through systematic review were only used to provide context to modeling results and not to quantify exposure estimates. Calculated fish tissue concentrations using the water solubility limit are one to five orders of magnitude higher than empirical values. Calculated fish tissue concentrations using the maximum modeled surface water concentration are up to two orders of magnitude greater than empirical values.

In addition, EPA calculated fish tissue concentrations using the highest measured DIBP concentrations in surface water. As described in Section 4.2.1, the maximum concentration was 3.3 µg/L (3.3×10^{-3} mg/L) from a U.S. study that assessed the spatial distribution of DIBP before, during, and after the opening of the Bonnet Carré Spillway in 2008 (Liu et al., 2013). DIBP was detected in 74 percent of the samples collected from the spillway and Lake Pontchartrain. Fish tissue concentrations calculated with monitored surface water concentrations are higher and lower than empirical values depending on the study of comparison.

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	Predicted BCF or BAF (Arnot-Gobas method) of 30.2 L/kg (U.S. EPA, 2025h)	Estimates of the water solubility limit for DIBP, which is approximately 6.20 mg/L	1.87E02 mg/kg ww
Modeled surface water concentration	Predicted BCF or BAF (Arnot-Gobas method) of 30.2 L/kg (U.S. EPA, 2025h)	1.21E−01 mg/L (harmonic mean, P50 flow distribution, no wastewater treatment)	3.65 mg/kg ww
Monitored surface water concentration	Highest measured concentration from a U.S. study (Liu et al., 2013) and predicted BAF (Arnot-Gobas method) of 30.2 L/kg (U.S. EPA, 2025h)	3.30E−03 mg/L	9.97E−02 mg/kg ww
Fish tissue monitoring data (wild-caught) *Concentrations reported as a dry weight were excluded from this table because conversions to wet weight were not possible.*	1 Canadian study collected 21 samples across 2 different species (McConnell, 2007)	N/A	3.6E−03 to 1.9E−01 mg/kg ww
	1 Chinese study collected 206 fish samples across 17 different species (Hu et al., 2020)		2.2E−02 to 7.77E−01 mg/kg ww
	1 Chinese study collected 69 fish samples across 3 species from 6 sampling sites (Cheng et al., 2018)		1.0E−02 to 5.0E−01 mg/kg ww

ww = wet weight
[a] These studies identified through systematic review that reported measured DIBP concentrations in fish tissue were not used as part of the analysis for quantifying exposure estimates; rather, they are provided to contextualize modeling results. Study quality varied for each study and can be found in the *Draft Data Quality Evaluation Information for General Population, Consumer, and Environmental Exposure for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025c).

7.1 General Population Fish Ingestion Exposure

EPA estimated exposure from fish consumption using age-specific 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 1 to 2 years who have the highest fish IR per kilogram of body weight among all lifestages in this screening level approach.

The ADR and ADD for non-cancer exposure 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 DIBP's carcinogenicity (U.S. EPA, 2025g). Estimated exposure to DIBP from fish ingestion was calculated according to 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 for mg/µg (1.0×10^{-3} mg/µg)
$CF2$	=	Conversion factor for kg/g (1.0×10^{-3} 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 Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025f). The number of 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 BAF are presented in Table 7-2. Corresponding screening level risk estimates are shown in Appendix E.1. 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	Adult ADR (mg/kg-day)	Young Toddler ADR (mg/kg-day)	Adult ADD (mg/kg-day)
Water solubility limit (6.20 mg/L mg/L)	5.20E−02	7.71E−02	1.18E−02

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 apply to only 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 the lack of reasonably available information on fish ingestion rates for the younger lifestages. 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.

Table 7-3 presents multiple exposure estimates for the subsistence fisher. Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark as shown in Appendix E.2. Therefore, EPA refined its evaluation by using modeled surface water concentrations that are based on the highest estimated 95th percentile release for the Plastic compounding OES, as well as the P50 flow metrics from the distribution and without consideration of wastewater treatment. The more refined exposure estimates did not result in risk estimates below the benchmark. Fish ingestion is not expected to be a pathway of concern for the subsistence fisher based on

the risk estimates shown in Appendix E.2 for the refined scenario.

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

Surface Water Concentration and Scenario	ADR/ADD (mg/kg-day)
Water solubility limit (6.20 mg/L)	3.33E−01
Plastic compounding, P50 flow, no wastewater treatment (1.21E−01 mg/L)	6.50E−03
ADD = average daily dose; ADR = acute dose rate	

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. However, EPA quantitatively evaluated only the tribal fish ingestion pathway for DIBP because of data limitations and recognizes that this overlooks many other unique exposure scenarios.

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 old) 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 not much lower 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. 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 tribal fish ingestion rate; 874 g/day (10.9 g/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 of age. 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 population. Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark as shown in Appendix E.3. Therefore, EPA refined its evaluation by using modeled surface water concentrations that are based on the highest estimated 95th percentile release for the Plastic compounding OES, as well as the P50 flow metrics from the distribution and without consideration of wastewater treatment. The more refined exposure estimates did not result in risk estimates below the benchmark. Overall, ingestion of fish potentially contaminated with DIBP is not expected to be 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 IR, Mean	Current IR, 95th Percentile	Heritage IR
Water solubility limit (6.20 mg/L)	5.06E−01	2.04	3.85
Plastic compounding, P50 flow, no wastewater treatment (1.21E−01 mg/L)	9.87E−03	3.98E−02	7.5E−02
Monitored surface water concentration (3.30E−03 mg/L) (Liu et al., 2013)	2.69E−04	1.09E−03	2.05E−03
ADD = average daily dose; ADR = acute dose rate; IR = ingestion rate			

7.4 Weight of the Scientific Evidence Conclusions

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. The water solubility limit resulted in risk estimates below the benchmark for only subsistence fisher and tribal populations (Appendix E.3). EPA refined its analysis by incorporating the Plastic compounding OES with the highest estimated release to surface water. The maximum modeled surface water concentration does not consider wastewater treatment and is based on the P50 flow distribution, which likely overestimates modeled concentrations. EPA expects high-end releases to discharge to surface waters with higher flow conditions like P75 or P90.

Lastly, it is critical to note that DIBP is expected to have low potential for bioaccumulation, biomagnification, and trophic transfer through food webs as described in Section 12. This is supported by the estimated BCF and BAF values of 30.2 L/kg for both, which does not meet the criteria to be considered bioaccumulative (BCF or BAF > 1,000).

As modeled surface water concentrations are biased toward over-estimation and bioconcentration, bioaccumulation, and trophic transfer of DIBP is not expected, EPA has robust confidence that fish ingestion is not a pathway of concern for all populations.

8 AMBIENT AIR CONCENTRATION

EPA considers both modeled and monitored concentrations of DIBP in the ambient air for this draft ambient air exposure assessment. The Agency's modeling estimates both short-term 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 DIBP.

8.1 Approach for Estimating Concentrations in and Deposition from Ambient Air

EPA used previously peer-reviewed methodology for fenceline communities (U.S. EPA, 2022b) to evaluate exposures and deposition via the ambient air pathway for this assessment. This methodology uses the Integrated Indoor/Outdoor Air Calculator (IIOAC) Model to estimate daily-average and annual-average concentrations of DIBP in the ambient air at three distances (*e.g.*, 100, 100–1,000, and 1,000 m) from the releasing facility. IIOAC also estimates dry, wet, and total deposition rates of DIBP from the ambient air to other media (*e.g.*, water and land) at those same distances. IIOAC is a spreadsheet-based tool that estimates outdoor air concentrations and deposition rates 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).

The Agency uses the maximum EPA-estimated daily releases of DIBP across all OES/COUs as direct inputs to the IIOAC Model. These Agency-estimated releases are based on production volumes from facilities that manufacture, process, repackage, or dispose of DIBP as described in the *Draft Environmental Release and Occupational Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025e).

The maximum EPA estimated daily release value for fugitive and stack releases for DIBP was 8.82 kg/site-day and categorized under the Plastic compounding OES with an unknown media of release (could be releases to air, land, water, or incineration, or any combination and could be either fugitive, stack, or any combination). Since the release type is unknown, under the methodology used, EPA assumed the entire release was either all fugitive or all stack releases and models the entire release as each type. Although this assumption captures the highest release of each type possible, it also limits the analysis to exposure from an individual release type rather than both at the same time and may overestimate ambient concentrations of DIBP for either release type.

8.1.1 Release and Exposure Scenarios Evaluated

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

- Release: Maximum Daily Release (kg/site-day)
- Release Dataset: Engineering Estimate (no TRI or NEI release data reported)
- Release Type: Stack and Fugitive
- Release Pattern: Consecutive
- Distances Evaluated: 100, 100 to 1,000, and 1,000 m
- Meteorological Stations:
  - South (Coastal): Surface and Upper Air Stations at Lake Charles, Louisiana
- Operating Scenario: 365 and 216 days/year; 24 hours/day
- 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 as shown in the *Draft Ambient Air Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025a). A description of select outputs relied on in this 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 Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025a). EPA used the highest estimated concentrations 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-averaged and Annual-Averaged IIOAC-Modeled Concentrations at 100 m from Releasing Facility

Source Type	Daily-Average Concentration ($\mu g/m^3$)	Annual-Average Concentration ($\mu g/m^3$)
Fugitive	16.68	15.81
Stack	0.91	0.64
Total	17.59	16.45

The source apportioned wet and dry deposition rates and the total deposition rates for the scenario used in the ecological assessment (U.S. EPA, 2025i) are provided in Table 8-3.

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

Source Type	Total Annual-Average Deposition Rates (g/m^2)		
	Total	Wet	Dry
Fugitive	2.65E−04	2.62E−04	3.81E−06
Stack	5.17E−05	4.98E−05	3.08E−06
Total	3.17E−04	3.12E−04	6.89E−06

8.2 Measured Concentrations in Ambient Air

EPA reviewed published literature as described in the *Draft Systematic Review Protocol for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025j) to identify studies where ambient air concentrations of DIBP were measured. The monitoring studies identified were not used as part of the analysis for quantifying exposure estimates. Rather, they were used to provide context for modeled concentrations.

EPA identified a single Chinese study (Zhu et al., 2016) that measured concentrations of several phthalates including DIBP. A simple plot of the measured concentrations is provided in Appendix F. This study received an overall data quality rating of medium under EPA's systematic review.

Measured concentrations of DIBP in this study were low, generally in the ng/m^3 range. However, whether these data reflect conditions in the United States or TSCA COUs 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, regulations of emissions standards often vary between the United States and foreign countries.

EPA also reviewed EPA's Ambient Monitoring Technology Information Center database but did not find any monitored DIBP concentrations in ambient air (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 DIBP ambient air concentrations were estimated using the maximum EPA estimated daily 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 away), 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.

DIBP did not have any reported releases in the databases EPA typically relies upon for facility reported release data (*e.g.*, TRI or NEI). Therefore, DIBP releases were estimated and used as direct inputs to the IIOAC Model. Any limitations and uncertainties of these estimated releases, as described in the *Draft Environmental Release and Occupational Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025e), 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 present a limitation on the modeled results for DIBP. 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 and number of operating days to calculate daily average releases assumes operations are continuous and releases are the same for each day of operation. This can underestimate short-term or daily exposure and deposition rates because results may miss actual peak release (and associated exposures) if higher and lower releases occur on different days.
As described in Section 8.1, for this ambient air assessment, EPA assumes the entire 8.22 kg/site-day is released to ambient air and is either entirely fugitive or entirely stack releases. This provides a conservative assumption for each individual release type (fugitive or stack) ensuring possible exposure pathways are not missed and is health protective for this screening analysis. However, since EPA assumes the entire release is either fugitive or stack, modeled concentrations and deposition rates for fugitive and stack releases are not additive as they cannot happen at the same time. Nonetheless, EPA still provides a total exposure and deposition rate from both release types as if they occurred at the same time for this screening level assessment. This provides low confidence in the exposure scenario (cannot

occur at same time under assumptions modeled) and an overestimate of ambient concentrations and deposition rates at the evaluated distances. However, if results indicate the total exposure or deposition rate under this scenario still does not indicate an exposure or risk concern, EPA has high confidence that exposure to and deposition rates of DIBP via the ambient air pathway do not pose an exposure or risk concern and no further analysis is needed. If results indicated an exposure or risk concern, the Agency would have low confidence in the results and refine the analysis to be more representative of a real exposure scenario (*e.g.,* only determine exposures and derive risk estimates based on a single release type).

8.4 Weight of Scientific Evidence Conclusions

EPA has slight confidence in the exposure scenario modeled for this assessment since emissions are assumed to be either all fugitive or all stack and are not additive (exposure to fugitive or stack releases cannot occur at the same time under the assumptions modeled) and the Agency still adds results together as if they occur at the same time. EPA has moderate confidence in the IIOAC-modeled results used to characterize exposures and deposition rates since EPA used conservative inputs, considers a series of exposure scenarios under varying operating scenarios, multiple particle sizes, is based on previously peer reviewed methodology, and incorporates recommendations received during previous peer review and public comment. Despite the limitations and uncertainties described in Section 8.3, this screening level analysis presents an upper-bound value from which exposures can be characterized and risk estimates derived. The conservative inputs and assumptions lead to overestimation of exposure and deposition rates, providing a high confidence the exposure estimates are health protective. Based on the results presented here and risk estimates described in the *Draft Risk Evaluation for Diisobutyl phthalate (DIBP)* (U.S. EPA, 2025i), EPA has robust confidence the ambient air pathway is not a pathway of concern for either exposure to or deposition rates of DIBP.

9 AMBIENT AIR EXPOSURE TO GENERAL POPULATION

9.1 Exposure Calculations

Modeled ambient air concentrations from IIOAC need to be converted to estimates of exposures to derive risk estimates. For this exposure assessment, EPA assumes the general population is continuously exposed (*i.e.*, 24 hours per day, 365/216 days per year) to outdoor ambient air concentrations. Therefore, daily average modeled ambient air concentrations are equivalent to daily average exposure concentrations, and annual average modeled ambient air concentrations are equivalent to annual average 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 Exposure Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025a).

9.2 Overall Conclusions

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 Risk Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025g), EPA does not expect an inhalation risk from ambient air nor ingestion risk from air to soil deposition to result from exposures to DIBP from industrial releases. Since 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 DIBP through inhalation of ambient air or ingestion from air to soil deposition are not necessary.

10 HUMAN MILK EXPOSURES TO GENERAL POPULATION

Infants are potentially 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 DIBP is a developmental and reproductive toxicant (U.S. EPA, 2025d). EPA considered exposure (Section 10.1) and hazard (Section 10.3) information, as well as pharmacokinetic models (Section 10.2), to determine the most appropriate approach to evaluate infant exposure to DIBP from human milk ingestion. EPA 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 gestation and/or the perinatal window. In other words, infant exposure and risk estimates from maternal exposure are expected to be protective of nursing infants as well.

10.1 Biomonitoring Information

DIBP has the potential to accumulate in human milk because of its small mass (278.35 Daltons or g/mol) and lipophilicity (log K_{OW} = 4.34). EPA identified eight biomonitoring studies, of which one is a U.S. study, from reasonably available information that investigated if DIBP or its metabolites were present in human milk. These studies provide evidence of DIBP or its metabolites in human milk and were not used as part of the analysis for quantifying exposure estimates. Study quality can be found in the *Draft Data Quality Evaluation Information for General Population, Consumer, and Environmental Exposure for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025c). DIBP or its metabolites were detected in human milk samples in each of these studies. None of the studies characterized if any of the study participants may be occupationally exposed to DIBP.

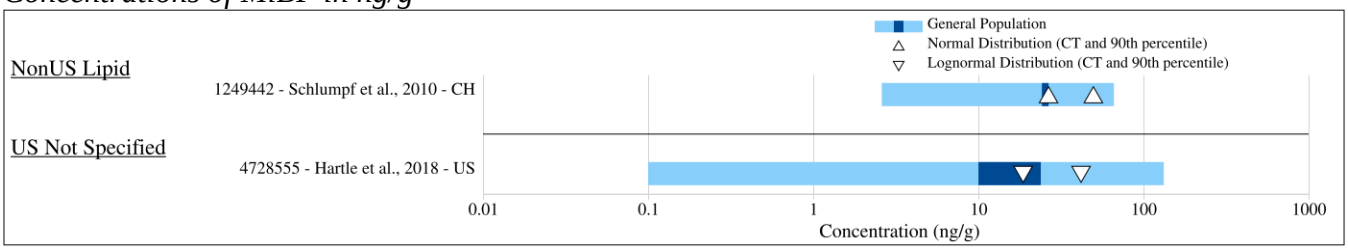
In a U.S. study, DIBP's primary metabolite, mono-isobutyl phthalate (MIBP) was measured in 21 samples collected in the Mother's Milk Bank in California. The concentrations ranged from 0.10 to 132.7 ng/g lipid weight with a mean concentration of 23.88 ng/g (Hartle et al., 2018).

None of the reported concentrations of DIBP or its metabolites in human milk from non-U.S. studies can be compared with the U.S. study. Six of the non-U.S. studies measured DIBP or MIBP as a wet weight rather than a lipid weight. Those studies collected samples from Italy, Germany, Switzerland, Korea, and Taiwan, and concentrations ranged from less than 0.1 to 66.2 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). One German study reported a lipid weight concentration of DIBP (median 1 ng/g; maximum 5.8 ng/g) and not MIBP (Zimmermann et al., 2012).

It is important to note that biomonitoring data do not distinguish between exposure routes or pathways and does not allow for source apportionment. While they provide important empirical evidence that human milk ingestion is a potential exposure pathway for nursing infants, EPA cannot isolate the contribution of specific TSCA uses to the measured levels in human milk. There is no evidence in any of the studies that the measured levels of DIBP or its metabolites can be attributed solely or partially to TSCA uses. The use of biomonitoring data to characterize a nursing infant's exposure to DIBP represents an aggregate exposure from all DIBP sources and pathways, which may contribute to the presence of DIBP in human milk, including both TSCA and non-TSCA uses. In other words, biomonitoring data reflect total infant exposure through human milk ingestion, and the contribution of specific TSCA COUs to overall exposure cannot be determined.

Concentrations of DIBP in ng/L

Concentrations of MiBP in ng/g



Concentrations of MiBP in ng/L

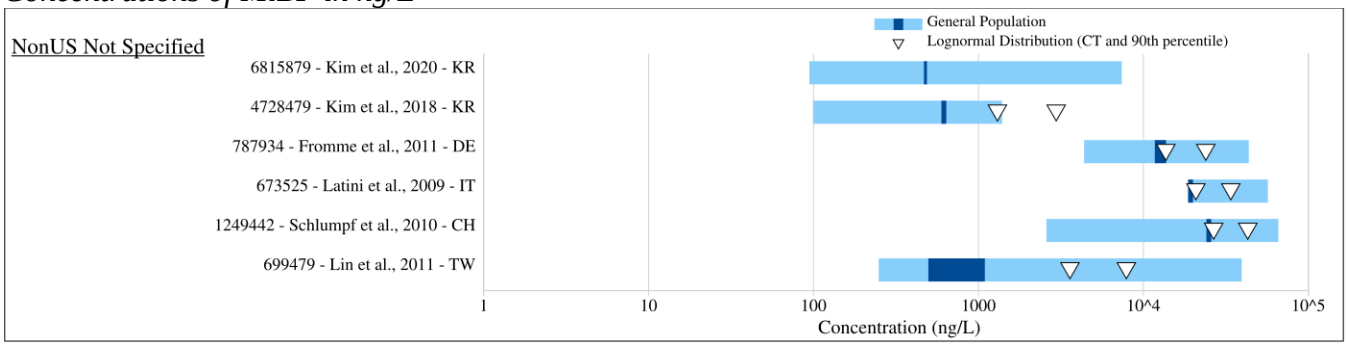


Figure 10-1. Concentrations of DIBP or MIBP in Human Milk in Either Lipid (ng/g) or Wet Weight (ng/L)

Notes: These studies provide evidence of DIBP or MIBP in human milk and were not used as part of the analysis for quantifying exposure estimates. Study quality varied and can be found in the *Draft Data Quality Evaluation Information for General Population, Consumer, and Environmental Exposure for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025c).

10.2 Modeling Information

EPA explored the potential to model DIBP 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 DIBP and concluded that uncertainties in establishing an appropriate half-life value precludes using the model to quantify lactational transfer and exposure from TSCA COUs. DIBP is rapidly hydrolyzed to its primary monoester metabolite MIBP, which undergoes further oxidation reactions to produce secondary metabolites. This rapid hydrolysis does not make the parent compound a reliable biomarker of exposure because its measurement in organs, tissues, and matrices is transient and may be prone to error and contamination from sampling materials. MIBP is readily detectable in urine because of its relatively short side chain of three carbons. However, it too can be an environmental or laboratory contaminant as simple hydrolysis of the ubiquitous DIBP can then generate MIBP (Koch and Calafat, 2009). The secondary oxidized metabolite, 2OH-MIBP, is less prone to analytical contamination. However, 2OH-MIBP by itself accounts for 20 percent of the eliminated

DIBP, and 70 percent is excreted as MIBP (Koch et al., 2012). This indicates neither the primary nor secondary metabolite is a reliable biomarker of DIBP exposure.

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 developmental and reproductive toxicity studies of experimental animals that evaluated the effects of oral exposures to DIBP. 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 were observed in 13 oral exposure studies following gestational or perinatal exposures to DIBP and are attributable to antiandrogenic effects during gestation (see *Draft Human Health Hazard Assessment for Diisobutyl Phthalate* (U.S. EPA, 2025g)). No one- or two-generation reproductive studies of DIBP are available for any routes of exposure. Furthermore, no studies were identified that evaluated only lactational exposure (*i.e.*, from birth to weaning) from quantified levels of DIBP or its metabolites in milk. However, the hazard values are based on developmental and reproductive toxicity in the offspring following maternal exposures during gestation. Because these values designed to be protective of infants are expressed in terms of maternal exposure levels 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 DIBP through human milk because the available biomonitoring data demonstrate that DIBP can be present in human milk and hazard data demonstrate that the developing male reproductive system can be particularly susceptible to the effects of DIBP. 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. However, 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 assessment is based on male reproductive effects resulting from maternal exposures throughout sensitive phases of development. EPA therefore has confidence that the risk estimates calculated based on maternal exposures are protective of a nursing infant.

11 URINARY BIOMONITORING

The use of human biomonitoring data is an important tool for determining total dose (or aggregate exposure) to a chemical for real world populations. Reverse dosimetry uses biomonitoring data, as shown in Figure 11-1, to estimate an external exposure or intake dose to a chemical responsible for the measured biomarker (U.S. EPA, 2019b). Intake doses estimated using reverse dosimetry are not source apportionable and are therefore not directly comparable to the exposure estimates presented throughout this document associated with specific COUs. However, the total intake dose estimated from reverse dosimetry can help contextualize the exposure estimates from TSCA COUs as being potentially underestimated or overestimated. This section discusses urinary biomonitoring data that provide total exposure from all sources for different life stages.

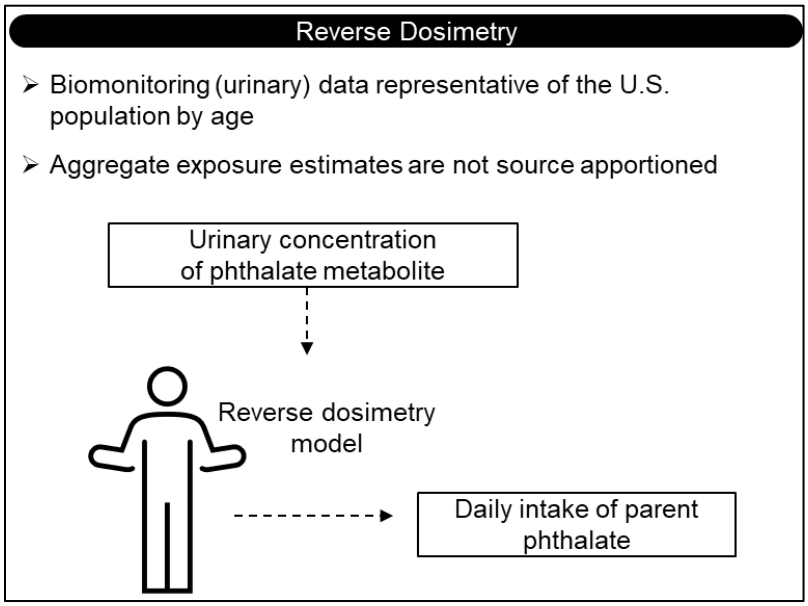


Figure 11-1. Reverse Dosimetry Approach for Estimating Daily Intake

11.1 Approach for Analyzing Biomonitoring Data

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.

NHANES reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. EPA analyzed data for two metabolites of DIBP: (2) mono-2-methyl-2-hydroxypropyl phthalate (MHiBP), measured in the 2013 to 2018 NHANES cycles; and (2) mono-isobutyl phthalate (MiBP), measured in the 2001 to 2018 NHANES cycles. Although MHiBP 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 MHiBP data from the 2015 to 2018 NHANES cycles (sampling details can be found in Appendix G).

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

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. Over time, statistical analyses of DIBP metabolite trends were performed with PROC DESCRIPT using SAS-callable SUDAAN.

11.1.1 Temporal Trend of MiBP

Figure 11-2 through Figure 11-7 show urinary MiBP concentrations plotted over time for the various populations to visualize the temporal exposure trends. Overall, median and 95th percentile MiBP urinary concentrations significantly increased over time for all lifestages.

From 2001 to 2018, median and 95th percentile MiBP concentrations significantly increased among all children 3 to less than 16 years ($p < 0.001$ for both percentiles of exposure) (Figure 11-4), as well as for children 6 to less than 11 years ($p < 0.001$ for both percentiles of exposure) (Figure 11-6) and adolescents 11 to less than 16 years ($p < 0.001$ for both percentiles of exposure) (Figure 11-7). MiBP concentrations also significantly increased among toddlers 3 to less than 6 years at the 95th percentile ($p < 0.001$) (Figure 11-5). Similarly, median and 95th percentile MiBP concentrations significantly increased among all adults age 16 and older ($p < 0.001$ for both percentile exposures), adult males ($p < 0.001$ for both percentile exposures), adult females ($p < 0.001$ for both percentile exposures) (Figure 11-2), and women of reproductive age 16 to 49 years ($p < 0.001$ for both percentile exposures) (Figure 11-3).

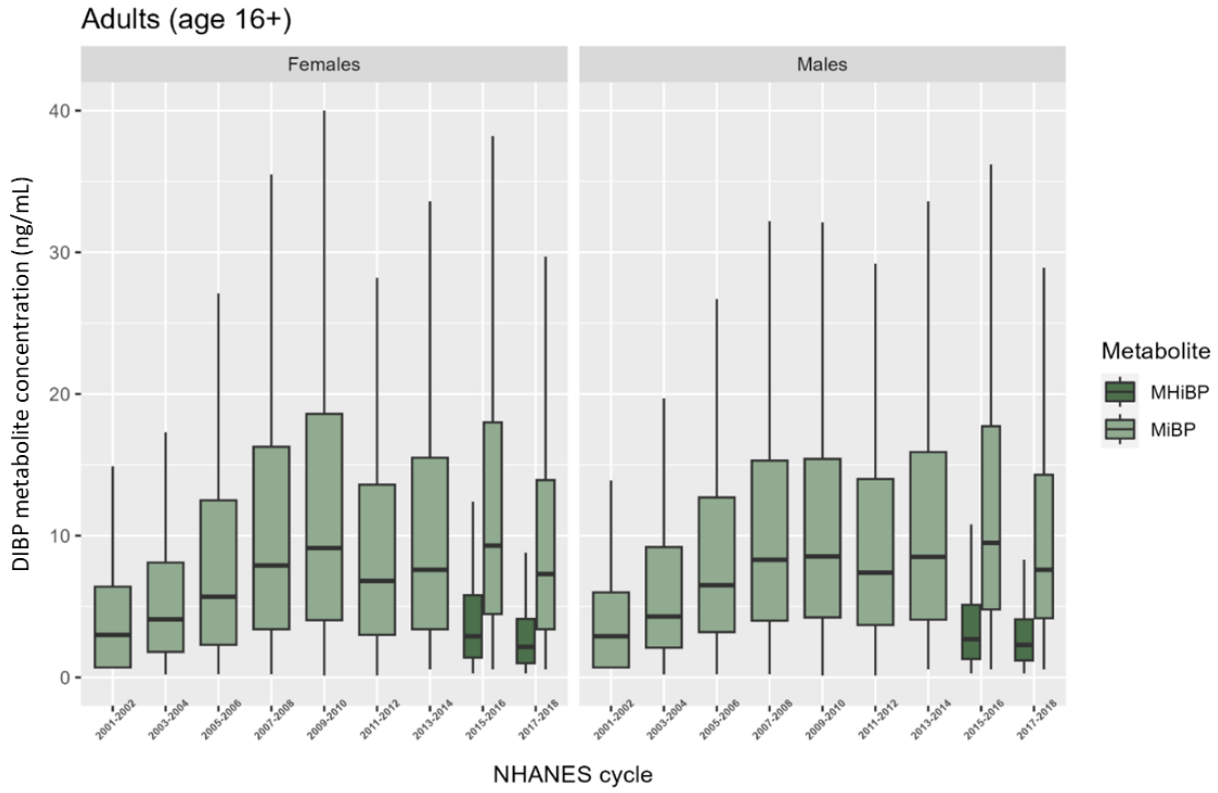


Figure 11-2. Urinary DIBP Metabolite Concentrations for Adults (Aged 16+ Years)

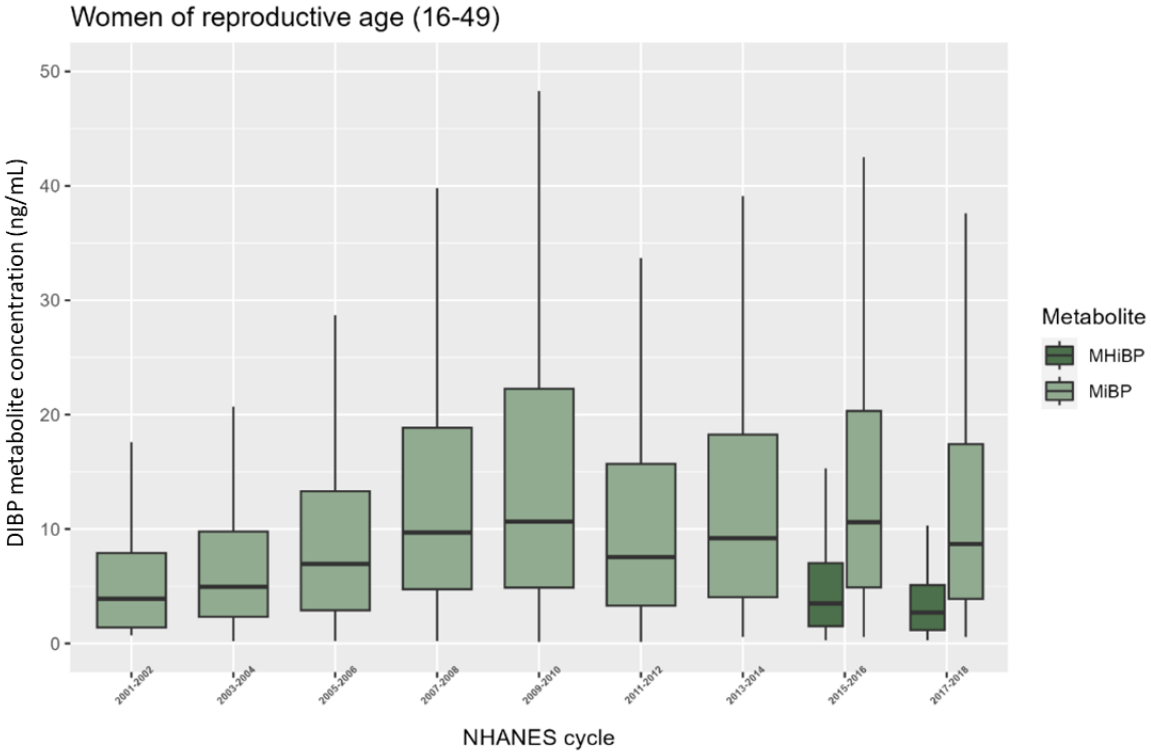


Figure 11-3. Urinary DIBP Metabolite Concentrations for Women of Reproductive Age (16–49 Years)

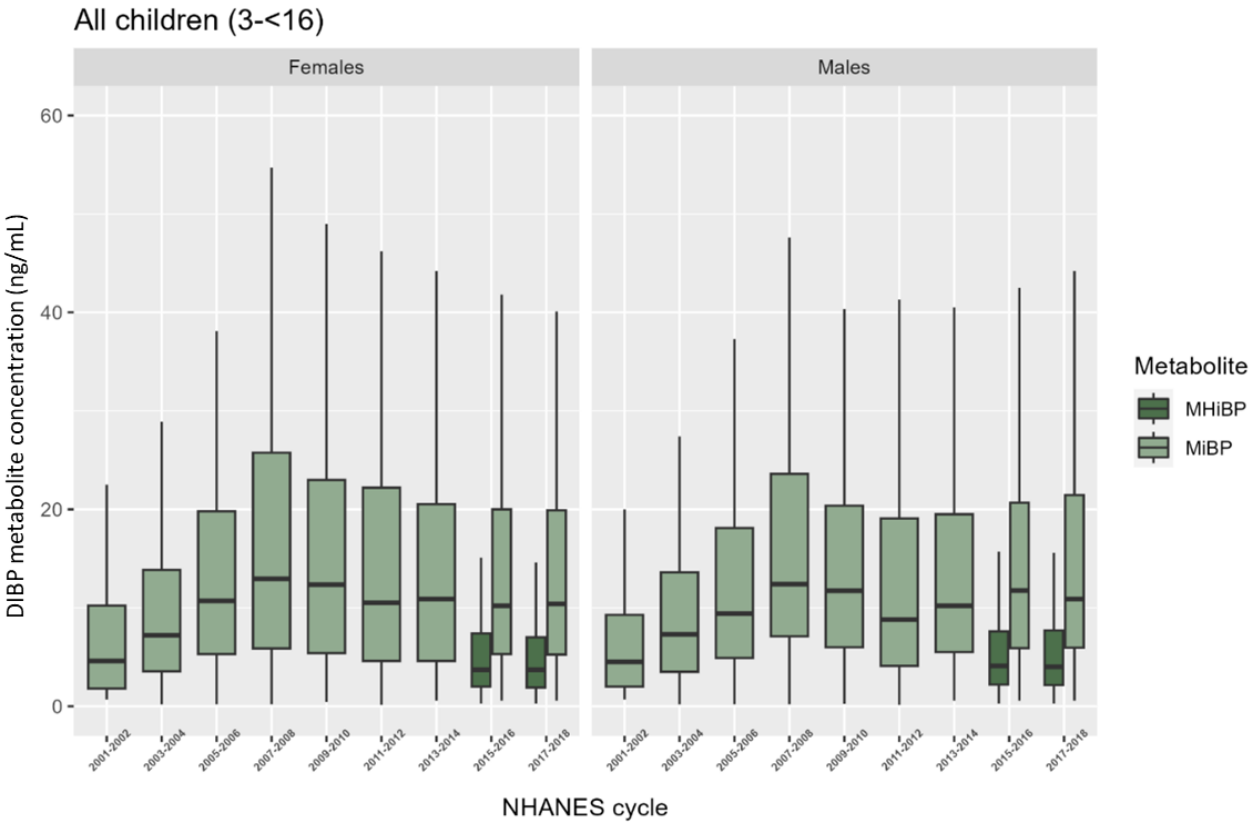


Figure 11-4. Urinary DIBP Metabolite Concentrations for All Children (Aged 3 to <16 Years) by Sex

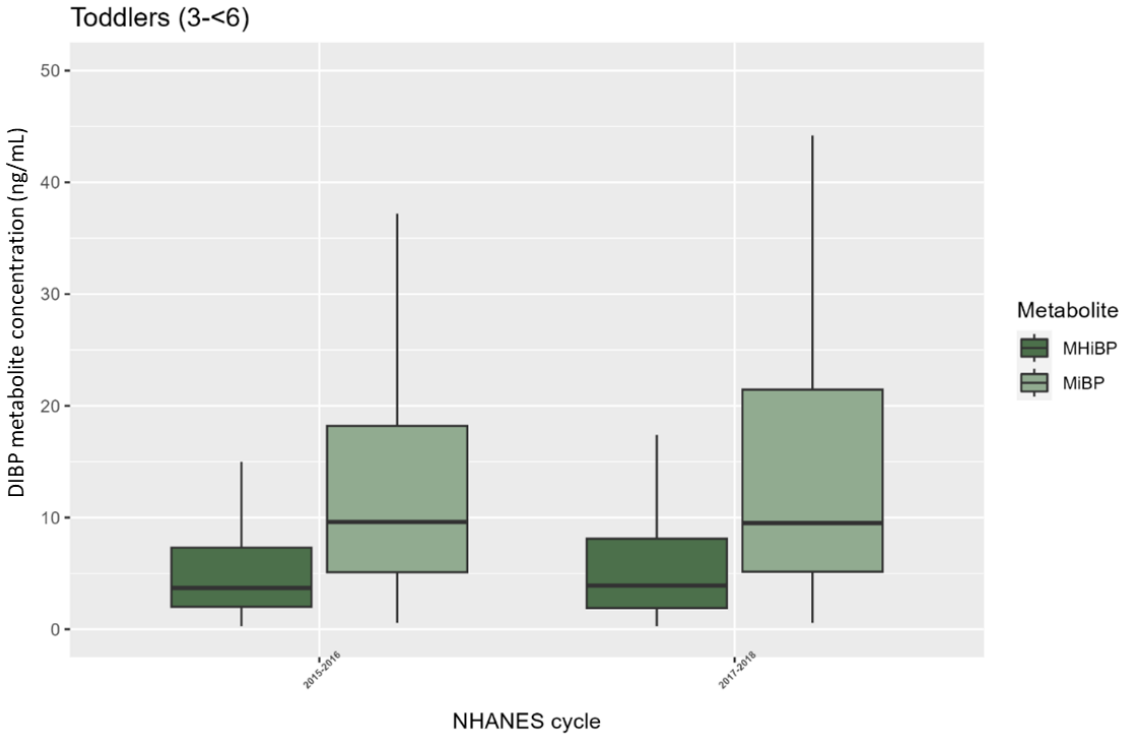


Figure 11-5. Urinary DIBP Metabolite Concentrations for Toddlers (Aged 3 to <6 Years)

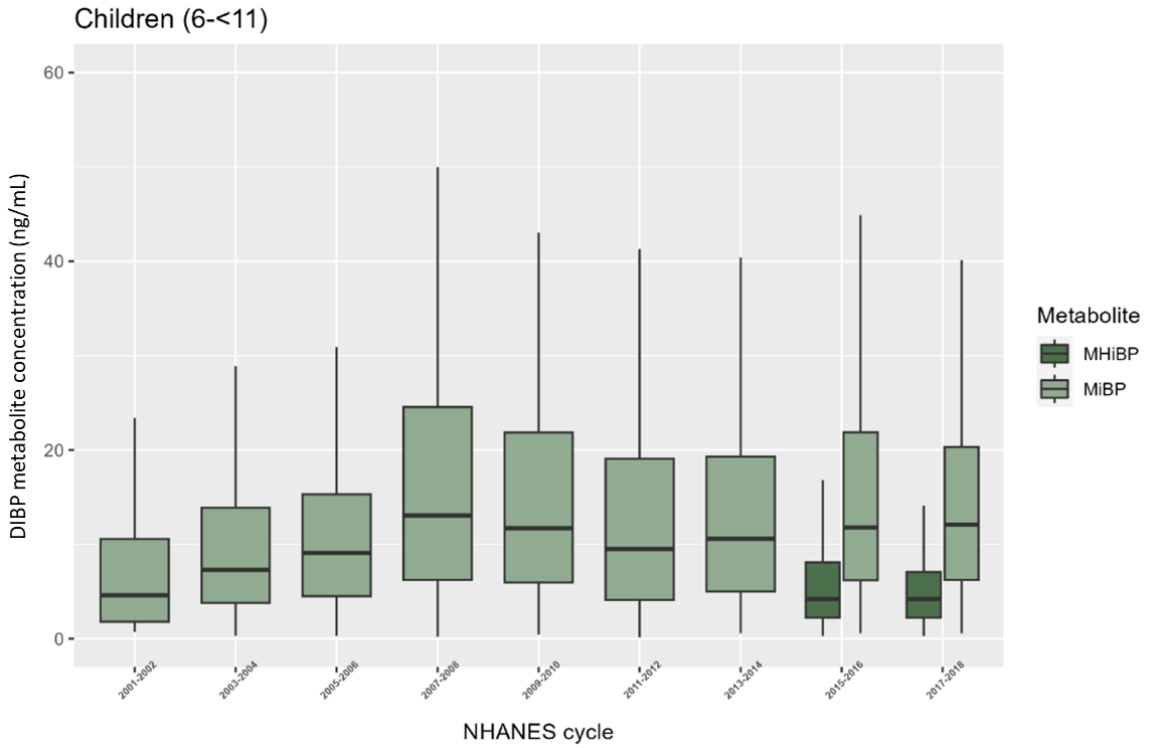


Figure 11-6. Urinary DIBP Metabolite Concentrations for Children (Aged 6 to <11 Years)

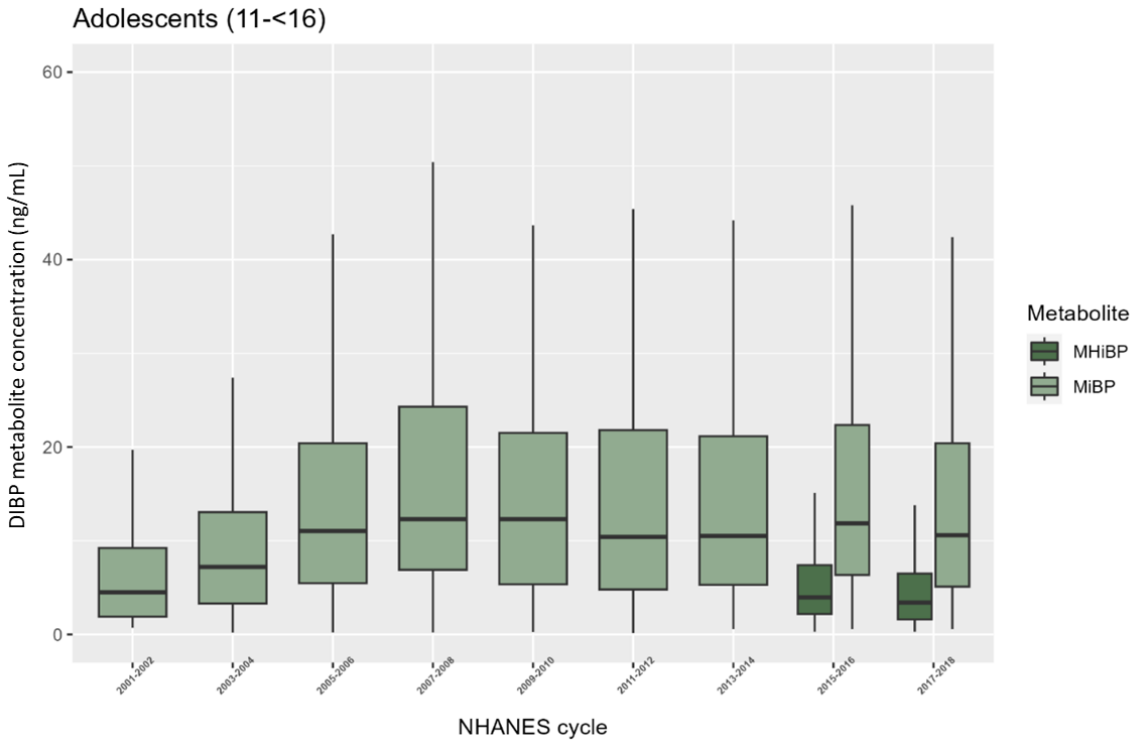


Figure 11-7. Urinary DIBP Metabolite Concentrations for Adolescents (Aged 11 to <16 Years)

11.1.2 Changes in MHiBP Concentration

As mentioned in Section 11.1, only data from the 2015 to 2018 cycles were analyzed for MHiBP resulting in the two data points shown for MHiBP concentrations in Figure 11-2 through Figure 11-7 .

Therefore, a temporal trend analysis was not conducted for MHiBP. However, a comparison of the metabolite concentrations between the 2015 to 2016 and 2017 to 2018 NHANES cycles show that median and 95th percentile MHiBP urinary concentrations decreased for most lifestages.

11.1.3 Daily Intake of DIBP from NHANES

Using DIBP metabolite concentrations measured in the most recently available sampling cycle (2017–2018), EPA estimated the daily intake of DIBP 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 (2014) and 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). For DIBP, the phthalate monoester metabolites are MIBP and MHiBP.

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

$$Phthalate\ DI = \frac{(UE_{Sum} \times CE)}{Fue_{sum}} \times MW_{Parent}$$

Where:

$Phthalate\ DI$	=	Daily intake (µg/kg$_{bw}$/day) value for the parent phthalate diester.
UE_{sum}	=	The 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 (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 reported 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 DIBP

Metabolite	F_{ue}[a] [b]	Reference	Study Population
MiBP	0.69	Anderson et al. (2011)	n = 13 volunteers (age and sex not specified)

[a] F_{ue} values presented on a molar basis and estimated by study authors based on metabolite excretion over a 24-hour period.
[b] F_{ue} value of 0.69 based on excretion of DBP urinary metabolite MnBP.

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 DIBP are reported in Table 11-2.

Fractional urinary excretion (F_{ue}) values can be determined through controlled human exposure studies. One controlled human exposure study of DIBP was identified in which one volunteer (36-year-old male) was dosed with 60 µg/kg body weight of deuterated DIBP (D4-DIBP) (total dose of 5.380 mg DIBP) and then urine samples were collected over 48 hours following dosing (Koch et al., 2012). Given that this study evaluated a single individual, EPA instead used the F_{ue} values for DBP, an isomer of DIBP, estimated from a controlled human exposure study of 13 volunteers (Anderson et al., 2011). Anderson et al. (2011) estimated a F_{ue} value of 0.69 for mono-n-butyl phthalate (MnBP), a metabolite of DBP, which EPA selected as surrogate data for MiBP. The use of MnBP as a surrogate for MiBP is supported by the structural similarity of DIBP and DBP, which are isomers. Further, the F_{ue} value estimated for MnBP by Anderson et al. (2011) in the study of 13 volunteers is similar to the F_{ue} value estimated by Koch et al. (2012) for MiBP from a single volunteer (*i.e.*, 0.69 for MnBP versus 0.703 for MiBP). Additionally, U.S. CPSC (2014) also used the F_{ue} value from MnBP as a surrogate for MIBP in their 2014 phthalate risk assessment.

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

Demographic	50th percentile Daily Intake Value (95% CI) (µg/kg-day)	95th percentile Daily Intake Value (95% CI) (µg/kg-day)
All	0.25 (0.23–0.28)	1.16 (0.97–1.35)
Females	0.26 (0.22–0.31)	0.96 (0.77–1.15)
Males	0.25 (0.21–0.28)	1.35 (1.01–1.69)
White non-Hispanic	0.24 (0.2–0.29)	0.99 (0.74–1.23)
Black non-Hispanic	0.24 (0.2–0.29)	1.38 (1.05–1.71)
Mexican American	0.25 (0.21–0.29)	1.13 (0.52–1.73)
Other	0.28 (0.23–0.34)	1.23 (0.83–1.63)
Above poverty level	0.31 (0.25–0.37)	1.1 (0.77–1.43)
Below poverty level	0.25 (0.21–0.28)	1.16 (0.9–1.41)
Toddlers (3 to <6 years)	0.51 (0.45–0.57)	1.98 (1.42–2.54)
Children (6 to <11 years)	0.32 (0.26–0.37)	1.19 (0.68–1.71)
Adolescents (12 to <16 years)	0.2 (0.17–0.23)	0.86 (0.35–1.37)
Adults (16+ years)	0.19 (0.16–0.22)	0.59 (0.23–0.96)
Male toddlers (3 to <6 years)	0.57 (0.48–0.65)	2.12 (1.56–2.67)
Male children (6 to <11 years)	0.33 (0.26–0.39)	1.62 (0.69–2.56)
Male adolescents (12 to <16 years)	0.21 (0.18–0.23)	0.59 (0.12–1.05)
Male adults (16+ years)	0.16 (0.12–0.21)	0.49 (–0.03 to 1)
Female toddlers (3 to <6 years)	0.4 (0.33–0.47)	1.52 (0.53–2.51)
Female children (6 to <11 years)	0.31 (0.23–0.38)	0.88 (0.32–1.44)
Female adolescents (12 to <16 years)	0.18 (0.09–0.27)	0.86[a]
Women of reproductive age (16–49 years)	0.2 (0.15–0.25)	0.57[a]
Female adults (16+ years)	0.25 (0.23–0.28)	1.16 (0.97–1.35)

[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 (2020). The daily intake values in the present analysis are calculated with all available NHANES data between 1999 to 2018, while the CPSC report only contains estimates for DIBP calculated using MiBP data from the 2005 to 2006 NHANES cycle and the Health Canada analysis used data from the 2009–2011 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 as 0.19 µg/kg-day and a 95th percentile daily intake value of 0.78 µg/kg-day.

The Health Canada (2020) assessment reports mean daily intake values for male children aged 6 to 11 as 1.5 µg/kg-day and median and 95th percentile intakes of 0.76 and 5.3 µg/kg bw/day, respectively. For adult females (age 20–49), the reported median daily intake was 0.46 µg/kg-day and the 95th percentile was 1.4 µg/kg-day.

As described earlier, 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. The U.S. CPSC (2014) states that DIBP exposures were highest among toddlers, and that toddlers were primarily exposed to DIBP through food and beverages. As the outdoor environment did not contribute to DIBP exposures, the exposures to the general population ambient air, surface water, and drinking water quantified in this document 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.

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 (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 (2020). However, there are challenges and sources of uncertainty associated with the use of reverse dosimetry approaches. 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 the following: (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 (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, 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) for further discussion).

11.3 Weight of Scientific Evidence Conclusions

For the urinary biomonitoring data, despite the uncertainties discussed in Section 11.2, overall 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 presented in this document. 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 the Agency has robust confidence in the use of its total daily intake value to contextualize the exposure estimates from TSCA COUs as being overestimated as described in Section 11.1.3.

12 ENVIRONMENTAL BIOMONITORING AND TROPHIC TRANFER

EPA assessed the available studies related to the biomonitoring of DIBP collected in accordance with the *Draft Systematic Review Protocol Supporting TSCA Risk Evaluations for Chemical Substances* (U.S. EPA, 2021b) and *Draft Systematic Review Protocol for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025j). Chemicals can be transferred from contaminated media and diet to biological tissue and accumulate throughout an organisms' lifespan (bioaccumulation) if they are not readily excreted or metabolized. Through dietary consumption of prey, a chemical can subsequently be transferred from one trophic level to another. If biomagnification occurs, higher trophic level predators will contain greater body burdens of a contaminant compared to lower trophic level organisms. EPA reviewed biomonitoring studies and provided qualitative descriptions of the potential dietary exposures to aquatic and terrestrial organisms via feeding (trophic) relationships.

12.1 Aquatic Environmental Biomonitoring

Measured DIBP concentrations stemmed from studies examining phthalate ester levels in aquatic ecosystems. Multiple aquatic species had DIBP wet weight concentrations reported from one study. Upon examining the highest geometric mean DIBP wet weight concentration at each trophic level, it was determined that DIBP generally decreases in concentration as it transfers up trophic levels.

DIBP wet weight concentrations have been reported for three species of primary consumers (*e.g.*, crustaceans and mollusks). The hepatopancreas of the dungeness crab (*Cancer magister*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a geometric mean DIBP concentration of 0.002 mg/kg wet weight (ww) (McConnell, 2007). For two mollusk species in the same location, the geometric mean DIBP concentrations were 0.00046 and 0.00078 mg/kg ww in the whole bodies of the softshell clam (*Mya arenaria*) and the blue mussel (*Mytilus edulis*), respectively (McConnell, 2007). Primary consumers thus had geometric mean DIBP concentrations ranging from 0.00046 to 0.002 mg/kg ww.

DIBP wet weight concentrations were reported for one species of omnivorous finfish, which are secondary and tertiary consumers. The shiner perch (*Cymatogaster aggregata*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a whole body geometric mean DIBP concentration of 0.0018 mg/kg ww (McConnell, 2007).

12.2 Trophic Transfer

Trophic transfer is the process by which chemical contaminants can be taken up by organisms through dietary and media exposures and be transferred from one trophic level to another. Due to its physical and chemical properties, environmental fate, and exposure parameters, DIBP is not expected to persist in surface water, groundwater, or air. Based on its solubility (6.2 mg/L) and organic carbon:water adsorption coefficent (log K_{OC} = 4.34), DIBP readily sorbs to organic matter such as sediment and suspended solids suggesting limited bioavailability. DIBP is expected to have an environmental biodegradation half-life in aerobic environments on the order of days to weeks. While DIBP is not anticipated to persist within air with a half-life of 1.15 days, the octanol:air partition coefficient (log K_{OA}) of 9.47 estimated from EPI Suite™ (U.S. EPA, 2017a) indicates adsorption to organic carbon within airborne particles with limited atmospheric oxidation. Within aerobic soils, DIBP is expected to have a half-life of approximately 10 days and results from EPI Suite™ suggest that DIBP will not degrade rapidly in anaerobic environments. For further information on the sources of these values, please see the *Draft Chemistry, Fate, and Transport Assessment for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025h).

The available data suggests that DIBP has low bioaccumulation potential in aquatic and terrestrial organisms (Kim et al., 2016; Teil et al., 2012), and no biomagnification across trophic levels in the aquatic food web (Mackintosh et al., 2004). The estimated fish upper trophic level BCF for DIBP is 30.2 L/kg (U.S. EPA, 2017a), which is well below the criteria to be considered bioaccumulative (estimated BCF/BAF > 1,000 L/kg) (U.S. EPA, 2012). In the Orge River, France, reported fish aquatic biota-sediment accumulation factors (BSAF) in roach (*Rutilus rutilus*), chub (*Leuciscus cephalus*), and perch (*Perca fluviatilis*) were 62.5 ± 26.5, 41.4 ± 13.3, and 123.5 ± 75.3, respectively (Teil et al., 2012). Reported Trophic Magnification Factors (TMF) of 0.11 and 1.8 and aquatic food web magnification factor (FWMF) of 0.81 indicate trophic dilution of DIBP from lower to higher trophic levels within the food web (Kim et al., 2016; Mackintosh et al., 2004). In terrestrial environments, there is limited information on the bioconcentration and bioaccumulation of DIBP. While DIBP is expected to be bioavailable in soils, the reported DIBP BCF value (2.23) on the edible fraction of several fruits and vegetables suggest low uptake potential of DIBP in soils (Li et al., 2016). The available estimated BCF and measured BSAF values in higher trophic level piscivorous fish and the FWMF study conducted by Mackintosh (2004) provide evidence that trophic transfer of DIBP is not a likely source of significant DIBP exposure. Due to the low persistence, limited bioavailability, and low bioaccumulation potential of DIBP, EPA did not conduct a quantitative analysis of DIBP trophic transfer.

12.3 Weight of Scientific Evidence Conclusions

Based on the reasonably available data, EPA has robust confidence that DIBP is found in relatively low concentrations in aquatic organism tissues, especially at higher trophic levels. Additionally, DIBP has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms and therefore DIBP is expected to go through trophic dilution as it passes through the food web.

13 CONCLUSION OF ENVIRONMENTAL EXPOSURE AND GENERAL POPULATION SCREENING LEVEL ANALYSIS

13.1 Environmental Exposure Conclusions

DIBP is expected to be released to the environment via air, water, and biosolids and landfills. 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 Chemistry, Fate, and Transport Assessment for Diisobutyl phthalate (DIBP)* (U.S. EPA, 2025h).

EPA conducted modeling with VVWM-PSC (U.S. EPA, 2019c) to estimate concentrations of DIBP within surface water and sediment. PSC inputs include physical and chemical properties of DIBP (*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) allowing EPA to model predicted surface water concentrations. 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 DIBP-releasing facilities, all releases are assumed initially to be released to surface water without treatment. The resulting surface water and sediment concentrations are presented in Table 4-5 and Table 4-6, respectively and will be utilized within the environmental risk characterization for DIBP.

There are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered for DIBP. However, based on high-quality physical and chemical property data, EPA determined that DIBP will have low persistence potential in soils. Therefore, groundwater concentrations resulting from releases to the landfill or to agricultural lands via biosolids applications are not quantified but are discussed qualitatively in Section 3. Modeled soil DIBP concentrations from air deposition to soil (Table 8-2) and modeled DIBP concentrations in biosolids-amended soils (Table 3-2) from OESs with the resulting highest concentrations to soil are used to assess risk quantitatively in conjunction with hazard thresholds (U.S. EPA, 2024b) for relevant soil dwelling organisms and plants within the Environmental Risk Characterization section of the *Draft Risk Evaluation for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025i).

EPA conducted a qualitative trophic transfer assessment by evaluating the chemical and physical properties, fate, and exposure of DIBP and preliminarily determined that DIBP does not bioaccumulate. Therefore, the Agency did not conduct a quantitative analysis of the trophic transfer of DIBP through food webs. EPA has robust confidence that DIBP has limited bioaccumulation and bioconcentration potential based on physical chemical and fate properties, biotransformation, and empirical metrics of bioaccumulation metrics presented in Section 12.

13.2 Weight of Scientific Evidence Conclusions for Environmental Exposure Conclusion

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), landfills (Section 3.2), surface water (Section 4.1), ambient air (Section 8), and environmental biomonitoring and trophic transfer (Section 12). EPA summarized its weight of scientific evidence using confidence descriptors: robust, moderate, slight, or indeterminate. 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 has robust confidence that DIBP has limited bioaccumulation and bioconcentration potential based on physical, chemical, and fate properties, biotransformation, and empirical metrics of bioaccumulation.

13.3 General Population Exposure Conclusions

The general population can be exposed to DIBP from various exposure pathways. As shown in Table 1-3, 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. Using 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.

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

OES[a]	Release Media	Environmental Media	DIBP Concentration
Plastic compounding *without wastewater treatment*	Water	Surface water (30Q5, median flow)	174 µg/L
		Surface water (harmonic mean, median flow)	121 µg/L
Plastic compounding *with wastewater treatment*	Water	Surface water (30Q5, median flow)	55.7 µg/L
		Surface water (harmonic mean, median flow)	38.7 µg/L
Plastic compounding (fugitive and stack)	Ambient air	Daily-averaged total (fugitive and stack, 100 m)	17.59 µg/m^3
		Annual-averaged total (fugitive and stack, 100 m)	16.45 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 exposure and incidental ingestion from swimming in surface water, drinking water ingestion, fish ingestion, and exposure from 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 DIBP for the highest exposed populations.

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

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Pathway of Concern[b]
All	Biosolids (Section 3.1)	All considered qualitatively			No
All	Landfills (Section 3.2)	All considered qualitatively			No
Plastic compounding	Surface water	Dermal	Dermal exposure to DIBP in surface water during swimming (Section 5.1.1)	Adults (21+ years)	No
		Oral	Incidental ingestion of DIBP in surface water during swimming (Section 5.1.2)	Youth (11–15 years)	No

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Pathway of Concern[b]
Plastic compounding	Drinking water	Oral	Ingestion of drinking water (Section 6.1.1)	Infant (<1 year)	No
All	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	Adult (21+ years)	No
			Ingestion of fish for subsistence fishers (Section 7.2)	Adult (21+ years)	No
			Ingestion of fish for tribal populations (Section 7.3)	Adult (21+ years)	No
Plastic compounding (fugitive and stack)	Ambient air	Inhalation	Inhalation of DIBP in ambient air resulting from industrial releases (Section 9)	All	No
		Oral	Ingestion from air to soil deposition resulting from industrial releases (Section 9)	Infant and Children (6 months to 12 years)	No

[a] Table 1-1 provides a crosswalk of industrial and commercial COUs to OESs.
[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 4.4), drinking water (Section 6.4), fish ingestion (Section 7.4), ambient air (Section 8.3.1), human milk (Section 10.4), and urinary biomonitoring (Section 11.2 and 11.3). The strengths, limitations, and uncertainties associated with the reverse dosimetry approach is available in Section 11.2.

EPA summarized its weight of scientific evidence using the following confidence descriptors: robust, moderate, slight, or indeterminate. 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 of 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. For its quantitative exposure assessment of surface water (Section 5.2), drinking water (Section 6.4), fish ingestion (Section 7.4), ambient air (Section 8.4), human milk (Section 10), and urinary biomonitoring (Section 11.3), EPA has robust confidence that the screening level analysis was appropriately conservative to determine that no environmental pathway has the potential for non-cancer or cancer risk to the general population. Despite slight and 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 DIBP 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 data. Furthermore, risk estimates for high-end exposure scenarios were still consistently above the benchmarks, adding to confidence that non-cancer and 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)

2452 **Table_Apx A-3. Recommended Default Values for Common Exposure Factors**

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source(s)
		Occupational	Residential	
ED	Exposure duration (hours/day)	8	24	
EF	Exposure frequency (days/year)	250	365	
EY	Exposure years (years)	40	Varies for adults 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) 0.195 Toddler (1–5 years) 0.294 Child (6–10 years) 0.315 Youth (11–15 years) 0.436 Youth (16–20 years)	Chapter 3 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 3-9 per capita mean values; weighted averages for adults (years 21 to 49 and 50+), for toddlers (years 1–2, 2–3, and 3 to <6).
IR_{inc}	Incidental water ingestion rate (L/hr)		0.025 Adult 0.05 Child (6 to <16 years)	Evaluation of Swimmer Exposures Using the SWIMODEL Algorithms and Assumptions (U.S. EPA, 2015a)

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source(s)
		Occupational	Residential	
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, 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/hr) 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"
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)[a]	
	Mean	Upper (95th percentile)
Birth to <1 month	150	220

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

[a] Values were converted from Table 15-1 of U.S. EPA (2011a) using the density of human milk of 1.03 g/mL

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 (2021a)
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 (hr/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 (2021a)
AT	Averaging time (years for ADD)	57	5	5	Number of years in age group,	U.S. EPA (2021a)
K_p	Permeability coefficient (cm/hr)	0.0071 cm/hr			CEM estimate aqueous K_p	U.S. EPA; ICF Consulting (2022)

2459 **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/hr)	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 (2021a)
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 (2021a)
AT	Averaging time (years for ADD)	57	5	5	Number of years in age group,	U.S. EPA (2021a)
CF1	Conversion factor (mg/µg)	1.00E−03				
CF2	Conversion factor (days/year)	365				

2460

Appendix B ESTIMATING HYDROLOGICAL FLOW DATA FOR SURFACE WATER MODELING

A distribution of flow metrics was generated by collecting flow data for facilities across one North American Industry Classification System (NAICS) code associated with DIBP-releasing facilities (Table_Apx B-1). EPA's Enforcement and Compliance History Online (ECHO) database was accessed via the API and queried for facilities regulated under the Clean Water Act within the one relevant NAICS code. All available National Pollutant Discharge Elimination System (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 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 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 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 E−FAST to convert between these flow metrics and solved for the 7Q10 using Equation_Apx B-1. In previous assessments, the EPA has selected the 7Q10 flow as a representative low flow scenario for biological impacts due to effluent in 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)
cfs	=	Cubic feet per second
$30Q5$	=	Lowest monthly average flow from NHD, in MLD

Further, the harmonic mean (HM) flow was calculated using Equation_Apx B-1, derived from the relevant E−FAST regression.

Equation_Apx B-2. Calculating the Harmonic Mean Flow

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

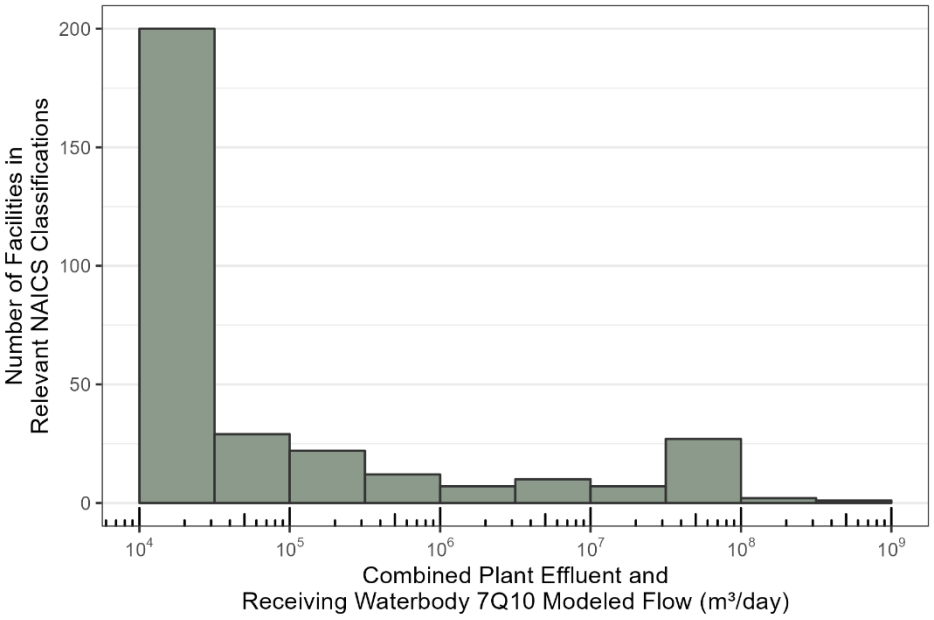
Where:

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

Table_Apx B-1. Relevant NAICS Codes for Facilities Associated with DIBP Releases

NAICS Code	NAICS Name
325199	All Other Basic Organic Chemical Manufacturing

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. A minimum effluent flow rate of six cubic feet per second, derived from the average reported effluent flow rate across facilities, was applied. The receiving waterbody 7Q10 flow was then calculated as the sum of the hydrologic 7Q10 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 7Q10 flow rate was selected to be 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

Quantified release estimates to surface water were evaluated with PSC modeling, applying the receiving waterbody flows estimated from the developed distribution. For each COU with surface water releases of wastewater effluent, the highest estimated release to surface water was modeled. The total days of release associated with the highest OES surface water releases was applied as continuous days of release per year (for example, 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 (*e.g.*, 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 GENERAL POPULATION SURFACE WATER RISK SCREENING RESULTS

C.1 Incidental Dermal Exposures (Swimming)

Based on the estimated dermal doses in Table 5-1., 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. *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 for the High-End Release Estimate 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
Plastic compounding[a] *without wastewater treatment*	174	121	2,800	3,657	6,028
Plastic compounding[a] *with wastewater treatment*	55.7	38.7	8,750	11,000	19,000
Highest monitored surface water[b]	3.30	3.30	150,000	190,000	320,000

30Q5 = 30 consecutive days of lowest flow over a 5-year period
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] Liu et al. (2013) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

C.2 Incidental Ingestion

Based on the estimated incidental ingestion doses in Table 5-2, EPA screened for risk to adults, youth, and children. 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. *Based on the conservative modeling parameters for surface water concentration and exposure factor parameters, risk for non-cancer health effects for incidental ingestion through swimming is not expected.*

Table_Apx C-2. Risk Screen for Modeling Incidental Ingestion Doses for Adults, Youths, and Children, for the High-End Release Estimate 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
Plastic compounding[a] *without wastewater treatment*	174	121	9,500	6,120	11,000
Plastic compounding[a] *with wastewater treatment*	55.7	38.7	29,700	19,100	34,000
Highest monitored surface water[b]	3.30	3.30	500,000	320,000	570,000

30Q5 = 30 consecutive days of lowest flow over a 5-year period
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] Liu et al. (2013) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

Appendix D GENERAL POPULATION DRINKING WATER 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. *Based on the conservative modeling parameters for drinking water concentration and exposure factor 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 DIBP 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, for the High-End Release Estimate 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
Plastic compounding[a] *without wastewater treatment*	174	121	814	7,141	232	2,796	652	6,523
Plastic compounding[a] *with wastewater treatment*	55.7	38.7	2,544	22,000	725	8,736	2,039	20,000
Highest monitored surface water[b]	3.30	3.30	43,000	230,000	12,000	90,000	34,000	210,000

30Q5 = 30 consecutive days of lowest flow over a 5-year period
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] Liu et al. (2013) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

Appendix E FISH INGESTION RISK SCREENING RESULTS

E.1 General Population

Using the water solubility limit as the surface water concentration, acute and chronic non-cancer risk estimates for the general population were above the benchmark of 30 (Table_Apx E-1). In comparison, the risk estimates using the highest monitored surface water concentration exceed the benchmark by three orders of magnitude. These results indicate that fish ingestion is not an exposure pathway of concern for the general population.

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

	Acute Non-Cancer MOE UFs = 30		Chronic Non-Cancer MOE UFs = 30
	Adult	Young Toddler	
Water solubility limit (6.20 mg/L)	110	70	480
Monitored surface water concentration (3.30E−03 mg/L) (Liu et al., 2013)	206,110	138,820	907,850

E.2 Subsistence Fishers

Using the water solubility limit as the surface water concentration, acute and chronic non-cancer risk estimates for subsistence fishers were below the benchmark of 30 (Table_Apx E-2). EPA then refined its evaluation of this exposure pathway by modeling surface water concentrations based on the highest modeled 95th percentile release for the Plastic compounding OES and the 50th percentile flow. The acute and chronic non-cancer risk estimates are one order of magnitude above their corresponding benchmarks using modeled data. EPA also included the highest monitored surface water concentrations from Liu et al. (2013) (Section 4.2.1). In comparison, the risk estimates using the highest monitored surface water concentration exceed the benchmark by four orders of magnitude. These results indicate that the modeled concentrations are conservative, as discussed in Section 4.1 and that fish ingestion is not an exposure pathway of concern for subsistence fishers.

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

	Acute and Chronic Non-Cancer MOE UFs = 30
Water solubility limit (6.20 mg/L)	20
Modeled surface water concentration for Plastic compounding, P50 flow (1.28E−01 mg/L)	876
Monitored surface water concentration (3.30E−03 mg/L) (Liu et al., 2013)	32,130
Note: The acute and chronic MOEs are identical because the exposure estimates and POD do not change between acute and chronic.	

E.3 Tribal Populations

Acute and chronic non-cancer risk estimates were below their benchmarks using the water solubility limit as the surface water concentration (Table_Apx E-3). EPA then refined its analysis by using modeled surface water concentrations based on the highest modeled 95th percentile release for the Plastic compounding OES and 50th percentile flow rate. EPA also included the highest monitored surface water concentrations from Liu et al. (2013) (Section 4.2.1). Non-cancer risk estimates are above

benchmark using both modeled and monitored surface water concentrations. These results indicate that the modeled concentrations are conservative, as discussed in Section 4.1, and that fish ingestion is not an exposure pathway of concern for tribal populations.

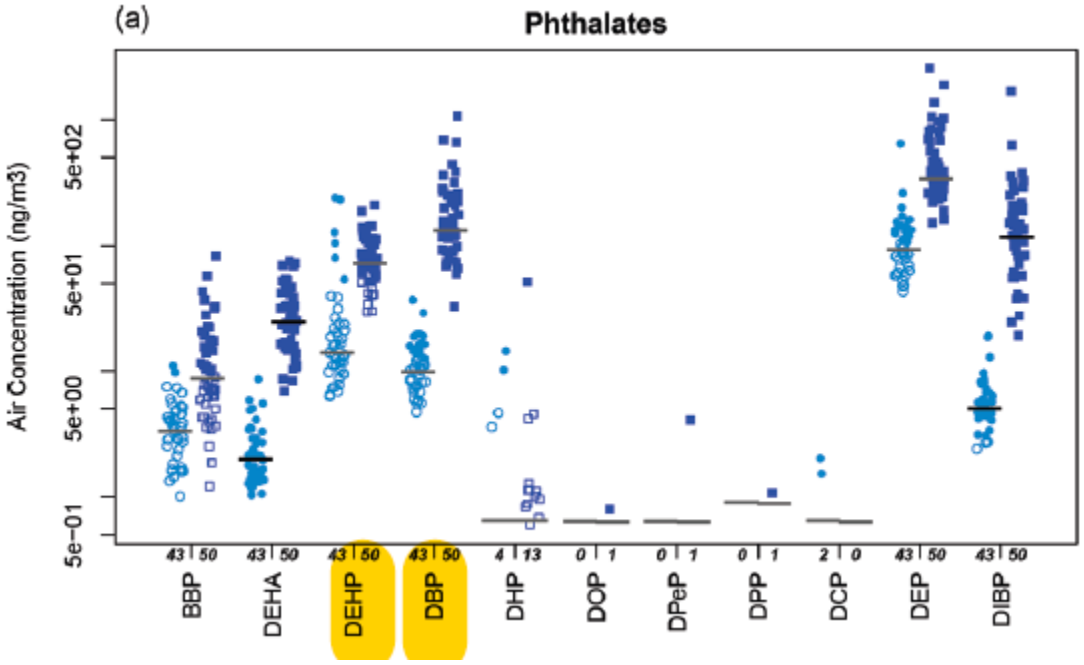
Table_Apx E-3. Risk Estimates for Fish Ingestion Exposure for Tribal Populations

	Acute and Chronic Non-Cancer MOE UFs = 30		
	Current IR, Mean	**Current IR, 95th Percentile**	**Heritage IR**
Water solubility limit (6.20 mg/L)	10	0	0
Modeled surface water concentration for Plastic compounding, P50 flow (1.28E−01 mg/L)	578	143	76
Monitored surface water concentration (3.30E−05 mg/L) (Liu et al., 2013)	21,180	5,250	2,780
Note: The acute and chronic MOEs are identical because the exposure estimates and 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 DIBP, DEHP, and DBP 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.

Appendix G URINARY BIOMONITORING METHODS AND RESULTS

EPA analyzed urinary biomonitoring data from the U.S. Centers for Disease Control and Prevention (CDC) National Health and Nutrition Evaluation Surveys (NHANES), which reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. Two metabolites of DIBP, mono-2-methyl-2-hydroxypropyl phthalate (MHiBP) and mono-isobutyl phthalate (MiBP), have been reported in the NHANES data. MiBP has been reported starting in the 2001 to 2002 NHANES cycle and has been measured in 24,199 participants, including 6,617 children and 17,582 adults. Beginning with the 2015 to 2016 cycle, NHANES began reporting data on MHiBP, which has been measured in 5,737 members of the general public, including 1,961 children aged 15 and under and 3,776 adults aged 16 years and over. Urinary MiBP and MHiBP concentrations were quantified using high performance liquid chromatography-electrospray ionization-tandem mass spectrometry. Limits of detection (LOD) for each cycle of NHANES are provided in Table_Apx G-1. Values below the LOD were replaced by the lower limit of detection divided by the square root of two (NCHS, 2021).

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

NHANES Cycle	MiBP	MHiBP
2001–2002	0.94	–
2003–2004	0.26	–
2005–2006	0.3	–
2007–2008	0.3	–
2009–2010	0.2	–
2011–2012	0.2	–
2013–2014	0.8	–
2015–2016	0.8	0.4
2017–2018	0.8	0.4

2641 **Table_Apx G-2. Summary of Urinary DIBP 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	MHiBP	Adults	All adults	1,896	1,896 (93.57%)	2.3 (1.9–2.4)	10.7 (8–23.2)	2.13 (1.87–2.41)	10.44 (8.36–13.3)
2017–2018	MHiBP	Adults	All adults	1,896	1,896 (97.31%)	2.3 (1.9–2.4)	10.7 (8–23.2)	2.13 (1.87–2.41)	10.44 (8.36–13.3)
2017–2018	MHiBP	Adults	At or above poverty level	467	467 (94%)	2.3 (1.8–2.5)	9.5 (7.1–28.2)	2.04 (1.79–2.33)	9.74 (7.92–13.59)
2017–2018	MHiBP	Adults	At or above poverty level	467	467 (97.22%)	2.3 (1.8–2.5)	9.5 (7.1–28.2)	2.04 (1.79–2.33)	9.74 (7.92–13.59)
2017–2018	MHiBP	Adults	Below poverty level	337	337 (94.07%)	2.4 (1.6–3.3)	14.5 (7.1–26.5)	2.62 (2.24–3.06)	14.71 (10.85–18.6)
2017–2018	MHiBP	Adults	Below poverty level	337	337 (97.63%)	2.4 (1.6–3.3)	14.5 (7.1–26.5)	2.62 (2.24–3.06)	14.71 (10.85–18.6)
2017–2018	MHiBP	Adults	Black non-Hispanic	438	438 (95.89%)	2.7 (2–3.3)	11.1 (6–12.2)	1.94 (1.73–2.2)	9.09 (7.12–11.77)
2017–2018	MHiBP	Adults	Black non-Hispanic	438	438 (98.63%)	2.7 (2–3.3)	11.1 (6–12.2)	1.94 (1.73–2.2)	9.09 (7.12–11.77)
2017–2018	MHiBP	Adults	Females	952	952 (91.91%)	2.6 (2.3–2.9)	11.3 (9.1–16.2)	2.67 (2.14–2.99)	10.52 (9.17–13.42)
2017–2018	MHiBP	Adults	Females	952	952 (96.95%)	2.6 (2.3–2.9)	11.3 (9.1–16.2)	2.67 (2.14–2.99)	10.52 (9.17–13.42)
2017–2018	MHiBP	Adults	Males	944	944 (95.23%)	2.3 (1.9–2.4)	10.6 (7.5–28.2)	1.88 (1.67–2.07)	10.24 (7.21–14.72)
2017–2018	MHiBP	Adults	Males	944	944 (97.67%)	2.3 (1.9–2.4)	10.6 (7.5–28.2)	1.88 (1.67–2.07)	10.24 (7.21–14.72)
2017–2018	MHiBP	Adults	Mexican American	278	278 (95.68%)	2.3 (1.8–3.1)	9 (6.1–99.2)	2.61 (2.06–2.98)	13.11 (8.75–23.28)
2017–2018	MHiBP	Adults	Mexican American	278	278 (98.92%)	2.3 (1.8–3.1)	9 (6.1–99.2)	2.61 (2.06–2.98)	13.11 (8.75–23.28)
2017–2018	MHiBP	Adults	Other	532	532 (91.54%)	2.1 (1.6–2.4)	10.8 (5.1–34.8)	2.55 (2.22–3.06)	13.55 (9.55–17.89)
2017–2018	MHiBP	Adults	Other	532	532 (96.05%)	2.1 (1.6–2.4)	10.8 (5.1–34.8)	2.55 (2.22–3.06)	13.55 (9.55–17.89)
2017–2018	MHiBP	Adults	Unknown income	840	840 (93.21%)	2.1 (1.5–3.6)	21.7 (4.1–34.8)	2.31 (1.65–2.81)	8.89 (6.01–16.65)
2017–2018	MHiBP	Adults	Unknown income	840	840 (96.9%)	2.1 (1.5–3.6)	21.7 (4.1–34.8)	2.31 (1.65–2.81)	8.89 (6.01–16.65)
2017–2018	MHiBP	Adults	White non-Hispanic	648	648 (92.75%)	2.1 (1.8–2.4)	10.7 (6.1–48.4)	2 (1.75–2.31)	9.8 (7.89–13.59)
2017–2018	MHiBP	Adults	White non-Hispanic	648	648 (96.76%)	2.1 (1.8–2.4)	10.7 (6.1–48.4)	2 (1.75–2.31)	9.8 (7.89–13.59)
2017–2018	MiBP	Adults	All adults	1,896	1,896 (93.57%)	7.5 (6.6–8.8)	33.6 (25.7–83.9)	6.81 (6.16–7.44)	32.27 (26.06–38.35)
2017–2018	MiBP	Adults	All adults	1,896	1,896 (97.31%)	7.5 (6.6–8.8)	33.6 (25.7–83.9)	6.81 (6.16–7.44)	32.27 (26.06–38.35)
2017–2018	MiBP	Adults	At or above poverty level	467	467 (94%)	7.5 (6.5–9.1)	32.1 (24.5–83.9)	6.67 (6.08–7.13)	31.5 (23.51–36.67)
2017–2018	MiBP	Adults	At or above poverty level	467	467 (97.22%)	7.5 (6.5–9.1)	32.1 (24.5–83.9)	6.67 (6.08–7.13)	31.5 (23.51–36.67)
2017–2018	MiBP	Adults	Below poverty level	337	337 (94.07%)	8 (5–11)	54.8 (23.5–95.6)	8.4 (7.12–9.71)	45.64 (32.63–63.74)
2017–2018	MiBP	Adults	Below poverty level	337	337 (97.63%)	8 (5–11)	54.8 (23.5–95.6)	8.4 (7.12–9.71)	45.64 (32.63–63.74)
2017–2018	MiBP	Adults	Black non-Hispanic	438	438 (95.89%)	10.3 (7.4–14.2)	33.5 (22.8–61.4)	6.94 (6.23–7.43)	28.75 (23.51–40.57)
2017–2018	MiBP	Adults	Black non-Hispanic	438	438 (98.63%)	10.3 (7.4–14.2)	33.5 (22.8–61.4)	6.94 (6.23–7.43)	28.75 (23.51–40.57)
2017–2018	MiBP	Adults	Females	952	952 (91.91%)	8.1 (7.4–9)	42.4 (31.9–53.6)	7.7 (6.67–9)	31.82 (26.18–38.18)
2017–2018	MiBP	Adults	Females	952	952 (96.95%)	8.1 (7.4–9)	42.4 (31.9–53.6)	7.7 (6.67–9)	31.82 (26.18–38.18)
2017–2018	MiBP	Adults	Males	944	944 (95.23%)	7.5 (6.6–9.1)	33.4 (24.9–83.9)	6.21 (5.66–6.92)	32.27 (23.52–46.74)
2017–2018	MiBP	Adults	Males	944	944 (97.67%)	7.5 (6.6–9.1)	33.4 (24.9–83.9)	6.21 (5.66–6.92)	32.27 (23.52–46.74)

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	MiBP	Adults	Mexican American	278	278 (95.68%)	7.9 (4.8–12)	26.8 (17.5–367.4)	8.55 (7.65–9.15)	38.35 (26.32–56.56)
2017–2018	MiBP	Adults	Mexican American	278	278 (98.92%)	7.9 (4.8–12)	26.8 (17.5–367.4)	8.55 (7.65–9.15)	38.35 (26.32–56.56)
2017–2018	MiBP	Adults	Other	532	532 (91.54%)	7.5 (4.7–10.2)	30.2 (21–143.4)	8.24 (7.36–8.99)	38.27 (27.22–54.59)
2017–2018	MiBP	Adults	Other	532	532 (96.05%)	7.5 (4.7–10.2)	30.2 (21–143.4)	8.24 (7.36–8.99)	38.27 (27.22–54.59)
2017–2018	MiBP	Adults	Unknown income	840	840 (93.21%)	6.6 (4.2–11.6)	62.3 (16.6–143.4)	7 (5.29–8.24)	29.75 (19.17–55.64)
2017–2018	MiBP	Adults	Unknown income	840	840 (96.9%)	6.6 (4.2–11.6)	62.3 (16.6–143.4)	7 (5.29–8.24)	29.75 (19.17–55.64)
2017–2018	MiBP	Adults	White non-Hispanic	648	648 (92.75%)	7 (6–8.8)	33.4 (22.7–188.2)	6.25 (5.66–6.99)	30.52 (22.52–36.67)
2017–2018	MiBP	Adults	White non-Hispanic	648	648 (96.76%)	7 (6–8.8)	33.4 (22.7–188.2)	6.25 (5.66–6.99)	30.52 (22.52–36.67)
2015–2016	MHiBP	Adults	All adults	1,880	1,880 (95.16%)	2.7 (2.3–2.9)	15.3 (10.7–19.2)	2.55 (2.24–2.87)	11.43 (9.92–13.72)
2015–2016	MHiBP	Adults	All adults	1,880	1,880 (98.09%)	2.7 (2.3–2.9)	15.3 (10.7–19.2)	2.55 (2.24–2.87)	11.43 (9.92–13.72)
2015–2016	MHiBP	Adults	At or above poverty level	461	461 (95.01%)	2.4 (1.9–2.9)	15.3 (10.4–18.5)	2.5 (2.16–2.91)	10.2 (9.19–12.86)
2015–2016	MHiBP	Adults	At or above poverty level	461	461 (99.13%)	2.4 (1.9–2.9)	15.3 (10.4–18.5)	2.5 (2.16–2.91)	10.2 (9.19–12.86)
2015–2016	MHiBP	Adults	Below poverty level	399	399 (95.74%)	2.9 (2.5–3.6)	16.8 (8.5–30.4)	2.92 (2.55–3.5)	15.87 (13.74–19.2)
2015–2016	MHiBP	Adults	Below poverty level	399	399 (97.99%)	2.9 (2.5–3.6)	16.8 (8.5–30.4)	2.92 (2.55–3.5)	15.87 (13.74–19.2)
2015–2016	MHiBP	Adults	Black non-Hispanic	427	427 (96.49%)	3 (2.4–4.3)	11.8 (8.7–15.8)	2.6 (2.3–2.96)	11.54 (9.7–14.25)
2015–2016	MHiBP	Adults	Black non-Hispanic	427	427 (99.06%)	3 (2.4–4.3)	11.8 (8.7–15.8)	2.6 (2.3–2.96)	11.54 (9.7–14.25)
2015–2016	MHiBP	Adults	Females	984	984 (94.92%)	3.2 (2.7–3.5)	18.5 (12.7–19.6)	3.33 (2.97–3.66)	15.17 (11.3–20.31)
2015–2016	MHiBP	Adults	Females	984	984 (97.76%)	3.2 (2.7–3.5)	18.5 (12.7–19.6)	3.33 (2.97–3.66)	15.17 (11.3–20.31)
2015–2016	MHiBP	Adults	Males	896	896 (95.42%)	2.6 (2.2–2.9)	15.5 (10.5–19.6)	2.15 (1.94–2.47)	9.51 (8.92–10.2)
2015–2016	MHiBP	Adults	Males	896	896 (98.44%)	2.6 (2.2–2.9)	15.5 (10.5–19.6)	2.15 (1.94–2.47)	9.51 (8.92–10.2)
2015–2016	MHiBP	Adults	Mexican American	342	342 (95.61%)	2.7 (1.6–3.8)	12.8 (5.7–68.3)	2.97 (2.76–3.23)	14.33 (12.11–16.43)
2015–2016	MHiBP	Adults	Mexican American	342	342 (97.66%)	2.7 (1.6–3.8)	12.8 (5.7–68.3)	2.97 (2.76–3.23)	14.33 (12.11–16.43)
2015–2016	MHiBP	Adults	Other	540	540 (94.81%)	2.8 (2–3.7)	20.7 (10.5–29.2)	2.77 (2.34–3.16)	12.72 (10.22–14.93)
2015–2016	MHiBP	Adults	Other	540	540 (98.33%)	2.8 (2–3.7)	20.7 (10.5–29.2)	2.77 (2.34–3.16)	12.72 (10.22–14.93)
2015–2016	MHiBP	Adults	Unknown income	833	833 (94.96%)	3 (2–4.4)	10.7 (3.7–15.5)	2.18 (1.93–2.67)	9.67 (7.27–12.11)
2015–2016	MHiBP	Adults	Unknown income	833	833 (97.24%)	3 (2–4.4)	10.7 (3.7–15.5)	2.18 (1.93–2.67)	9.67 (7.27–12.11)
2015–2016	MHiBP	Adults	White non-Hispanic	571	571 (94.22%)	2.5 (1.9–2.8)	15.5 (8.5–19.6)	2.4 (2.06–2.8)	10.15 (9.04–13.74)
2015–2016	MHiBP	Adults	White non-Hispanic	571	571 (97.37%)	2.5 (1.9–2.8)	15.5 (8.5–19.6)	2.4 (2.06–2.8)	10.15 (9.04–13.74)
2015–2016	MiBP	Adults	All adults	1,880	1,880 (95.16%)	9.3 (7.9–10.7)	48 (34.3–57.2)	8.21 (7.31–8.91)	33.41 (28.33–39.91)
2015–2016	MiBP	Adults	All adults	1,880	1,880 (98.09%)	9.3 (7.9–10.7)	48 (34.3–57.2)	8.21 (7.31–8.91)	33.41 (28.33–39.91)
2015–2016	MiBP	Adults	At or above poverty level	461	461 (95.01%)	9.1 (7.6–10.7)	49 (33.4–57.2)	7.97 (7.12–9)	30.15 (26.84–34.62)
2015–2016	MiBP	Adults	At or above poverty level	461	461 (99.13%)	9.1 (7.6–10.7)	49 (33.4–57.2)	7.97 (7.12–9)	30.15 (26.84–34.62)
2015–2016	MiBP	Adults	Below poverty level	399	399 (95.74%)	9.4 (7.9–13)	42.5 (26.8–88.3)	9.19 (8–10.96)	47.5 (39.91–57.58)

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	MiBP	Adults	Below poverty level	399	399 (97.99%)	9.4 (7.9–13)	42.5 (26.8–88.3)	9.19 (8–10.96)	47.5 (39.91–57.58)
2015–2016	MiBP	Adults	Black non-Hispanic	427	427 (96.49%)	9.8 (7.9–14.4)	38.2 (27.1–51.2)	8.91 (8.11–9.87)	40 (28.57–52.83)
2015–2016	MiBP	Adults	Black non-Hispanic	427	427 (99.06%)	9.8 (7.9–14.4)	38.2 (27.1–51.2)	8.91 (8.11–9.87)	40 (28.57–52.83)
2015–2016	MiBP	Adults	Females	984	984 (94.92%)	9.7 (8.3–10.7)	44.9 (34.2–56.7)	9.78 (8.79–10.92)	44.53 (31.64–53.93)
2015–2016	MiBP	Adults	Females	984	984 (97.76%)	9.7 (8.3–10.7)	44.9 (34.2–56.7)	9.78 (8.79–10.92)	44.53 (31.64–53.93)
2015–2016	MiBP	Adults	Males	896	896 (95.42%)	9.2 (7.7–10.6)	48 (33.4–57.2)	7.26 (6.37–8.4)	28.33 (25–30.97)
2015–2016	MiBP	Adults	Males	896	896 (98.44%)	9.2 (7.7–10.6)	48 (33.4–57.2)	7.26 (6.37–8.4)	28.33 (25–30.97)
2015–2016	MiBP	Adults	Mexican American	342	342 (95.61%)	9.8 (5.7–13.3)	44.7 (23.7–88.3)	9.19 (8.67–10.55)	42.49 (34.52–48.08)
2015–2016	MiBP	Adults	Mexican American	342	342 (97.66%)	9.8 (5.7–13.3)	44.7 (23.7–88.3)	9.19 (8.67–10.55)	42.49 (34.52–48.08)
2015–2016	MiBP	Adults	Other	540	540 (94.81%)	9.3 (6.9–12.9)	67.2 (35.7–116.4)	8.74 (7.64–9.92)	38.96 (33–44.94)
2015–2016	MiBP	Adults	Other	540	540 (98.33%)	9.3 (6.9–12.9)	67.2 (35.7–116.4)	8.74 (7.64–9.92)	38.96 (33–44.94)
2015–2016	MiBP	Adults	Unknown income	833	833 (94.96%)	9.3 (8–19.1)	34.8 (21.2–62)	7.98 (6.34–8.82)	29.14 (20–39.41)
2015–2016	MiBP	Adults	Unknown income	833	833 (97.24%)	9.3 (8–19.1)	34.8 (21.2–62)	7.98 (6.34–8.82)	29.14 (20–39.41)
2015–2016	MiBP	Adults	White non-Hispanic	571	571 (94.22%)	8.9 (7.6–10.6)	47 (26.2–57.2)	7.48 (6.74–8.71)	29.02 (23.94–35.12)
2015–2016	MiBP	Adults	White non-Hispanic	571	571 (97.37%)	8.9 (7.6–10.6)	47 (26.2–57.2)	7.48 (6.74–8.71)	29.02 (23.94–35.12)
2013–2014	MiBP	Adults	All adults	2,040	2,040 (97.01%)	8.5 (7–9.8)	42.4 (29.5–49.9)	6.67 (6.24–7.01)	25.95 (22.16–30.21)
2013–2014	MiBP	Adults	At or above poverty level	484	484 (96.69%)	8.4 (6.5–9.9)	38.3 (28.4–52.1)	6.43 (5.98–6.81)	24.81 (20.22–28.41)
2013–2014	MiBP	Adults	Below poverty level	454	454 (98.46%)	9.2 (7.5–11.1)	45.8 (27.7–101.7)	7.87 (7.01–8.9)	33.75 (25–56.23)
2013–2014	MiBP	Adults	Black non-Hispanic	442	442 (97.29%)	11.8 (10.5–13.7)	58 (35.6–132.7)	7.09 (6.32–7.91)	29.39 (22.78–40.92)
2013–2014	MiBP	Adults	Females	1,076	1,076 (96.56%)	8.8 (7.3–10.7)	46.6 (33.1–58)	7.77 (7.21–8.35)	29.72 (25.32–39.12)
2013–2014	MiBP	Adults	Males	964	964 (97.51%)	8.5 (6.9–9.8)	42.4 (29.3–51.4)	6.07 (5.72–6.59)	23.24 (18.51–28.81)
2013–2014	MiBP	Adults	Mexican American	282	282 (98.58%)	6.5 (4.2–9.8)	35.5 (24.6–57.1)	8.39 (6.61–10)	31.48 (26.75–45.39)
2013–2014	MiBP	Adults	Other	496	496 (97.18%)	8.8 (7.2–10.2)	110.1 (29.5–180.4)	7.85 (6.77–9.26)	43.67 (30.46–63.97)
2013–2014	MiBP	Adults	Unknown income	921	921 (96.31%)	7.8 (4.4–13.8)	38.5 (21.1–149.2)	6.22 (5.45–7.8)	39.12 (19.47–45)
2013–2014	MiBP	Adults	White non-Hispanic	820	820 (96.22%)	7.7 (6.2–9.4)	27.8 (26.6–30.1)	6.16 (5.71–6.67)	20.9 (18.46–25.05)
2011-2012	MiBP	Adults	All adults	1,894	1,894 (98.84%)	7 (5.9–8.2)	42.9 (28.8–51.8)	6.48 (5.76–7.01)	27.34 (24.18–33.3)
2011–2012	MiBP	Adults	At or above poverty level	449	449 (99.11%)	6.7 (5.8–7.7)	42 (26.2–59.4)	6.27 (5.61–6.97)	27.34 (23.25–33.81)
2011–2012	MiBP	Adults	Below poverty level	441	441 (98.87%)	8.7 (5.9–10.7)	45 (22–71.4)	7.36 (6.29–8.52)	29.32 (21.42–46.52)
2011–2012	MiBP	Adults	Black non-Hispanic	499	499 (99.4%)	10.2 (8.2–12.5)	43.5 (37.2–57.4)	7.67 (7.02–8.29)	29.81 (22.21–38.09)
2011–2012	MiBP	Adults	Females	933	933 (98.61%)	6.2 (5.2–7.6)	33.7 (26–41.9)	7.58 (6.74–8.1)	28.46 (24.47–34.23)
2011–2012	MiBP	Adults	Males	961	961 (99.06%)	7.1 (6–8.4)	43.3 (28.8–57.4)	5.91 (5.12–6.88)	25.13 (22.21–33.32)
2011–2012	MiBP	Adults	Mexican American	186	186 (99.46%)	6 (4.2–9.6)	31.2 (13.9–95.9)	6.89 (5.26–8.57)	30.55 (18–67.77)
2011–2012	MiBP	Adults	Other	545	545 (98.17%)	7.8 (5.9–12.1)	36 (25.8–74.6)	8.08 (6.96–9.64)	33.24 (25.13–45)

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)
2011–2012	MiBP	Adults	Unknown income	821	821 (98.78%)	6 (3.6–11.2)	24.55 (14.3–38.6)	6.17 (4.86–7.5)	24.27 (15–37.67)
2011–2012	MiBP	Adults	White non-Hispanic	664	664 (98.8%)	6.3 (5.4–7.5)	43.3 (25.4–49.3)	5.71 (5.06–6.67)	25.83 (20.11–33.81)
2009–2010	MiBP	Adults	All adults	2,127	2,127 (99.76%)	8.64 (7.25–9.41)	38.69 (27.68–49.83)	7.3 (6.84–7.9)	26.37 (23.15–31.45)
2009–2010	MiBP	Adults	At or above poverty level	550	550 (99.82%)	8.19 (6.33–9.31)	33.04 (26.01–41.96)	7.06 (6.56–7.43)	23.7 (20.5–28.82)
2009–2010	MiBP	Adults	Below poverty level	469	469 (99.57%)	9.4 (8.1–10.49)	42.52 (31.44–52.31)	8.22 (7.19–9.27)	35.89 (27.13–48.94)
2009–2010	MiBP	Adults	Black non-Hispanic	400	400 (100%)	14.33 (9.96–18.6)	70.15 (37.4–108.26)	9.9 (8.59–11.25)	32.69 (25.17–41.24)
2009–2010	MiBP	Adults	Females	1,040	1,040 (99.9%)	10.2 (8.3–12.79)	46.01 (38.17–58.38)	8.72 (8.13–9.45)	30.18 (25.89–37.89)
2009–2010	MiBP	Adults	Males	1,087	1,087 (99.63%)	8.53 (7–9.41)	38 (26.93–49.84)	6.7 (6.26–7.25)	23.39 (20.91–27.25)
2009–2010	MiBP	Adults	Mexican American	393	393 (99.75%)	10.19 (6.35–15.4)	41.35 (23.48–60.71)	8.48 (7.33–9.59)	40.56 (28.81–48.09)
2009–2010	MiBP	Adults	Other	336	336 (99.7%)	10.52 (7.13–12.7)	47.21 (27.68–72.61)	9.67 (7.83–12.02)	43.7 (25.38–70.7)
2009–2010	MiBP	Adults	Unknown income	905	905 (99.78%)	11.94 (8.55–19.36)	52.63 (33.99–79.97)	9.41 (7.89–10.5)	30.5 (21.75–58.62)
2009–2010	MiBP	Adults	White non-Hispanic	998	998 (99.7%)	6.97 (5.36–8.64)	26.22 (21.72–32.28)	6.53 (6.18–7.02)	21.09 (18.58–24.9)
2007–2008	MiBP	Adults	All adults	2,021	2,021 (97.77%)	8.8 (7.8–9.4)	38.1 (26–44.2)	6.89 (6.49–7.34)	26.87 (23.94–33.02)
2007–2008	MiBP	Adults	At or above poverty level	505	505 (98.61%)	8.8 (7.8–9.5)	36.5 (24.1–46.5)	6.67 (6.26–7.08)	25.79 (21.3–32.42)
2007–2008	MiBP	Adults	Below poverty level	392	392 (98.21%)	9.7 (7.1–12.2)	44.2 (28.9–48.1)	8.67 (7.42–10.13)	36.89 (25.3–56.39)
2007–2008	MiBP	Adults	Black non-Hispanic	434	434 (98.62%)	11.6 (7.8–16.3)	46.3 (30.3–66.8)	8.06 (7.32–9.11)	27.01 (24.13–35.42)
2007–2008	MiBP	Adults	Females	1,030	1,030 (97.48%)	9.8 (7.8–12.6)	46.7 (36.1–54.8)	8.67 (7.73–9.62)	33.39 (24.93–52.6)
2007–2008	MiBP	Adults	Males	991	991 (98.08%)	8.8 (7.8–9.4)	37.8 (25.5–44.2)	6.36 (5.93–6.75)	24.12 (21.1–27.73)
2007–2008	MiBP	Adults	Mexican American	371	371 (99.46%)	10.2 (8.7–12.2)	47.6 (26–66)	8.7 (7.88–9.66)	33.49 (27.59–38.26)
2007–2008	MiBP	Adults	Other	294	294 (99.32%)	9.3 (5.8–14.5)	43.3 (14.6–74.4)	8.92 (7.44–11.39)	38.18 (25.83–56.39)
2007–2008	MiBP	Adults	Unknown income	948	948 (97.15%)	7.6 (4.7–10.1)	22.7 (14.6–38.9)	6.88 (5.58–8.3)	26 (19.31–121.09)
2007–2008	MiBP	Adults	White non-Hispanic	922	922 (96.2%)	8 (7.1–9.1)	25.6 (21.3–42.5)	6.33 (6.04–6.67)	24.46 (18.88–29.23)
2005–2006	MiBP	Adults	All adults	1,831	1,831 (97.21%)	6.5 (5.6–7.3)	34.1 (23.9–42.8)	4.9 (4.58–5.19)	18.98 (18.04–21.74)
2005–2006	MiBP	Adults	At or above poverty level	436	436 (96.79%)	6.4 (5.5–7.2)	32.1 (22.8–42.8)	4.77 (4.53–5)	18.78 (16.95–21.57)
2005–2006	MiBP	Adults	Below poverty level	340	340 (97.06%)	8 (5.7–10.4)	33.1 (22.3–45)	5.82 (5–6.95)	24.31 (18.06–32.84)
2005–2006	MiBP	Adults	Black non-Hispanic	464	464 (99.57%)	8 (6.9–9.6)	37.2 (23.7–73.8)	5.53 (4.82–6.7)	19.8 (15.06–28.07)
2005–2006	MiBP	Adults	Females	935	935 (96.68%)	6.3 (5.3–7.9)	35.1 (23.6–57.1)	6.15 (5.42–6.71)	23.3 (18.25–28.14)
2005–2006	MiBP	Adults	Males	896	896 (97.77%)	6.5 (5.6–7.4)	33.1 (23.8–42.8)	4.44 (4.21–4.79)	18.46 (16.78–19.81)
2005–2006	MiBP	Adults	Mexican American	390	390 (97.44%)	7.3 (5–9.8)	42.8 (22–60.3)	5.99 (5.05–7)	27.67 (23.13–36.26)
2005–2006	MiBP	Adults	Other	131	131 (99.24%)	10.2 (6.7–11.8)	45.8 (13.6–88.4)	6.43 (5.26–7.06)	24.33 (16.85–40.94)
2005–2006	MiBP	Adults	Unknown income	955	955 (97.49%)	6.9 (3.4–15.1)	36 (7.3–148.1)	5.12 (4.19–7.64)	18.65 (13.56–31.89)
2005–2006	MiBP	Adults	White non-Hispanic	846	846 (95.51%)	5.5 (4.3–6.5)	26.5 (20.3–37.5)	4.49 (4.29–4.75)	18.15 (16.74–18.98)
2003–2004	MiBP	Adults	All adults	1,889	1,889 (96.66%)	4.3 (3.7–5.1)	19.9 (16–27.8)	3.4 (3.04–3.95)	15.4 (12.97–18.72)

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	MiBP	Adults	At or above poverty level	474	474 (96.2%)	4 (3.4–4.7)	18.2 (15.4–23.3)	3.26 (2.99–3.71)	13.88 (11.54–17.3)
2003–2004	MiBP	Adults	Below poverty level	393	393 (96.95%)	4.9 (3.4–7.1)	21.8 (16.5–26)	4.07 (3.54–4.6)	24.6 (15.4–40.45)
2003–2004	MiBP	Adults	Black non-Hispanic	423	423 (99.29%)	7.05 (6.2–9.2)	32.9 (16.7–72.9)	4.59 (3.85–5.44)	18.72 (15.79–25.58)
2003–2004	MiBP	Adults	Females	980	980 (96.02%)	4.8 (4.2–5.5)	21.8 (18.8–26)	3.95 (3.33–4.54)	16.22 (13.89–20)
2003–2004	MiBP	Adults	Males	909	909 (97.36%)	4.1 (3.5–5.1)	19.9 (15.6–27.9)	3.19 (2.74–3.56)	13.78 (11.52–19.59)
2003–2004	MiBP	Adults	Mexican American	423	423 (96.93%)	4.9 (3.9–5.9)	19 (17.7–22.5)	4.47 (3.38–5.14)	22.65 (17.86–26.54)
2003–2004	MiBP	Adults	Other	142	142 (97.89%)	7 (4.3–11.9)	23.1 (16–34.3)	5.79 (3.75–8.82)	23.29 (18.46–40.45)
2003–2004	MiBP	Adults	Unknown income	904	904 (96.57%)	5.9 (3.5–6.9)	30.3 (8.5–183.2)	4.14 (2.66–5.8)	16.93 (8.92–31.3)
2003–2004	MiBP	Adults	White non-Hispanic	901	901 (95.12%)	3.5 (2.9–4)	16.5 (13.2–19.6)	3.06 (2.64–3.4)	11.96 (9.72–15.11)
2001–2002	MiBP	Adults	All adults	2,004	2,004 (98.1%)	3 (2.5–3.6)	16.2 (13.3–22)	2.41 (2.2–2.58)	11.88 (10.55–13.85)
2001–2002	MiBP	Adults	At or above poverty level	463	463 (96.98%)	3.1 (2.4–3.7)	14.4 (12.2–18.9)	2.28 (2.11–2.5)	10.5 (10–11.59)
2001–2002	MiBP	Adults	Below poverty level	361	361 (98.89%)	3.6 (2.4–5)	16.4 (10.2–34)	3.13 (2.69–3.63)	19.2 (10.39–54.07)
2001–2002	MiBP	Adults	Black non-Hispanic	414	414 (99.52%)	5.6 (4.8–6.6)	18.9 (14–40.8)	3.37 (2.81–3.64)	15.23 (12.5–17.59)
2001–2002	MiBP	Adults	Females	1,019	1,019 (98.14%)	2.8 (2.5–3.9)	22.6 (14.7–31.7)	2.95 (2.69–3.2)	13.82 (10.16–19.07)
2001–2002	MiBP	Adults	Males	985	985 (98.07%)	3.1 (2.5–3.6)	15.9 (12.6–22.2)	2.16 (1.97–2.37)	10.89 (10.11–12.5)
2001–2002	MiBP	Adults	Mexican American	445	445 (98.43%)	3.1 (2.3–4.4)	17.1 (11.2–31.3)	2.97 (2.46–3.71)	14 (11.96–19.37)
2001–2002	MiBP	Adults	Other	162	162 (96.91%)	3.7 (2.1–6)	17.8 (12.3–41.6)	2.92 (2.06–4.36)	13.5 (8–40.34)
2001–2002	MiBP	Adults	Unknown income	1,052	1,052 (98.29%)	3.2 (2.3–6.6)	55.3 (4.6–55.3)	2.31 (1.63–2.78)	11.94 (8.18–24.04)
2001–2002	MiBP	Adults	White non-Hispanic	983	983 (97.56%)	2.8 (1.9–3.3)	13.5 (9.6–24)	2.18 (1.96–2.41)	10.5 (9.23–12.03)

Table_Apx G-3. Regression Coefficients and P-Values for Statistical Analyses of DIBP Concentrations

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	p-value, 50th Percentile	Regression Coefficient, 95th Percentile	p-value, 95th Percentile
2013–2018	MHiBP	Adults	All adults	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Adults	All adults	Income	age sex race	–[a]	0.022	–[a]	<0.001
2013–2018	MHiBP	Adults	All adults	Race	age sex income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Adults	All adults	Sex	age race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Adults	All adults	Years	age sex race income	–0.3686	<0.001	–1.2150	<0.001
2013–2018	MHiBP	Adults	All adults	Years	age sex race income	–0.3686	<0.001	–1.2150	<0.001
2013–2018	MHiBP	Adults	At or above poverty level	Years	age sex race	–0.1791	0.0083	–0.1224	0.171
2013–2018	MHiBP	Adults	At or above poverty level	years	age sex race	–0.1791	0.0083	–0.1224	0.171

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th Percentile	p-value, 50th Percentile	Regression Coefficient, 95th Percentile	p-value, 95th Percentile
2013–2018	MHiBP	Adults	Below poverty level	Years	age sex race	1.36300	<0.001	0.77725	<0.001
2013–2018	MHiBP	Adults	Below poverty level	Years	age sex race	1.36300	<0.001	0.77725	<0.001
2013–2018	MHiBP	Adults	Black non-Hispanic	Years	age sex income	0.93895	<0.001	1.95162	<0.001
2013–2018	MHiBP	Adults	Black non-Hispanic	Years	age sex income	0.93895	<0.001	1.95162	<0.001
2013–2018	MHiBP	Adults	Females	Years	age race income	–0.2655	<0.001	0.12568	0.0941
2013–2018	MHiBP	Adults	Females	Years	age race income	–0.2655	<0.001	0.12568	0.0941
2013–2018	MHiBP	Adults	Males	Years	age race income	–0.1805	0.0136	–1.0822	<0.001
2013–2018	MHiBP	Adults	Males	Years	age race income	–0.1805	0.0136	–1.0822	<0.001
2013–2018	MHiBP	Adults	Mexican-American	Years	age sex income	–0.1788	0.0885	–1.0114	<0.001
2013–2018	MHiBP	Adults	Mexican-American	Years	age sex income	–0.1788	0.0885	–1.0114	<0.001
2013–2018	MHiBP	Adults	Other	Years	age sex income	–0.1050	0.3324	–1.7379	<0.001
2013–2018	MHiBP	Adults	Other	Years	age sex income	–0.1050	0.3324	–1.7379	<0.001
2013–2018	MHiBP	Adults	Unknown income	Years	age sex race	–4.5943	<0.001	–5.0401	<0.001
2013–2018	MHiBP	Adults	Unknown income	Years	age sex race	–4.5943	<0.001	–5.0401	<0.001
2013–2018	MHiBP	Adults	White non-Hispanic	Years	age sex income	–0.5763	<0.001	–0.6830	<0.001
2013–2018	MHiBP	Adults	White non-Hispanic	Years	age sex income	–0.5763	<0.001	–0.6830	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Income	age sex race	–[a]	0.9609	–[a]	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Race	age sex income	–[a]	0.0066	–[a]	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Sex	age race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Children	Adolescents (11–<16 years old)	Years	sex race income	0.31389	0.0167	–1.2537	<0.001
2013–2018	MHiBP	Children	Adolescents (11–<16 years old)	Years	sex race income	0.31389	0.0167	–1.2537	<0.001
2013–2018	MHiBP	Children	Toddlers (3–<6 years old)	Years	sex race income	0.13701	0.4935	–3.0511	<0.001
2013–2018	MHiBP	Children	Toddlers (3–<6 years old)	Years	sex race income	0.13701	0.4935	–3.0511	<0.001
2013–2018	MHiBP	Children	Children (6–<10 years old)	Years	sex race income	–0.5987	<0.001	–2.8074	<0.001
2013–2018	MHiBP	Children	Children (6–<10 years old)	Years	sex race income	–0.5987	<0.001	–2.8074	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Years	age sex race income	–0.214	0.0027	–1.3839	<0.001
2013–2018	MHiBP	Children	All children (<16 years old)	Years	age sex race income	–0.214	0.0027	–1.3839	<0.001
2013–2018	MHiBP	Children	At or above poverty level	Years	age sex race	–0.0023	0.9838	–2.4178	<0.001
2013–2018	MHiBP	Children	At or above poverty level	Years	age sex race	–0.0023	0.9838	–2.4178	<0.001
2013–2018	MHiBP	Children	Below poverty level	Years	age sex race	–0.1265	0.3384	–1.7698	<0.001
2013–2018	MHiBP	Children	Below poverty level	years	age sex race	–0.1265	0.3384	–1.7698	<0.001
2013–2018	MHiBP	Children	Black non-Hispanic	years	age sex income	–0.4374	0.1033	–4.2884	<0.001
2013–2018	MHiBP	Children	Black non-Hispanic	Years	age sex income	–0.4374	0.1033	–4.2884	<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
2013–2018	MHiBP	Children	Females	Years	age race income	0.21878	0.0742	–2.2196	<0.001
2013–2018	MHiBP	Children	Females	Years	age race income	0.21878	0.0742	–2.2196	<0.001
2013–2018	MHiBP	Children	Males	Years	age race income	–0.2000	0.089	–2.1054	<0.001
2013–2018	MHiBP	Children	Males	Years	age race income	–0.2000	0.089	–2.1054	<0.001
2013–2018	MHiBP	Children	Mexican-American	Years	age sex income	0.58504	0.0034	–1.9265	<0.001
2013–2018	MHiBP	Children	Mexican-American	Years	age sex income	0.58504	0.0034	–1.9265	<0.001
2013–2018	MHiBP	Children	Other	Years	age sex income	–1.3211	<0.001	–1.2658	<0.001
2013–2018	MHiBP	Children	Other	Years	age sex income	–1.3211	<0.001	–1.2658	<0.001
2013–2018	MHiBP	Children	Unknown income	Years	age sex race	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Children	Unknown income	Years	age sex race	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Children	White non-Hispanic	Years	age sex income	0.37433	0.0014	0.37390	0.0144
2013–2018	MHiBP	Children	White non-Hispanic	Years	age sex income	0.37433	0.0014	0.37390	0.0144
2013–2018	MHiBP	Women	All women of reproductive age	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Women	All women of reproductive age	Income	age sex race	–[a]	0.0959	–[a]	<0.001
2013–2018	MHiBP	Women	All women of reproductive age	Race	age sex income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Women	All women of reproductive age	Sex	age race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MHiBP	Women	All women of reproductive age	Years	age sex race income	–0.3137	0.0045	–0.7068	0.0457
2013–2018	MHiBP	Women	At or above poverty level	Years	age sex race	–0.0954	0.4658	–2.8884	0.0023
2013–2018	MHiBP	Women	Below poverty level	Years	age sex race	–1.0773	0.0055	–2.0736	0.1713
2013–2018	MHiBP	Women	Black non-Hispanic	Years	age sex income	0.10587	0.6953	0.32839	0.8179
2013–2018	MHiBP	Women	Females	Years	age race income	–0.3137	0.0045	–0.7068	0.0457
2013–2018	MHiBP	Women	Mexican-American	Years	age sex income	–0.5920	0.0827	–11.545	<0.001
2013–2018	MHiBP	Women	Other	Years	age sex income	0.25735	0.4627	2.15967	<0.001
2013–2018	MHiBP	Women	Unknown income	Years	age sex race	1.07762	0.0214	0.30840	0.4682
2013–2018	MHiBP	Women	White non-Hispanic	Years	age sex income	–0.1599	0.3169	–0.8214	0.6439
2001–2018	MiBP	Adults	All adults	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Adults	All adults	Income	age sex race	–[a]	0.0082	–[a]	<0.001
2001–2018	MiBP	Adults	All adults	Race	age sex income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Adults	All adults	Sex	age race income	–[a]	0.6048	–[a]	<0.001
2001–2018	MiBP	Adults	All adults	Years	age sex race income	0.20012	<0.001	0.33240	<0.001
2001–2018	MiBP	Adults	All adults	Years	age sex race income	0.20012	<0.001	0.33240	<0.001
2001–2018	MiBP	Adults	At or above poverty level	Years	age sex race	0.31524	<0.001	0.61478	<0.001
2001–2018	MiBP	Adults	At or above poverty level	Years	age sex race	0.31524	<0.001	0.61478	<0.001
2001–2018	MiBP	Adults	Below poverty level	Years	age sex race	0.06959	<0.001	0.19347	<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
2001–2018	MiBP	Adults	Below poverty level	Years	age sex race	0.06959	<0.001	0.19347	<0.001
2001–2018	MiBP	Adults	Black non-Hispanic	Years	age sex income	0.46794	<0.001	0.86700	<0.001
2001–2018	MiBP	Adults	Black non-Hispanic	Years	age sex income	0.46794	<0.001	0.86700	<0.001
2001–2018	MiBP	Adults	Females	Years	age race income	0.1047	<0.001	0.55412	<0.001
2001–2018	MiBP	Adults	Females	Years	age race income	0.1047	<0.001	0.55412	<0.001
2001–2018	MiBP	Adults	Males	Years	age race income	0.2729	<0.001	0.18563	<0.001
2001–2018	MiBP	Adults	Males	Years	age race income	0.2729	<0.001	0.18563	<0.001
2001–2018	MiBP	Adults	Mexican-American	Years	age sex income	0.01233	0.1804	0.00367	0.8029
2001–2018	MiBP	Adults	Mexican-American	Years	age sex income	0.01233	0.1804	0.00367	0.8029
2001–2018	MiBP	Adults	Other	Years	age sex income	0.2570	<0.001	1.25342	<0.001
2001–2018	MiBP	Adults	Other	Years	age sex income	0.2570	<0.001	1.25342	<0.001
2001–2018	MiBP	Adults	Unknown income	Years	age sex race	0.0409	0.0546	–0.192	<0.001
2001–2018	MiBP	Adults	Unknown income	Years	age sex race	0.0409	0.0546	–0.192	<0.001
2001–2018	MiBP	Adults	White non-Hispanic	Years	age sex income	0.322	<0.001	0.22987	<0.001
2001–2018	MiBP	Adults	White non-Hispanic	Years	age sex income	0.322	<0.001	0.22987	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Income	age sex race	–[a]	0.1759	–[a]	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Race	age sex income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Sex	age race income	–[a]	0.375	–[a]	0.2507
2001–2018	MiBP	Children	Adolescents (11–<16 years old)	Years	sex race income	0.28824	<0.001	0.1850	<0.001
2001–2018	MiBP	Children	Adolescents (11–<16 years old)	Years	sex race income	0.28824	<0.001	0.1850	<0.001
2001–2018	MiBP	Children	Toddlers (3–<6 years old)	Years	sex race income	–0.006	0.8001	0.48414	<0.001
2001–2018	MiBP	Children	Toddlers (3–<6 years old)	Years	sex race income	–0.006	0.8001	0.48414	<0.001
2001–2018	MiBP	Children	Children (6–<10 years old)	Years	sex race income	0.30894	<0.001	0.78373	<0.001
2001–2018	MiBP	Children	Children (6–<10 years old)	Years	sex race income	0.30894	<0.001	0.78373	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Years	age sex race income	0.14657	<0.001	0.50930	<0.001
2001–2018	MiBP	Children	All children (<16 years old)	Years	age sex race income	0.14657	<0.001	0.50930	<0.001
2001–2018	MiBP	Children	At or above poverty level	Years	age sex race	0.28505	<0.001	0.21288	<0.001
2001–2018	MiBP	Children	At or above poverty level	Years	age sex race	0.28505	<0.001	0.21288	<0.001
2001–2018	MiBP	Children	Below poverty level	Years	age sex race	0.16756	<0.001	0.6852	<0.001
2001–2018	MiBP	Children	Below poverty level	Years	age sex race	0.16756	<0.001	0.6852	<0.001
2001–2018	MiBP	Children	Black non-Hispanic	Years	age sex income	0.4272	<0.001	0.97083	<0.001
2001–2018	MiBP	Children	Black non-Hispanic	Years	age sex income	0.4272	<0.001	0.97083	<0.001
2001–2018	MiBP	Children	Females	Years	age race income	0.09830	<0.001	0.67921	<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
2001–2018	MiBP	Children	Females	Years	age race income	0.09830	<0.001	0.67921	<0.001
2001–2018	MiBP	Children	Males	Years	age race income	0.29792	<0.001	0.56441	<0.001
2001–2018	MiBP	Children	Males	Years	age race income	0.29792	<0.001	0.56441	<0.001
2001–2018	MiBP	Children	Mexican-American	Years	age sex income	0.17717	<0.001	0.50137	<0.001
2001–2018	MiBP	Children	Mexican-American	Years	age sex income	0.17717	<0.001	0.50137	<0.001
2001–2018	MiBP	Children	Other	Years	age sex income	–0.0771	0.0171	0.89832	<0.001
2001–2018	MiBP	Children	Other	Years	age sex income	–0.0771	0.0171	0.89832	<0.001
2001–2018	MiBP	Children	Unknown income	Years	age sex race	0.28458	<0.001	0.87504	<0.001
2001–2018	MiBP	Children	Unknown income	Years	age sex race	0.28458	<0.001	0.87504	<0.001
2001–2018	MiBP	Children	White non-Hispanic	Years	age sex income	0.33549	<0.001	0.6988	<0.001
2001–2018	MiBP	Children	White non-Hispanic	Years	age sex income	0.33549	<0.001	0.6988	<0.001
2001–2018	MiBP	Women	All women of reproductive age	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Women	All women of reproductive age	Income	age sex race	–[a]	0.004	–[a]	0.0768
2001–2018	MiBP	Women	All women of reproductive age	Race	age sex income	–[a]	0.0318	–[a]	<0.001
2001–2018	MiBP	Women	All women of reproductive age	Sex	age race income	–[a]	<0.001	–[a]	<0.001
2001–2018	MiBP	Women	All women of reproductive age	Years	age sex race income	0.30453	<0.001	0.94313	<0.001
2001–2018	MiBP	Women	At or above poverty level	Years	age sex race	0.35143	<0.001	1.35004	<0.001
2001–2018	MiBP	Women	Below poverty level	Years	age sex race	0.21431	<0.001	1.11566	<0.001
2001–2018	MiBP	Women	Black non-Hispanic	Years	age sex income	0.59144	<0.001	–0.4692	0.0431
2001–2018	MiBP	Women	Females	Years	age race income	0.30453	<0.001	0.94313	<0.001
2001–2018	MiBP	Women	Mexican-American	Years	age sex income	0.21859	<0.001	3.45999	<0.001
2001–2018	MiBP	Women	Other	Years	age sex income	0.12255	0.1234	0.00429	0.9854
2001–2018	MiBP	Women	Unknown income	Years	age sex race	0.27994	<0.001	0.88162	<0.001
2001–2018	MiBP	Women	White non-Hispanic	Years	age sex income	0.33024	<0.001	0.67859	<0.001

[a] Statistical test performed was a chi-square analysis and no regression coefficient was calculated