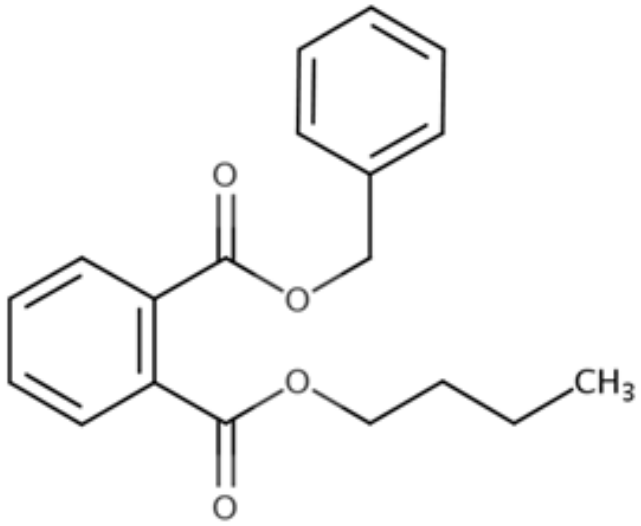
United States
Environmental Protection Agency

EPA-740-D-25-007
July 2025
Office of Chemical Safety and
Pollution Prevention

Draft Environmental Media and General Population and Environmental Exposure for Butyl Benzyl Phthalate (BBP)

Technical Support Document for the Draft Risk Evaluation

CASRN: 85-68-7



July 2025

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF APPENDIX TABLES

LIST OF APPENDIX FIGURES

ABBREVIATIONS AND ACRONYMS

7Q10	Lowest 7-day flow in a 10-year period
ADD	Average daily dose
ADR	Acute dose rate
AERMOD	American Meteorological Society (AMS)/EPA Regulatory Model
BAF	Bioaccumulation factor
BCF	Bioconcentration factor
CDC	Centers for Disease Control and Prevention (U.S.)
CEM	Consumer Exposure Model
COU	Condition of use
DAD	Dermal absorbed dose
DI	Daily intake
DIDP	Di-isodecyl phthalate
DINP	Di-isononyl phthalate
dw	Dry weight
ECHO	EPA's Enforcement and Compliance History Online Database
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
K_{OA}	Octanol:air coefficient
K_{OC}	Organic carbon:water partition coefficent
K_{OW}	Octanol:water partition coefficient
K_p	Dermal permeability coefficient
LADD	Lifetime average daily dose
MCNP	Mono-(carboxynonyl) phthalate
MBzP	Mono-benzyl phthalate
MOE	Margin of exposure
NAICS	North American Industry Classification System
NHANES	National Health and Nutrition Examination Survey
NPDES	National Pollutant Discharge Elimination System
OCSPP	Office of Chemical Safety and Pollution Prevention
OES	Occupational exposure scenario
OPPT	Office of Pollution Prevention and Toxics
PESS	Potentially exposed or susceptible subpopulation(s)
POD	Point of departure
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
UF	Uncertainty factor
ww	Wet weight
WWTP	Wastewater Treatment Plant

SUMMARY

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

EPA evaluated the reasonably available information for various environmental media concentrations and estimated exposure using a conservative scenario as a screening level approach. The conservative high-end exposure was assumed to result from the highest BBP 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 BBP in air, water, and land (soil, biosolids, and groundwater).
  - For the land pathway, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs. However, high-quality physical and chemical property data suggest that BBP will have low persistence potential and mobility in soils. Therefore, groundwater concentrations resulting from releases to the landfill or to agricultural lands via biosolids applications were not quantified but are discussed qualitatively.
  - For the water pathway, BBP in water releases is expected to predominantly partition into sediment. The high-end modeled total water column concentration of BBP for the acute human exposure scenarios was orders of magnitude above any monitored concentration likely due to conservative inputs. Therefore, EPA is confident that the use of the modelled concentration to estimate risk is protective.
  - For the ambient air pathway, modeled BBP concentrations are higher than measured concentrations by several orders of magnitude. This is an expected outcome since EPA's modeling uses high-end releases and conservative meteorological data. Further refinement of the modeled value was not conducted for inhalation because it was not identified as a pathway of concern.
- Screening-level risk estimates using high-end modeled water concentrations were above the benchmark for incidental dermal contact, ingestion from swimming, and ingestion of drinking water. The same is true using high-end modeled air concentrations for inhalation of ambient air. Therefore, no further refinement was necessary.
- For human exposure through fish ingestion, additional refinement of the modeled high-end release was conducted because the screening level analysis indicated potential risks. EPA conducted a refined analysis using the highest estimated 95th percentile release for the PVC plastic compounding Occupational Exposure Scenario (OES) and three additional flow scenarios. In the refined scenarios, which are expected to be more representative of exposures than the high-end screening analysis, no risk was identified.
- EPA concluded that there were no pathways of concern for the general population.
- BBP is not readily found (or if found, is in relatively low concentrations) in organism tissues, and has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms. Therefore, BBP has a low potential for trophic transfer through food webs.

1 ENVIRONMENTAL MEDIA CONCENTRATION OVERVIEW

This technical document supports the *Draft Risk Evaluation for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025j). BBP is a diester of phthalic acid under CASRN 85-68-7. The primary use for BBP is as a plasticizer in polyvinyl chloride (PVC) flooring and other materials, paints and coatings, and adhesives and sealants.

This document describes the use of reasonably available information to estimate environmental concentrations of BBP 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 BBP from facilities that use, manufacture, or process BBP under industrial and/or commercial COUs as detailed in the *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f). Table 1-1 provides a crosswalk between COUs and occupational exposure scenarios (OESs). Table 1-2 shows the types of releases to the environment by OES.

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

Life Cycle Stage	Category	Subcategory	OES
Manufacturing	Domestic manufacturing	Domestic manufacturing	Manufacturing
	Importing	Importing	Import and repackaging
Processing	Repackaging	Repackaging	Import and repackaging
	Incorporation into formulation, mixture, or reaction product	Fillers (*e.g.*, in custom compounding of purchased resin)	PVC plastics compounding; non-PVC materials compounding
		Plasticizers (adhesive manufacturing; paint and coating manufacturing; printing ink manufacturing; all other basic inorganic chemical manufacturing)	Incorporation into adhesives and sealants; paints and coatings; and into other formulation, mixture, or reaction products
		Laboratory chemicals manufacturing	Incorporation into other formulations, mixtures, or reaction products
		Biocide carrier manufacturing	Incorporation into other formulations, mixtures, or reaction products
	Incorporation into articles	Plasticizer in asphalt paving, roofing, and coating materials manufacturing; fabric, textile, and leather products not covered elsewhere manufacturing; floor coverings manufacturing; plastic product manufacturing; rubber product manufacturing; textiles, apparel, and leather manufacturing; transportation	Incorporation into paints and coatings; PVC plastics converting; non-PVC material converting

Life Cycle Stage	Category	Subcategory	OES
		equipment manufacturing	
	Recycling	Recycling	Recycling
Distribution in Commerce	Distribution in commerce	Distribution in commerce	Distribution in commerce
Industrial Uses	Adhesives and sealants	Adhesives and sealants	Application of adhesives and sealants
	Automotive, fuel, agriculture, outdoor use products	Automotive care products	Application of adhesives and sealants; application of paints and coatings
	Castings	Castings	Non-PVC material compounding; non-PVC material converting
	Construction, paint, electrical, and metal products	Building construction materials not covered elsewhere	Fabrication of final product from articles
	Floor coverings	Floor coverings	Application of paints and coatings
	Furnishing, cleaning, treatment/care products	Fabric, textile, and leather products not covered elsewhere	Application of paints and coatings
	Inks, toner and colorant products	Inks, toner and colorant products (*e.g.*, screen printing ink)	Application of paints and coatings
	Plastic and rubber products not covered elsewhere	Plastic and rubber products not covered elsewhere	PVC plastics converting; non-PVC material converting
	Other uses	Chemical intermediate	Incorporation into other formulations, mixtures, or reaction products
		Laboratory chemical	Use of laboratory chemicals
		Plastic and rubber products not covered elsewhere (*e.g.*, component of compound (resin) used to cast models)	PVC plastics converting; non-PVC material converting
	Hydraulic fluids	Hydraulic fluids	Use of lubricants and functional fluids
Commercial Uses	Adhesives and sealants	Adhesives and sealants	Application of adhesives and sealants
	Automotive, fuel, agriculture, outdoor use products	Automotive care products	Application of adhesives and sealants; application of paints and coatings

Life Cycle Stage	Category	Subcategory	OES
	Castings	Castings	Non-PVC material compounding; non-PVC material converting
	Floor coverings	Floor coverings	Application of paints and coatings
	Construction, paint, electrical, and metal products	Building/construction materials not covered elsewhere	Fabrication of final product from articles
	Furnishing, cleaning, treatment/care products	Fabric, textile, and leather products not covered elsewhere	Fabrication of final product from articles
	Inks, toner and colorant products	Inks, toner and colorant products	Application of paints and coatings
	Laboratory chemical	Laboratory chemical	Use of laboratory chemicals
	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
	Other uses	Chemical intermediate	Incorporation into other formulations, mixtures, or reaction products
		Plastic and rubber products not covered elsewhere (*e.g.*, component of compound (resin) used to cast models)	Non-PVC material converting
Disposal	Disposal	Disposal	Waste handling, treatment, and disposal

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
Import and repackaging – liquid	Fugitive air
	Wastewater to onsite treatment, discharge to POTW, or landfill
Import and repackaging – solid	Stack air
	Wastewater, incineration, or landfill
	Fugitive air, wastewater, incineration, or landfill
	Incineration or landfill
Incorporation into adhesives and sealants – liquid	Stack air
	Fugitive air
	Incineration or landfill
	Wastewater, incineration, or landfill
	Fugitive air, wastewater, incineration, or landfill
Incorporation into adhesives and sealants – solid	Stack air
	Fugitive air
	Incineration or landfill
	Wastewater, incineration, or landfill
	Fugitive air, wastewater, incineration, or landfill
Incorporation into paints and coatings	Stack air
	Fugitive air
	Wastewater, incineration, or landfill
Incorporation into other formulations, mixtures, and reaction products	Stack air
	Fugitive air
	Wastewater, incineration, or landfill
PVC plastics compounding	Fugitive or stack air
	Stack air
	Wastewater, incineration, or landfill
	Wastewater
	Fugitive air, wastewater, incineration, or landfill

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
	Incineration or landfill
PVC plastics converting	Fugitive or stack air
	Stack air
	Wastewater, incineration, or landfill
	Wastewater
	Fugitive air, wastewater, incineration, or landfill
	Incineration or landfill
Non-PVC materials compounding – liquid	Fugitive or stack air
	Stack air
	Wastewater, incineration, or landfill
	Wastewater
	Fugitive air, wastewater, incineration, or landfill
	Incineration or landfill
Non-PVC materials compounding – solid	Fugitive or stack air
	Stack air
	Wastewater, incineration, or landfill
	Wastewater
	Fugitive air, wastewater, incineration, or landfill
	Incineration or landfill
Non-PVC material converting	Fugitive or stack air
	Stack air
	Wastewater, incineration, or landfill
	Wastewater
	Fugitive air, wastewater, incineration, or landfill
	Incineration or landfill
Application of adhesives and sealants	Fugitive air
	Incineration, or landfill
	Wastewater, incineration, or landfill
Application of paints and coatings (control technology)	Fugitive air
	Stack air
	Incineration or landfill

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
	Wastewater, incineration, or landfill
Application of paints and coatings (no control technology)	Fugitive air
	Incineration or landfill
	Wastewater, incineration, or landfill
	Unknown (air, wastewater, incineration, or landfill)
Use of laboratory chemicals – liquid	Fugitive or stack air
	Wastewater, incineration, or landfill
Use of laboratory chemicals – solid	Stack air
	Unknown media (air, wastewater, incineration, or landfill)
	Wastewater, incineration, or landfill
	Incineration or landfill
Use of lubricants and functional fluids	Wastewater
	Landfill
	Recycling
	Fuel blending (incineration)
Fabrication of final product from articles – cutting, grinding, shaping, drilling, abrading, and similar activities	Fugitive or stack air, wastewater, incineration, or landfill
Fabrication of final product from articles – heating/plastic welding activities	Fugitive or stack air
Recycling	Stack air
	Fugitive air, wastewater, incineration, or landfill
	Wastewater
	Wastewater, incineration, or landfill
Waste handling, treatment, and disposal	Releases to all media are possible but non-quantifiable due to a lack of identified process- and product-specific data.

[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 BBP from the largest estimated releases for its screening level assessment of environmental and general population exposures. This means that EPA considered the concentration of BBP in a given environmental media resulting from the OES that had the highest release compared to the other OES(s).

The OES resulting in the highest environmental concentration of BBP varied by environmental media as shown in Table 2-1. Additionally, EPA relied on its fate assessment to determine which environmental pathways to consider. Details on the environmental partitioning and media assessment can be found in *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025i). Briefly, based on BBP's fate parameters (*e.g.*, Henry's Law constant, log K_{OC}, water solubility, and fugacity modeling estimates), EPA anticipates BBP to be predominantly in water, soil, and sediment. However, because BBP is released to the ambient air from industrial facilities and processes, inhalation of ambient air is a possible exposure pathway. EPA thus quantitatively assessed concentrations of BBP in surface water, sediment, and ambient air. Soil concentrations of BBP from land application of biosolids were not quantitatively assessed as BBP was expected to have limited persistence potential and mobility in soils receiving biosolids.

Environmental exposures calculated using the predicted concentrations of BBP are presented in Section 2.1. As BBP 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, 2025i). Additionally, EPA discusses the potential BBP dietary exposures to aquatic and terrestrial organisms in the environment in Section 12. EPA did not conduct a quantitative analysis of BBP trophic transfer, as BBP 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 BBP, please see the *Draft Chemistry, Fate, and Transport Assessment for Buty benzyl Phthalate (BBP)* (U.S. EPA, 2025i).

General population exposure is discussed using a risk screening approach detailed in Section 2. EPA used a margin of exposure (MOE) approach discussed in Section 2.2 using high-end exposure estimates (Section 2.1) to screen for potential non-cancer risks. EPA assumed that if there is no 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 pathway of concern for general population exposure 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/OES. Table 1-3 summarizes the exposure pathways assessed for the general population. For BBP, 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 BBP is not expected to be persistent or mobile in soils. Additionally, there are currently no U.S.-based studies reporting BBP concentration in biosolids or in soil following land application, therefore there is no current evidence of BBP-containing sludge and biosolids being used for surface land disposal or agricultural application. 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 BBP for highly exposed populations based on the OES leading to high-end concentrations of BBP in environmental media.

306 **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
Manufacturing	Surface water	Dermal	Dermal exposure to BBP in surface water during swimming (Section 5.1.1)	No
		Oral	Incidental ingestion of BBP in surface water during swimming (Section 5.1.2)	No
Manufacturing	Drinking water	Oral	Ingestion of drinking water (Section 6)	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
PVC plastics compounding			Ingestion of fish for tribal populations (Section 7.3)	No
Use of paints and coatings—no spray controls (Stack and fugitive)	Ambient air	Inhalation	Inhalation of BBP in ambient air resulting from industrial releases (Section 9.1)	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 OES
[b] Using the MOE approach, an exposure pathway was determined to not be a pathway of concern if the MOE was equal to or exceeded the benchmark MOE of 30.

307

2 SCREENING LEVEL ASSESSMENT OVERVIEW

Screening level assessments are useful when there is little facility location- or scenario-specific information available. EPA began its BBP exposure assessment using a screening level approach because of the limited environmental monitoring data and absence of location data for BBP 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 BBP per body weight were considered to be those at the upper end of the exposure distribution. Taken together, these exposure estimates are conservative because they were determined using the highest environmental media concentrations and greatest intake rate of BBP per kilogram of body weight. These exposure estimates are also protective of individuals having less exposure either due to lower intake rate or exposure to lower environmental media concentration. This is explained further in Section 2.1

For the general population screening level assessment, EPA used an MOE approach based on high-end exposure estimates to determine which exposure pathways were of potential concern for non-cancer risks. Using the MOE approach, an exposure pathway associated with a COU was determined to not be a pathway of concern if the MOE was equal to or exceeded the benchmark MOE of 30 (U.S. EPA, 2025h). Further details of the MOE approach are described in Section 2.2.

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

2.1 Estimating High-End Exposure

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

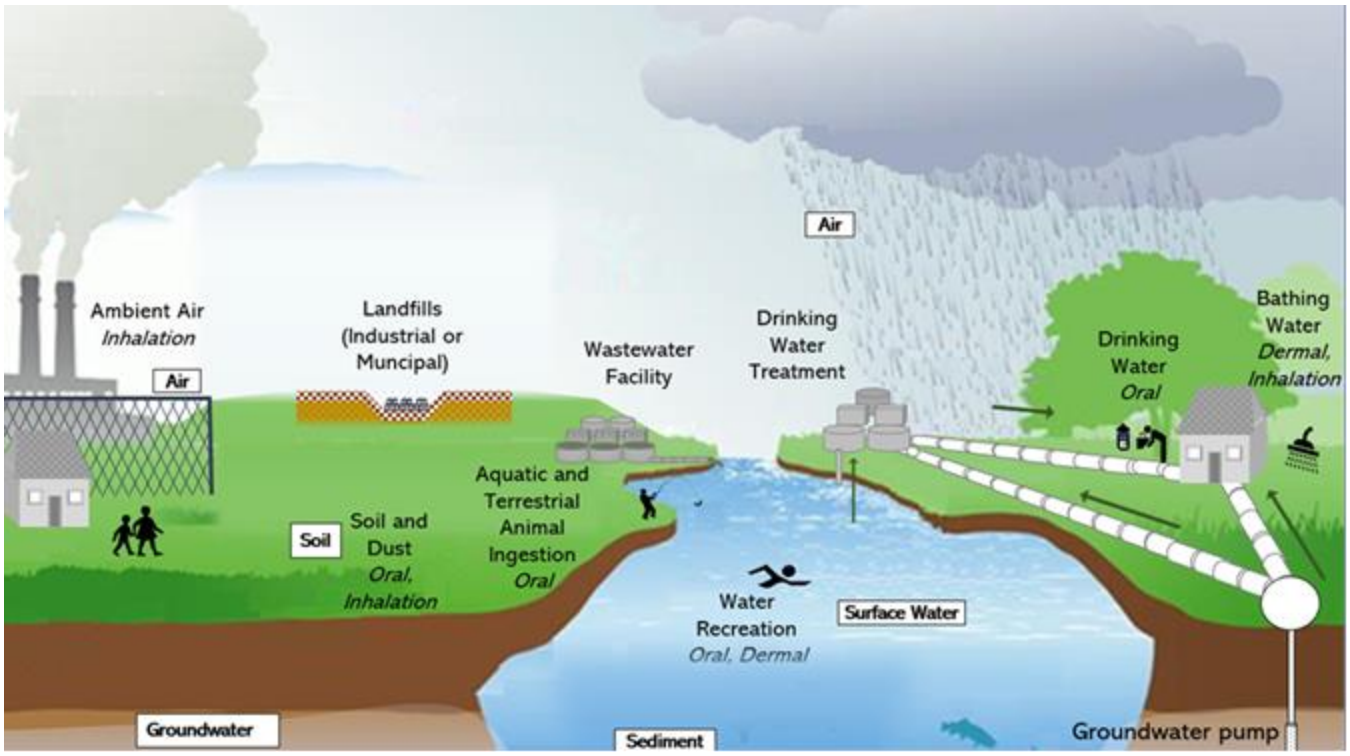


Figure 2-1. Potential Human Exposure Pathways for the General Population
The diagram presents the media (white text boxes) and routes of exposure (italics for oral, inhalation, or dermal) for the general population. Sources of drinking water from surface or water pipes are depicted with grey arrows.

For a screening level analysis, high-end exposures were estimated for each exposure pathway assessed. EPA's *Guidelines for Human Exposure Assessment* defined high-end exposure estimates as a "plausible estimate of individual exposure for those individuals at the upper end of an exposure distribution, the intent of which is to convey an estimate of exposure in the upper range of the distribution while avoiding estimates that are beyond the true distribution" (U.S. EPA, 2019b). If risk is not found for these individuals with high-end exposure, no risk is anticipated for central tendency exposure, 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 BBP per body weight were considered to be those at the upper end of the exposure distribution. 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 BBP environmental releases from biosolids and landfills (and therefore, resulting soil concentrations) were not quantified, exposure from soil or groundwater resulting from BBP release to the environment via biosolids or landfills was not quantitatively assessed. Instead, the scenarios were assessed qualitatively for exposures potentially resulting from biosolids and landfills.

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

OES	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Analysis (Quantitative or Qualitative)
All	Biosolids	No specific exposure scenarios were assessed for qualitative assessments			Qualitative, Section 3.1
All	Landfills	No specific exposure scenarios were assessed for qualitative assessments			Qualitative, Section 3.2
Manufacturing	Surface water	Dermal	Dermal exposure to BBP in surface water during swimming	All	Quantitative, Section 5.1.1
		Oral	Incidental ingestion of BBP in surface water during swimming	All	Quantitative, Section 5.1.2
Manufacturing	Drinking water	Oral	Ingestion of drinking water	All	Quantitative, Section 6.1.1
All	Fish ingestion	Oral	Ingestion of fish for general population	Adult	Quantitative, Section 7.1
All			Ingestion of fish for subsistence fishers	Adult	Quantitative, Section 7.2
PVC plastics compounding			Ingestion of fish for tribal populations	Adult	Quantitative, Section 7.3
Use of paints and coatings—no spray controls (Stack)	Ambient air	Inhalation	Inhalation of BBP in ambient air resulting industrial releases	All	Quantitative, Section 9.1
		Oral	Ingestion from air to soil deposition resulting from industrial releases	Infant and Children (6 months to 12 years)	

As part of the general population exposure assessment, EPA considered fenceline populations in proximity to releasing facilities as part of the ambient air exposure assessment by utilizing screening methodology described in EPA's *Draft TSCA Screening Level Approach for Assessing Ambient Air and Water Exposures to Fenceline Communities (Version 1.0)* (U.S. EPA, 2022b). For other exposure pathways, EPA's screening method assessing high-end exposure scenarios used release data that 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 oral drinking water exposures (Section 6), incidental dermal exposures (Section 5.1.1), and incidental oral exposures (Section 5.1.2) for the general population. Modeled ambient air concentrations (Section 8.1) were used to estimate inhalation exposures.

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

2.2 Margin of Exposure Approach

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

Equation 2-1. Margin of Exposure Calculation

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

Where:

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

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

The non-cancer hazard values used to screen for risk are described in detail in the *Draft Non-Cancer Human Health Hazard Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025h). 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	HEC[a] (mg/m^3) [ppm]	HED[a] (mg/kg-day)	Benchmark MOE[b]	Reference[c]
Acute, intermediate, and chronic	Developing male reproductive toxicity	Rat	Multi-generational or 5 days during gestation	NOAEL = 50	Phthalate syndromE−related effects (*e.g.*, ↓ AGD; ↓ fetal testicular testosterone; ↓ reproductive organ weights; Leydig cell effects; ↓ mRNA and/or protein expression of steroidogenic genes; ↓INSL3)	64.2 [5.03]	12	UF_A = 3 UF_H = 10 *Total UF = 30*	Furr et al. (2014); Aso et al. (2005); Tyl et al. (2004)

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] HED and HEC values were calculated based on the most sensitive NOAEL of 50 mg/kg-day

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

[c] Tyl et al. (2004) support a statistically significant effects at NOAEL = 50 mg/kg-day of decreased AGD and decreased reproductive organ weights in a multi-generational study; the remaining effects listed reached statistical significance at higher doses (most of which are not considered adverse in isolation). Furr et al. (2014) and Aso et al. (2005) reflect supporting phthalate syndromE−related effects (*e.g.*, reduced ex vivo testicular testosterone production or testicular histopathological changes, respectively) at LOAEL = 100 mg/kg-day.

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

3 LAND PATHWAY

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data identified during systematic review to obtain concentrations of BBP in terrestrial land pathways (*i.e.*, biosolids, wastewater sludge, agricultural soils, landfills, and landfill leachate). No monitoring data was available from a review of government regulatory and reporting databases related to soil, landfills, or biosolids (*e.g.*, California Environmental Data Exchange Network [CEDEN], Water Quality Portal [WQP]). Several academic experimental and field studies, however, have identified BBP in various media including leachate, activated sludge, and biosolids (Wu et al., 2019; Zhu et al., 2019; Salaudeen et al., 2018a, b; Wu et al., 2017; Gani and Kazmi, 2016; Gao et al., 2014; Meng et al., 2014; Tran et al., 2014; Ikonomou et al., 2012; Oppenheimer et al., 2007; Stephenson, 2007; Fauser et al., 2003; U.S. EPA, 1982). EPA cannot correlate monitoring levels from the reviewed studies and identified databases to specific releases associated with BBP TSCA COUs. That is, EPA does not have any facility specific BBP release data since facilities do not report releases of BBP to the terrestrial environment from TSCA COUs. As such, the present assessment of BBP exposure via potential land pathways is qualitative in nature relying on the fate and physical-chemical characteristics of BBP. When possible, data from the existing literature including experimental and field data was used to support the qualitative 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. BBP may be introduced to biosolids by the absorption or adsorption of BBP to particulate or organic material during wastewater treatment. Wastewater treatment is expected to remove between 40 to 97 percent of BBP via both biodegradation and sorption processes (Wu et al., 2019; Salaudeen et al., 2018a, b; Wu et al., 2017; Gani and Kazmi, 2016; Gao et al., 2014; Tran et al., 2014; Oppenheimer et al., 2007; Stephenson, 2007; Fauser et al., 2003; U.S. EPA, 1982). The STPWIN™ model in EPI Suite™ predicts 99.9 percent removal of BBP in wastewater treatment in a high biodegradation scenario, with 18 percent removal due to sorption to sludge (U.S. EPA, 2017a). The proportion sorbed to sludge is expected to increase appreciably under scenarios with less efficient biodegradation treatment. Therefore, the relative contributions of biodegradation and sorption may vary dramatically among treatment systems, as discussed in Section 7.2 in the *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate* (U.S. EPA, 2025i).

BBP 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 BBP in sludge at concentrations ranging from 1.8 to 693 ng/g dry weight (dw) (Ikonomou et al., 2012). Beyond North America, BBP has been identified in sludge at various concentrations in wastewater plants located in China (Zhu et al., 2019; Meng et al., 2014).

Anaerobic sludge digestion can potentially reduce BBP concentrations in biosolids, with one study reporting a 74.3 to 76.4 percent decrease in BBP solids concentrations following anaerobic digestion for two WWTPs (Armstrong et al., 2018). However, anaerobic sludge did not consistently result in the reduction of BBP concentrations with two plants reporting no change in concentrations following treatment. Anaerobic digestion may be an effective treatment process, but the efficiency will depend on the specific operating conditions of the digester and microbial community present.

There are currently no U.S.-based studies reporting BBP concentration in biosolids or in soil following land application. BBP-containing sludge and biosolids have not been reported for uses in surface land disposal or agricultural application.

BBP is not expected to be persistent in topsoil if it is applied to land through biosolids applications. No field studies were identified during systematic review related to aerobic or anaerobic degradation of BBP in soil. However, two laboratory studies reported by the European Commission using synthetic soil did show a degree of the capacity for aerobic biodegradation in microbial communities. The half-life of BBP in aerobic synthetic soil ranged from 3.5 to 20 days in composting conditions and up to 59 days in synthetic soil (ECJRC, 2008). There were no studies evaluating the potential for biodegradation under anaerobic conditions in soil. While not soil, studies involving biodegradation of BBP in sediments reported half-lives ranging from 0.5 to 11 days under aerobic conditions and half-lives from 1.5 to 26 days under anaerobic conditions (Kickham et al., 2012; Lertsirisopon et al., 2006; Yuan et al., 2002). It is likely that degradation in soils experience a similar trend with comparable rates of biodegradation in both aerobic and anaerobic systems.

Other sources of BBP in biosolids-amended soils may include atmospheric deposition to soil. While long-range transport and deposition of BBP in the atmosphere has not been directly monitored, Net et al. (2015) noted possible atmospheric deposition of similar phthalates in agricultural settings. A 2008 study noted concentrations up to 896 ng/L of BBP in precipitation samples (Peters et al., 2008) while a 2010 study on atmospheric deposition of phthalates notes bulk wet and dry deposition of BBP and other phthalates from the atmosphere (Zeng et al., 2010).

BBP 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 BBP to sorb strongly to organic media and soil (log K_{OW} = 4.7; log K_{OC} = 3.4–4.2), potential leaching is limited. Any leaching which does occur in the uppermost soil layers will sorb to soil lower in the column and show minimal potential to interact with groundwater systems. BBP is not readily taken up by agricultural crop or cover crops planted in soils fertilized with biosolids. A study evaluating the potential for BBP to be taken up by crops demonstrated the largest concentration of BBP was on the surface of crop leaves resulting from volatilization of BBP from the soil and subsequent deposition onto the plants shoots and leaves (Müller and Kördel, 1993). Exposed plants do not readily absorb BBP from the soil nor do they incorporate BBP into the roots, shoots, leaves, or fruiting bodies (Müller and Kördel, 1993). BBP may be present on the surface of any plants growing in the vicinity resulting from localized atmospheric deposition of BBP blown up by the wind or volatizing out of the top layer of soil. While possible, no studies identified thus far in systematic review have reported that BBP is susceptible to longer range atmospheric transport resulting in land application of BBP containing biosolids beyond the immediate region of initial application.

Concentrations of BBP 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, BBP concentrations in soil were estimated using the concentrations identified in wastewater sludge (1.8 to 693 ng/g dw) (Ikonomou et al., 2012).

Using the EPA recommended application rate and volume and application limitation in accordance with the *Standards for the Use of Disposal of Sewage Sludge* (40 CFC Part 503) (U.S. EPA, 1993). Biosolids application rates and frequencies were selected using EPA's recommendation to the public in *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 five years. Preliminary conservative calculations assumed no significant degradation from abiotic transformation processes in between annual applications. While biosolids are often prE−treated and stabilized using processes including thermal pyrolysis, anaerobic digestion, aerobic digestion, or composting, there are very few studies examining the capacity of such biosolids stabilization treatments to remove BBO from wastewater sludges. At least one study demonstrated that anaerobic digestion of sludge may remove up to 76% of BBP in the unstabilized solids (Armstrong et al., 2018). However, given the limited research into the remaining sludge treatment technologies, EPA assumed there was no significant reduction of BBP in from the use of sludge stabilization treatments.

Table 3-1. Typical Biosolids Application Scenarios

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

Soil surface concentrations and incorporated concentrations were calculated from the minimum and maximum recommended application rates for each agricultural crop cover (Table 3-2). Minimum (1.8 ng/g dw) and maximum (693 ng/g dw) concentrations of BBP 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 by the EPA was determined to have a high confidence level during systematic review. BBP concentrations in sludge selected from the wastewater sludge monitoring study was not used to quantify exposures estimates in the BBP risk evaluation document. The information instead provides general insight on the concentrations which may result if biosolids containing BBP is applied to agricultural land at the recommended application rates at the observed concentrations.

Under ideal composting conditions, BBP present in applied biosolids may or may not pose the potential for accumulation in applied fields. In ideal composting conditions (*e.g.*, actively aerated, turned, watered, and temperature maintained), applied BBP would likely degrade fully within a standard 30 to 90-day composting window; Using the fastest observed composting half-life of 0.5 days, less than 1 percent of BBP would be present at the end of 30 and 90 days (Kickham et al., 2012; ECJRC, 2008; Lertsirisopon et al., 2006; Yuan et al., 2002). However, using the more conservative 20-day half-life, 35 percent of BBP would remain at 30 days and 4.5 percent of BBP would remain at 90 days (ECJRC, 2008). Under nonideal conditions more representative of what would be expected in agricultural processes, like that of a static pile or windrow passively aerated composting system, BBP would likely not fully degrade in a typical composting or agricultural period before fertilizer reapplication is required. Using the conservative half-life of 59-days extracted from ECJRC study (ECJRC, 2008), which more accurately represents the passive conditions of an agricultural field, 70 percent of BBP would remain at the end of a 30-day period and 35 percent at the end of a 90-day period; While passively managed

composting operations can remain active for up to one year with appropriate monitoring (Brodie et al., 2000), 1.5 percent of BBP would still remain at the end of a one−year period. Under passively aerated conditions, BBP may be at risk of continued accumulation if applied as a biosolids-amended soil treatment if applied at a higher frequency. Typical application frequency for various crops in commercial agricultural operations ranges from one to four times a year at the discretion of the farmer and soil conditions (U.S. EPA, 2000a). Application at higher frequencies, even at less conservative degradation rates, may allow for the potential for accumulation of BBP in biosolids amended soils if sufficient time is not provided between applications.

Table 3-2. Estimated BBP 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	0.0018	5,080	1	2.3E−03	9.1E−06
Corn	0.0018	10,161	1	4.5E−03	1.8E−05
Corn	0.693	5,080	1	8.7E−01	3.5E−03
Corn	0.693	10,161	1	1.7	7.0E−03
Hay	0.0018	20,321	1	9.0E−04	3.6E−06
Hay	0.0018	5,080	3	6.8E−03	2.7E−05
Hay	0.693	20,321	1	3.5E−01	1.4E−03
Hay	0.693	5,080	3	2.6	1.0E−02
Small grains	0.0018	20,321	1	9.0E−04	3.6E−06
Small grains	0.0018	5,080	3	6.8E−03	2.7E−05
Small grains	0.693	20,321	1	3.5E−01	1.4E−03
Small grains	0.693	5,080	3	2.6	1.0E−02
Soybeans	0.0018	20,321	1	2.3E−03	9.1E−06
Soybeans	0.0018	5,080	1	9.0E−03	3.6E−05
Soybeans	0.693	5,080	1	8.7E−01	3.5E−03
Soybeans	0.693	20,321	1	3.5	1.4E−02

[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 BBP in biosolids may range by several orders of magnitude depending largely on the source material and method of application. The surface loading rate for spray or near surface injection applications range from 9.0×10^{-5} to 3.4 mg/m^2, while the surface loading rate for mixing applications (assuming a 7-inch [18-cm] tilling depth) may range from 4.0×10^{-6} to 0.01 mg/m^3 depending on the application rate, frequency, and applied biosolids concentration. As mentioned previously, the concentrations in the applied stabilized biosolids may be reduced up to 75% through the anaerobic digestions (Armstrong et al., 2018), which would result in a 75% reduction in the surface and topsoil concentrations.

Once in the soil, BBP is expected to have a high affinity to particulate (log K_{OC} = 3.4–4.2) and organic media (log K_{OW} = 4.7) which would limit mobility from biosolids or biosolid amended soils. Similarly, high sorption to particulate and organics would likely lead to high retardation which would limit infiltration to and mobility within surrounding groundwater systems. BBP is soluble in water (2.69 mg/L) and does have 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. Since BBP does have high hydrophobicity and a high affinity for soil sorption, it is unlikely that BBP will migrate from potential biosolids-amended soils via groundwater infiltration. BBP has been detected in surface runoff originating from landfills containing BBP (IARC, 2013). However, the limited mobility and high sorption to soil suggests that infiltration of such stormwater runoff would be of minimal concern to deeper groundwater systems.

There is limited information available related to the uptake and bioavailability of BBP in land applied soils. BBP'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 BBP. Based on the solubility (2.69 mg/L) and hydrophobicity (log K_{OW} = 4.7; log K_{OC} = 3.4–4.2), BBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of BBP into crops planted in BBP containing soils found that BBP was not found in any of the plant tissues (*i.e.*, roots, shoots, leaves) resulting from uptake via soil or water. Another phthalate with similar chemical properties, dibutyl phthalate (DBP), was found, however, on the surface of the plants due to localized atmospheric transport and deposition but is not readily absorbed by plants directly through the soil (Müller and Kördel, 1993). BAF and BCF were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 1.6 to 1.8 (upper-lower trophic levels) and log BAF ranging from 1.6 to 1.8 (upper-lower trophic levels) (U.S. EPA, 2017a).

There is limited measured data on concentrations of BBP 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 BBP 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 modeling software to estimate concentrations of BBP in biosolids. There is data regarding the concentration of BBP in wastewater solids, however, limited information is available related to the treatment capacity for the removal of BBP from wastewater sludge through the application of sludge

stabilization. Additionally, there is uncertainty in the relevancy of the biosolids monitoring data to the COUs considered in this evaluation. Overall, due to the high confidence in the biodegradation rates and physical and chemical data, there is robust confidence that BBP in soils will not be mobile and will have low persistence potential. The existing literature suggests that BBP present in biosolid amended soils will likely not be absorbed by any plants or crops growing in the soil. While experimental and monitoring data are limited, preliminary analysis suggests that soil dwelling organisms may be exposed to BBP. However, there is no evidence to suggest that BBP will accumulate in exposed organisms resulting from the direct application of BBP through biosolids applied in fertilizers.

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.

For this assessment, landfills will be 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 BBP; 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 may still be present in the subsurface, conditions may still be favorable for aerobic biodegradation. However, photolysis is not considered to be a significant source of degradation in this zone. In the lower-landfill zone, conditions are assumed to be anoxic, and temperatures present in this zone are likely to inhibit anaerobic biodegradation of BBP. 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).

BBP may be deposited into landfills through various waste streams including consumer waste, residential waste, industrial waste, and municipal waste including dewatered wastewater biosolids. No studies were identified in systematic review determining the concentration of BBP in waste entering landfills in the United States. A 1997 study of German refuse identified during systematic review with an overall confidence rating of high, however, identified phthalates in mixed waste residential refuse. BBP was identified in residential refuse with the highest concentrations of BBP present in "compound materials" (*e.g.*, plastic products) (30–344 µg/g) and "other plastics" (0.5–130 µg/g) (Bauer and Herrmann, 1997). No data has been provided by TRI relating to the release of BBP to landfills.

No studies were identified which reported the concentration of BBP in landfills or in the surrounding areas. There is limited information regarding BBP in dewatered biosolids, which may be sent to landfills for disposal. As mentioned previously, BBP has been identified in a high-quality U.S.-based and international surveys of wastewater sludge (Ikonomou et al., 2012). A 2012 survey of North American wastewater plants (Canada and United States) identified BBP in sludge at concentrations ranging from 1.8 to 693 ng/g dw (Ikonomou et al., 2012). Beyond North America, BBP has been identified in sludge at various concentrations in wastewater plants located in China (Zhu et al., 2019; Meng et al., 2014).

BBP is capable of leaching from bioreactors simulating landfill conditions using residential waste. One 1997 study evaluating a variety of phthalates, including BBP, reported a leaching potential of 1.1 grams of phthalates per ton of refuse from benchtop-scale reactors using 50 kg of unaltered residential mixed waste (Bauer and Herrmann, 1997). The generated leachate was composed of approximately 2.0 to 2.9

percent of total phthalates (4 to 8 µg/L of DBP per 1 ton of residential refuse) (Bauer and Herrmann, 1997).

No studies have directly evaluated the presence of BBP in landfill or waste leachate. However, BBP is expected to have a high affinity to particulate (log K_{OC} = 3.4–4.2) and organic media (log K_{OW} = 4.7) which would cause significant retardation in groundwater and limit leaching to groundwater. Because of its high hydrophobicity and high affinity for soil sorption, it is unlikely that BBP will migrate from landfills via groundwater infiltration. Nearby surface waters, however, may be susceptible to BBP contamination via surface water runoff if it is not captured before interacting with surface water.

While persistence in landfills has not been directly measured, BBP can undergo abiotic degradation via carboxylic acid ester hydrolysis to form 2-butyl phthalate and 1-butanol (U.S. EPA, 2024a). Hydrolysis is not expected to be a significant degradation pathway in landfills with an estimated half-life of 3.4 years under standard environmental conditions (at pH 7 and 20 °C) (U.S. EPA, 2017a). Temperature in lower landfills, however, often exceed 70 °C in very complex matrices. In such matrices, temperature, pressure, ionic strength, and chemical activity may all effect the hydrolysis rate of BBP. With the very limited data available, the hydrolysis rate of BBP 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, BBP is shielded from light and photolysis is not considered a significant abiotic degradation pathway.

In both the upper and lower landfills, BBP is shielded from light, and photolysis is not considered a significant abiotic degradation pathway. In the lower landfill, high temperatures (exceeding 60 °C) and low water content may partially or completely inhibit biological degradation (Huang et al., 2013). Aerobic and anaerobic degradation of BBP has not been directly measured. Aerobic degradation of BBP, however, has been measured directly in landfills or landfill leachate. BBP is degradable in aerobic soil conditions with a half-life ranging less than 3.5 to 59 days (ECJRC, 2008). BBP may also degrade under anaerobic conditions such as those that would exist in lower landfills. While anaerobic biodegradation of BBP has not been directly measured in soil, it is expected to undergo rates of biodegradation similar to that of BBP under aerobic conditions, as is the case in sediment aerobic and anaerobic degradation (Kickham et al., 2012; ECJRC, 2008; Lertsirisopon et al., 2006; Yuan et al., 2002). In landfills with high leachate production, BBP may be more persistent with areas saturated with leachate are likely in the lowest sections of the landfill in locations with temperatures exceeding the habitable zones for most microorganisms capable of degrading.

BBP's sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms adjacent to landfills. BBP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. Studies evaluating the uptake of BBP into crops planted in BBP containing soils found that BBP was not found in any of the plant tissues (*i.e.*, roots, shoots, leaves) resulting from the uptake via soil or water. BBP was found, however, on the surface of the plants due to localized atmospheric transport and deposition but is not readily absorbed by plants directly through the soil (Müller and Kördel, 1993). BAF and BCF were modeled using the BCFBAF™ model in EPI Suite™ with an estimated log BCF ranging from 1.6 to 1.8 (upper-lower trophic levels) and log BAF ranging from 1.6 to 1.8 (upper-lower trophic levels) (U.S. EPA, 2017a).

3.2.1 Weight of Scientific Evidence Conclusions

There is uncertainty in the relevancy of the landfill leachate monitoring data to the COUs considered in this evaluation. Based on the biodegradation and hydrolysis data for conditions relevant to landfills,

there is high confidence that BBP will be persistent in landfills. Overall, due to high-quality physical and chemical property data, there is robust confidence that BBP is unlikely to be present in landfill leachates. The existing literature suggests that BBP present in landfills will likely not be absorbed by any nearby plants. While experimental and monitoring data are limited, preliminary analysis suggests that soil dwelling organisms may be exposed to BBP. However, there is no evidence to suggest that BBP will accumulate in exposed organisms resulting from the from the leaching or otherwise transport of BBP from landfill material to surrounding soil or groundwater.

4 SURFACE WATER CONCENTRATION

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of BBP in ambient surface water and aquatic sediments. Though the available monitoring data were limited, BBP was detected in surface water, finished drinking water, and aquatic sediments. However, EPA cannot correlate monitoring levels with any releases associated with BBP TSCA COUs. In addition, BBP is not a listed as a 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 BBP release data since facilities do not report releases of BBP 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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f). 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 using the U.S. EPA's Variable Volume Water Model (VVWM) in Point Source Calculator tool (PSC) (U.S. EPA, 2019c) to estimate surface water and sediment concentrations of BBP resulting from TSCA COU releases. PSC inputs include physical and chemical properties of BBP (*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. PSC was also used to estimate BBP 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. BBP has a log K_{OC} of 4.86, indicating a strong potential to sorb to suspended particles in the water column and settled sediment in the benthic environment (U.S. EPA, 2017a).

Physical and chemical, and environmental fate properties selected by EPA for this assessment were applied as inputs to the PSC model (Table 4-1). Selected values are described in detail in the *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025i). 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]
K_{OC}	72,444 mL/g
Water Column Half-life	14 days at 25 °C
Photolysis Half-life	18 days at 30N
Hydrolysis Half-life	1,500 days at 25 °C

Parameter	Value[a]
Benthic Half-life	19.3 days at 25 °C
Molecular Weight	312.37 g/mol
Vapor Pressure	0.00000825 torr
Solubility	2.69 mg/L
Henry's Law Constant	0.000000761 atm·m^3/mol at 25 °C
Heat of Henry	74,826 J/mol
Reference Temp	25 °C
[a] For details on selected values, see *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025i).	

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

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}	0.04
Water column DOC	5.0 mg/L
Water column biomass	0.4 mg/L
Benthic depth	0.05 m
Benthic porosity	0.50
Benthic bulk density	1.35 g/cm³
Benthic f_{oc}	0.04
Benthic doc	5.0 mg/L
Benthic biomass	0.006 g/m²

Parameter	Value
Mass transfer coefficient	0.00000001 m/s

A required input for the PSC model is the hydrologic flow rate of the receiving water body. EPA used modeling approaches to assess releases of BBP to water for all OESs because there were no reported data from available sources (*e.g.*, TRI and Discharge Monitoring Report [DMR]) (U.S. EPA, 2024b). Without TRI and DMR data, EPA cannot identify the receiving water bodies and their location-specific hydrological flow data. EPA instead 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 BBP-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 is 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.

Different hydrological flow rates were used for different exposure scenarios. 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 concern for aquatic life (U.S. EPA, 2007). The regression equations for deriving the harmonic mean and 7Q10 flows are provided in Appendix B. Hydrologic flows in the receiving waterbodies were added to facility effluent flows, as the rate of effluent contributes a substantial amount of flow to receiving waterbodies in many cases. The median, 75th percentile, and 90th percentile (P50, P75, P90, respectively) flows from the distribution were applied to represent variation in the potential receiving waterbodies.

A screening analysis was conducted using the highest surface water concentrations derived from facility releases. The generic release scenario for the Manufacturing OES estimates a combined release to wastewater, incineration, or landfill. Because the proportion of the release from Manufacturing OES to just surface water could not be determined from reasonably available information, and the discharge as wastewater includes the possibility of direct discharge without further treatment, for screening purposes EPA conservatively assumed that all of the release would be directly discharged to surface water, to represent an upper-bound of surface water concentrations. The tiered exposure approach utilized the highest resulting environmental concentrations from this release scenario as the basis of a screening analysis for general population exposure. Additionally, surface water concentrations derived from the PVC plastics compounding OES (the OES with the highest estimated release to only surface water) were incorporated into the screening analysis for reference (Table 4-3). EPA's process for selecting the Manufacturing and PVC Plastics Compounding OESs is detailed in Section 4.4 along with the confidence in using the surface water concentrations for the purpose of a screening level assessment.

Table 4-3 below shows the surface water concentration modeled from the selected OESs using the 7Q10 flow. The total days of release associated with the selected OESs were applied as continuous days of release per year as a conservative approach (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). The highest water column concentration averaged over the number of release days (*i.e.*, 250) was used to estimate general population and aquatic exposure. In most cases, the number of averaging days did not substantially impact waterbody concentration estimates. Appendix B describes the methods to calculate the rolling averages.

When considering the release quantity for the Manufacturing and PVC plastics 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 BBP, releases of the phthalates DEHP and DBP were examined. Across the facilities with known release data from various OES, but predominantly PVC plastics compounding, there appears to be a general trend of larger amounts of loading being released to larger receiving waterbodies (Figure 4-1). While there is notable variability in receiving waterbody flows, an overall tendency for greater releases to be paired with receiving waterbodies with greater flows is suggested, particularly with respect to the minimum flows reported from the distribution across release amounts. Due to the uncertainty associated with the receiving waterbody for the generic scenario, EPA models the Manufacturing and PVC plastics 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 Manufacturing OES being orders of magnitude greater than any of the known direct discharge phthalate releases of DBP and DEHP reported to EPA programs, and the assumption of the largest releases tending to be released in larger waterbodies, EPA 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 flows for this high-end release scenario. Given the strong effect the receiving waterbody flow has on environmental concentrations, and the wide variability across the generic flow distributions, this is a source of uncertainty for the generic release scenarios.

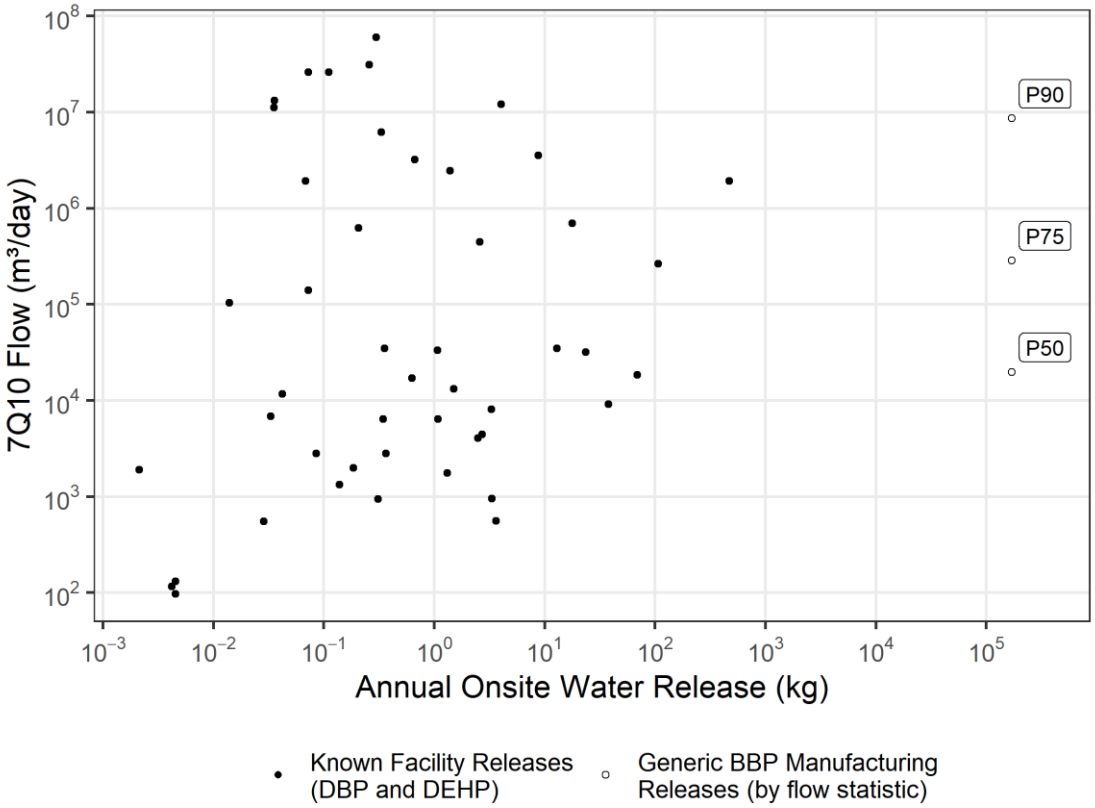


Figure 4-1. Plot of known phthalate facility releases of DEHP and DBP with high-end generic scenario estimations for BBP.

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 BBP-releasing facilities, all releases are assumed initially 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 BBP from the water column prior to discharge, with treated effluent showing up to a 96 percent reduction in one study (Tran et al., 2014). Release modeling is shown in

Table 4-3. This first-tier analysis includes some notably high estimated concentrations in the receiving waterbody and sediment. These likely represent a mismatch of higher release amounts with lower flows, due to the generic nature of the release assessment and hydrologic flow data and lack of sitE−specific data. 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 the *Draft Environmental Hazard Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025e).

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)
Manufacturing	300	566	30,700	14,900

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)
Without wastewater treatment (P50 flow rate with high-end release)				
PVC plastics compounding *Without wastewater treatment* (P50 flow rate with high-end release)	254	65.7	4,340	2,100

[a] Details on operating days and daily releases are provided in *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f)

The OESs with the highest total water column concentrations (Manufacturing and PVC plastics compounding) were additionally 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 BBP concentrations (as in the modeling shown previously in this section), and another with a wastewater treatment removal efficiency of 62 percent applied, reducing the modeled concentrations in the receiving waterbody. The BBP surface water concentration after application of the removal efficiency are more likely to represents human exposure to BBP in drinking water, where additional removal from drinking water treatment would also be expected.

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

Scenario	Release Estimate (kg/day)[a]	Harmonic Mean Flow (m^3/d)	30Q5 Flow (m^3/d)	Removal Efficiency Applied (%)	Harmonic Mean Concentration (µg/L)	30Q5 Concentration (µg/L)
Manufacturing *Without wastewater treatment* (P50 flow rate with high-end release)	566	32,034	22,966	0	17,600	24,500
Manufacturing *With wastewater treatment* (P50 flow rate with high-end	566	32,034	22,966	62	6,670	9,310

Scenario	Release Estimate (kg/day)[a]	Harmonic Mean Flow (m^3/d)	30Q5 Flow (m^3/d)	Removal Efficiency Applied (%)	Harmonic Mean Concentration (µg/L)	30Q5 Concentration (µg/L)
release)						
Manufacturing *Without wastewater treatment* (P75 flow rate with high-end release)	566	427,829	294,373	0	1,320	1,920
PVC plastics compounding *Without wastewater treatment* (P50 flow rate with high-end release)	65.7	32,034	22,966	0	2,049	2,852
PVC plastics compounding *With wastewater treatment* (P50 flow rate with high-end release)	65.7	32,034	22,966	62	779	1,084
PVC plastics compounding *Without wastewater treatment* (P75 flow rate with high-end release)	65.7	427,829	294,373	0	153	223

[a] Details on operating days and daily releases are provided in *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f)

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. Two studies were identified from the United States that reported BBP in surface water (NWQMC, 2021; Liu et al., 2013) (Table 4-5). EPA STOrage and RETrieval (STORET) data were

obtained through 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. Since 2004, the maximum monitored level in water was in a Arizona DEQ groundwater sample with 40 µg/L of BBP. Where the media subdivision was specified as surface water, the maximum monitored level was 2.65 µg/L, from urban floodwater in Florida after Hurricane Rita.

In March 2008 through June 2009, Liu et al. (2013) assessed the spatial distribution of phthalates in Lake Pontchartrain, LA, before, during, and after opening of the Bonnet Carré Spillway that occurred April to May of 2008. Forty-two freshwater samples were collected from the Bonnet Carré Spillway at six sites located about one mile apart. 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. BBP was only discussed for the central lake area samples. The study reported that BBP was found at one location, but the level of BBP was not reported.

Two additional studies, both from France, reported levels of BBP in surface water. Valton et al. (2014) examined levels of phthalates in the Orge River, a suburban tributary of the Seine River. The study 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 mean BBP concentrations of 59 ng/L. Sampling year, number of samples, and detection frequency were not reported. A study conducted by Schmidt et al. (2020) quantified phthalate concentrations in the Rhône River in Arles city, France. This river exports water to the Gulf of Lion, which is 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. BBP was detected in approximately 60 percent of samples with a mean concentration of 0.5 ng/L.

Two additional studies measuring BBP in surface water were identified, but no detections were found (Bach et al., 2020; Lee et al., 2019).

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

Reference	Sampling Location	BBP Concentrations	Sampling Notes	Study Quality Rating
WQP (NWQMC, 2021)[a]	United States	Water overall: ND–40 µg/L (n = 45,854) Maximum levels by media subdivision (µg/L): 10 (unspecified); 40 (groundwater); 2.65 (surface water); 4.25 (stormwater); ND (wastewater)	U.S. STOrage and RETrieval (STORET) water quality data, 2004-2024	Medium
Liu et al. (2013)	United States	Bonnet Carré Spillway (6 locations; n = 42) NR Central lake area (6 locations; n = 54)	Freshwater samples from Lake Pontchartrain, LA, before, during, and after opening of the Bonnet Carré	Medium

Reference	Sampling Location	BBP Concentrations	Sampling Notes	Study Quality Rating
		FOD: 1.9%*, concentration NR *Calculated	Spillway that occurred April/May 2008, March 2008–June 2009	
Valton et al. (2014)	France	FOD and sample number NR Mean ± SD = 59 ± 82 ng/L	Freshwater samples from the outlet of the Orge River basin, date NR	Medium
Schmidt et al. (2020)	France	FOD ~60%* (n = 22) Median, mean ± SD (range) = 0.5, 0.5 ± 0.1 (ND–0.6) ng/L LOQ = 0.09 ng/L *Determined from Figure S2 in the study.	Monthly Rhône River samples, May 2017–April 2018	High

ND = non-detect; FOD = frequency of detection; NR = not reported; LOQ = limit of quantification; SD = standard deviation

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

4.2.2 Measured Concentrations in Sediment

EPA 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 or subsequent risk estimates. EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of BBP in sediment. One reference from the United States was available. EPA STORET sediment data (surface, subsurface, or unspecified matrices) reported by various local, state, and federal agencies around the country were obtained through WQP (NWQMC, 2021). Since 2004, the maximum level in sediment (311,000 µg/kg) came from a single a sample of sediment deposited by urban floodwater in Louisiana from Hurricane Katrina(Table 4-6). A study from South Korea was also identified that reported BBP levels in sediment; however, in all samples collected, no BBP was found (Lee et al., 2019).

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

Reference	Sampling Location	BBP Concentrations	Sampling Notes	Study Quality Rating
WQP (NWQMC, 2021)[a]	United States	Overall: ND–311,000 µg/kg (n = 7,792) Maximum levels by media subdivision (µg/kg): 311,000 (unspecified); 10,400 (surface); 610	U.S. STOrage and RETrieval (STORET) water quality data, 2004-2024	Medium

Reference	Sampling Location	BBP Concentrations	Sampling Notes	Study Quality Rating
		(subsurface, dw)		

dw = dry weight; ND = non-detect

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

4.3 Evidence Integration for Surface Water and Sediment

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

EPA conducted modeling with PSC to estimate concentrations of BBP within surface water and sediment using estimated 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 BBP (*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 sediment concentrations. The use of physical and chemical properties of BBP gathered and evaluated through the systematic review process increases confidence in the application of the PSC model. Only the chemical release amount, days-on of chemical release, and the receiving water body hydrologic flow were changed for each COU/OES. 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 BBP releases. 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 are actively releasing BBP to surface water, while the reported measured concentrations represent sampled ambient water concentrations of BBP. Because the release of BBP 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. Differences in magnitude between modeled and measured concentrations may be due to measured concentrations not being geographically or temporally close to known releases of BBP. In addition, when modeling with PSC, EPA assumed all releases were directly discharged to surface waters without prior treatment, and that no releases were routed through publicly owned treatment works prior to release. EPA recognizes that this is a conservative assumption that results in no removal of BBP prior to release to surface water.

Concentrations of BBP within the sediment were estimated using the highest 2015 to 2020 annual releases and estimates of 7Q10 hydrologic flow data for the receiving water body that were derived from National Hydrography Dataset (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 the EPA ECHO API and the NHDPlus V2.1 EROM flow database include self-reported hydrologic reach codes on NPDES permits and the best available flow estimations from the EROM flow data. 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 which percentiles from the distributions of flow statistics are most representative for the generic

scenarios represented in the BBP release modeling, as discussed in Section 4.1. 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 considering the uncertainties.

4.4 Weight of Scientific Evidence Conclusions

As detailed in *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f), EPA had DMR data for some OES, but due to limited release data for facilities discharging BBP to surface waters for all OES, releases were modeled as generic scenarios, and the high-end estimate for each COU was used as an input for surface water modeling. Additionally, due to 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. For the screening level assessment, EPA utilized releases associated with the Manufacturing and PVC Plastics Compounding OESs as they resulted in the highest surface water concentrations for use in environmental risk and general population risk. EPA determined the surface water concentration associated with these OES represented a conservative high-end exposure scenario and was appropriate to use in its screening level assessment to assess all other OESs and their associated COUs.

EPA utilized daily release information to estimate surface water concentrations for use in general population and environmental exposure assessment. As detailed in *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f), EPA estimated a range for daily releases for each OES. EPA was not able to estimate site−specific releases for the Fabrication of final products from articles OES. EPA assessed releases from the Distribution in commerce OES as part of the individual OESs where the relevant activities occur. Disposal sites handling post-consumer, end-use BBP were not quantifiable due to the wide and disperse use of BBP in PVC and other products. EPA assumed that releases during consumer waste handling, treatment, and disposal are captured in the upstream OESs.

For BBP, daily releases for each OES was estimated using generic scenarios. EPA summarized the overall weight of scientific evidence conclusions for its BBP release estimate for each OES in the *Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f). Overall EPA concluded the weight of scientific conclusion was generally moderate for releases that use GSs/ESDs.

Daily releases to water for each OES was reported to the following categories for BBP:

- Wastewater
- Wastewater, Incineration, or Landfill
- Fugitive Air, Wastewater, Incineration, or Landfill

Only the discharge type categorized as Wastewater is known to be discharged only to water. For the other categories categorized as releasing to multiple media types, EPA could not differentiate the proportion of BBP released only to surface water. For these generic scenario OES, there was insufficient data to quantify what portion of a release may be discharged specifically to surface water. Therefore, EPA proceeded with a conservative estimation of environmental concentrations and exposures under the assumption that the total amount released for these OES were directly discharged to surface water (for the Manufacturing; Import and repackaging; Incorporation into adhesives and sealants; Incorporation into paints and coatings; Incorporation into other formulations, mixtures, or reaction products; Application of adhesives and sealants; Application of paints and coatings OES).

Table 4-7 below identifies the data available for use in modeling surface water concentrations for each OES and EPA's confidence in the estimated surface water concentrations used for exposure assessment. In considering the various OES for use in a screening assessment, EPA identified Manufacturing and PVC plastics compounding OESs for use in environmental exposure and general population exposure. EPA determined these OES as most appropriate for use in screening as it resulted in a high-end surface water concentration based on many conservative assumptions, such as the assumption that there is no removal of BBP prior to release in surface water, and that in the case of the Manufacturing OES, the total multimedia release is assumed to be discharged directly to surface water. Due to the lower flow rates selected from the generated distributions, coupled with high-end release scenarios, EPA has moderate confidence in the modeled concentrations from the PVC plastics compounding OES as being representative of actual releases, with a slight bias toward over-estimation. EPA has only slight confidence in the high-end estimated concentrations for the Manufacturing OES, with a bias toward over-estimation, due to the uncertainty around the portion of the total estimated release being discharged to surface water. The incorporation of higher percentile flows (P75) with the high-end release estimates increase confidence in the representativeness of the concentrations presented. Additionally, EPA has robust confidence that no surface water release scenarios exceed the highest concentrations presented in this evaluation. This is because of conservative assumptions that include use of high-end releases for each COU and coupling those with lower flow rates from the generated distributions. 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 "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 at least one order of magnitude. Additionally, surface water concentrations estimated using P50 flow exceeded the water solubility of 2.69 mg/L. This confirms EPA's expectation that modeled concentrations presented here are biased toward overestimation, to be applied as a screening evaluation.

Overall, EPA has robust confidence that the high-end estimated surface water concentration modeled using the Manufacturing and PVC plastics compounding OES is appropriate to use in its screening level assessment for surface water exposure and fish ingestion exposure to the general population to assess all other OESs and their associated COUs, including OESs and COUs with releases that could not be quantified.

Table 4-7. BBP Release Data Used for Modeling Surface Water Concentrations

OES	Water Release Data Type	Weight of Scientific Evidence for Surface Water Concentrations
Manufacturing	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water. Due to the uncertainty around the portion of the release going to surface water, EPA has only slight confidence in the estimated value for this OES, but robust confidence that the estimated

OES	Water Release Data Type	Weight of Scientific Evidence for Surface Water Concentrations
		concentration represents a high-end value appropriate to supplement the screening analysis for general population exposure.
Import and repackaging	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
PVC plastics compounding	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were available to model a surface water-specific release, and the high-end estimated concentrations were applied in the screening analysis. EPA has greater confidence in the representativeness of this OES releasing to actual surface water concentrations compared to the Manufacturing OES. EPA has moderate confidence in the surface water concentration but robust confidence that this OES represents a conservative surface water concentration appropriate for screening.
Non-PVC materials compounding	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening.
Incorporation into adhesives and sealants	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.

OES	Water Release Data Type	Weight of Scientific Evidence for Surface Water Concentrations
Incorporation into paints and coatings	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Incorporation into other formulations, mixtures, or reaction products	Generic Scenario (multimedia) and DMR	EPA had reported releases for this OES from DMR. Release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
PVC plastics converting	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Non-PVC material converting	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Recycling	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening.

OES	Water Release Data Type	Weight of Scientific Evidence for Surface Water Concentrations
Distribution in commerce	No release data	Release to surface water expected to be negligible. EPA has robust confidence that the OES selected for screening will cover this OES.
Application of adhesives and sealants	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Application of paints and coatings	Generic Scenario (multimedia) and DMR	EPA had reported releases for this OES from DMR. Release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Fabrication of final product from articles	No release data	Release to surface water expected to be negligible or captured in other up-stream OES. EPA has robust confidence that the OES selected for screening will cover this OES.
Use of laboratory chemicals	Generic Scenario (multimedia)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Because EPA was unable to model releases to just surface water, EPA calculated a surface water concentration based on the assumption that the total multimedia release was directed to surface water, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Use of lubricants and functional fluids	Generic Scenario (water-specific)	No facilities reported releases for this OES, so EPA modeled releases using generic scenarios. Sufficient release data were

OES	Water Release Data Type	Weight of Scientific Evidence for Surface Water Concentrations
		available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Repackaging	DMR	EPA had reported releases for this OES from DMR. Release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.
Waste handling, treatment, and disposal	DMR	EPA had reported releases for this OES from DMR. Release data were available to model a surface water-specific release, and the resulting range of estimated concentrations were below the high-end releases applied for screening. EPA has robust confidence that the OES selected for screening will cover this OES.

5 SURFACE WATER EXPOSURE TO GENERAL POPULATION

Concentrations of BBP 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 BBP in surface water based on highest releasing OESs (Manufacturing and PVC plastics 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 are affected by BBP 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.1. EPA used the Consumer Exposure Model (CEM) (U.S. EPA; ICF Consulting, 2022) to estimate the dermal permeability coefficient (K_p) of 0.0071 cm/hr.

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 end release value from the Manufacturing and PVC plastics compounding OESs (Table 4-4) as the surface water concentration. Dose values are presented both with and without a wastewater treatment removal efficiency of 62 percent applied. In addition to these modeled concentrations, the monitored concentrations from NWQMC (2021)were included for comparison. The monitored water column concentration are roughly two orders of magnitude less than the high-end modeled counterparts. Doses calculated using the surface water monitoring data are one to two orders of magnitude lower than corresponding doses modeled using the high-end Manufacturing and PVC plastics compounding OESs.

Table 5-1. Modeled Dermal (Swimming) Doses for Adults, Youths, and Children, for the High-End Release Estimate from Modeling and Monitoring Results[1]

Scenario	Water Column Concentrations		Adult (21+ Years)		Youth (11–15 Years)		Child (6–10 Years)	
	30Q5[a] Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing *Without wastewater treatment* (P50 flow rate)	24,500	17,600	2.47E−01	1.46E−01	1.89E−01	1.12E−01	1.15E−01	6.77E−02
PVC plastics compounding *Without wastewater treatment* (P50 flow rate)	2,852	2,049	9.8E–03	4.9E−03	1.53E–02	7.6E−03	8.6E–03	4.3E−03
Highest monitored surface water NWQMC (2021)	40	40	4.0E–04	3.32E−04	3.1E–04	2.54E−04	1.9E–04	1.54E−04

[a] 30Q5 = 30 consecutive days of lowest flow over a 5-year period; POT = potential

5.1.2 Oral Ingestion Exposure

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

The following equations were used to calculate incidental oral (swimming) doses for adults, youth, and children using the Manufacturing and PVC plastics 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)

[1] Doses are calculated using Equation 5-1 and Equation 5-2.

Equation 5-4. Average Daily Incidental Calculation

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

Where:

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

A summary of inputs utilized for these estimates are present 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. Modeled 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)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing *Without wastewater treatment*	24,500	17,600	8.5E−02	5.0E−02	1.3E−01	7.7E−02	7.4E−02	4.4E−02
PVC plastics compounding *Without wastewater treatment*	2,852	2,049	9.84E−03	4.92E−03	1.53E−02	7.63E−03	8.61E−03	4.30E−03
Highest monitored surface water NWQMC (2021)	40	40	1.4E–04	1.13E−04	2.1E–04	1.76E−04	1.2E–04	9.93E−05

5.2 Weight of Scientific Evidence Conclusions

No facility- or site-specific information was reasonably available when estimating release of BBP to the environment. Environmental releases to water were estimated using generic scenarios (U.S. EPA, 2025f). 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 for PVC plastics compounding and slight confidence for Manufacturing 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 (people being exposed dermally and through incidental ingestion while swimming in surface water) were assessed as high-end potential exposures to BBP in surface waters. EPA's *Exposure Factors Handbook* provided detailed information on the 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. BBP concentrations are expected to decrease further downstream from this point of release due to further dilution, partitioning, and degradation, when released to surface waters, and the point-of-release exposure modeling conducted in this section is protective of the potential downstream exposures.

6 DRINKING WATER EXPOSURE TO GENERAL POPULATION

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

Very limited information is available on the removal of BBP in drinking water treatment plants. As stated in the *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate* (U.S. EPA, 2025i), no data were identified by the EPA for BBP in drinking water in the United States. Based on its water solubility (2.69 mg/L) and log K_{OW} (4.7), BBP in water is expected to mainly partition to suspended solids present in water. The available information suggest that the use of flocculants and filtering media could potentially help remove BBP during drinking water treatment by sorption into suspended organic matter, settling, and physical removal. However, as a conservative assumption, EPA did not assume a drinking water removal rate in estimating potential exposures to BBP via drinking water. No monitoring data reporting detectable levels of BBP were identified by the EPA for in drinking water in the United States.

6.1 Modeling Approach for Estimating Concentrations in Drinking Water

6.1.1 Drinking Water Ingestion

Drinking Water Intake Estimates via Modeled Surface Water Concentrations

Modeled surface water concentrations estimated in Section 4.1 were used to estimate drinking water exposures. For this screening exercise, only the highest modeled facility release was included in the drinking water exposure analysis, alongside the highest monitored surface water concentration. For reference, these high-end concentration estimates were considered with and without wastewater treatment prior to discharge to the receiving waterbody. When applied, a wastewater treatment efficiency of 62 percent removal efficiency (U.S. EPA, 1982) was assumed for treatment of facility The drinking water scenarios presented here no further drinking water treatment applied, are expected to be overestimations of actual high-end 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 BBP)
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 BBP)
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 for chronic non-cancer were calculated using the 95th percentile ingestion rate for drinking water. Table 6-1 summarizes the drinking water doses for adults, youth, and toddlers for water under scenarios with and without applying wastewater treatment. These estimates do not incorporate additional dilution beyond the point of discharge and in this case, it is assumed that the surface water outfall is located very close (within a few km) to the drinking water intake location. Applying dilution factors would decrease the dose for all scenarios.

Table 6-1. Modeled Drinking Water Doses for Adults, Toddlers, and Infants for the High-end Release Estimate from Modeling and Monitoring Results

Scenario	Surface Water Concentrations		Adult (21+ Years)		Toddler (1–5 Years)		Infant (Birth to <1 Year)	
	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)
PVC plastics compounding *Without wastewater treatment*	2,852	2,049	1.1E–01	1.57E−02	1.43E−01	1.72E−02	4.03E−01	4.00E−02
PVC plastics compounding *With wastewater treatment*	1,084	779	4.4E−02	5.96E−03	5.44E−02	6.52E−03	1.53E−01	1.52E−02

Scenario	Surface Water Concentrations		Adult (21+ Years)		Toddler (1–5 Years)		Infant (Birth to <1 Year)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Manufacturing *Without wastewater treatment*	24,500	17,600	9.86E−01	1.6E−01	1.23	1.7E−01	3.46	4.1E−01
Manufacturing *With wastewater treatment*	9,310	6,690	3.75E−01	6.04E−02	4.67E−01	6.62E−02	1.31	1.5E−01
Highest monitored surface water NWQMC (2021)	40	40	1.6E–03	3.61E−04	2.0E–03	3.96E−04	2.0E–03	9.23E−04

6.2 Measured Concentrations in Drinking Water

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of BBP in drinking water. Three references provided information related to BBP in drinking water (CA Water Board, 2022; Bach et al., 2020; Sulentic et al., 2018), but none reported detectable levels of BBP in drinking water.

6.3 Evidence Integration for Drinking Water

EPA estimates low potential exposure to BBP via drinking water, even when considering high-end release scenarios without applying drinking water removal efficiencies. Additional qualitative considerations suggest that actual measured concentrations in raw and finished water would decrease further. Available finished drinking water concentrations reported from the U.S. were below the limit of detection, corroborating the expectation of very little exposure to the general population via treated drinking water.

6.4 Weight of Scientific Evidence Conclusions

No facility- or site-specific information was reasonably available when estimating release of BBP to the environment. Environmental releases to water were estimated using generic scenarios (U.S. EPA, 2025f). 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 for PVC plastics compounding and slight confidence for Manufacturing 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 D and demonstrate no risk estimates to the general population below the benchmark. EPA has moderate confidence in the treated surface water as drinking water exposure scenario. 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 2.69 mg/L and (2) the maximum modeled concentration among OESs with only wastewater as the discharge type. (U.S. EPA, 2025i)). The range of water solubility values was 0.67 to 2.8 mg/L, and 2.69 mg/L was selected as the most environmentally relevant. While not the maximum, it is still near the upper bound and thus appropriate for use in a screening approach. For the modeled surface water concentrations, values were estimated for the PVC plastics compounding OES at the P50, P75, and P90 flow rates. Modeled concentrations for the PVC plastics compounding OES are not the highest across all the OESs if the ones discharging to multiple media types are also considered. The Manufacturing OES will result in the maximum modeled surface water concentration across all OESs. As described in Section 4.4, however, uncertainties inherent in assuming what proportion of a release discharges to water leads to low confidence in the results. EPA estimated the exposure and risks associated with the Manufacturing OES for informational purposes only (see *Draft Fish Ingestion Risk Calculator for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025g). The Agency did not use those results to assess the fish ingestion pathway. The remainder of this section discusses modeled surface water concentrations for only the PVC plastics compounding OES.

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 BBP, the estimated BAF and BCF values using the Arnot-Gobas method for upper trophic organisms are both 40.1 L/kg (see *Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025i)). Table 7-1 compares the fish tissue concentration calculated using a BAF/BCF and various surface water concentrations 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 are three orders of magnitude higher than empirical fish tissue concentrations reported within published literature.

In addition, EPA calculated fish tissue concentrations using the highest measured BBP concentrations in U.S. surface water for contextual purposes. As described in Section 4.2.1, the maximum concentration measured in U.S. surface water was 2.65 µg/L (2.65×10^{-3} mg/L) from WQP (NWQMC, 2021). The maximum surface water concentration among all reasonably available literature was slightly higher at 4 µg/L in France (Tran et al., 2014). Fish tissue concentrations calculated with the predicted BAF and monitored surface water concentrations are within the same order of magnitude as that reported within published literature (Table 7-1).

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

Approach	Data Description	Surface Water Concentration	Fish Tissue Concentration
Water solubility limit	Predicted BAF (Arnot-Gobas method) of 40.1 L/kg (U.S. EPA, 2024d)	Estimates of the water solubility limit for BBP, which is approximately 2.69 mg/L (Howard et al., 1985)	1.08E02 mg/kg ww

Approach	Data Description	Surface Water Concentration	Fish Tissue Concentration
Modeled surface water concentration	Predicted BAF (Arnot-Gobas method) of 40.1 L/kg (U.S. EPA, 2024d)	2.50 mg/L for PVC plastics compounding, P50 flow	1.00E02 mg/kg ww
Monitored surface water concentration	Highest measured concentration in the U.S. (NWQMC, 2021) and predicted BAF (Arnot-Gobas method) of 40.1 L/kg (U.S. EPA, 2024d)	2.65E–03 mg/L	1.06E–01 mg/kg ww
Fish tissue monitoring data (wild-caught)[a]	One U.S. study collected samples across 11 species (Camanzo et al., 1987)	N/A	BBP was not detected in any of the samples.
	Three Canadian studies collected samples across five species and in non-specified species (Cao et al., 2015; McConnell, 2007; Lin et al., 2003)		2.1E–01 to 1.2 to mg/kg ww
	Seven studies collected samples across 30 species and four countries (Taiwan (Huang et al., 2008), China (He et al., 2020; Hu et al., 2020; Cheng et al., 2018), France (Teil et al., 2014; Valton et al., 2014) Italy (Panio et al., 2020))		All seven studies reported only a dry weight and not wet weight concentration to enable comparison with the calculated fish tissue concentrations.

ww = wet weight

[a] These studies identified through systematic review that reported measured BBP 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 Extraction Information for General Population, Consumer, and Environmental Exposure for Butyl Benzyl Phthalate (BBP)* (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 fish IR per kilogram of body weight. This section estimates exposure and risks for adults and toddlers 1–2 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. Exposure estimates via fish ingestion were 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 (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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025g). 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. Because calculated fish tissue concentrations and thus exposure estimates are higher based on the water solubility limit than modeled concentrations for the PVC plastics compounding OES, EPA did not further refine the screening level analysis. 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 calculated with the water solubility limit.

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 (2.69 mg/L)	2.99E–02	4.44E–02	6.80E–03

7.2 Subsistence Fish Ingestion Exposure

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

The exposures calculated using the water solubility limit and predicted BAF are presented in Table 7-3. Corresponding screening level risk estimates are shown in Appendix E.2. Because calculated fish tissue concentrations and thus exposure estimates are higher based on the water solubility limit than modeled

concentrations for the PVC plastics compounding OES, EPA did not further refine the screening level analysis. Fish ingestion is not expected to be a pathway of concern for the subsistence fisher based on the conservative screening level risk estimates using an upper-bound of exposure calculated with the water solubility limit.

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

Surface Water Concentration	ADR/ADD (mg/kg-day)
Water solubility limit (2.69 mg/L)	1.92E–01

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 creates many unique exposure scenarios that can expose tribal members to higher doses of contaminants in the environment. EPA used the reasonably available information to quantitatively evaluate the tribal fish ingestion pathway for BBP but lacks reasonably available data to assess other unique exposure scenarios unique to tribal populations.

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

In addition to the current mean fish ingestion rate, EPA reviewed literature and surveys to identify a high-end (*i.e.*, 90th or 95th percentile) current 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) current 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.

Current ingestion rates are considered more representative of contemporary rates of fish consumption. However, because current fish consumption rates are suppressed by contamination, degradation, or loss

of access, EPA also reviewed existing literature for 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). They are less relevant than current ingestion rates. 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, with the exception of the ingestion rate. Three ingestion rates were used: 216 g/day (2.7 g/kg-day) for a central tendency current consumption rate; 874 g/day (10.9 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 old. 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 IR focused on adults (Table 7-4).

Table 7-4 presents multiple exposure estimates for the tribal populations. Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark (see Appendix E.3). Therefore, EPA refined its evaluation by using modeled surface water concentrations based on 1) the highest estimated 95th percentile release for the PVC plastics compounding OES as described in U.S. EPA (2024b) and 2) the median (50th percentile, P50), 75th percentile (P75), and 90th percentile (P90) flow metrics from the distribution. While the modeled surface water concentration using the P50 flow metric resulted in screening level risk estimates below the benchmark, the higher flow metrics (*i.e.*, P75 and P90) are expected to be more representative of the flow conditions associated with high-end releases. The more refined exposure estimates did not result in risk estimates below the benchmarks (see Appendix E.3). In addition, exposure estimates using modeled surface water concentrations are up to three orders of magnitude higher than those using the monitored surface water concentration. This indicates that modeled concentrations are conservative. Overall, ingestion of fish potentially contaminated with BBP is not expected to be a pathway of concern for the tribal population.

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

Surface Water Concentration and Scenario	ADR/ADD (mg/kg-day)		
	Current Mean IR	Current IR, 95th Percentile	Heritage IR
Water solubility limit (2.69 mg/L)	2.91E–01	1.18	2.22
PVC plastics compounding, P50 flow (2.05 mg/L)	2.22E−01	8.96E−01	1.69
PVC plastics compounding, P75 flow (1.53E–01 mg/L)	1.66E−02	6.69E−02	1.26E−01
PVC plastics compounding, P90 flow (3.48E–03 mg/L)	3.77E−04	1.52E−03	2.87E−03
Monitored surface water concentration (2.65E–03 mg/L) (NWQMC, 2021)	2.87E–04	1.16E–03	2.19E–03

7.4 Weight of Scientific Evidence Conclusions

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

To account for the variability in fish consumption across the United States, fish intake estimates were considered for both general population, subsistence fishing populations, and tribal populations. Conservative exposure estimates based on the water solubility limit resulted in screening level risk estimates below the benchmark for only tribal populations. EPA refined its analysis for tribal populations by using modeled surface water concentrations based on the highest estimated 95th percentile release for the PVC plastics compounding OES as described in U.S. EPA (2024b) and the P50, P75, and P90 flow metrics from the distribution. The higher flow metrics (*i.e.*, P75 and P90) are expected to be more representative of the flow conditions associated with high-end releases.

BBP is expected to have low potential for bioaccumulation, biomagnification, and trophic transfer through food webs (Section 12). This is supported by the estimated BCF/BAF value of 40.1 L/kg, respectively (U.S. EPA, 2024d), which does not meet the criteria to be considered bioaccumulative (BCF/BAF > 1,000). Furthermore, EPA did not find reasonably available data sources that report the aquatic bioconcentration, bioaccumulation, and trophic transfer of BBP through food webs.

As modeled surface water concentrations are biased toward overestimation, and bioconcentration, bioaccumulation, and trophic transfer of BBP 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 in the ambient air for this draft ambient air exposure assessment for BBP. EPA'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 BBP.

8.1 Approach for Estimating Concentrations in 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 BBP in the ambient air at three distances (*e.g.,* 100; 100 to 1,000, and 1,000 meters) from the releasing facility. IIOAC also estimates dry, wet, and total deposition rates of BBP 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 run results from a suite of dispersion scenarios in a variety of meteorological and land-use settings within EPA's American Meteorological Society/EPA Regulatory Model (AERMOD). Additional information on IIOAC can be found in the user guide (U.S. EPA, 2019d).

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

The maximum EPA estimated daily release value for BBP was 231 kg/site−day and categorized under the "Use of paints and coatings – no spray controls" 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. While 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 which may overestimate ambient concentrations of BBP.

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 meters, 100–1,000 meters, and 1,000 meters
- Meteorological Stations:
  - South (Coastal): Surface and Upper Air Stations at Lake Charles, Louisiana
- Operating Scenario: 365 and 287 days per year; 24 hrs/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 (see *BBP Draft Ambient Air IIOAC Exposure and Risk Calculator* (U.S. EPA, 2025a)). A description of select outputs relied upon in this assessment are provided below.

Fenceline Average: represents the daily-average and annual-average concentrations at 100 meters 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 meters.

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

High-end, Total Annual-average Deposition: 95th percentile annual-average total deposition rate across the entire distribution of modeled total deposition rates at 100 meters.

8.1.3 Modeled Results from IIOAC

All results for each scenario described in Section 8.1.1 are included in the *BBP Draft Ambient Air IIOAC Exposure and Risk Calculator* (U.S. EPA, 2025a). EPA utilized the highest estimated concentrations and deposition rates across all modeled scenarios to evaluate exposures and total 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 meters 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 Meters from Releasing Facility

Source Type	Daily-Average Concentration ($\mu g/m^3$)	Annual-Average Concentration ($\mu g/m^3$)
Fugitive	150.0	150.0
Stack	19.76	16.86
Total	169.76	166.86

The source apportioned wet and dry deposition rates and the total deposition rates for the scenario used in the *Draft Environmental Hazard Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025e) are provided in Table 8-3.

Table 8-3. Source Apportioned and Total Annual-average IIOAC Modeled Deposition Rates at 100 Meters from Releasing Facility

Source Type	Total Annual Deposition Rate (g/m^2)		
	Total	Wet	Dry
Fugitive	6.94E−03	6.86E−03	9.99E−05
Stack	1.35E−03	1.31E−03	8.06E−05
Total	8.30E−03	8.17E−03	1.81E−04

8.2 Measured Concentrations in Ambient Air

EPA reviewed published literature as described in the *Draft Data Quality Evaluation Information for General Population, Consumer, and Environmental Exposure for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025d) to identify studies where ambient concentrations of BBP 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) which measured concentrations of several phthalates including BBP. 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 BBP in this study were low, generally in the ng/m^3 range. How these data do or do not 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 BBP concentrations (U.S. EPA, 2022a).

8.3 Evidence Integration

EPA relied on the IIOAC modeled concentrations and deposition rates to characterize human and ecological exposures for the ambient air exposure assessment. Modeled BBP 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 meters away), and it is unknown if the sampling sites are located at a similar distance from a site. Additionally, measured concentrations represent all sources (TSCA and other sources) contributing BBP to the ambient air, while modelled concentrations are specific to TSCA sources.

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.

BBP did not have any reported releases in databases EPA typically relies upon for facility reported release data (*e.g.*, TRI or NEI). Therefore, BBP 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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025f), 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 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 BBP. 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 meters distance from releasing facilities, the size of the facility, and the release point within a facility. For larger facilities, 100 meters 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 meters 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 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 231 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. None−the−less, 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 BBP via the ambient air pathway does not pose an exposure or risk concern and no further analysis is needed. If results indicated an exposure or risk concern, EPA 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 low 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 EPA 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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025j) EPA has high confidence the ambient air pathway is not a pathway of concern for either exposure to or deposition rates of BBP.

9 AMBIENT AIR EXPOSURE TO GENERAL POPULATION

9.1 Exposure Calculations

Modeled ambient air concentration outputs from IIOAC need to be converted to estimates of exposures to derive risk estimates. For this exposure assessment, EPA assumes the general population is continuously exposed (*i.e.*, 24 hours per day, 365/287 days per year) to outdoor ambient air concentrations. Therefore, daily average modeled ambient air concentrations are equivalent to acute exposure concentrations, and annual average modeled ambient air concentrations are equivalent to chronic exposure concentrations used to derive risk estimates (Section 8.1.3). Calculations for general population exposure to ambient air via inhalation and ingestion from air to soil deposition for lifestages expected to be highly exposed based on exposure factors can be found in *Draft Ambient Air IIOAC Exposure Results and Risk Calculations For Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025b).

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 Hazard Assessment for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025h), EPA does not expect an inhalation risk from ambient air nor ingestion risk from air to soil deposition to result from exposures to BBP 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 BBP through inhalation of ambient air or ingestion from air to soil deposition are not necessary.

10 HUMAN MILK EXPOSURE

Infants are potentially susceptible because of their higher exposure per body weight, immature metabolic systems, and the potential for chemical toxicants to disrupt sensitive developmental processes, among other reasons. Reasonably available information from studies of experimental animal models also indicates that BBP is a developmental toxicant (U.S. EPA, 2025h). EPA considered exposure (Section 10.1) and hazard (Section 10.2) information, as well as pharmacokinetic models (Section 10.3), to determine the most appropriate approach to evaluate infant exposure to BBP 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 the gestational and/or perinatal period. In other words, exposure and risk estimates from maternal exposure are expected to be protective of nursing infants as well.

10.1 Biomonitoring Information

BBP has the potential to accumulate in human milk because of its small mass (312.4 Daltons or g/mol) and lipophilicity (log K_{OW} = 4.73). EPA identified nine biomonitoring studies through systematic review, of which one is a U.S. study ((Hartle et al., 2018)), from reasonably available information that investigated if BBP or its metabolites were present in human milk. These nine studies provide evidence of BBP 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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025d). Three of the nine studies did not detect any of the compounds (Zimmermann et al., 2012; Fromme et al., 2011; Schlumpf et al., 2010). A summary of the studies is provided in Figure 10-1. None of the studies characterized if any of the study participants may be occupationally exposed to BBP.

BBP was measured in all 21 samples collected from the Mother's Milk Bank in California. The concentrations ranged from 1.59 to 83.2 ng/g lipid weight (lw) with a mean of 25.08 ng/g (Hartle et al., 2018). Five non-U.S. studies measured BBP or its primary metabolite, MzBP (monobenzyl phthalate) as wet weight. In those studies, the concentrations in human milk ranged from less than 0.06 to 26 µg/L ww (Kim et al., 2018; Lin et al., 2011; Latini et al., 2009; Hogberg et al., 2008; Main et al., 2006).

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 BBP or their metabolites can be attributed solely or partially to TSCA uses. The use of biomonitoring data to characterize a nursing infant's exposure to BBP represents an aggregate exposure from all BBP sources and pathways which may contribute to the presence of BBP 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.

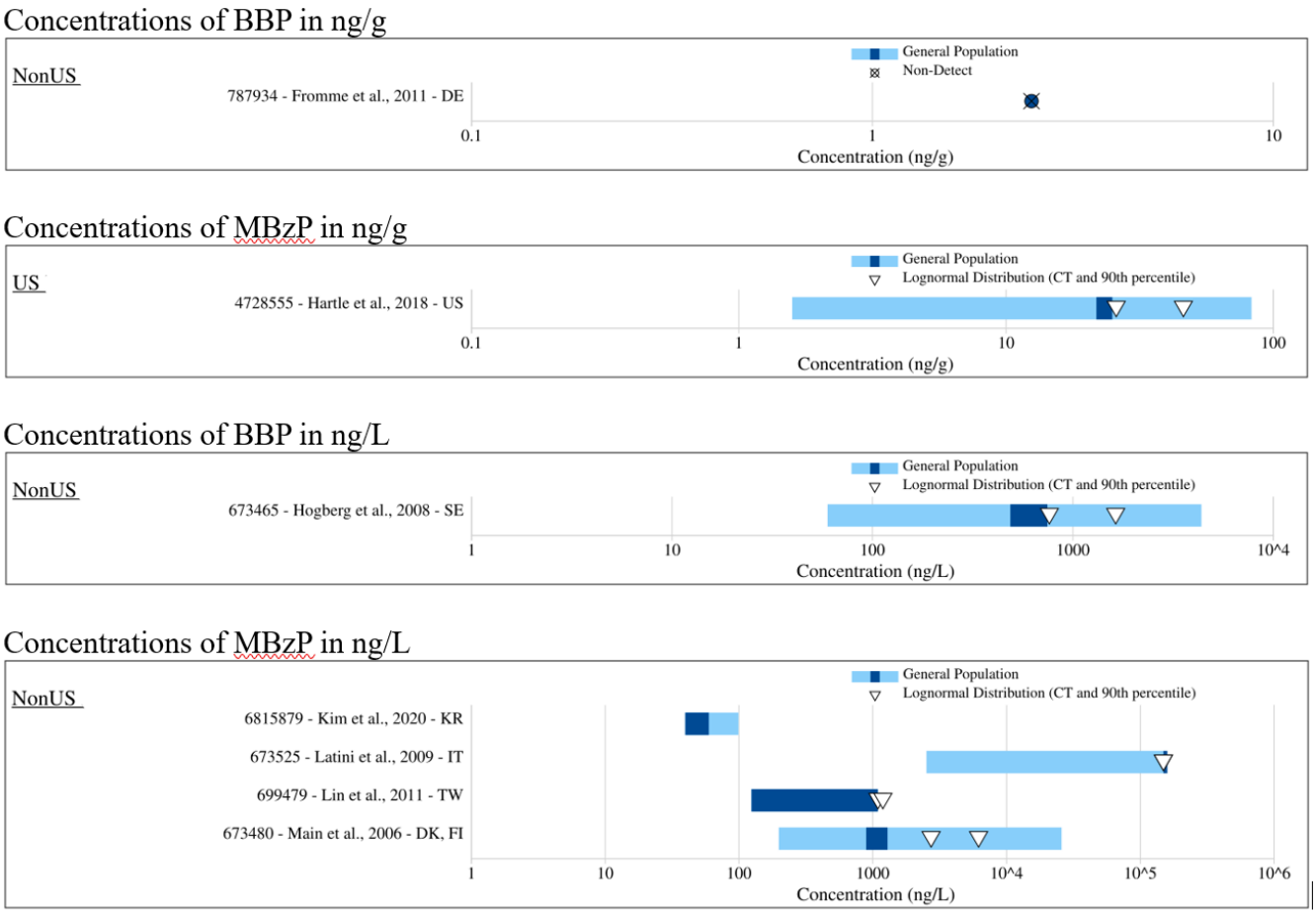


Figure 10-1. Concentrations of BBP or MBzP in Human Milk in Either Lipid (ng/g) or Wet (ng/L) Weight

These studies provide evidence of BBP or MzBP in human milk and were not used as part of the analysis for quantifying exposure estimates. 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 Butyl Benzyl Phthalate (DBP)* (U.S. EPA, 2025d).

10.2 Modeling Information

EPA explored the potential to model BBP 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. EPA 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. However, due to significant uncertainties in establishing an appropriate half-life value for BBP, use of the model to quantify lactational transfer and exposure for BBP was not supported.

EPA considered the model input data available for BBP and concluded that uncertainties in establishing an appropriate half-life value precludes using the model to quantify lactational transfer and exposure from TSCA COUs. Measurement of the parent phthalate (*i.e.*, BBP) in organs, tissues, and matrices is prone to error and contamination from sampling materials because of its rapid hydrolysis (Koch and Calafat, 2009). BBP is predominantly excreted in urine as the monoester metabolite, mono-benzyl

phthalate (MBzP), as well a minor amount as mono-n-butyl phthalate (MnBP). MnBP is also the major metabolite of dibutyl phthalate (DBP) (see the toxicokinetics summary in the *Draft Human Health Hazard Assessment for Butyl Benzyl Phthalate* (U.S. EPA, 2025h). This indicates that neither the parent compound nor the primary metabolite is a sensitive biomarker of exposure to DBP. As a result, measured half-life values for BBP and MBzP reported by Eigenberg et al. (1986) and Kim et al. (2015) were not considered. No data were available for secondary oxidized metabolites in humans. These uncertainties in establishing an appropriate half-life value for BBP do not support using the model to quantify lactational transfer and exposure for TSCA COUs.

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

10.3 Hazard Information

EPA considered developmental and reproductive toxicity studies of rats that evaluated the effects of oral exposures to BBP resulting from maternal exposures. The critical effect is disruption to androgen action during the critical window of male reproductive development, leading to a spectrum of effects on the developing male reproductive system that is consistent with phthalate syndrome. These effects follow gestational and/or perinatal oral exposures to BBP (see *Draft Non-cancer Human Health Hazard Assessment for Butyl Benzyl Phthalate* (U.S. EPA, 2025h)). No studies have evaluated only lactational exposure (*i.e.*, from birth to weaning) from quantified levels of BBP or its metabolites in milk. However, the hazard values are based on developmental and reproductive toxicity in the offspring following maternal exposure during gestation and the perinatal period. Because these values designed to be protective of infants are expressed in terms of maternal exposure 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 be uninformative.

10.4 Weight of Scientific Evidence Conclusions

EPA considered infant exposure to BBP through human milk because the available biomonitoring data demonstrate that BBP can be present in human milk and hazard data demonstrate that the developing male reproductive system may be particularly susceptible to the effects of BBP. While 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, EPA 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 in multigenerational studies. EPA therefore has confidence that the risk estimates calculated based on maternal exposures are protective of a nursing infant's greater susceptibility during this unique lifestage whether due to sensitivity or greater exposure per body weight.

11 URINARY BIOMONITORING

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

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

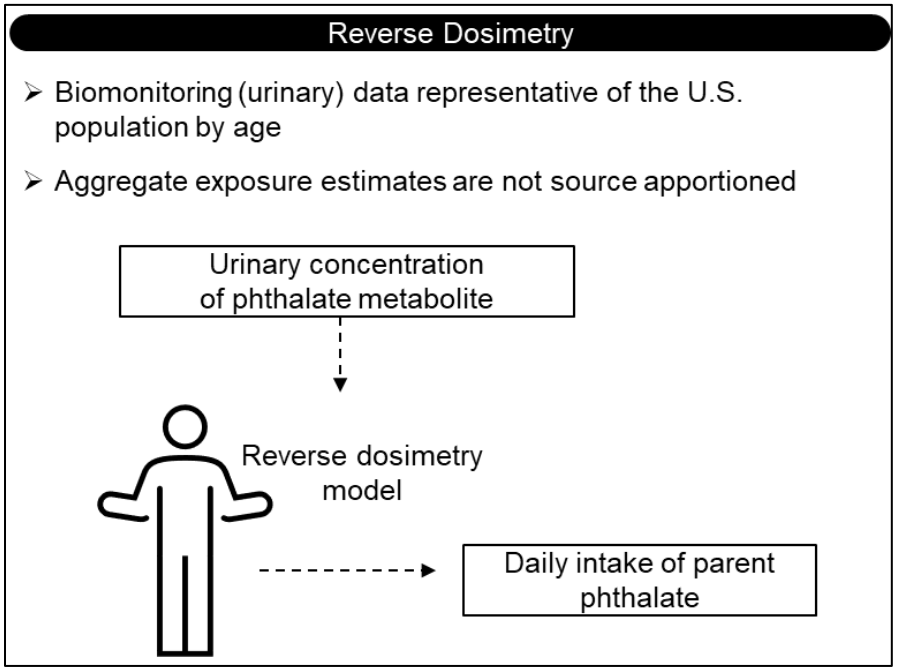


Figure 11-1. Reverse Dosimetry Approach for Estimating Daily Intake

11.1.1 Approach for Analyzing Biomonitoring Data

EPA analyzed urinary biomonitoring data from NHANES, which reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. Specifically, EPA analyzed data for MBzP, a metabolite of BBP, which has been reported in the 1999–2018 NHANES cycles. Sampling details can be found in Appendix B. Urinary concentrations of MBzP 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 MBzP were analyzed for all available

NHANES survey years to examine the temporal trend of BBP 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. Statistical analyses of BBP metabolite trends over time were performed with PROC DESCRIPT using SAS-callable SUDAAN.

11.1.1.1 Temporal Trend of MBzP

The figures below show urinary MBzP concentrations plotted over time for the various populations to visualize the temporal exposure trends. Overall, MBzP urinary concentrations have decreased over time across all life stages.

From 1999 to 2018, 50th percentile MBzP concentrations decreased significantly for all children under 16 ($p<0.001$), as well as for male children ($p<0.001$) and female children ($p<0.001$) (Figure 11-4). This trend held for all age groups: 3 to less than 6 years ($p<0.001$) (Figure 11-5), 6 to less than 11 ($p<0.001$) (Figure 11-6), and 11 to less than 16 years ($p<0.001$) (Figure 11-7). The 50th percentile MBzP urinary concentrations also decreased significantly amongst all adults ($p<0.001$), adult males ($p<0.001$), and adult females ($p<0.001$) (Figure 11-2).

From 1999 to 2018, 95th percentile MBzP concentrations also decreased significantly for all children under 16 ($p<0.001$), as well as for male children ($p<0.001$), female children ($p<0.001$) (Figure 11-5), and children ages 11 to less than 16 years ($p<0.001$) (Figure 11-7). The 95th percentile MBzP concentrations decreased significantly amongst all adults ($p<0.001$), as well as amongst adult males ($p<0.001$) and adult females ($p<0.001$) over time (Figure 11-2).

From 1999 to 2018, both 50th and 95th percentile MBzP urinary concentrations decreased amongst women of reproductive age ($p<0.001$ for 50th and 95th percentile) (Figure 11-3).

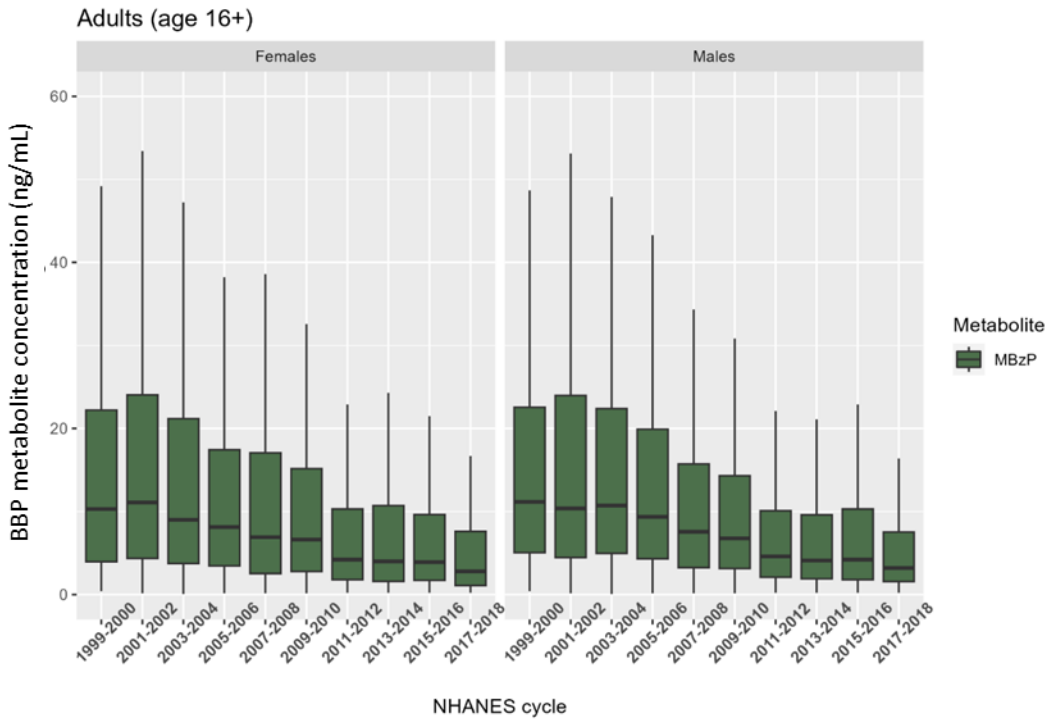


Figure 11-2. Urinary BBP Metabolite Concentrations for Adults (16+ Years Old)

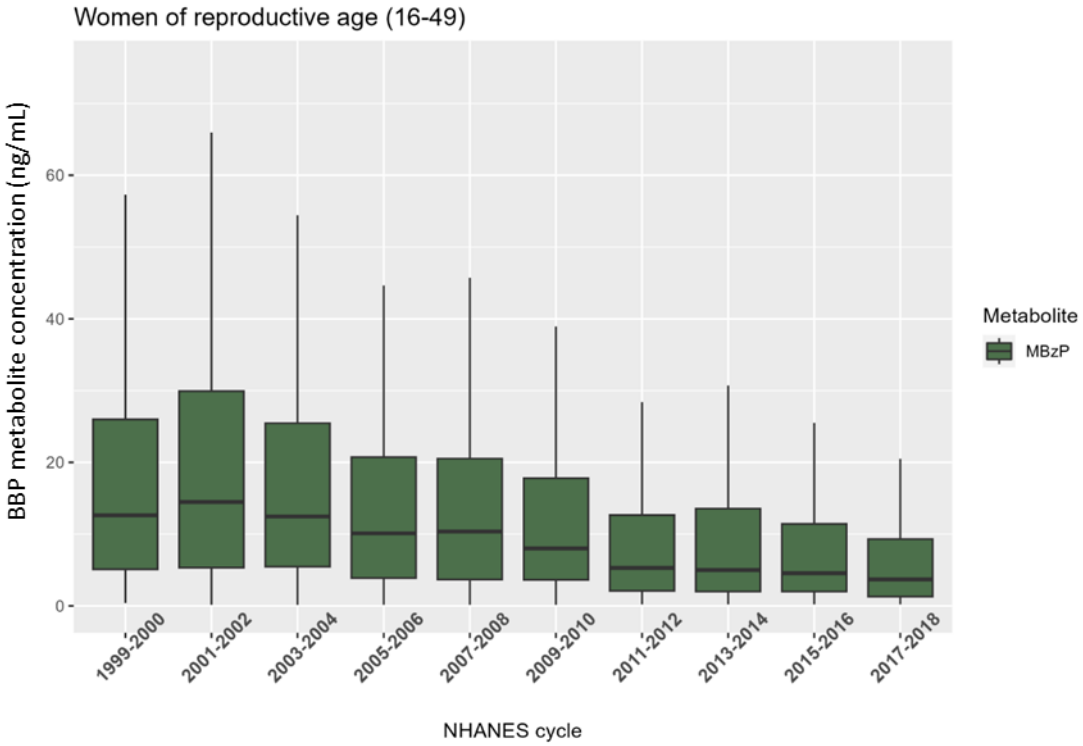


Figure 11-3. Urinary BBP Metabolite Concentrations for Women of Reproductive Age (16–49 Years Old)

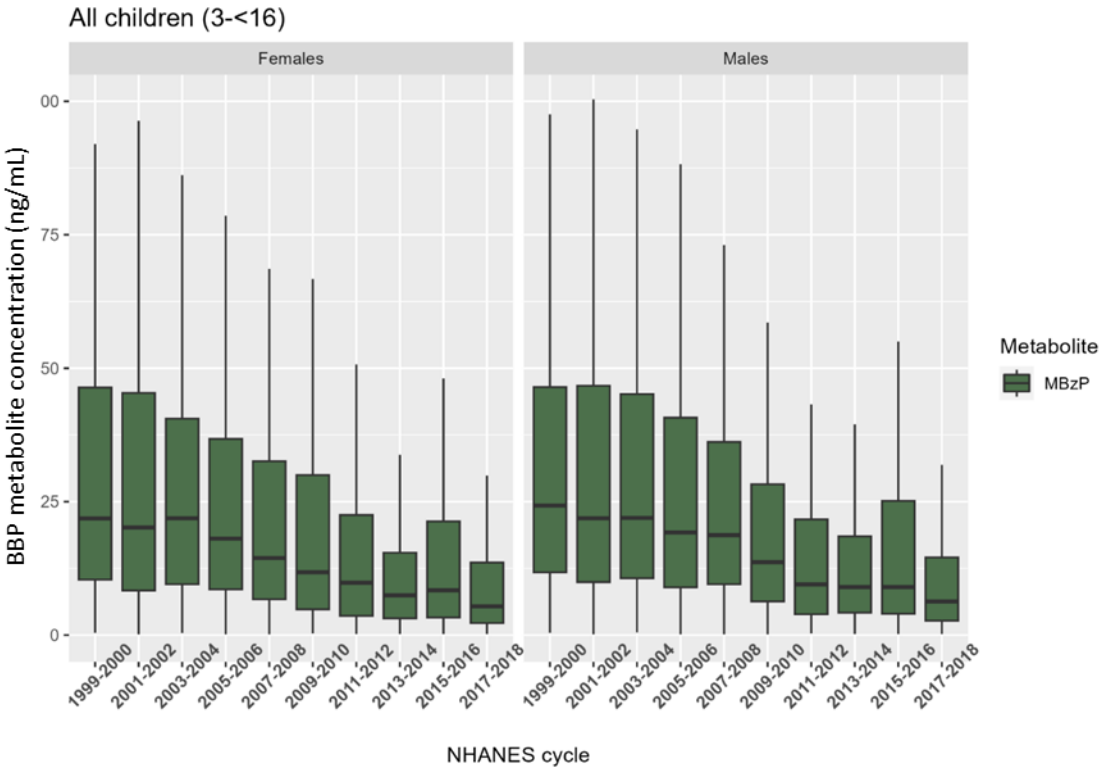


Figure 11-4. Urinary BBP Metabolite Concentrations for All Children (3 to <16 Years Old) by Sex

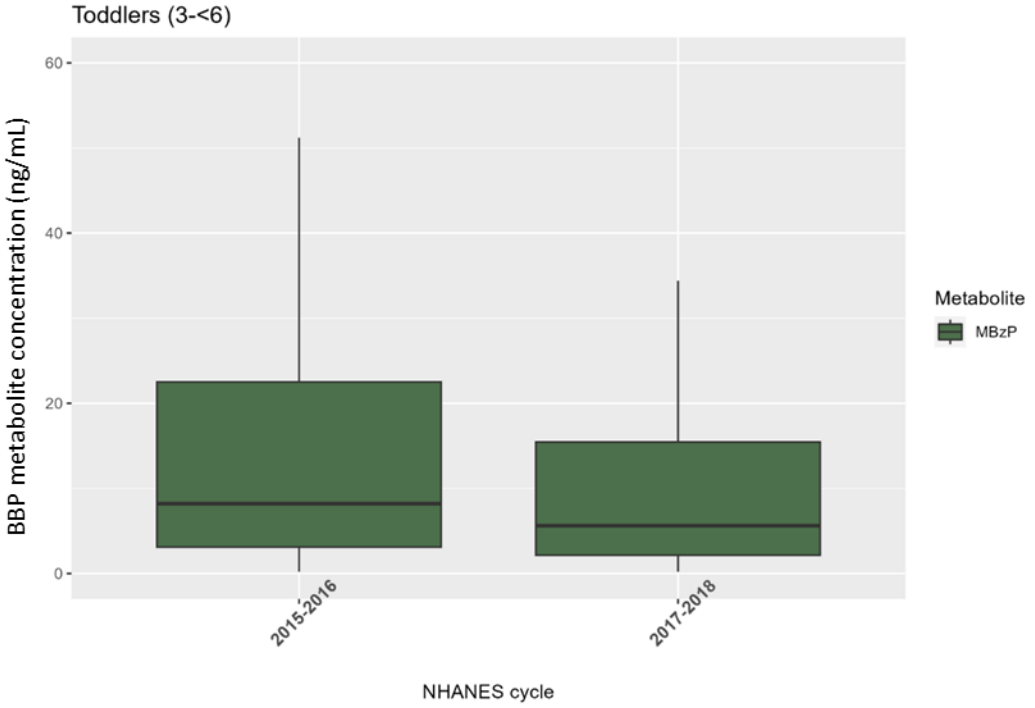


Figure 11-5. Urinary BBP Metabolite Concentrations for Toddlers (3 to <6 Years Old)

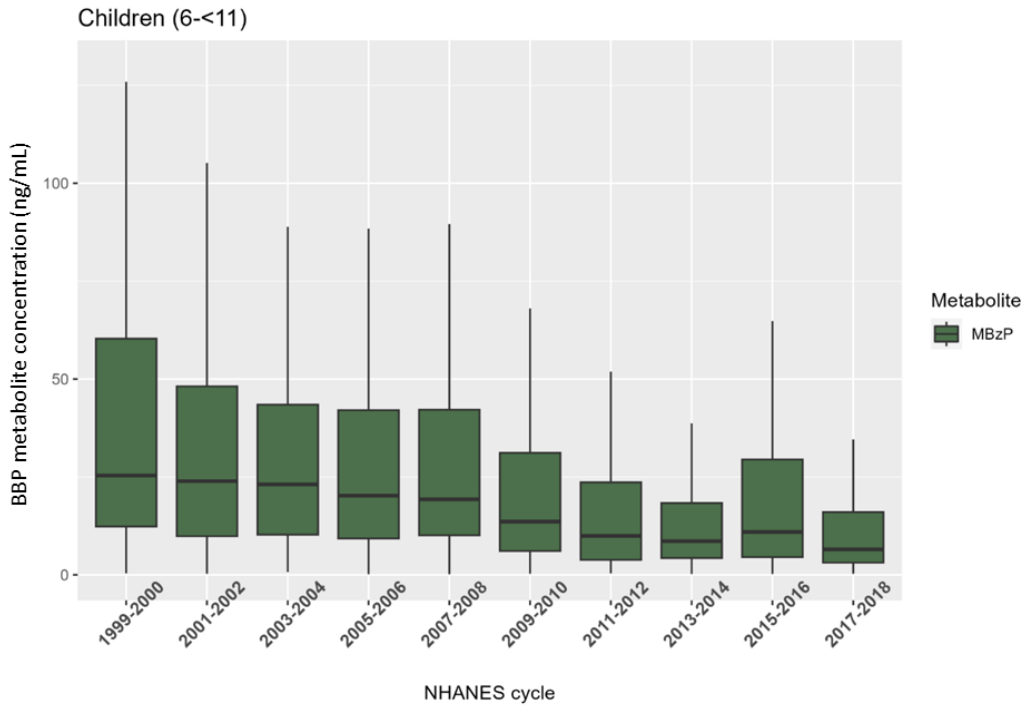


Figure 11-6. Urinary BBP Metabolite Concentrations for Children (6 to <11 Years Old)

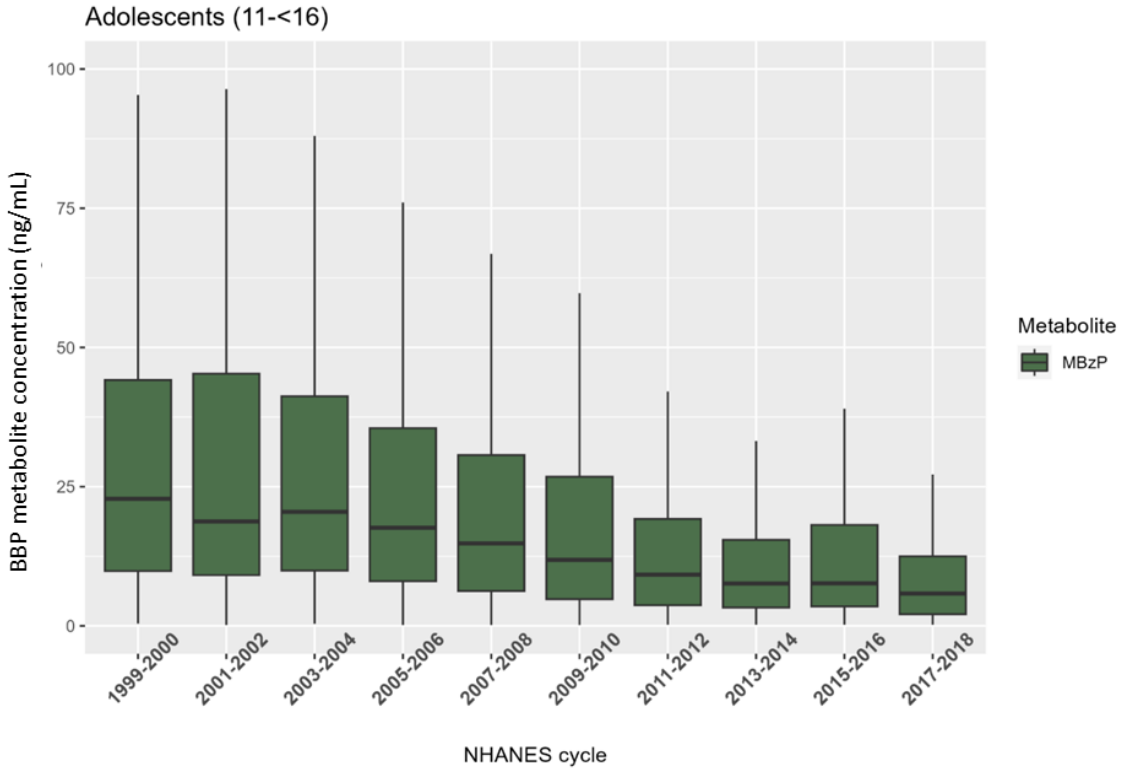


Figure 11-7. Urinary BBP Metabolite Concentrations for Adolescents (11 to <16 Years Old) by Sex

11.1.1.2 Daily Intake of BBP from NHANES

Using BBP metabolite concentrations measured in the most recently available sampling cycle (2017–2018), EPA estimated the daily intake of BBP 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. CPSC (2014) and Health Canada (Health Canada, 2020) to estimate daily intake values for exposure assessment. For phthalates, reverse dosimetry can be used to estimate a daily intake (DI) value for a parent phthalate diester based on phthalate monoester metabolites measured in human urine using Equation 11-1 (Koch et al., 2007). For BBP, the phthalate monoester metabolite is MBzP.

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

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

Metabolite	F_{ue}[a]	Reference	Study Population
MBzP	0.73	Anderson et al. (2011)	n = 14 volunteers (sex and age not provided)

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

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

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

Demographic	50th Percentile Daily Intake Value (Median [95% CI]) (µg/kg-day)	50th Percentile Daily Intake Value (Median [95% CI]) (µg/kg-day)
All	0.12 (0.11–0.14)	0.85 (0.68–1.01)
Females	0.12 (0.1–0.14)	0.83 (0.54–1.11)
Males	0.13 (0.11–0.15)	0.85 (0.57–1.12)
White non-Hispanic	0.15 (0.12–0.18)	0.87 (0.54–1.2)
Black non-Hispanic	0.14 (0.11–0.16)	0.94 (0.63–1.24)
Mexican-American	0.11 (0.08–0.13)	0.78 (0.57–0.99)
Other	0.11 (0.09–0.13)	0.62 (0.23–1)
Above poverty level	0.18 (0.15–0.22)	1.15 (0.7–1.6)
Below poverty level	0.12 (0.1–0.14)	0.79 (0.56–1.01)
Toddlers (3 to <6 years old)	0.22 (0.18–0.26)	1.74 (0.88–2.6)
Children (6 to <11 years old)	0.14 (0.11–0.17)	0.85 (0.49–1.22)
Adolescents (12 to <16 years old)	0.12 (0.07–0.16)	0.55 (0.26–0.83)
Adults (16+ years old)	0.09 (0.07–0.11)	0.37 (0.28–0.47)
Male toddlers (3 to <6 years old)	0.22 (0.15–0.3)	2.46 (0.84–4.07)
Male children (6 to <11 years old)	0.16 (0.11–0.2)	0.84 (0.4–1.29)
Male adolescents (11 to <16 years old)	0.14 (0.1–0.18)	0.64[a]
Male adults (16+ years old)	0.1 (0.07–0.12)	0.34[a]
Female toddlers (3 to <6 years old)	0.2 (0.14–0.27)	1.61 (0.72–2.5)
Female children (6 to <11 years old)	0.12 (0.08–0.17)	0.88 (0.27–1.48)
Female adolescents (11 to <16 years old)	0.1 (0.03–0.17)	0.53 (N/A)[a]
Women of reproductive age (16–49 years old)	0.08 (0.06–0.11)	0.42 (0–0.85)
Female adults (16+ years old)	0.08 (0.06–0.11)	0.42 (0–0.85)

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

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

Median and 95th percentile daily intake values in the U.S. CPSC (2014) report were estimated for men and women of reproductive age (15–45). U.S. CPSC reports a median daily intake value for adults aged 15 to 45 as 0.29 µg/kg-day and a 95th percentile daily intake value of 1.3 µg/kg-day.

The Health Canada (Health Canada, 2020) assessment reports median daily intake values for male children and female children aged 6 to 11 as 1.3 µg/kg-day. Among 12 to 19 year-old males, the median daily intake value was 0.36 µg/kg-day and the 95th percentile was 1.4 µg-kg/day, and among 12 to 19 year-old females, the median daily intake value was 0.28 µg/kg-day and the 95th percentile was 1.6 µg/kg-day The reported median and 95th percentile daily intake values for adults (age 20–49) were 0.2 and 0.97 µg/kg-day for males and 0.19 and 1.2 µg/kg-day for females.

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. Taken together with results from U.S. CPSC (2014) stating that BBP exposure comes primarily from diet and indoor exposures for infants, toddlers, children, and women and that outdoor environment did not contribute to BBP exposures, general population exposures via ambient air, surface water, and drinking water quantified in this document are likely overestimates. The estimates from individual pathways exceed the total intake values measured even at the 95th percentile of the U.S. population for all ages.

11.1.2 Limitations and Uncertainties of Reverse Dosimetry Approach

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

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

In addition to some of the limitations and uncertainties discussed above and outlined by U.S. CPSC (2014), the short half-lives of phthalates can be a challenge when using a reverse dosimetry approach. Phthalates have elimination half-lives on the order of several hours and are quickly excreted from the

body in urine and to some extent feces (ATSDR, 2022; EC/HC, 2015). Therefore, spot urine samples, as collected through NHANES and many other biomonitoring studies, are representative of relatively recent exposures. Spot urine samples were used by Health Canada (Health Canada, 2020) and U.S. CPSC (2014) to estimate daily intake values. However, due to the short half-lives of phthalates, a single spot sample may not be representative of average urinary concentrations that are collected over a longer term or calculated using pooled samples (Shin et al., 2019; Aylward et al., 2016). Multiple spot samples provide a better characterization of exposure, with multiple 24-hour samples potentially leading to better characterization but are less feasible to collect for large studies (Shin et al., 2019). Due to rapid elimination kinetics, U.S. CPSC concluded that spot urine samples collected at a short time (2 to 4 hours) since last exposure may overestimate human exposure, while samples collected at a longer time (greater than 14 hours) since last exposure may underestimate exposure (see Section 4.1.3 of U.S. CPSC (2014) for further discussion).

11.1.3 Weight of Scientific Evidence Conclusions

For the urinary biomonitoring data, despite the uncertainties discussed in Section 11.1.2, 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. Reverse dosimetry modeling does not distinguish between routes or pathways of exposure and does not allow for source apportionment (*i.e.*, exposure from TSCA COUs cannot be isolated), but EPA has robust confidence in the use of its total daily intake value to contextualize the exposure estimates from TSCA COUs as being overestimated as described in Section 11.1.1.2.

12 ENVIRONMENTAL BIOMONITORING AND 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. EPA has assessed the available studies 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 Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2024c) relating to the biomonitoring of BBP. 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 the descriptions of BBP content in biotic tissue via biomonitoring studies and provides qualitative descriptions of the potential dietary exposures to aquatic and terrestrial organisms via feeding (trophic) relationships.

12.1 Environmental Biomonitoring

Four studies reported BBP in organism tissues as wet weight concentrations. Measured BBP concentrations were reported from studies examining phthalate ester levels in aquatic ecosystems and in organisms at multiple trophic levels from primary producers (algae) to top predators.

BBP concentrations were only reported for one primary producer from aquatic ecosystems (McConnell, 2007). In Vancouver, British Columbia, Canada, the green algae *Prasiola meridionalis* from the urban False Creek Harbor had a geometric mean whole body BBP concentration of 0.0099 mg/kg wet weight (ww) (McConnell, 2007).

BBP concentrations were reported for four species of primary consumers (*e.g.*, crustaceans and mollusks) (Sánchez-Avila et al., 2013; Blair et al., 2009; McConnell, 2007). The hepatopancreas of the dungeness crab (*Cancer magister*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a geometric mean BBP concentration of 0.010 mg/kg ww (McConnell, 2007). For four mollusk species, geometric mean BBP concentrations ranged from approximately 0.0013 to 0.0031 mg/kg ww in blue mussel (*Mytilus edulis*), the softshell clam (*Mya arenaria*), Pacific oysters (*Crassostrea gigas*), and geoduck clams (*Panope abrupta*) which were sampled from the urban False Creek Harbor in Vancouver, British Columbia, Canada (Blair et al., 2009; McConnell, 2007; Mackintosh et al., 2004). Together, primary consumers had geometric mean BBP concentrations ranging from 0.0013 to 0.010 mg/kg ww (Blair et al., 2009; McConnell, 2007; Mackintosh et al., 2004).

One U.S. study collected samples across eleven fish species but did not find BBP in any fish tissue samples (Camanzo et al., 1987). Omnivorous and piscivorous finfish are secondary/tertiary consumers that had BBP wet weight concentrations reported for twelve species (McConnell, 2007; Camanzo et al., 1987). For three omnivorous finfish, the shiner perch (*Cymatogaster aggregata*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a geometric mean BBP concentration in its whole body at 0.0079 mg/kg ww (McConnell, 2007). For nine piscivorous finfish, the spiny dogfish (*Squalus acanthias*) from the urban False Creek Harbor in Vancouver, British Columbia, Canada had a geometric mean BBP concentration in its liver at 0.18 mg/kg ww (McConnell, 2007).

12.2 Trophic Transfer

EPA does not expect BBP to persist in surface water, groundwater, or air. BBP may persist in sediment, soil, biosolids, or landfills after release to these environments, but BBP's bioavailability is expected to

be limited (U.S. EPA, 2025i). BBP has a log K_{OW} of 4.73, which indicates high hydrophobicity and possible potential for bioconcentration. However, BBP has a log K_{OC} of 4.82 which indicates a strong affinity for BBP to sorb onto organic matter reducing BBP's availability in the water. The reasonably available BCFs reported in the literature range from 12.4 to 663 (U.S. EPA, 2025i; Carr et al., 1997; Barrows et al., 1980), and are below the Canadian Environmental Protection Act bioaccumulation criterion of 5,000 (Government of Canada, 2000). Modeling results from the BCFBAF™ module in EPI Suite™ predict a BAF of 40 for BBP (U.S. EPA, 2017a). The modeled BAF of BBP (40) based on simple lipid-water partitioning is in the same order of magnitude and in reasonable agreement with the measured BCF of intact BBP (12.4) (U.S. EPA, 2025i) (Carr et al., 1997). The similarity between the BAF, which estimates BBP uptake in the organism through all routes of chemical exposure (*e.g.*, dietary absorption, transport across the respiratory surface, dermal absorption) and BCF, which estimates BBP uptake only through transport across respiratory surfaces and dermal absorption, suggests that the bioaccumulation of these substances is mainly the result of chemical exchange between the organism and the water via the respiratory surfaces. Dietary uptake, metabolic transformation, and growth dilution appear to play a secondary role, but may contribute to the variability in the observed BAFs among the different organisms (Mackintosh et al., 2004; Gobas et al., 2003).

The empirically derived trophic magnification factor (TMF) of BBP is 0.77 from a marine food web in False Creek Harbor (Vancouver, British Columbia), indicating trophic dilution from lower to higher trophic levels and no biomagnification (Mackintosh et al., 2004). Additionally, two studies reported BSAF values of 2 to 20 for five species of fish from rivers in Taiwan (Huang et al., 2008) and 2.8 to 4.3 for three species of fish from the Orge river in France (Teil et al., 2012). Overall, the empirically measured data suggest that BBP will have a low bioaccumulation and trophic magnification potential in aquatic organisms. This conclusion is consistent with the observations made for other phthalates with measured BCF/BAFs such as di-isodecyl phthalate (DIDP), di-isononyl phthalate (DINP), and di-ethylhexyl phthalate (DEHP) (Mackintosh et al., 2004).

EPA identified two studies that report BAF values for BBP in terrestrial environments. One study reported values of 6.79 to 35.75 for wheat and 1.41 to 2.90 for maize (Li et al., 2018). The other study measuring concentrations of BBP in vegetables did not detect BBP in any of the vegetables sampled (n = 16), which indicates no terrestrial bioaccumulation potential (Li et al., 2016). The measured data suggest that BBP will have a low bioaccumulation and biomagnification potential in terrestrial organisms.

Overall, EPA conducted qualitative assessments of the physical properties, fate, and exposure of BBP and preliminarily determined that BBP has low bioaccumulation potential, and trophic transfer is unlikely to occur in aquatic or terrestrial food webs. Thus, EPA did not conduct a quantitative modeling analysis of the trophic transfer of BBP through food webs.

12.3 Weight of Scientific Evidence Conclusions

Given the reasonably available data, EPA has robust confidence that BBP is not readily found or if found is in relatively low concentrations in organism tissues, and that BBP has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms, and thus low potential for trophic transfer through food webs.

The conclusion that BBP is not readily detected in organism tissue is supported by the few studies reporting biomonitoring data. This conclusion is weakened because only one of these studies was conducted in the United States. The conclusion that BBP has low bioaccumulation and biomagnification potential is supported by the laboratory and field estimates of BCF values and modeled BCF/BAF

values, the relatively low concentrations detected in fish species, and an empirical study indicating food web biodilution (Mackintosh et al., 2004).

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

13.1 Environmental Exposure Conclusions

BBP 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 Butyl benzyl phthalate (BBP)* (U.S. EPA, 2025i).

EPA conducted modeling with VVWM-PSC (U.S. EPA, 2019c) to estimate concentrations of BBP within surface water and sediment. PSC inputs include physical and chemical properties of BBP (*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 BBP-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 BBP.

There are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered for BBP. However, based on high-quality physical and chemical property data, EPA determined that BBP 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 BBP concentrations from air deposition to soil (Table 8-3) and modeled BBP 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, 2025e) for relevant soil dwelling organisms and plants within the Environmental Risk Characterization section of the *Draft Risk Evaluation for Butyl Benzyl Phthalate (BBP)* (U.S. EPA, 2025j).

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

13.3 General Population Exposure Conclusions

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

Table 13-1 summarizes the conclusions for the exposure pathways and lifestages that were assessed for the general population based on starting with a screening level approach using high-end environmental media concentrations and refining estimates as needed. EPA conducted a quantitative evaluation for the following: incidental dermal and incidental ingestion from swimming in surface water, drinking water ingestion, fish ingestion, and ambient air inhalation. Biosolids and landfills were assessed qualitatively in Sections 3.1 and 3.2, respectively. Exposure results are found in the sections linked in Table 13-1 and risk results are found in Appendix C, Appendix D, and Appendix E. Results indicate that no pathways were of concern for BBP for the highest exposed populations.

Table 13-1. 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 exposure scenarios were assessed for qualitative assessments			No
All	Landfills (Section 3.2)	All exposure scenarios were assessed for qualitative assessments			No
Manufacturing	Surface water	Dermal	Dermal exposure to BBP in surface water during swimming (Section 5.1.1 and Appendix C)	All	No
		Oral	Incidental ingestion of BBP in surface water during swimming (Section 5.1.2 and Appendix C)	All	No
Manufacturing	Drinking water	Oral	Ingestion of drinking water (Section 6 and Appendix D)	All	No
All	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1 and	Adult (21+ years)	No

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Pathway of Concern[b]
			Appendix E)		
All			Ingestion of fish for subsistence fishers (Section 7.2 and Appendix E)	Adult (21+ years)	No
PVC plastics compounding			Ingestion of fish for tribal populations (Section 7.3 and Appendix E)	Adult (21+ years)	No
Use of paints and coatings—no spray controls (Stack and fugitive)	Ambient air	Inhalation	Inhalation of BBP in ambient air resulting from industrial releases (Section 9.1)	All	No
		Oral	Ingestion of soil from air to soil deposition resulting from industrial releases (Section 9)	Infants and Children (6 months to 12 years)	No

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

13.4 Weight of Scientific Evidence Conclusions for General Population Exposure

The weight of scientific evidence supporting the exposure estimate is decided based on the strengths, limitations, and uncertainties associated with the exposure estimates, which are discussed in detail for biosolids (Section 3.1.1), landfills (Section 3.2.1), surface water (Sections 4.3.1 and 4.4), drinking water (Section 6.4), fish ingestion (Section 7.4.1), ambient air (Sections 8.3.1 and 8.4), and human milk (Section 10.4). EPA conducted reverse dosimetry to calculate daily intake values for BBP using biomonitoring data from NHANES. However, in contrasting the screening level analyses for general population exposures 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 strengths, limitations, and uncertainties associated with the reverse dosimetry approach is available in Section 11.1.2.

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

EPA determined robust confidence in its qualitative assessment and conclusions pertaining to exposures from biosolids (Section 3.1.1) and landfills (Section 3.2.1). For its quantitative assessment, EPA modeled exposure due to various exposure scenarios resulting from different pathways of exposure. Exposure estimates used high-end inputs for the purpose of a screening level analysis. When available, monitoring data were compared to modeled estimates to evaluate overlap, magnitude, and trends to

inform confidence in the quantitative exposure assessment of surface water (Sections 4 and 5), drinking water (Section 6), fish ingestion (Section 7), ambient air (Sections 8 and 9), and human milk (Section 10). EPA has robust confidence that the screening level analysis was appropriately conservative to determine that no environmental pathway has the potential for non-cancer risks to the general population. Despite slight 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 which yielded modeled values exceeding those of monitored values. Furthermore, risk estimates for high-end exposure scenarios were still consistently above the benchmarks, adding to confidence that non-cancer risks are not expected.

REFERENCES

Anderson, WA; Castle, L; Hird, S; Jeffery, J; Scotter, MJ. (2011). A twenty-volunteer study using deuterium labelling to determine the kinetics and fractional excretion of primary and secondary urinary metabolites of di-2-ethylhexylphthalate and di-iso-nonylphthalate. Food Chem Toxicol 49: 2022-2029. http://dx.doi.org/10.1016/j.fct.2011.05.013

Armstrong, DL; Rice, CP; Ramirez, M; Torrents, A. (2018). Fate of four phthalate plasticizers under various wastewater treatment processes. J Environ Sci Health A Tox Hazard Subst Environ Eng 53: 1075-1082. http://dx.doi.org/10.1080/10934529.2018.1474580

Aso, S; Ehara, H; Miyata, K; Hosyuyama, S; Shiraishi, K; Umano, T; Minobe, Y. (2005). A two-generation reproductive toxicity study of butyl benzyl phthalate in rats. J Toxicol Sci 30: S39-S58. http://dx.doi.org/10.2131/jts.30.S39

ATSDR. (2022). Toxicological profile for di(2-ethylhexyl)phthalate (DEHP) [ATSDR Tox Profile]. (CS274127-A). Atlanta, GA. https://www.atsdr.cdc.gov/ToxProfiles/tp9.pdf

Aylward, LL; Hays, SM; Zidek, A. (2016). Variation in urinary spot sample, 24 h samples, and longer-term average urinary concentrations of short-lived environmental chemicals: implications for exposure assessment and reverse dosimetry. J Expo Sci Environ Epidemiol 27: 582-590. http://dx.doi.org/10.1038/jes.2016.54

Bach, C; Rosin, C; Munoz, JF; Dauchy, X. (2020). National screening study investigating nine phthalates and one adipate in raw and treated tap water in France. Environ Sci Pollut Res Int 27: 36476-36486. http://dx.doi.org/10.1007/s11356-020-09680-6

Barrows, ME; Petrocelli, SR; Macek, KJ; Carroll, JJ. (1980). Bioconcentration and elimination of selected water pollutants by bluegill sunfish (Lepomis macrochirus). In R Haque (Ed.), Dynamics, Exposure and Hazard Assessment of Toxic Chemicals (pp. 379-392). Ann Arbor, MI: Ann Arbor Science.

Bauer, MJ; Herrmann, R. (1997). Estimation of the environmental contamination by phthalic acid esters leaching from household wastes. Sci Total Environ 208: 49-57. http://dx.doi.org/10.1016/S0048-9697(97)00272-6

Blair, JD; Ikonomou, MG; Kelly, BC; Surridge, B; Gobas, FA. (2009). Ultra-trace determination of phthalate ester metabolites in seawater, sediments, and biota from an urbanized marine inlet by LC/ESI-MS/MS. Environ Sci Technol 43: 6262-6268. http://dx.doi.org/10.1021/es9013135

Brodie, HL; Carr, LE; Condon, P. (2000). A comparison of static pile and turned windrow methods for poultry litter compost production. Compost Science and Utilization 8: 178–189. http://dx.doi.org/10.1080/1065657X.2000.10701990

CA Water Board. (2022). EDT library and water quality analyses data and download page. Available online at https://www.waterboards.ca.gov/drinking_water/certlic/drinkingwater/EDTlibrary.html

Camanzo, J; Rice, CP; Jude, DJ; Rossmann, R. (1987). Organic priority pollutants in nearshore fish from 14 Lake Michigan USA tributaries and embayments 1983. J Great Lakes Res 13: 296-309. http://dx.doi.org/10.1016/S0380-1330(87)71653-0

Cao, XL; Zhao, W; Dabeka, R. (2015). Di-(2-ethylhexyl) adipate and 20 phthalates in composite food samples from the 2013 Canadian total diet study. Food Addit Contam Part A Chem Anal Control Expo Risk Assess 32: 1893-1901. http://dx.doi.org/10.1080/19440049.2015.1079742

Carr, KH; Coyle, GT; Kimerle, RA. (1997). Bioconcentration of (14C)butyl benzyl phthalate in bluegill sunfish (Lepomis macrochirus). Environ Toxicol Chem 16: 2200-2203. http://dx.doi.org/10.1002/etc.5620161030

Cheng, Z; Li, HH; Yu, L; Yang, ZB; Xu, XX; Wang, HS; Wong, MH. (2018). Phthalate esters distribution in coastal mariculture of Hong Kong, China. Environ Sci Pollut Res Int 25: 17321-17329. http://dx.doi.org/10.1007/s11356-018-1735-6

CPSC. (2014). Chronic Hazard Advisory Panel on phthalates and phthalate alternatives (with appendices). Bethesda, MD: U.S. Consumer Product Safety Commission, Directorate for Health

Sciences. https://www.cpsc.gov/s3fs-public/CHAP-REPORT-With-Appendices.pdf

CPSC. (2015). Estimated phthalate exposure and risk to pregnant women and women of reproductive age as assessed using four NHANES biomonitoring data sets (2005/2006, 2007/2008, 2009/2010, 2011/2012). Rockville, Maryland: U.S. Consumer Product Safety Commission, Directorate for Hazard Identification and Reduction. https://web.archive.org/web/20190321120312/https://www.cpsc.gov/s3fs-public/NHANES-Biomonitoring-analysis-for-Commission.pdf

David, RM. (2000). Exposure to phthalate esters [Letter]. Environ Health Perspect 108: A440. http://dx.doi.org/10.1289/ehp.108-a440a

Duncan, M. (2000). Fish consumption survey of the Suquamish Indian Tribe of the Port Madison Indian Reservation, Puget Sound Region. Suquamish, WA: The Suquamish Tribe, Port Madison Indian Reservation. http://www.deq.state.or.us/wq/standards/docs/toxics/suquamish2000report.pdf

EC/HC. (2015). State of the science report: Phthalate substance grouping: Medium-chain phthalate esters: Chemical Abstracts Service Registry Numbers: 84-61-7; 84-64-0; 84-69-5; 523-31-9; 5334-09-8;16883-83-3; 27215-22-1; 27987-25-3; 68515-40-2; 71888-89-6. Gatineau, Quebec: Environment Canada, Health Canada. https://www.canada.ca/content/dam/eccc/migration/ese-ees/4d845198-761d-428b-a519-75481b25b3e5/sos_phthalates-20-medium-chain-_en.pdf

ECJRC. (2008). European Union Summary Risk Assessment Report: Benzyl butyl phthalate (BBP) (CAS No: 85-68-7, EINECS: 201-622-7). (EUR 22773 EN/2). Luxembourg: Office for Official Publications of the European Communities. https://publications.jrc.ec.europa.eu/repository/handle/JRC46623

Eigenberg, DA; Bozigian, HP; Carter, DE; Sipes, IG. (1986). Distribution, excretion, and metabolism of butylbenzyl phthalate in the rat. J Toxicol Environ Health 17: 445-456. http://dx.doi.org/10.1080/15287398609530839

Fauser, P; Vikelsoe, J; Sorensen, PB; Carlsen, L. (2003). Phthalates, nonylphenols and LAS in an alternately operated wastewater treatment plant--fate modelling based on measured concentrations in wastewater and sludge. Water Res 37: 1288-1295. http://dx.doi.org/10.1016/S0043-1354(02)00482-7

Fromme, H; Gruber, L; Seckin, E; Raab, U; Zimmermann, S; Kiranoglu, M; Schlummer, M; Schwegler, U; Smolic, S; Völkel, W. (2011). Phthalates and their metabolites in breast milk - Results from the Bavarian Monitoring of Breast Milk (BAMBI). Environ Int 37: 715-722. http://dx.doi.org/10.1016/j.envint.2011.02.008

Furr, JR; Lambright, CS; Wilson, VS; Foster, PM; Gray, LE, Jr. (2014). A short-term in vivo screen using fetal testosterone production, a key event in the phthalate adverse outcome pathway, to predict disruption of sexual differentiation. Toxicol Sci 140: 403-424. http://dx.doi.org/10.1093/toxsci/kfu081

Gani, KM; Kazmi, AA. (2016). Comparative assessment of phthalate removal and risk in biological wastewater treatment systems of developing countries and small communities. Sci Total Environ 569-570: 661-671. http://dx.doi.org/10.1016/j.scitotenv.2016.06.182

Gao, D; Li, Z; Wen, Z; Ren, N. (2014). Occurrence and fate of phthalate esters in full-scale domestic wastewater treatment plants and their impact on receiving waters along the Songhua River in China. Chemosphere 95: 24-32. http://dx.doi.org/10.1016/j.chemosphere.2013.08.009

Gobas, FAP; Mackintosh, CE; Webster, G; Ikonomou, M; Parkerton, TF; Robillard, K. (2003). Bioaccumulation of phthalate esters in aquatic food-webs. In CA Staples (Ed.), Phthalate esters (pp. 201-225). Berlin, Germany: Springer Verlag. http://dx.doi.org/10.1007/b11467

Government of Canada. (2000). Canadian Environmental Protection Act, 1999: Persistence and bioaccumulation regulations (pp. 607-612). (SOR/2000-107). Ottawa: Queen's Printer, Canada Gazette. https://canadagazette.gc.ca/rp-pr/p2/2000/2000-03-29/pdf/g2-13407.pdf

Harper, B; Harding, A; Harris, S; Berger, P. (2012). Subsistence Exposure Scenarios for Tribal

Applications. Hum Ecol Risk Assess 18: 810-831. http://dx.doi.org/10.1080/10807039.2012.688706

Hartle, JC; Cohen, RS; Sakamoto, P; Barr, DB; Carmichael, SL. (2018). Chemical contaminants in raw and pasteurized human milk. J Hum Lact 34: 340-349. http://dx.doi.org/10.1177/0890334418759308

He, MJ; Lu, JF; Wang, J; Wei, SQ; Hageman, KJ. (2020). Phthalate esters in biota, air and water in an agricultural area of western China, with emphasis on bioaccumulation and human exposure. Sci Total Environ 698: 134264. http://dx.doi.org/10.1016/j.scitotenv.2019.134264

Health Canada. (2020). Screening assessment - Phthalate substance grouping. (En14-393/2019E-PDF). Environment and Climate Change Canada. https://www.canada.ca/en/environment-climate-change/services/evaluating-existing-substances/screening-assessment-phthalate-substance-grouping.html

Hogberg, J; Hanberg, A; Berglund, M; Skerfving, S; Remberger, M; Calafat, AM; Filipsson, AF; Jansson, B; Johansson, N; Appelgren, M; Hakansson, H. (2008). Phthalate diesters and their metabolites in human breast milk, blood or serum, and urine as biomarkers of exposure in vulnerable populations. Environ Health Perspect 116: 334-339. http://dx.doi.org/10.1289/ehp.10788

Howard, PH; Banerjee, S; Robillard, KH. (1985). Measurement of water solubilities octanol-water partition coefficients and vapor pressures of commercial phthalate esters. Environ Toxicol Chem 4: 653-662. http://dx.doi.org/10.1002/etc.5620040509

Hu, H; Mao, L; Fang, S; Xie, J; Zhao, M; Jin, H. (2020). Occurrence of phthalic acid esters in marine organisms from Hangzhou Bay, China: Implications for human exposure. Sci Total Environ 721: 137605. http://dx.doi.org/10.1016/j.scitotenv.2020.137605

Huang, J; Nkrumah, PN; Li, Y; Appiah-Sefah, G. (2013). Chemical behavior of phthalates under abiotic conditions in landfills [Review]. Rev Environ Contam Toxicol 224: 39-52. http://dx.doi.org/10.1007/978-1-4614-5882-1_2

Huang, PC; Tien, CJ; Sun, YM; Hsieh, CY; Lee, CC. (2008). Occurrence of phthalates in sediment and biota: Relationship to aquatic factors and the biota-sediment accumulation factor. Chemosphere 73: 539-544. http://dx.doi.org/10.1016/j.chemosphere.2008.06.019

IARC. (2013). Some chemicals present in industrial and consumer products, food and drinking-water [Review]. In IARC Monographs on the Evaluation of Carcinogenic Risks to Humans (pp. 9-549). Lyon, France: World Health Organization. http://monographs.iarc.fr/ENG/Monographs/vol101/mono101.pdf

Ikonomou, MG; Kelly, BC; Blair, JD; Gobas, FA. (2012). An interlaboratory comparison study for the determination of dialkyl phthalate esters in environmental and biological samples. Environ Toxicol Chem 31: 1948-1956. http://dx.doi.org/10.1002/etc.1912

Kapraun, D, ustin F.; Zurlinden, T, odd J.; Verner, M, arc-André; Chiang, C, atheryne; Dzierlenga, M, ichael W.; Carlson, L, aura M.; Schlosser, P, aul M.; Lehmann, G, eniece M. (2022). A generic pharmacokinetic model for quantifying mother-to-offspring transfer of lipophilic persistent environmental chemicals. Toxicol Sci 2022: kfac084. http://dx.doi.org/10.1093/toxsci/kfac084

Kickham, P; Otton, SV; Moore, MM; Ikonomou, MG; Gobas, FAP, C. (2012). Relationship between biodegradation and sorption of phthalate esters and their metabolites in natural sediments. Environ Toxicol Chem 31: 1730-1737. http://dx.doi.org/10.1002/etc.1903

Kim, MG; Kim, TH; Shin, BS; Lee, YB; Lee, JB; Choi, HG; Lee, Y; Yoo, SD. (2015). Sensitive liquid chromatography-tandem mass spectrometry method for the simultaneous determination of benzyl butyl phthalate and its metabolites, monobenzyl phthalate and monobutyl phthalate, in rat plasma, urine, and various tissues collected from a toxicokinetic study. Anal Bioanal Chem 407: 7391-7400. http://dx.doi.org/10.1007/s00216-015-8901-1

Kim, S; Eom, S; Kim, HJ; Lee, JJ; Choi, G; Choi, S; Kim, S; Kim, SY; Cho, G; Kim, YD; Suh, E; Kim,

SK; Kim, S; Kim, GH; Moon, HB; Park, J; Kim, S; Choi, K; Eun, SH. (2018). Association between maternal exposure to major phthalates, heavy metals, and persistent organic pollutants, and the neurodevelopmental performances of their children at 1 to 2 years of age–CHECK cohort study. Sci Total Environ 624: 377-384. http://dx.doi.org/10.1016/j.scitotenv.2017.12.058

Koch, HM; Becker, K; Wittassek, M; Seiwert, M; Angerer, J; Kolossa-Gehring, M. (2007). Di-n-butylphthalate and butylbenzylphthalate - urinary metabolite levels and estimated daily intakes: Pilot study for the German Environmental Survey on children. J Expo Sci Environ Epidemiol 17: 378-387. http://dx.doi.org/10.1038/sj.jes.7500526

Koch, HM; Calafat, AM. (2009). Human body burdens of chemicals used in plastic manufacture [Review]. Philos Trans R Soc Lond B Biol Sci 364: 2063-2078. http://dx.doi.org/10.1098/rstb.2008.0208

Koch, HM; Drexler, H; Angerer, J. (2003). An estimation of the daily intake of di(2-ethylhexyl)phthalate (DEHP) and other phthalates in the general population. Int J Hyg Environ Health 206: 77-83. http://dx.doi.org/10.1078/1438-4639-00205

Latini, G; Wittassek, M; Del Vecchio, A; Presta, G; De Felice, C; Angerer, J. (2009). Lactational exposure to phthalates in Southern Italy. Environ Int 35: 236-239. http://dx.doi.org/10.1016/j.envint.2008.06.002

Lee, YM; Lee, JE; Choe, W; Kim, T; Lee, JY; Kho, Y; Choi, K; Zoh, KD. (2019). Distribution of phthalate esters in air, water, sediments, and fish in the Asan Lake of Korea. Environ Int 126: 635-643. http://dx.doi.org/10.1016/j.envint.2019.02.059

Lertsirisopon, R; Soda, S; Sei, K; Ike, M; Fujita, M. (2006). Biodegradability of four phthalic acid esters under anaerobic condition assessed using natural sediment. J Environ Sci 18: 793-796.

Li, C; Chen, J; Wang, J; Han, P; Luan, Y; Ma, X; Lu, A. (2016). Phthalate esters in soil, plastic film, and vegetable from greenhouse vegetable production bases in Beijing, China: Concentrations, sources, and risk assessment [Supplemental Data]. Sci Total Environ 568: 1037-1043. http://dx.doi.org/10.1016/j.scitotenv.2016.06.077

Li, Y, an; Huang, G; Gu, H, ua; Huang, Q; Lou, C; Zhang, L, ei; Liu, H. (2018). Assessing the Risk of Phthalate Ester (PAE) Contamination in Soils and Crops Irrigated with Treated Sewage Effluent. Water 10: 999. http://dx.doi.org/10.3390/w10080999

Lin, S; Ku, H; Su, P; Chen, J; Huang, P; Angerer, J; Wang, S. (2011). Phthalate exposure in pregnant women and their children in central Taiwan. Chemosphere 82: 947-955. http://dx.doi.org/10.1016/j.chemosphere.2010.10.073

Lin, ZP; Ikonomou, MG; Jing, H; Mackintosh, C; Gobas, FA. (2003). Determination of phthalate ester congeners and mixtures by LC/ESI-MS in sediments and biota of an urbanized marine inlet. Environ Sci Technol 37: 2100-2108. http://dx.doi.org/10.1021/es026361r

Liu, P; Tian, T; Barreto, J; Chou, J. (2013). Assessment and analysis of phthalate esters, in Lake Pontchartrain, by SPME combining with GC-MS. Environ Technol 34: 453-462. http://dx.doi.org/10.1080/09593330.2012.698653

Mackintosh, CE; Maldonado, J; Hongwu, J; Hoover, N; Chong, A; Ikonomou, MG; Gobas, FA. (2004). Distribution of phthalate esters in a marine aquatic food web: Comparison to polychlorinated biphenyls. Environ Sci Technol 38: 2011-2020. http://dx.doi.org/10.1021/es034745r

Mage, DT; Allen, RH; Kodali, A. (2008). Creatinine corrections for estimating children's and adult's pesticide intake doses in equilibrium with urinary pesticide and creatinine concentrations. J Expo Sci Environ Epidemiol 18: 360-368. http://dx.doi.org/10.1038/sj.jes.7500614

Main, KM; Mortensen, GK; Kaleva, MM; Boisen, KA; Damgaard, IN; Chellakooty, M; Schmidt, IM; Suomi, AM; Virtanen, HE; Petersen, JH; Andersson, AM; Toppari, J; Skakkebaek, NE. (2006). Human breast milk contamination with phthalates and alterations of endogenous reproductive hormones in infants three months of age. Environ Health Perspect 114: 270-276. http://dx.doi.org/10.1289/ehp.8075

McConnell, ML. (2007) Distribution of phthalate monoesters in an aquatic food web. (Master's Thesis). Simon Fraser University, Burnaby, Canada. Retrieved from http://summit.sfu.ca/item/2603

Meng, XZ; Wang, Y; Xiang, N; Chen, L; Liu, Z; Wu, B; Dai, X; Zhang, YH; Xie, Z; Ebinghaus, R. (2014). Flow of sewage sludge-borne phthalate esters (PAEs) from human release to human intake: implication for risk assessment of sludge applied to soil. Sci Total Environ 476-477: 242-249. http://dx.doi.org/10.1016/j.scitotenv.2014.01.007

Müller, J; Kördel, W. (1993). Occurrence and fate of phthalates in soil and plants. Sci Total Environ 134: 431-437. http://dx.doi.org/10.1016/S0048-9697(05)80044-0

NCHS. (2021). National Health and Nutrition Examination Survey - 2017-2018 Data Documentation, Codebook, and Frequencies: Phthalates and Plasticizers Metabolites - Urine (PHTHTE_J). Available online at https://wwwn.cdc.gov/Nchs/Nhanes/2017-2018/PHTHTE_J.htm (accessed February 9, 2024).

Net, S; Sempéré, R; Delmont, A; Paluselli, A; Ouddane, B. (2015). Occurrence, fate, behavior and ecotoxicological state of phthalates in different environmental matrices [Review]. Environ Sci Technol 49: 4019-4035. http://dx.doi.org/10.1021/es505233b

Northcote, TG. (1973). Some impacts of man on Kootenay Lake and its salmonoids. (Technical Report No. 25). Great Lakes Fishery Commission. http://glfc.org/pubs/TechReports/Tr25.pdf

NWQMC. (2021). Water quality portal [Database]. Washington, DC. Retrieved from https://www.waterqualitydata.us/

Oppenheimer, J; Stephenson, R; Burbano, A; Liu, L. (2007). Characterizing the passage of personal care products through wastewater treatment processes. Water Environ Res 79: 2564-2577. http://dx.doi.org/10.2175/106143007X184573

Panio, A; Fabbri Corsarini, S; Bruno, A; Lasagni, M; Labra, M; Saliu, F. (2020). Determination of phthalates in fish fillets by liquid chromatography tandem mass spectrometry (LC-MS/MS): A comparison of direct immersion solid phase microextraction (SPME) versus ultrasonic assisted solvent extraction (UASE). Chemosphere 255: 127034. http://dx.doi.org/10.1016/j.chemosphere.2020.127034

Peters, RJB; Beeltje, H; van Delft, RJ. (2008). Xeno-estrogenic compounds in precipitation. J Environ Monit 10: 760-769. http://dx.doi.org/10.1039/b805983g

Polissar, NL; Salisbury, A; Ridolfi, C; Callahan, K; Neradilek, M; Hippe, D; Beckley, WH. (2016). A fish consumption survey of the Shoshone-Bannock Tribes: Vols. I-III. Polissar, NL; Salisbury, A; Ridolfi, C; Callahan, K; Neradilek, M; Hippe, D; Beckley, WH. https://www.epa.gov/sites/production/files/2017-01/documents/fish-consumption-survey-shoshone-bannock-dec2016.pdf

RIDOLFI. (2016). Heritage fish consumption rates of the Kootenai Tribe of Idaho. Washington, DC: U.S. Environmental Protection Agency. https://www.epa.gov/sites/default/files/2017-01/documents/heritage-fish-consumption-rates-kootenai-dec2016.pdf

Salaudeen, T; Okoh, O; Agunbiade, F; Okoh, A. (2018a). Fate and impact of phthalates in activated sludge treated municipal wastewater on the water bodies in the Eastern Cape, South Africa. Chemosphere 203: 336-344. http://dx.doi.org/10.1016/j.chemosphere.2018.03.176

Salaudeen, T; Okoh, O; Agunbiade, F; Okoh, A. (2018b). Phthalates removal efficiency in different wastewater treatment technology in the Eastern Cape, South Africa. Environ Monit Assess 190: 299. http://dx.doi.org/10.1007/s10661-018-6665-8

Sánchez-Avila, J; Vicente, J; Echavarri-Erasun, B; Porte, C; Tauler, R; Lacorte, S. (2013). Sources, fluxes and risk of organic micropollutants to the Cantabrian Sea (Spain). Mar Pollut Bull 72: 119-132. http://dx.doi.org/10.1016/j.marpolbul.2013.04.010

Schlumpf, M; Kypke, K; Wittassek, M; Angerer, J; Mascher, H; Mascher, D; Vökt, C; Birchler, M; Lichtensteiger, W. (2010). Exposure patterns of UV filters, fragrances, parabens, phthalates, organochlor pesticides, PBDEs, and PCBs in human milk: correlation of UV filters with use of

cosmetics. Chemosphere 81: 1171-1183. http://dx.doi.org/10.1016/j.chemosphere.2010.09.079

Schmidt, N; Castro-Jimenez, J; Fauvelle, V; Ourgaud, M; Sempere, R. (2020). Occurrence of organic plastic additives in surface waters of the Rhone River (France). Environ Pollut 257: 113637. http://dx.doi.org/10.1016/j.envpol.2019.113637

Shin, HM; Bennett, DH; Barkoski, J; Ye, X; Calafat, AM; Tancredi, D; Hertz-Picciotto, I. (2019). Variability of urinary concentrations of phthalate metabolites during pregnancy in first morning voids and pooled samples. Environ Int 122: 222-230. http://dx.doi.org/10.1016/j.envint.2018.11.012

Stephenson, R. (2007). Fate of pharmaceuticals and personal care products through wastewater treatment processes. London, United Kingdom: IWA Publishing. http://dx.doi.org/10.2166/9781780403830

Sulentic, RO; Dumitrascu, I; Deziel, NC; Gurzau, AE. (2018). Phthalate Exposure from Drinking Water in Romanian Adolescents. Int J Environ Res Public Health 15: 2109. http://dx.doi.org/10.3390/ijerph15102109

Teil, MJ; Tlili, K; Blanchard, M; Chevreuil, M; Alliot, F; Labadie, P. (2012). Occurrence of Polybrominated Diphenyl Ethers, Polychlorinated Biphenyls, and Phthalates in Freshwater Fish From the Orge River (Ile-de France). Arch Environ Contam Toxicol 63: 101-113. http://dx.doi.org/10.1007/s00244-011-9746-z

Teil, MJ; Tlili, K; Blanchard, M; Labadie, P; Alliot, F; Chevreuil, M. (2014). Polychlorinated biphenyls, polybrominated diphenyl ethers, and phthalates in roach from the Seine River Basin (France): Impact of densely urbanized areas. Arch Environ Contam Toxicol 66: 41-57. http://dx.doi.org/10.1007/s00244-013-9955-8

Tran, BC; Teil, MJ; Blanchard, M; Alliot, F; Chevreuil, M. (2014). BPA and phthalate fate in a sewage network and an elementary river of France. Influence of hydroclimatic conditions. Chemosphere 119C: 43-51. http://dx.doi.org/10.1016/j.chemosphere.2014.04.036

Tyl, RW; Myers, CB; Marr, MC; Fail, PA; Seely, JC; Brine, DR; Barter, RA; Butala, JH. (2004). Reproductive toxicity evaluation of dietary butyl benzyl phthalate (BBP) in rats. Reprod Toxicol 18: 241-264. http://dx.doi.org/10.1016/j.reprotox.2003.10.006

U.S. EPA. (1982). Fate of priority pollutants in publicly owned treatment works, Volume i. (EPA 440/1-82/303). Washington, DC: Effluent Guidelines Division. http://nepis.epa.gov/exe/ZyPURL.cgi?Dockey=000012HL.txt

U.S. EPA. (1989). Risk assessment guidance for superfund, volume I: Human health evaluation manual (Part A). Interim final. (EPA/540/1-89/002). Washington, DC. https://www.epa.gov/sites/production/files/2015-09/documents/rags_a.pdf

U.S. EPA. (1992). Dermal exposure assessment: Principles and applications (interim report) [EPA Report]. (EPA/600/8-91/011B). Washington, DC: Office of Health and Environmental Assessment. http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=12188

U.S. EPA. (1993). Standards for the use or disposal of sewage sludge: Final rules [EPA Report]. (EPA 822/Z-93-001). Washington, DC.

U.S. EPA. (2000a). Biosolids technology fact sheet: Land application of biosolids. (EPA 832-F-00-064).

U.S. EPA. (2000b). Methodology for deriving ambient water quality criteria for the protection of human health (2000). (EPA/822/B-00/004). Washington, DC: U.S. Environmental Protection Agency, Office of Water. https://www.nj.gov/drbc/library/documents/EPA_human-health-criteria2000.pdf

U.S. EPA. (2004). Risk Assessment Guidance for Superfund (RAGS), volume I: Human health evaluation manual, (part E: Supplemental guidance for dermal risk assessment). (EPA/540/R/99/005). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www.epa.gov/risk/risk-assessment-guidance-superfund-rags-part-e

U.S. EPA. (2007). Exposure and Fate Assessment Screening Tool (E-FAST), Version 2.0 [Computer

Program]. Washington, DC.
U.S. EPA. (2009). Targeted national sewage sludge survey sampling and analysis technical report [EPA Report]. (EPA-822-R-08-016). Washington, DC: U.S. Environmental Protection Agency, Office of Water. http://nepis.epa.gov/exe/ZyPURL.cgi?Dockey=P1003RL8.txt
U.S. EPA. (2011a). Exposure factors handbook: 2011 edition [EPA Report]. (EPA/600/R-090/052F). Washington, DC: U.S. Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment. https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100F2OS.txt
U.S. EPA. (2011b). Recommended use of body weight 3/4 as the default method in derivation of the oral reference dose. (EPA100R110001). Washington, DC. https://www.epa.gov/sites/production/files/2013-09/documents/recommended-use-of-bw34.pdf
U.S. EPA. (2014). Estimated fish consumption rates for the U.S. population and selected subpopulations (NHANES 2003-2010) [EPA Report]. (EPA-820-R-14-002). Washington, DC. https://www.epa.gov/sites/production/files/2015-01/documents/fish-consumption-rates-2014.pdf
U.S. EPA. (2015a). Evaluation of Swimmer Exposures Using the SWIMODEL Algorithms and Assumptions. Available online at https://www.epa.gov/sites/production/files/2016-11/documents/swimodel_final.pdf
U.S. EPA. (2015b). Guidance for using the volatilization algorithm in the pesticide in water calculator and water exposure models. Washington, DC: Environmental Fate and Effects Division. https://www.epa.gov/pesticide-science-and-assessing-pesticide-risks/guidance-using-volatilization-algorithm-pesticide
U.S. EPA. (2016). Guidance for conducting fish consumption surveys. (823B16002). https://www.epa.gov/sites/production/files/2017-01/documents/fc_survey_guidance.pdf
U.S. EPA. (2017a). Estimation Programs Interface Suite™ v.4.11. Washington, DC: U.S. Environmental Protection Agency, Office of Pollution Prevention Toxics. Retrieved from https://www.epa.gov/tsca-screening-tools/download-epi-suitetm-estimation-program-interface-v411
U.S. EPA. (2017b). Update for Chapter 5 of the Exposure Factors Handbook: Soil and dust ingestion [EPA Report]. (EPA/600R-17/384F). Washington, DC: National Center for Environmental Assessment, Office of Research and Development. https://nepis.epa.gov/Exe/ZyPURL.cgi?Dockey=P100TTX4.txt
U.S. EPA. (2019a). Exposure factors handbook chapter 3 (update): Ingestion of water and other select liquids [EPA Report]. (EPA/600/R-18/259F). Washington, DC. https://cfpub.epa.gov/ncea/efp/recordisplay.cfm?deid=343661
U.S. EPA. (2019b). Guidelines for human exposure assessment [EPA Report]. (EPA/100/B-19/001). Washington, DC: Risk Assessment Forum. https://www.epa.gov/sites/production/files/2020-01/documents/guidelines_for_human_exposure_assessment_final2019.pdf
U.S. EPA. (2019c). Point Source Calculator: A Model for Estimating Chemical Concentration in Water Bodies. Washington, DC: U.S. Environmental Protection Agency, Office of Chemical Safety and Pollution Prevention.
U.S. EPA. (2019d). User's Guide: Integrated Indoor-Outdoor Air Calculator (IIOAC). Washington, DC: U.S. EPA.
U.S. EPA. (2021a). About the Exposure Factors Handbook. Available online at https://www.epa.gov/expobox/about-exposure-factors-handbook
U.S. EPA. (2021b). Draft systematic review protocol supporting TSCA risk evaluations for chemical substances, Version 1.0: A generic TSCA systematic review protocol with chemical-specific methodologies. (EPA Document #EPA-D-20-031). Washington, DC: Office of Chemical Safety and Pollution Prevention. https://www.regulations.gov/document/EPA-HQ-OPPT-2021-0414-0005

U.S. EPA. (2022a). Ambient Monitoring Technology Information Center (AMTIC) - Ambient Monitoring Archive for HAPs [Database]. Washington, DC. Retrieved from https://www.epa.gov/amtic/amtic-ambient-monitoring-archive-haps

U.S. EPA. (2022b). Draft TSCA screening level approach for assessing ambient air and water exposures to fenceline communities (version 1.0) [EPA Report]. (EPA-744-D-22-001). Washington, DC: Office of Chemical Safety and Pollution Prevention, U.S. Environmental Protection Agency. https://www.epa.gov/system/files/documents/2022-01/draft-fenceline-report_sacc.pdf

U.S. EPA. (2022c). Enforcement and Compliance History Online (ECHO) [Database]. Washington, DC. Retrieved from https://echo.epa.gov/

U.S. EPA. (2024a). Chemical Transformation Simulator (CTS) data: DEHP, DCHP, DIBP, DBP, and BBP transformation products. Washington, DC. Retrieved from https://qed.epa.gov/cts/gentrans/

U.S. EPA. (2024b). Draft environmental release and occupational exposure assessment for Dicyclohexyl phthalate (DCHP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2024c). Draft Systematic Review Protocol for Dicyclohexyl Phthalate (DCHP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2024d). Fate Assessment for Diisononyl Phthalate (DINP). Washington, DC: Office of Pollution Prevention and Toxics. https://www.regulations.gov/docket/EPA-HQ-OPPT-2018-0436

U.S. EPA. (2025a). Draft Ambient Air Exposure Assessment for Benzyl Butyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025b). Draft Ambient Air IIOAC Exposure Results and Risk Calculations for Dibutyl Phthalate (DBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025c). Draft Data Extraction Information for General Population, Consumer, and Environmental Exposure for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025d). Draft Data Quality Evaluation and Data Extraction Information for Environmental Release and Occupational Exposure for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025e). Draft Environmental Hazard Assessment for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025f). Draft Environmental Release and Occupational Exposure Assessment for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025g). Draft Fish Ingestion Risk Calculator for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025h). Draft Non-Cancer Human Health Hazard Assessment for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025i). Draft Physical Chemistry and Fate and Transport Assessment for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025j). Draft Risk Evaluation for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA. (2025k). Draft Surface Water Human Exposure Risk Calculator for Butyl Benzyl Phthalate (BBP). Washington, DC: Office of Pollution Prevention and Toxics.

U.S. EPA; ICF Consulting. (2022). Consumer Exposure Model (CEM) user guide, Version 3.0. (EPA Contract #EP-W-12-010). Washington, DC: U.S. Environmental Protection Agency, Office of Pollution Prevention and Toxics.

USDA-ARS. (1963). Composition of foods: Raw, processed, prepared: U.S. Department of Agriculture, Agriculture Handbook No. 8. Washington, DC.

USDA NRCS. (2008). Soil quality indicators: Bulk density. USDA Natural Resources Conservation Service. https://www.nrcs.usda.gov/sites/default/files/2022-10/nrcs142p2_051591.pdf

Valton, AS; Serre-Dargnat, C; Blanchard, M; Alliot, F; Chevreuil, M; Teil, MJ. (2014). Determination of phthalates and their by-products in tissues of roach (Rutilus rutilus) from the Orge river (France). Environ Sci Pollut Res Int 21: 12723-12730. http://dx.doi.org/10.1007/s11356-014-3213-0

Wu, J; Ma, T; Zhou, Z; Yu, N, a; He, Z; Li, B; Shi, Y; Ma, D. (2019). Occurrence and fate of phthalate esters in wastewater treatment plants in Qingdao, China. Hum Ecol Risk Assess 25: 1547-1563. http://dx.doi.org/10.1080/10807039.2018.1471341

Wu, Q; Lam, JCW; Kwok, KY; Tsui, MMP; Lam, PKS. (2017). Occurrence and fate of endogenous steroid hormones, alkylphenol ethoxylates, bisphenol A and phthalates in municipal sewage treatment systems. J Environ Sci 61: 49-58. http://dx.doi.org/10.1016/j.jes.2017.02.021

Yuan, SY; Liu, C; Liao, CS; Chang, BV. (2002). Occurrence and microbial degradation of phthalate esters in Taiwan river sediments. Chemosphere 49: 1295-1299. http://dx.doi.org/10.1016/s0045-6535(02)00495-2

Zeng, F; Lin, Y; Cui, K; Wen, J; Ma, Y; Chen, H; Zhu, F; Ma, Z; Zeng, Z. (2010). Atmospheric deposition of phthalate esters in a subtropical city. Atmos Environ 44: 834-840. http://dx.doi.org/10.1016/j.atmosenv.2009.11.029

Zhu, Q; Jia, J; Zhang, K; Zhang, H; Liao, C. (2019). Spatial distribution and mass loading of phthalate esters in wastewater treatment plants in China: An assessment of human exposure. Sci Total Environ 656: 862-869. http://dx.doi.org/10.1016/j.scitotenv.2018.11.458

Zhu, ZY; Ji, YQ; Zhang, SJ; Zhao, JB; Zhao, J. (2016). Phthalate Ester Concentrations, Sources, and Risks in the Ambient Air of Tianjin, China. Aerosol Air Qual Res 16: 2294-2301. http://dx.doi.org/10.4209/aaqr.2015.07.0473

Zimmermann, S; Gruber, L; Schlummer, M; Smolic, S; Fromme, H. (2012). Determination of phthalic acid diesters in human milk at low ppb levels. Food Addit Contam Part A Chem Anal Control Expo Risk Assess 29: 1780. http://dx.doi.org/10.1080/19440049.2012.704529

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)

2617 **Table_Apx A-3. Recommended Default Values for Common Exposure Factors**

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
ED	Exposure duration (hrs/day)	8	24	
EF	Exposure frequency (days/year)	250	365	
EY	Exposure years (years)	40	Varies for Adult (chronic non-cancer) 78 (Lifetime) 1 Infant (birth to <1 year) 5 Toddler (1–5 years) 5 Child (6–10 years) 5 Youth (11–15 years) 5 Youth (16–20 years)	Number of years in age group. Note: These age bins may vary for different measurements and sources
AT	Averaging time non-cancer	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	See pg. 6–23 of Risk assessment guidance for superfund, volume I: Human health evaluation manual (Part A). (U.S. EPA, 1989)
	Averaging time cancer	78 years (28,470 days)	78 years (28,470 days)	See Table 18-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)
BW	Body weight (kg)	80	80 Adult 7.83 Infant (birth to <1 year) 16.2 Toddler (1–5 years) 31.8 Child (6–10 years) 56.8 Youth (11–15 years) 71.6 Youth (16–20 years) 65.9 Adolescent woman of childbearing age (16 to <21) – apply to all developmental exposure scenarios	See Table 8-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a) (Refer to Figure 31 for agE−specific BW) Note: These age bins may vary for different measurements and sources See Table 8-5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
$IR_{dw\text{-}acute}$	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)
IR_{fish}	Fish ingestion rate (g/day)		22 Adult	Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (U.S. EPA, 2014) This represents the 90th percentile consumption rate of fish and shellfish from inland and nearshore waters for the U.S. adult population 21 years of age and older, based on NHANES data from 2003–2010

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
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 Kow 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)

2618
2619

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

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

A.1 Surface Water Exposure Activity Parameters

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

Input	Description (Units)	Adult (21+ Years)	Youth (11–15 Years)	Child (6–10 Years)	Notes	Reference
BW	Body weight (kg)	80	56.8	31.8	Mean body weight. Chapter 8 of the *Exposure Factors Handbook*, Table 8-1	U.S. EPA (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)

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)

Input	Description (Units)	Adult (21+ Years)	Youth (11–15 Years)	Child (6–10 Years)	Notes	Reference
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				

2626

Appendix B ESTIMATING HYDROLOGICAL FLOW DATA FOR SURFACE WATER MODELING

A distribution of flow metrics was generated by collecting flow data for facilities across 3 North American Industry Classification System (NAICS) codes associated with BBP-releasing facilities (Table_Apx B-1). EPA's Enforcement and Compliance History Online (ECHO) database was accessed via the Application Programming Interface (API) and queried for facilities regulated under the Clean Water Act within each of the 20 relevant NAICS codes (U.S. EPA, 2022c). 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 the National Hydrography Dataset Plus (NHDPlus) reach code associated with the receiving waterbody for each available facility. Modeled flow metrics were then extracted for the retrieved reach codes, from the NHDPlus V2.1 Flowline Network's Enhanced Runoff Method (EROM) Flow database. The EROM database provides modeled monthly average flows for each month of the year. While the EROM flow database represents averages across a 30-year time period, the lowest of the monthly average flows was selected as a substitute for the 30Q5 flow used in modeling, as both approximate the lowest observed monthly flow at a given location. The substitute 30Q5 flow was then plugged into the regression equation used by the EPA's Exposure and Fate Assessment Tool, Version 2014 (E−FAST) (U.S. EPA, 2007) to convert between these flow metrics and solved for the 7Q10 using Equation B-1. E−FAST is a dilution-based model that estimates chemical concentrations in surface water concentrations for use in general population and aquatic exposure assessments. 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 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 B-2, derived from the relevant E−FAST regression.

Equation B-2. Calculating the Harmonic Mean Flow

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

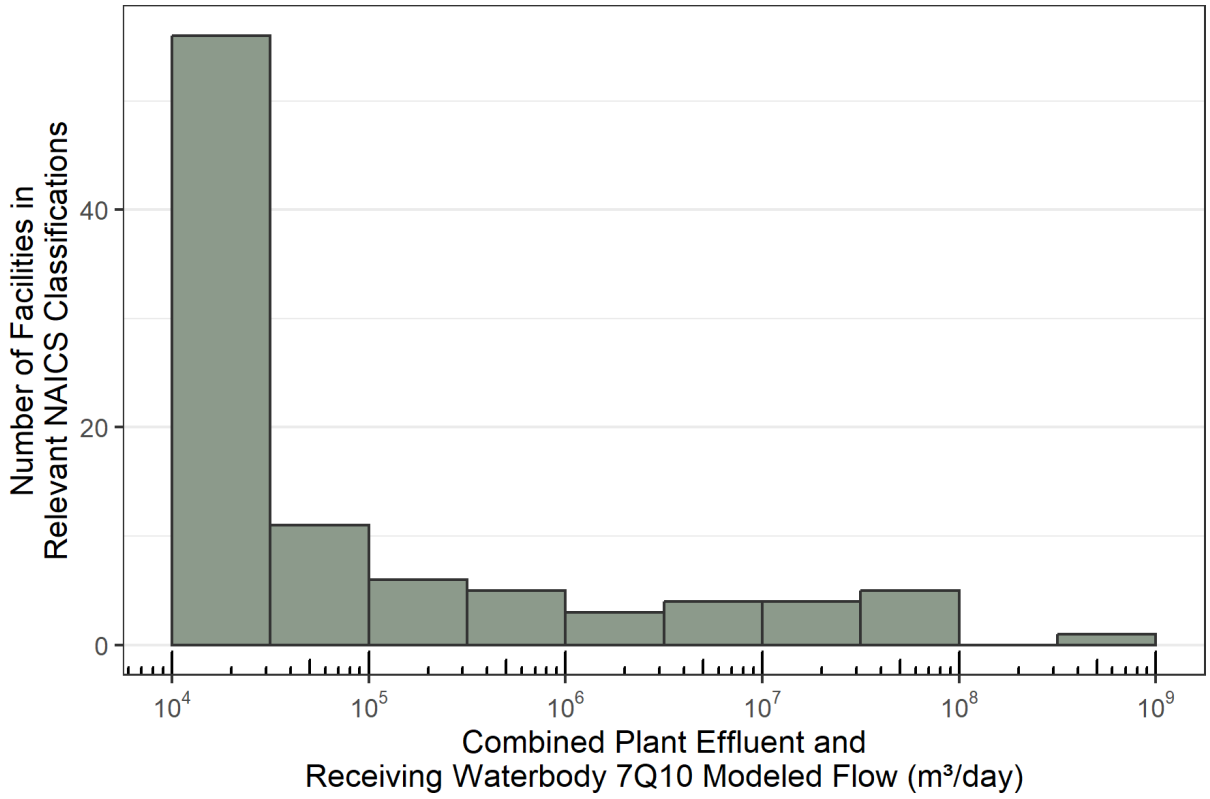
Where:

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 BBP Releases

NAICS Code	NAICS Name
325520	Adhesive Manufacturing
325199	All Other Basic Organic Chemical Manufacturing
325998	All Other Miscellaneous Chemical Product and Preparation

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. For the COU with surface water releases, the highest estimated release to surface water was modeled. The total days of release associated with the highest COU release 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 (for example, the first average will be for values 1- total days of release and the second average will be for values 2- total days of release +1).

Appendix C GENERAL POPULATION SURFACE WATER RISK SCREENING RESULTS

C.1 Incidental Dermal Exposure (Swimming)

Based on the estimated dermal doses in Table 5-1, EPA screened for risk to adults, youth, and children. Table_Apx C-1 summarizes the acute MOEs based on the dermal doses. Using the total acute dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 (U.S. EPA, 2025h). Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects from 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
PVC plastics compounding *Without Wastewater Treatment* (P50 flow rate with high-end release)	2,852	2,049	417	545	898
Manufacturing *Without Wastewater Treatment* (P50 flow rate with high-end release)	24,500	17,600	49	63	105
Highest monitored surface water NWQMC (2021)	40	40	30,000	39,000	64,000

C.2 Incidental Ingestion Exposure (Swimming)

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 (U.S. EPA, 2025h). Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects from 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
PVC plastics compounding *Without wastewater treatment*	2,852	2,049	1,220	786	1,394
Manufacturing *Without Wastewater Treatment*	24,500	17,600	142	92	162
Highest monitored surface water NWQMC (2021)	40	40	870,000	560,000	990,000

Appendix D GENERAL POPULATION DRINKING WATER RISK SCREENING RESULTS

Based on the estimated drinking water doses in Table 6-1, EPA screened for risk to adults, infants, and toddlers. Table_Apx D-1 summarizes the acute and chronic MOEs based on the drinking water doses. For the highest modeled surface water concentrations in the Manufacturing and PVC plastic compounding OESs, applying the high-end release data, paired with the P50 flow, some of the most sensitive acute and chronic MOEs are less than the benchmark of 30 (U.S. EPA, 2025h). While these conservative scenarios were applied as a screening assessment for risk, further refinement of the scenarios, which included modeling the high-end releases against the P75 and P90 flow rates, with and without wastewater treatment, resulted in MOEs orders of magnitude above the benchmark. Based on the conservative modeling parameters for drinking water concentration and exposure factors parameters, and MOEs below the benchmark only occurring for the highest end of exposure scenarios including an unlikely confluence of factors, risk for non-cancer health effects from drinking water ingestion is not expected. The full distribution of MOEs estimated are presented in the *Draft Surface Water Human Exposure Risk Calculator for Diisobutyl Phthalate (DIBP)* (U.S. EPA, 2025k).

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 practice, however, 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 BBP 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
Manufacturing *Without wastewater treatment*	24,500	17,600	12	75	3	29.5	10	68.9
Manufacturing *With wastewater treatment*	9,310	6,690	32	199	9	78	25.7	181
PVC plastics compounding *Without wastewater treatment*	2,852	2,049	105	765	30	300	84	699
PVC plastics compounding *With*	1,322	950	105	765	78	789	221	1,840

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
wastewater treatment								
Highest monitored surface water NWQMC (2021)	40	40	7,455	33,000	2,125	13,000	5,975	30,000

Appendix E FISH INGESTION RISK SCREENING RESULTS

E.1 General Population

Using conservative exposure estimates based on the water solubility limit as the surface water concentration, acute and chronic non-cancer risk estimates for the general population were above the benchmark of 30 (Table_Apx E-1). These results indicate that fish ingestion is not a pathway of concern for BBP for the general population.

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

Surface Water Concentration and Scenario	Acute Non-cancer MOE UFs = 30		Adult Chronic Non-cancer MOE UFs = 30
	Adult	Young Toddler	
Water solubility limit (2.69 mg/L)	401	270	1,766
Monitored surface water concentration (2.65E–03 mg/L) NWQMC (2021)	406,938	274,090	1,792,464

E.2 Subsistence Fishers

Using conservative exposure estimates based on the water solubility limit as the surface water concentration, acute and chronic non-cancer risk estimates for subsistence fishers were above the benchmark of 30 (Table_Apx E-2). These results indicate that fish ingestion is not a pathway of concern for BBP for subsistence fishers.

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

Surface Water Concentration and Scenario	Acute and Chronic Non-cancer MOE UFs = 30
Water solubility limit (2.69 mg/L)	62
Monitored surface water concentration (2.65E–03 mg/L) (NWQMC, 2021)	63,441
Note: The acute and chronic MOEs are identical because the exposure estimates and the POD (point of departure) do not change between acute and chronic.	

E.3 Tribal Populations

Using conservative exposure estimates based on the water solubility limit as the surface water concentration, screening level non-cancer risk estimates for tribal populations were below the benchmark of 30 (Table_Apx E-3). Therefore, EPA refined its evaluation by using modeled surface water concentrations based on the 1) highest estimated 95th percentile release for the PVC plastics compounding OES as described in U.S. EPA (2024b) and 2) the P50, P75, and P90 flow metrics from the distribution. The higher flow metrics (*i.e.*, P75 and P90) are expected to be more representative of the flow conditions associated with high-end releases. The more refined exposure estimates did not result in risk estimates below the benchmarks. Overall, ingestion of fish potentially contaminated with BBP is not expected to be a pathway of concern for the tribal population.

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

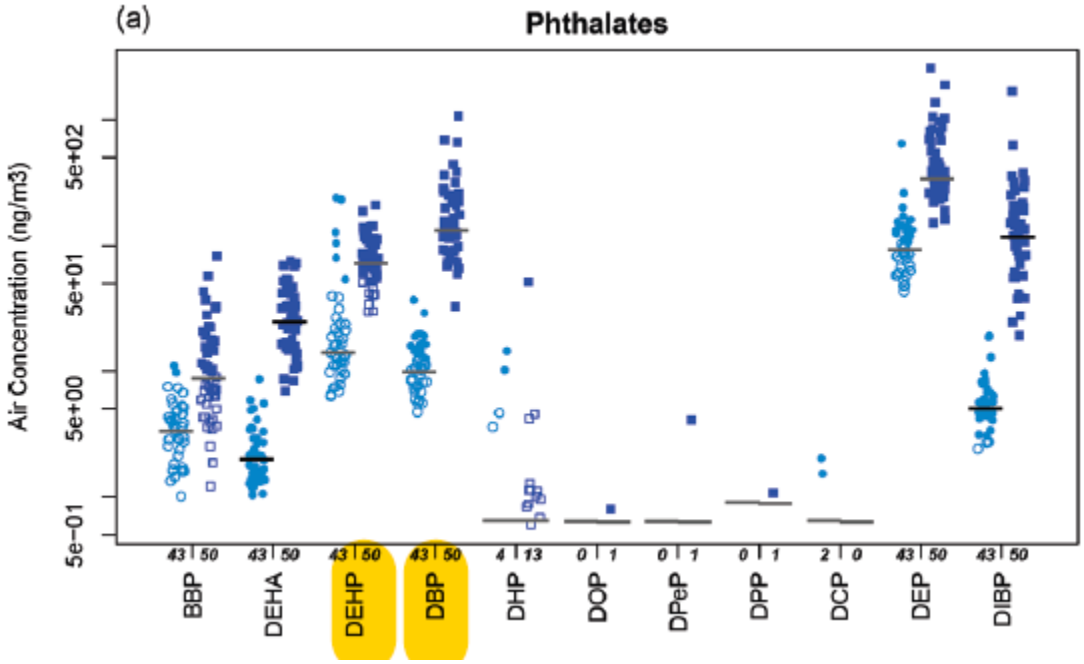
Surface Water Concentration and Scenario	Acute and Chronic Non-cancer MOE[a] UFs = 30		
	Current IR, Mean	Current IR, 95th Percentile	Heritage IR
Water solubility limit (2.69 mg/L)	41	10	5
PVC plastics compounding, P50 flow (2.05 mg/L)	54	13	7
PVC plastics compounding, P75 flow (1.53E–01 mg/L)	724	179	95
PVC plastics compounding, P90 flow (3.48E–03 mg/L)	31,849	7,889	4,178
Monitored surface water concentration (2.65E–03 mg/L) NWQMC (2021)	41,824	10,360	5,487

[a] The acute and chronic MOEs are identical because the exposure estimates and the POD (point of departure) 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 3.51E−08 to 9.75E−11, well below 1E−06.



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

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. The metabolite of BBP, Mono-benzyl phthalate (MBzP), has been reported in the NHANES data. MBzP has been reported in NHANES beginning with the 1999 cycle and measured in 26,740 members of the general public, including 7,331 children aged 15 and under and 19,409 adults aged 16 and over. Urinary MBzP concentrations were quantified using high performance liquid chromatography-electrospray ionization-tandem mass spectrometry. Limits of detection (LOD) for each cycle on NHANES are provided in Table_Apx G-1. Values below the LOD were replaced by the lower limit of detection divided by the square root of two (NCHS, 2021).

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

NHANES Cycle	MBzP (ng/mL)
1999–2000	0.47
2001–2002	0.47
2003–2004	0.11
2005–2006	0.3
2007–2008	0.3
2009–2010	0.216
2011–2012	0.3
2013–2014	0.3
2015–2016	0.3
2017–2018	0.3

2804 **Table_Apx G-2. Summary of Urinary BBP 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	MBzP	Adults	All adults	1,896	1,896 (94.83%)	3 (2.5–3.9)	32.5 (21.2–49.4)	3.02 (2.62–3.49)	23.37 (20.65–28.28)
2017–2018	MBzP	Adults	At or above poverty level	467	467 (97%)	3.1 (2.5–4.6)	33.4 (21.7–55.3)	2.73 (2.37–3.28)	21.47 (17.85–24.54)
2017–2018	MBzP	Adults	Below poverty level	337	337 (95.25%)	3.6 (2.3–5.7)	30.3 (18.5–52.8)	5 (4.29–5.88)	38.57 (30.17–52.4)
2017–2018	MBzP	Adults	Black non-Hispanic	438	438 (96.12%)	4.5 (3–5.6)	32.5 (15.8–52.8)	2.99 (2.63–3.56)	25.86 (17.98–33.51)
2017–2018	MBzP	Adults	Females	952	952 (92.96%)	3.2 (2.1–4.7)	30 (22.1–50.4)	3.52 (2.71–4.85)	26 (20.22–33.72)
2017–2018	MBzP	Adults	Males	944	944 (96.72%)	3.1 (2.5–3.9)	33 (21.1–50)	2.73 (2.34–3.2)	22.27 (18.45–25.85)
2017–2018	MBzP	Adults	Mexican American	278	278 (96.04%)	2.4 (1.3–3.5)	14.2 (7.9–33.4)	2.69 (2.18–3.2)	22.73 (16.41–30.71)
2017–2018	MBzP	Adults	Other	532	532 (91.17%)	2.1 (1.5–3.1)	38.2 (7.5–55.3)	2.69 (2.11–3.37)	22.7 (15–34.17)
2017–2018	MBzP	Adults	Unknown income	840	840 (93.57%)	3.1 (1.6–4.3)	21.9 (7.9–38.2)	2.98 (2.17–4.07)	22.99 (17.14–30.97)
2017–2018	MBzP	Adults	White non-Hispanic	648	648 (96.45%)	3.2 (2.5–4.8)	32.2 (21.1–57.5)	3.1 (2.6–3.92)	23.37 (20.22–29.29)
2015–2016	MBzP	Adults	All adults	1,880	1,880 (97.07%)	4.5 (3.8–5.1)	40 (24.8–59.8)	4.22 (3.56–5)	37.3 (31.25–47.56)
2015–2016	MBzP	Adults	At or above poverty level	461	461 (97.83%)	4.4 (3.8–5.2)	32.8 (23.8–44.8)	3.93 (3.21–4.88)	34.62 (28.6–41.34)
2015–2016	MBzP	Adults	Below poverty level	399	399 (98.5%)	6.3 (3.7–8.5)	43.3 (24.5–77.8)	5.87 (5.1–8.21)	74.93 (54.72–116)
2015–2016	MBzP	Adults	Black non-Hispanic	427	427 (99.06%)	7.3 (4.3–11.8)	46.1 (26.5–431.2)	5.09 (4.18–6.21)	65.31 (49.52–102.8)
2015–2016	MBzP	Adults	Females	984	984 (97.05%)	5.05 (3.6–6.1)	54.4 (30.4–75.7)	5.09 (4.35–6.13)	44.62 (33.4–51.17)
2015–2016	MBzP	Adults	Males	896	896 (97.1%)	4.4 (3.8–5.2)	38.8 (24.5–62.7)	3.71 (3.09–4.3)	35.4 (27.48–45.36)
2015–2016	MBzP	Adults	Mexican American	342	342 (96.78%)	3.6 (2.4–6.2)	37.8 (18.5–83.6)	4.83 (3.3–6.64)	51.25 (34.64–75.06)
2015–2016	MBzP	Adults	Other	540	540 (95.37%)	4.7 (3.3–7.7)	32.6 (16.4–59.8)	4.03 (2.87–5.32)	31.84 (23.28–44.55)
2015–2016	MBzP	Adults	Unknown income	833	833 (95.68%)	4.6 (2.4–13.9)	121.9 (6.6–160.3)	4.17 (2.99–6.86)	31.31 (20–58.53)
2015–2016	MBzP	Adults	White non-Hispanic	571	571 (97.37%)	4.2 (3.5–5.5)	32.8 (15.2–78.8)	4 (3.2–5.1)	35.5 (27.61–45.22)
2013–2014	MBzP	Adults	All adults	2,040	2,040 (97.11%)	4.9 (4.4–5.7)	30.4 (24.6–37.6)	3.98 (3.6–4.44)	25.57 (21.22–31.22)
2013–2014	MBzP	Adults	At or above poverty level	484	484 (98.14%)	4.7 (3.8–5.5)	26.2 (20.3–34.9)	3.68 (3.33–4.08)	22.27 (19.44–25.83)
2013–2014	MBzP	Adults	Below poverty level	454	454 (98.24%)	7.3 (3.8–13.5)	55 (23.1–115.3)	6.67 (5.04–7.85)	39.7 (32.71–62.98)
2013–2014	MBzP	Adults	Black non-Hispanic	442	442 (98.19%)	6.1 (4.8–7.9)	58.9 (37.6–100.9)	4.74 (4.08–5.37)	28.78 (23.87–37.68)
2013–2014	MBzP	Adults	Females	1,076	1,076 (96.93%)	5.45 (4.2–6.7)	39.7 (27.4–61.7)	4.56 (3.86–5.42)	30.78 (24.21–51.3)
2013–2014	MBzP	Adults	Males	964	964 (97.3%)	4.9 (4.3–5.6)	30.1 (23.8–37.1)	3.71 (3.33–4.08)	22.96 (19.39–26.74)
2013–2014	MBzP	Adults	Mexican American	282	282 (97.87%)	3.8 (2.5–5.7)	19.9 (13.5–58.6)	3.75 (3.1–5.21)	26.88 (18.95–36.86)
2013–2014	MBzP	Adults	Other	496	496 (95.36%)	4.1 (3.4–4.9)	24.7 (22.5–30.4)	3.6 (3.14–4.27)	23.87 (17.77–25.65)
2013–2014	MBzP	Adults	Unknown income	921	921 (95.77%)	5.5 (3.2–8.9)	29.4 (16.9–58)	4.07 (3.06–6.59)	23.87 (14.17–30.83)
2013–2014	MBzP	Adults	White non-Hispanic	820	820 (97.32%)	5.1 (4.3–6.6)	28.2 (22.9–37.6)	3.96 (3.52–4.62)	25.08 (20.28–35.05)
2011–2012	MBzP	Adults	All adults	1,894	1,894 (97.62%)	4.3 (3.8–5.2)	37.4 (25.2–65.1)	4.71 (4.27–5.2)	27.95 (24.47–31.49)
2011–2012	MBzP	Adults	At or above poverty level	449	449 (98.89%)	4.2 (3.5–5.2)	36.4 (22.7–48.3)	4.46 (4.04–4.84)	23.24 (18.89–28.71)

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	MBzP	Adults	Below poverty level	441	441 (97.73%)	5.7 (3.9–7)	63.4 (20.4–85.7)	6.44 (5.48–7.86)	51.57 (37.09–59.77)
2011–2012	MBzP	Adults	Black non-Hispanic	499	499 (98.6%)	8.2 (5.3–11.6)	48.8 (27.2–74.3)	5.12 (4.12–6.08)	39.94 (26.67–54.06)
2011–2012	MBzP	Adults	Females	933	933 (97.43%)	4.7 (4–5.3)	39.4 (24.8–73.4)	5.5 (4.62–6.57)	31.06 (27.1–49.28)
2011–2012	MBzP	Adults	Males	961	961 (97.81%)	4.3 (3.8–5.2)	37 (24.3–65.3)	4.26 (3.91–4.73)	24.66 (18.32–35.64)
2011–2012	MBzP	Adults	Mexican American	186	186 (98.39%)	4.7 (2.9–6.6)	20.6 (13.4–29.8)	5.77 (5–7.06)	31.06 (17.07–56.59)
2011–2012	MBzP	Adults	Other	545	545 (95.96%)	3.8 (2.9–4.9)	37.4 (25.5–67.9)	4.33 (3.61–4.94)	38.28 (18.89–90.96)
2011–2012	MBzP	Adults	Unknown income	821	821 (96.83%)	4.3 (2.4–5.9)	20.3 (15–21.5)	4.86 (3.86–6.43)	29.88 (15–80.38)
2011–2012	MBzP	Adults	White non-Hispanic	664	664 (98.04%)	3.9 (3.1–5.4)	37.5 (18.4–79.2)	4.62 (4.17–5.05)	24.67 (19.68–28.71)
2009–2010	MBzP	Adults	All adults	2,127	2,127 (99.39%)	6.89 (5.77–7.96)	46.01 (34.93–62.85)	6.24 (5.53–7.18)	36.71 (31.26–45.14)
2009–2010	MBzP	Adults	At or above poverty level	550	550 (99.27%)	6.7 (5.48–8.23)	44.6 (30.36–64.45)	5.98 (5.28–6.9)	32.53 (27.91–38.63)
2009–2010	MBzP	Adults	Below poverty level	469	469 (99.57%)	7.94 (6.13–8.62)	59.86 (33.29–132.29)	8.4 (6.79–10.21)	67.16 (55.68–99.57)
2009–2010	MBzP	Adults	Black non-Hispanic	400	400 (100%)	7.85 (6.08–10.21)	65.52 (37.99–92.91)	6.5 (5.03–7.77)	34.36 (25.87–51.01)
2009–2010	MBzP	Adults	Females	1,040	1,040 (99.13%)	7.33 (6.42–9.81)	43.2 (32.47–51.26)	7.17 (5.86–8.43)	46.27 (32.58–64.47)
2009–2010	MBzP	Adults	Males	1,087	1,087 (99.63%)	6.76 (5.73–8.03)	48.33 (34.91–63.99)	5.88 (5.12–6.72)	31.65 (27.61–35.17)
2009–2010	MBzP	Adults	Mexican American	393	393 (99.24%)	7.33 (4.9–10.97)	37.94 (23.7–74.58)	7.16 (6.25–8.15)	60.4 (32.57–72.68)
2009–2010	MBzP	Adults	Other	336	336 (99.11%)	4.99 (3.4–7.66)	39.74 (24.7–59.14)	5.38 (4.41–7.52)	30 (19.74–44.87)
2009–2010	MBzP	Adults	Unknown income	905	905 (99.34%)	5.55 (3.17–8.75)	42.49 (24–125.8)	5.38 (4.87–7.37)	32.57 (23.59–40.67)
2009–2010	MBzP	Adults	White non-Hispanic	998	998 (99.3%)	6.98 (5.73–8.56)	45.66 (33.65–64.45)	6.16 (5.39–7.34)	34.69 (28.49–47.63)
2007–2008	MBzP	Adults	All adults	2,021	2,021 (97.87%)	8.496 (7.128–9.576)	46.008 (37.368–62.136)	6.78 (6.12–7.61)	42.94 (35.74–49.41)
2007–2008	MBzP	Adults	At or above poverty level	505	505 (99.41%)	8.496 (7.128–9.576)	43.272 (33.624–66.888)	6.53 (5.87–7.2)	40.26 (34.13–46.38)
2007–2008	MBzP	Adults	Below poverty level	392	392 (98.72%)	10.584 (7.992–13.968)	46.728 (32.976–58.176)	9.42 (7.89–11.44)	56.4 (40.17–93.05)
2007–2008	MBzP	Adults	Black non-Hispanic	434	434 (98.62%)	9.072 (6.336–11.664)	69.336 (31.32–78.84)	7.03 (5.94–7.75)	42.58 (34.83–50.87)
2007–2008	MBzP	Adults	Females	1,030	1,030 (97.09%)	12.312 (10.008–13.896)	70.488 (48.888–127.8)	7.36 (6.31–8.74)	52.1 (44.49–69.31)
2007–2008	MBzP	Adults	Males	991	991 (98.69%)	8.568 (7.128–9.576)	43.272 (34.272–60.048)	6.65 (5.68–7.46)	36.25 (30.62–45.5)
2007–2008	MBzP	Adults	Mexican American	371	371 (98.65%)	9.144 (6.408–11.664)	40.536 (34.632–73.872)	7.78 (5.45–10.83)	42.94 (33.66–56.23)
2007–2008	MBzP	Adults	Other	294	294 (97.62%)	6.768 (3.24–10.656)	24.768 (18–60.048)	6.1 (3.78–9.4)	35.43 (28.88–50.87)
2007–2008	MBzP	Adults	Unknown income	948	948 (96.41%)	7.128 (5.04–12.888)	49.32 (10.944–112.464)	6.66 (5.49–8.36)	31.09 (23.37–90)

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)
2007–2008	MBzP	Adults	White non-Hispanic	922	922 (97.29%)	8.784 (7.128–10.44)	44.208 (32.616–66.888)	6.71 (6.1–7.66)	43.79 (33.23–53.26)
2005–2006	MBzP	Adults	All adults	1,831	1,831 (98.58%)	10.512 (9.288–12.312)	63.72 (56.664–67.104)	8.43 (7.32–9.49)	51.04 (44.86–59.31)
2005–2006	MBzP	Adults	At or above poverty level	436	436 (99.31%)	10.44 (8.856–12.816)	63.576 (55.512–68.76)	8.51 (7.31–9.4)	48.23 (40–59.56)
2005–2006	MBzP	Adults	Below poverty level	340	340 (98.82%)	11.088 (8.712–13.968)	63.72 (32.76–113.976)	9.53 (7.6–11.41)	61.5 (57.76–86.4)
2005–2006	MBzP	Adults	Black non-Hispanic	464	464 (98.92%)	12.024 (8.64–15.12)	77.688 (56.664–114.048)	7.77 (6.47–10.49)	55.38 (41.16–62.8)
2005–2006	MBzP	Adults	Females	935	935 (97.97%)	9.864 (6.552–11.448)	67.536 (42.48–83.52)	9.43 (8.83–10.48)	72.67 (53.79–91.23)
2005–2006	MBzP	Adults	Males	896	896 (99.22%)	10.584 (9.288–12.6)	63.72 (55.512–67.104)	7.58 (6.65–8.91)	43.66 (36.26–48.45)
2005–2006	MBzP	Adults	Mexican American	390	390 (99.23%)	10.224 (7.416–12.744)	58.104 (32.76–88.776)	8.43 (7.08–9.02)	45.15 (30.49–54.49)
2005–2006	MBzP	Adults	Other	131	131 (99.24%)	12.96 (4.824–21.744)	64.44 (24.984–139.032)	8.59 (5.57–14.98)	47.02 (36.62–93.94)
2005–2006	MBzP	Adults	Unknown income	955	955 (98.12%)	12.024 (3.096–32.472)	45.432 (12.672–94.608)	6.09 (5.33–9.92)	23.25 (18.14–92.21)
2005–2006	MBzP	Adults	White non-Hispanic	846	846 (97.99%)	10.152 (8.064–13.176)	59.184 (50.544–68.76)	8.53 (7.2–9.77)	51.44 (42.75–65.28)
2003–2004	MBzP	Adults	All adults	1,889	1,889 (99.52%)	11.16 (9.216–13.464)	61.344 (51.696–74.736)	8.48 (7.65–9.31)	51.06 (43.75–62.86)
2003–2004	MBzP	Adults	At or above poverty level	474	474 (99.37%)	10.512 (8.064–13.464)	61.344 (51.192–75.816)	7.99 (7.2–9.04)	45.85 (40.5–54.9)
2003–2004	MBzP	Adults	Below poverty level	393	393 (99.49%)	12.6 (9.432–14.112)	54.504 (39.024–102.672)	11.59 (10.25–13.95)	78.84 (65.77–97.75)
2003–2004	MBzP	Adults	Black non-Hispanic	423	423 (99.76%)	16.2 (11.952–21.672)	78.768 (56.592–93.024)	10.02 (8.45–11.77)	53.93 (42.81–69.6)
2003–2004	MBzP	Adults	Females	980	980 (99.69%)	12.024 (9.144–15.12)	65.736 (50.688–92.232)	9.82 (8.37–11.57)	61.66 (49.58–71.65)
2003–2004	MBzP	Adults	Males	909	909 (99.34%)	10.944 (9.216–13.464)	61.416 (51.696–74.736)	7.98 (6.99–9.03)	44.92 (37.42–58.73)
2003–2004	MBzP	Adults	Mexican American	423	423 (99.53%)	10.512 (8.856–13.896)	62.712 (36.072–94.68)	9.08 (8.14–10.07)	66.6 (41.08–100.08)
2003–2004	MBzP	Adults	Other	142	142 (100%)	10.224 (7.56–13.536)	34.992 (19.08–44.856)	9.02 (7.03–10.9)	78.84 (37.38–246.45)

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)
2003–2004	MBzP	Adults	Unknown income	904	904 (99.56%)	12.888 (4.464–25.2)	47.232 (25.2–74.736)	7.86 (6.61–9.99)	49.44 (23.84–97.2)
2003–2004	MBzP	Adults	White non-Hispanic	901	901 (99.33%)	10.656 (7.776–13.824)	61.416 (47.88–75.816)	8.03 (7.2–9.31)	46.31 (39.86–54.9)

Table_Apx G-3. Regression Coefficients and P-values for Statistical Analyses of MBzP 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	MBzP	Adults	All adults	Age	sex race income	–[a]	<0.001	–[a]	<0.001
2013–2018	MBzP	Adults	All adults	Income	age sex race	–[a]	<0.001	–[a]	<0.001
2013–2018	MBzP	Adults	All adults	Race	age sex income	–[a]	<0.001	–[a]	<0.001
2013–2018	MBzP	Adults	All adults	Sex	age race income	–[a]	0.1119	–[a]	<0.001
2013–2018	MBzP	Adults	All adults	Years	age sex race income	–0.3290	<0.001	–1.0039	<0.001
2013–2018	MBzP	Adults	All adults	Years	age sex race income	–0.3290	<0.001	–1.0039	<0.001
2013–2018	MBzP	Adults	At or above poverty level	Years	age sex race	–0.3953	<0.001	–1.1898	<0.001
2013–2018	MBzP	Adults	At or above poverty level	Years	age sex race	–0.3953	<0.001	–1.1898	<0.001
2013–2018	MBzP	Adults	Below poverty level	Years	age sex race	–0.4518	<0.001	–1.162	<0.001
2013–2018	MBzP	Adults	Below poverty level	Years	age sex race	–0.4518	<0.001	–1.162	<0.001
2013–2018	MBzP	Adults	Black non-Hispanic	Years	age sex income	–0.2435	<0.001	–1.2106	<0.001
2013–2018	MBzP	Adults	Black non-Hispanic	Years	age sex income	–0.2435	<0.001	–1.2106	<0.001
2013–2018	MBzP	Adults	Females	Years	age race income	–0.4089	<0.001	–0.7033	<0.001
2013–2018	MBzP	Adults	Females	Years	age race income	–0.4089	<0.001	–0.7033	<0.001
2013–2018	MBzP	Adults	Males	Years	age race income	–0.3725	<0.001	–0.9019	<0.001
2013–2018	MBzP	Adults	Males	Years	age race income	–0.3725	<0.001	–0.9019	<0.001
2013–2018	MBzP	Adults	Mexican-American	Years	age sex income	–0.3155	<0.001	–0.8723	<0.001
2013–2018	MBzP	Adults	Mexican-American	Years	age sex income	–0.3155	<0.001	–0.8723	<0.001
2013–2018	MBzP	Adults	Other	Years	age sex income	–0.3768	<0.001	–0.4164	<0.001
2013–2018	MBzP	Adults	Other	Years	age sex income	–0.3768	<0.001	–0.4164	<0.001
2013–2018	MBzP	Adults	Unknown income	Years	age sex race	–0.0408	0.1193	–0.6250	<0.001
2013–2018	MBzP	Adults	Unknown income	Years	age sex race	–0.0408	0.1193	–0.6250	<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	MBzP	Adults	White non-Hispanic	Years	age sex income	–0.542	<0.001	–1.1074	<0.001
2013–2018	MBzP	Adults	White non-Hispanic	Years	age sex income	–0.542	<0.001	–1.1074	<0.001
2013–2018	MBzP	Children	All children (<16 years old)	Age	sex race income	– [a]	<0.001	– [a]	<0.001
2013–2018	MBzP	Children	All children (<16 years old)	Income	age sex race	– [a]	0.0128	– [a]	<0.001
2013–2018	MBzP	Children	All children (<16 years old)	Race	age sex income	– [a]	<0.001	– [a]	<0.001
2013–2018	MBzP	Children	All children (<16 years old)	Sex	age race income	– [a]	0.3294	– [a]	0.0038
2013–2018	MBzP	Children	Adolescents (11–<16 years old)	Years	sex race income	–0.5365	<0.001	–1.5899	<0.001
2013–2018	MBzP	Children	Adolescents (11–<16 years old)	Years	sex race income	–0.5365	<0.001	–1.5899	<0.001
2013–2018	MBzP	Children	Toddlers (3–<6 years old)	Years	sex race income	–0.3907	<0.001	–1.2566	<0.001
2013–2018	MBzP	Children	Toddlers (3–<6 years old)	Years	sex race income	–0.3907	<0.001	–1.2566	<0.001
2013–2018	MBzP	Children	Children (6–<10 years old)	Years	sex race income	–0.2917	<0.001	0.07718	0.0308
2013–2018	MBzP	Children	Children (6–<10 years old)	Years	sex race income	–0.2917	<0.001	0.07718	0.0308
2013–2018	MBzP	Children	All children (<16 years old)	Years	age sex race income	–0.3539	<0.001	–1.0296	<0.001
2013–2018	MBzP	Children	All children (<16 years old)	Years	age sex race income	–0.3539	<0.001	–1.0296	<0.001
2013–2018	MBzP	Children	At or above poverty level	Years	age sex race	–0.7878	<0.001	–2.1812	<0.001
2013–2018	MBzP	Children	At or above poverty level	Years	age sex race	–0.7878	<0.001	–2.1812	<0.001
2013–2018	MBzP	Children	Below poverty level	Years	age sex race	–0.315	<0.001	–0.80	<0.001
2013–2018	MBzP	Children	Below poverty level	Years	age sex race	–0.315	<0.001	–0.80	<0.001
2013–2018	MBzP	Children	Black non-Hispanic	Years	age sex income	–0.9670	<0.001	–3.4386	<0.001
2013–2018	MBzP	Children	Black non-Hispanic	Years	age sex income	–0.9670	<0.001	–3.4386	<0.001
2013–2018	MBzP	Children	Females	Years	age race income	–0.2911	<0.001	–0.2691	<0.001
2013–2018	MBzP	Children	Females	Years	age race income	–0.2911	<0.001	–0.2691	<0.001
2013–2018	MBzP	Children	Males	Years	age race income	–0.5372	<0.001	–1.8356	<0.001
2013–2018	MBzP	Children	Males	Years	age race income	–0.5372	<0.001	–1.8356	<0.001
2013–2018	MBzP	Children	Mexican-American	Years	age sex income	–0.4697	<0.001	–0.8363	<0.001
2013–2018	MBzP	Children	Mexican-American	Years	age sex income	–0.4697	<0.001	–0.8363	<0.001
2013–2018	MBzP	Children	Other	Years	age sex income	–0.1035	<0.001	–0.2434	<0.001
2013–2018	MBzP	Children	Other	Years	age sex income	–0.1035	<0.001	–0.2434	<0.001
2013–2018	MBzP	Children	Unknown Income	Years	age sex race	0.04313	0.4129	0.32618	<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	MBzP	Children	Unknown Income	Years	age sex race	0.04313	0.4129	0.32618	<0.001
2013–2018	MBzP	Children	White non-Hispanic	Years	age sex income	–0.6311	<0.001	–1.3746	<0.001
2013–2018	MBzP	Children	White non-Hispanic	Years	age sex income	–0.6311	<0.001	–1.3746	<0.001
2013–2018	MBzP	Women	All women of reproductive age	Age	sex race income	– [a]	<0.001	– [a]	<0.001
2013–2018	MBzP	Women	All women of reproductive age	Income	age sex race	– [a]	0.265	– [a]	0.0652
2013–2018	MBzP	Women	All women of reproductive age	Race	age sex income	– [a]	0.1267	– [a]	<0.001
2013–2018	MBzP	Women	All women of reproductive age	Sex	age race income	– [a]	<0.001	– [a]	<0.001
2013–2018	MBzP	Women	All women of reproductive age	Years	age sex race income	–0.5212	<0.001	–1.2797	<0.001
2013–2018	MBzP	Women	At or above poverty level	Years	age sex race	–0.4526	<0.001	–2.2884	<0.001
2013–2018	MBzP	Women	Below poverty level	Years	age sex race	–0.7034	<0.001	0.20487	0.5073
2013–2018	MBzP	Women	Black non-Hispanic	Years	age sex income	–0.5845	<0.001	0.3356	0.1941
2013–2018	MBzP	Women	Females	Years	age race income	–0.5212	<0.001	–1.2797	<0.001
2013–2018	MBzP	Women	Mexican-American	Years	age sex income	–0.5409	<0.001	–1.6176	<0.001
2013–2018	MBzP	Women	Other	Years	age sex income	–0.3347	<0.001	–0.9468	<0.001
2013–2018	MBzP	Women	Unknown income	Years	age sex race	–0.4815	<0.001	–1.3442	<0.001
2013–2018	MBzP	Women	White non-Hispanic	Years	age sex income	–0.5967	<0.001	–0.6889	<0.001

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

2807