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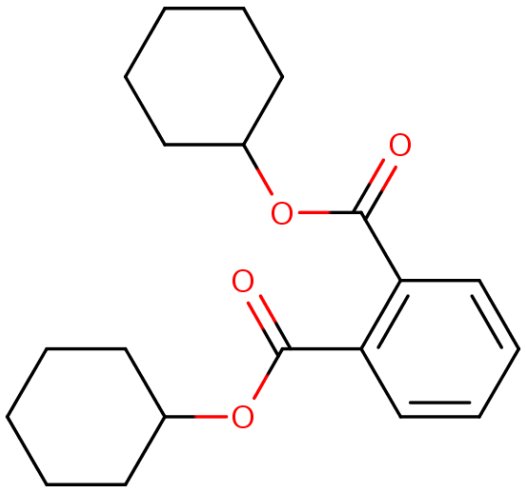
Environmental Media and General Population and Environmental Exposure Assessment for Dicyclohexyl Phthalate (DCHP)

Technical Support Document for the Risk Evaluation

CASRN 84-61-7



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KEY ABBREVIATIONS AND ACRONYMS

7Q10	Lowest 7-day average flow in a 10-year period
30Q5	Lowest 30-day average flow in a 5-year period
ADD	Average daily dose
ADR	Acute dose rate
AERMOD	American Meteorological Society (AMS)/EPA Regulatory Model
BAF	Bioaccumulation factor
BBP	Butyl benzyl phthalate
BCF	Bioconcentration factor
CASRN	Chemical Abstracts Service Registry Number
CDC	Centers for Disease Control and Prevention (U.S.)
CEM	Consumer Exposure Model
COU	Condition of use
DBP	Dibutyl phthalate
DCHP	Dicyclohexyl phthalate
DEHP	Diethylhexyl phthalate
ECHO	EPA's Enforcement and Compliance History Online (database)
EPA	Environmental Protection Agency (U.S.)
EROM	Enhanced Runoff Method (flow database)
HEC	Human equivalent concentration
HED	Human equivalent dose
IIOAC	Integrated Indoor-Outdoor Air Calculator Model

IR	Ingestion rate
K_{OC}	Organic carbon:water partition coefficient
K_{OW}	Octanol:water partition coefficient
K_p	Dermal permeability coefficient
LADD	Lifetime average daily dose
MOE	Margin of exposure
NAICS	North American Industry Classification System
NHANES	National Health and Nutrition Examination Survey
NPDES	National Pollutant Discharge Elimination System
OES	Occupational exposure scenario
PESS	Potentially exposed or susceptible subpopulation(s)
POD	Point of departure
PSC	Point Source Calculator tool
SD	Standard deviation
TRI	Toxics Release Inventory
TSCA	Toxic Substances Control Act
TSD	Technical support document
UF	Uncertainty factor
U.S.	United States

SUMMARY

Dicyclohexyl Phthalate (DCHP) – Environmental Media Concentration and General Population Exposure: Key Points

The U.S. Environmental Protection Agency (EPA or the Agency) evaluated the reasonably available information for various environmental media concentrations and estimated exposure using a worst-case exposure scenario as a screening level approach. The conservative worst-case exposure was assumed to result from the highest DCHP releases associated with the corresponding Toxic Substances Control Act (TSCA) condition of use (COU) via different exposure pathways. The key points of this assessment are summarized below:

- EPA assessed environmental concentrations of DCHP in air, surface and groundwater, as well as land (soil, biosolids, and groundwater) for use in environmental exposure and general population exposure assessment.
    - For the land pathway, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered. However, based on high-quality physical and chemical property data, EPA determined that DCHP 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 surface water pathway, DCHP released into water is expected to predominantly partition into sediment. The high-end, modeled, total water column concentration of DCHP was orders of magnitude above any monitored value.
    - For the ambient air pathway, modeled DCHP concentrations are higher than measured concentrations by several orders of magnitude. This is an expected outcome because EPA's modeling uses high-end releases and conservative meteorological data.
    - Although DCHP may persist in sediment, soil, biosolids, or landfills after release to these media/environments, DCHP's bioavailability is expected to be limited.
- Screening level risk estimates using high-end, modeled surface water concentrations exceeded 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.
- For human exposure through fish ingestion, additional refinements of the highest modeled surface water concentration were conducted because screening level risk estimates indicated potential risks. In these refined scenarios, which are expected to be more representative of DCHP exposures than the high-end screening analysis, no risk was identified.
- EPA concludes that there are no exposure DCHP pathways of concern for the general population.
- DCHP is not readily found in aquatic or terrestrial organisms and has low bioaccumulation and biomagnification potential. Therefore, DCHP has low potential for trophic transfer through food webs.

1 ENVIRONMENTAL MEDIA CONCENTRATION OVERVIEW

This technical support document (TSD) accompanies the TSCA *Risk Evaluation for Dicyclohexyl Phthalate (DCHP)* (also called the "risk evaluation") (U.S. EPA, 2025l). DCHP is a common chemical name for the category of chemical substances under one Chemical Abstracts Service Registry Number (CASRN; 84-61-7): 1,2-benzenedicarboxylic acid, dicyclohexyl ester; phthalic acid, dicyclohexyl ester; and dicyclohexyl 1,2-benzenedicarboxylate. DCHP is a white, crystalline solid commonly used as a plasticizer in the production of plastics and other polymers.

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

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

Life Cycle Stage	Category	Subcategory	OES(s)
Manufacturing	Domestic manufacturing	Domestic manufacturing	Manufacturing
	Importing	Importing	Import and repackaging
Processing	Repackaging	Repackaging (*e.g.,* laboratory chemicals)	Import and repackaging
	Incorporation into formulation, mixture, or reaction product	Adhesives manufacturing	Incorporation into adhesives and sealants
		Plasticizer in manufacturing adhesive, paint and coating, plastics product, printing ink, rubber product, and plastic material and resin	Incorporation into adhesives and sealants Incorporation into paints and coatings PVC plastics compounding Non-PVC material compounding
		Stabilizing agent in manufacturing plastics product, paint and coating, asphalt, paving, roofing, and coating materials, and adhesive	Incorporation into adhesives and sealants Incorporation into paints and coatings Incorporation into other formulations, mixtures, or reaction products PVC plastics compounding Non-PVC material compounding
	Incorporation into articles	Plasticizer in plastic product manufacturing and rubber product manufacturing	PVC plastics converting Non-PVC material converting
	Recycling	Recycling	Recycling

Life Cycle Stage	Category	Subcategory	OES(s)
Disposal	Disposal	Disposal	Waste handling, treatment, and disposal
Distribution in Commerce	Distribution in commerce	Distribution in commerce	Distribution in commerce
Industrial Uses	Adhesive and sealants	Adhesives and sealants in transportation equipment manufacturing, computer and electronic product manufacturing	Application of adhesives and sealants
	Finishing agent	Cellulose film production	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 in transportation equipment manufacturing	Fabrication or use of final products or articles
	Paints and coatings	Paints and coatings	Application of paints and coatings
Commercial Uses	Adhesives and sealants	Adhesives and sealants	Application of adhesives and sealants
	Building/construction materials not covered elsewhere	Building/construction materials not covered elsewhere	Fabrication or use of final products or articles
	Inks, toner, and colorant products	Inks, toner, and colorant products (*e.g.,* screen printing ink)	Application of paints and coatings
	Laboratory chemical	Laboratory chemical	Use of laboratory chemicals
	Paints and coatings	Paints and coatings	Application of paints and coatings
	Plasticizer in other articles with routine direct contact during normal use including rubber articles; plastic articles (hard)	Plasticizer in other articles with routine direct contact during normal use including rubber articles; plastic articles (hard)	Fabrication or use of final products or articles

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

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d]
Manufacturing	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Import and repackaging	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Incorporation into adhesives and sealants	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Incorporation into paints and coatings	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Incorporation into other formulations, mixtures, and reaction products not covered elsewhere	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
PVC plastics compounding	Fugitive or stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Water
	Incineration or landfill
PVC plastics converting	Fugitive or stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Water
	Incineration or landfill

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d]
Non-PVC material compounding	Fugitive or stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Water
	Incineration or landfill
Non-PVC material converting	Fugitive or stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Water
	Incineration or landfill
Application of adhesives and sealants	Fugitive air
	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Application of paints and coatings	Fugitive air
	Stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Incineration or landfill
Use of laboratory chemicals – liquid	Fugitive or stack air
	Water, incineration, or landfill
Use of laboratory chemicals – solid	Stack air
	Unknown media (air, water, incineration, or landfill)
	Water, incineration, or landfill
	Incineration or landfill
Fabrication or use of final products or articles – dust generation	Fugitive or stack air, water, incineration, or landfill
Fabrication or use of final products or articles – vapor generation	Fugitive or stack air
Recycling	Stack air
	Fugitive air, water, incineration, or landfill
	Wastewater
	Water, incineration, or landfill
Waste handling, treatment, and	Releases to all media are possible but non-quantifiable due to a lack

OES[a]	Type of Discharge,[b] Air Emission,[c] or Transfer for Disposal[d]
disposal	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 DCHP from the largest estimated releases for its screening level assessment of environmental and general population exposures. This means that the Agency considered the environmental concentration of DCHP in a given environmental media resulting from the OES that had the highest release compared to the other OES for the same releasing media. The OES resulting in the highest environmental concentration of DCHP 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 *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k). Briefly, based on DCHP's fate parameters (*e.g.*, Henry's Law constant, organic carbon:water partition coefficient (log K_{OC}), water solubility, fugacity modeling), EPA anticipates DCHP to be predominantly in surface water, soil, and sediment. However, because DCHP 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 DCHP in surface water, sediment, and ambient air. Soil concentrations of DCHP from land application of biosolids were not quantitatively assessed as DCHP was expected to have limited persistence potential and mobility in soils receiving biosolids. Additionally, DCHP in groundwater from landfills was not quantified because of its high hydrophobicity and high affinity for soil sorption, making unlikely that DBP will migrate from landfills via groundwater infiltration.

Environmental exposures using the predicted concentrations of DCHP are presented in Section 1. 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. If no risk was identified for an individual with 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 major pathway of general population exposure and not pursued further. For those pathways identified as exposure pathways of concern for the general population, further exposure assessments were conducted to include higher tiers of modeling when available, refinement of exposure estimates, and exposure estimates for additional subpopulations and COUs or OESs.

Table 1-3 summarizes the exposure pathways assessed for the general population. For DCHP, exposures to the general population via surface water, drinking water sourced from surface 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, landfills, and groundwater) were qualitatively assessed because DCHP is not expected to be persistent or mobile in soils. Only limited and non-U.S. data on biosolids were identified, which detected DCHP in biosolids at very low concentrations comprising less than 1 percent of total phthalates concentrations in biosolids; no monitoring data for DCHP in landfill were available. Further description of the qualitative and quantitative assessments for each exposure pathway can be found in the sections linked in Table 1-3. As summarized in that table, biosolids application to soil, waste disposal into landfills and subsequent leaching into groundwater, surface water, drinking water, fish ingestion, and ambient air are not

pathways of concern for DCHP for highly exposed populations based on the OES that may result in the highest concentrations of DCHP in environmental media.

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

OES[a]	Exposure Pathway	Exposure Route	Exposure Scenario	Pathway of Concern[b]
All	Biosolids (Section 3.1)	No specific exposure scenarios were assessed for qualitative assessments		No
All	Landfills (Section 3.2)	No specific exposure scenarios were assessed for qualitative assessments		No
Application of paints and coatings; PVC plastics compounding	Surface water	Dermal	Dermal exposure to DCHP in surface water during swimming (Section 5.1.1)	No
		Oral	Incidental ingestion of DCHP in surface water during swimming (Section 5.1.2)	No
Application of paints and coatings; PVC plastics compounding	Drinking water	Oral	Ingestion of drinking water sourced from surface water (Section 6.1.1)	No
PVC plastics compounding; Application of paints and coatings	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	No
			Ingestion of fish for subsistence fishers (Section 7.2)	No
			Ingestion of fish for tribal populations (Section 7.3)	No
Application of paints and coatings-no engineering controls	Ambient air	Inhalation	Inhalation of DCHP in ambient air resulting from industrial releases (Section 8)	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.

2 SCREENING LEVEL ASSESSMENT OVERVIEW

Screening level assessments are useful when there is little facility location- or scenario-specific information available. EPA began its DCHP exposure assessment using a screening level approach because of the limited environmental monitoring data and absence of location data for DCHP 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 DCHP per body weight were considered to be those at the upper end of the exposure. Taken together, these exposure estimates are conservative because they were determined using the highest environmental media concentrations and greatest intake rate of DCHP 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. Additional 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 OES/COUs.

2.1 Estimating High-End Exposure

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

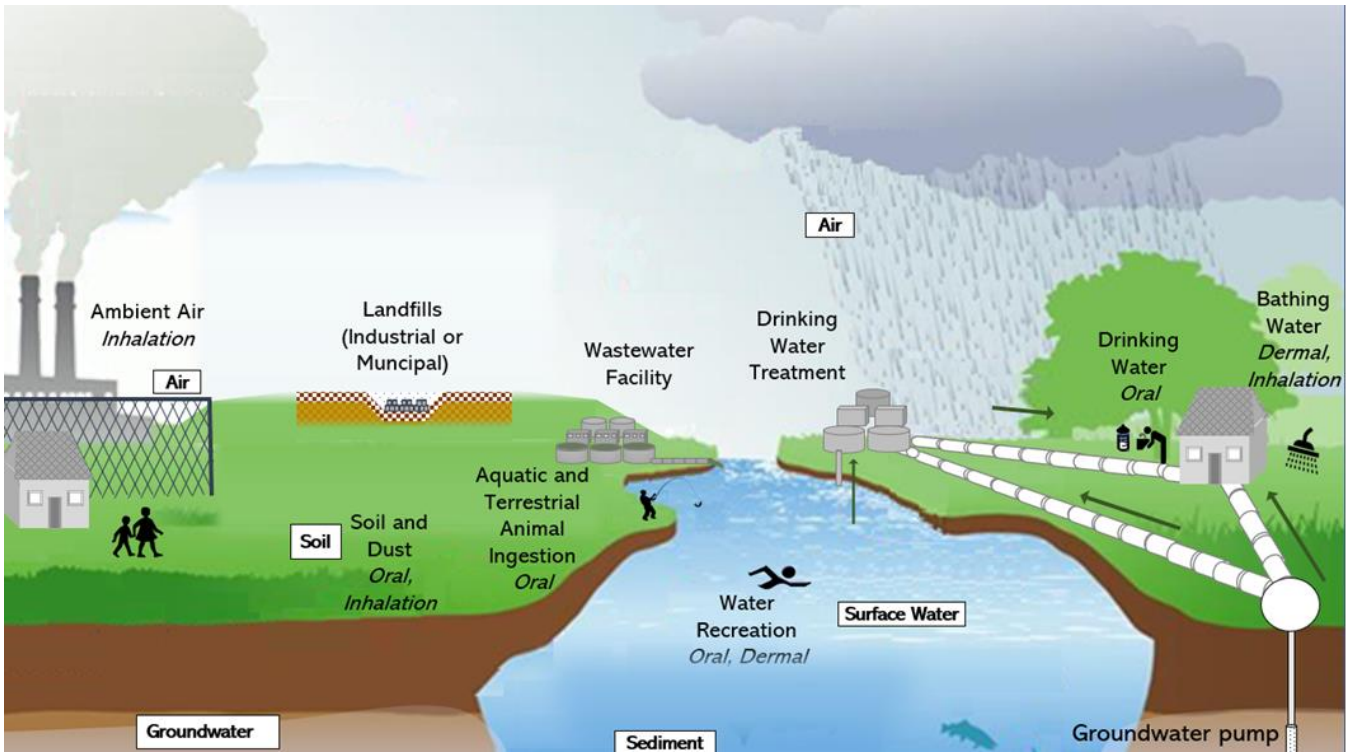

Figure 2-1. Potential Human Exposure Pathways for the General Population
The diagram presents the media (white text boxes) and routes of exposure (italics for oral, inhalation, or dermal) for the general population. Sources of drinking water from surface or water pipes are depicted with grey arrows.

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

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

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

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

OES	Exposure Pathway	Exposure Route	Exposure Scenario	Lifestage	Analysis (Quantitative or Qualitative) and Section
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
PVC plastics compounding; application of paints and coatings	Surface water	Dermal	Dermal exposure to DCHP in surface water during swimming	Adults, youths, and children	Quantitative, Section 5.1.1
		Oral	Incidental ingestion of DCHP in surface water during swimming	Adults, youths, and children	Quantitative, Section 5.1.2
PVC plastics compounding; application of paints and coatings	Drinking water	Oral	Ingestion of drinking water sourced from surface water	Adults, youths, and children	Quantitative, Section 6.1.1
PVC plastics compounding; application of paints and coatings	Fish ingestion	Oral	Ingestion of fish for General Population	Adults and children	Quantitative, Section 7.1
			Ingestion of fish for subsistence fishers	Adults	Quantitative, Section 7.2
			Ingestion of fish for tribal populations	Adults	Quantitative, Section 7.3
Application of paints and coatings – no engineering controls	Ambient air	Inhalation	Inhalation of DCHP in ambient air resulting from industrial releases	All	Quantitative, Section 9.1

As part of the general population exposure assessment, EPA considered fenceline populations in proximity to releasing facilities as part of the ambient air exposure assessment by utilizing pre-screening methodology described in EPA's *Draft TSCA Screening Level Approach for Assessing Ambient Air and Water Exposures to Fenceline Communities (Version 1.0)* (U.S. EPA, 2022b). For other exposure pathways, EPA's screening method assessing high-end exposure scenarios used release data that 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 6.1.1), incidental dermal exposures (Section 5.1.1), incidental oral exposures (Section 5.1.2), and fish ingestion exposure (Section 7). Modeled ambient air concentrations (Section 8.1) were used to estimate inhalation exposures.

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

available as well as 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, 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, it is important to emphasize that calculated risk estimates are not "bright-line" indicators of unreasonable risk—EPA has 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 *Non-Cancer Human Health Hazard Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025j). Briefly, after considering hazard identification and evidence integration, dose-response evaluation, and weight of scientific evidence of POD candidates, EPA chose one non-cancer POD for acute, intermediate, and chronic exposure scenarios (Table 2-2). HECs are based on daily continuous (24-hour) exposure while and HEDs are daily values.

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.

Table 2-2. Non-Cancer HECs and HEDs Used to Estimate Risks

Exposure Scenario	Target Organ System	Species	Duration	POD (mg/kg-day)	Effect	HED [a] (mg/kg-day)	HEC [a] (mg/m^3) [ppm]	Benchmark MOE[b]	Reference
Acute, intermediate, chronic	Developmental toxicity	Rat	10 days during gestation	NOAEL (LOEL) [c] = 10	Phthalate syndrome-related effects (*e.g.,* ↓ fetal testicular testosterone; ↓ AGD; Leydig cell effects; ↓ mRNA and/or protein expression of steroidogenic genes; ↓ INSL3)	2.4	13 [0.95]	$UF_A = 3$ $UF_H = 10$ *Total UF = 30*	Li et al. (2016)

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

[a] HED and HEC values calculated based on the most sensitive LOAEL of 10 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] Statistically significant effects at 10 mg/kg-day are limited to fetal Leydig cell effects, decreased expression of genes and proteins involved in steroidogenesis, and decreased protein expression of INSL3 (all of which are not considered adverse in isolation). The remaining effects listed reached statistical significance at higher doses.

3 LAND PATHWAY

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data identified during systematic review to obtain concentrations of DCHP in terrestrial land pathways (*i.e.*, biosolids, wastewater sludge, agricultural soils, landfills, and landfill leachate). No monitoring data were 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 DCHP in various media, including leachate, activated sludge, and biosolids (Wu et al., 2019). EPA cannot correlate monitoring levels with any releases associated with DCHP TSCA COUs. That is, EPA does not have any facility-specific DCHP release data because facilities do not report releases of DCHP to surface waters from TSCA COUs. As such, the present assessment of DCHP exposure via potential land pathways is qualitative in nature relying on the physical and chemical properties and fate characteristics of DCHP. When possible, data from the existing literature, including experimental and field data, were used to support the qualitative assessment.

3.1 Biosolids

The term "biosolids" refers to treated sludge that meets 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 is discussed in Section 3.2. DCHP may be introduced to biosolids by the absorption or adsorption of DCHP to particulate or organic material during wastewater treatment. Wastewater treatment is expected to remove up to 98 percent of DCHP via sorption of DCHP to biosolids (Wu et al., 2019). The STPWIN™ Model in EPI Suite™ predicts that sorption will account for a total of 71.2 percent removal of DCHP in wastewater treatment, with 70.6 percent attributed to biosolid sorption and the remaining 0.6 percent attributed to biological treatment (U.S. EPA, 2017).

There are currently no U.S.-based studies reporting DCHP concentration in biosolids or in soil following land application. However, three Chinese studies provided data related to DCHP in biosolids. A 2019 survey of wastewater removal of phthalates in China identified DCHP in two of the three sludge samples collected with an average concentration and standard deviation (SD) of 0.31 ± 0.20 mg/kg dry weight (dw) (Wu et al., 2019). This study received an overall data quality rating of medium under EPA's systematic review. A separate 2019 Chinese survey of wastewater sludge from 46 wastewater treatment plants found DCHP in 57 percent of samples with a mean DCHP concentration of 0.0093 mg/kg (range: 0.0014–0.0836 mg/kg), comprising less than 1 percent of the total phthalate concentration in biosolids (Zhu et al., 2019). An earlier, 2013 survey of 25 Chinese wastewater treatment plants identified DCHP in 100 percent of sludge samples (n = 25) with a mean concentration of 0.10 mg/kg (range: 0.039–0.19 mg/kg), accounting for 0.08 percent of total phthalates present in sludge samples (total phthalates mean: 123 mg/kg, total phthalates range: 22.6–1,350 mg/kg) (Meng et al., 2014). The latter study received an overall data quality rating of medium under EPA's systematic review.

Other sources of DCHP in biosolids-amended soils may include atmospheric or wet deposition to soil. DCHP may be present in rain, with one 2008 Dutch survey reporting DCHP at concentrations up to 0.196 µg/L in precipitation (Peters et al., 2008). The study received an overall data quality rating of medium under EPA's systematic review. DCHP may be deposited to biosolid-amended soils directly from the atmosphere with one 2010 Chinese study reporting a mean deposition flux of DCHP from outdoor air to soil in the range of 0.088 to 0.433 µg/m^2-day (urban) and 0.033 µg/m^2-day (suburban)

(Zeng et al., 2010). That study received an overall data quality rating of high under EPA's systematic review. However, as in precipitation, it is likely that direct deposition of DCHP to biosolid-amended soils would be severely limited by the low persistence of DCHP in the atmosphere.

No data were available reporting or estimating the DCHP concentrations in biosolids or biosolid-applied soils in the United States. A conservative estimate of 0.71 mg/kg dw was calculated from the 95th percentile[1] of the highest reported average concentration of DCHP in biosolids (mean and SD 0.31 ± 0.20 mg/kg dw reported by Wu et al. (2019)). A DCHP soil concentration calculated from the 95th percentile of the highest reported average concentration of DCHP in dewatered biosolids, 0.71 mg/kg dry weight, was be used as the conservative soil concentration of DCHP in biosolid-amended soils. High-end release scenarios were considered not to be applicable to the evaluation of land application of biosolids. More specifically, high-end releases of DCHP from industrial facilities are typically not discharged directly to municipal wastewater treatment plants without pretreatment, and biosolids from industrial facilities not expected to be directly applied to land following on-site treatment. No industrial facilities have reported release of DCHP-containing water to POTW facilities, nor have they reported biosolid production or land application of DCHP-containing biosolids to the Toxics Release Inventory (TRI).

DCHP is expected to have a high affinity to particulate (log K_{OC} = 4.47) and organic media (octanol:water partition coefficient [log K_{OW}] = 4.82), 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. DCHP is slightly soluble in water (1.48 mg/L) and does have limited potential to leach from biosolids and infiltrate into biosolids. However, the high-end concentration estimates of DCHP and high sorption to biosolids suggest that potential leaching from biosolids-amended soils will not be solubility-limited but instead will be limited by high sorption and high retardation. Because DCHP does have high hydrophobicity and a high affinity for soil sorption, it is unlikely that DCHP will migrate from potential biosolids-amended soils via groundwater infiltration or surface runoff. As such, EPA did not simulate surface water runoff or groundwater infiltration resulting from the land application of biosolids.

DCHP is readily biodegradable in soil with an aerobic half-life of 8.1 to 16.8 days in shallow, moist soils (NCBI, 2020; EC/HC, 2015). In anaerobic conditions, DCHP may be slightly more persistent with an anoxic half-life of 26.4 days (Yuan et al., 2002). There is limited information available related to the uptake and bioavailability of DCHP in land applied soils. DCHP's solubility and sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms. Further, no studies were identified evaluating the bioaccumulation potential of DCHP. Based on the solubility (1.48 mg/L) and hydrophobicity (log K_{OW} = 4.82; log K_{OC} = 4.47), DCHP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms (U.S. EPA, 2025k). A bioaccumulation factor (BAF) and bioconcentration factor (BCF) were modeled using the BCFBAF™ model in EPI Suite™ with an estimated BCF of 708 and BAF of 67 (log BCF = 2.85; log BAF = 1.83) (U.S. EPA, 2017).

There are limited measured data on concentrations of DCHP in biosolids or soils receiving biosolids. However, the high-quality biodegradation rates and physical and chemical properties suggest that DCHP will have limited persistence potential and mobility in soils receiving biosolids.

[1] The 95th percentile may be calculated by the following equation, assuming normal distribution:
$95th\ percentile = mean + 1.96 \times SD$

3.1.1 Weight of Scientific Evidence Conclusions

There is considerable uncertainty in the applicability of using generic release scenarios and wastewater treatment plant modeling software to estimate concentrations of DCHP in biosolids. 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 DCHP in soils will not be mobile and will have low persistence potential. The limited available data for bioavailability suggests that soil dwelling organisms may be exposed in regions in which DCHP-containing biosolids was applied but is not expected to substantially bioaccumulate DCHP.

3.2 Landfills

For this assessment, landfills will be considered to be divided into two zones: (1) "upper-landfill" zone with normal environmental temperatures and pressures, where biotic processes are the predominant route of degradation for DCHP; and (2) "lower-landfill" zone where elevated temperatures and pressures exist, and abiotic degradation is the predominant route of degradation. In the upper-landfill zone where oxygen might still be present in the subsurface, conditions can 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 DCHP. 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).

DCHP 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 which reported the concentration of DCHP in landfills or in the surrounding land. There is limited information regarding DCHP in dewatered biosolids, which may be sent to landfills for disposal. No U.S. studies were identified which report DCHP concentration in wastewater biosolids or sludge. Several Chinese studies reported in Section 3.1 reported DCHP in Chinese wastewater plant biosolids. Since no data were available estimating the concentration of DCHP in biosolids, a conservative estimate of 0.71 mg/kg dw was calculated from the 95th percentile of the highest reported average concentration of DCHP in dewatered biosolids.

DCHP is slightly soluble in water (1.48 mg/L) and does have limited potential to leach from landfills into nearby groundwater or surface water systems. However, DCHP is expected to have a high affinity to particulate (log K_{OC} = 4.47) and organic media (log K_{OW} = 4.82), which would cause significant retardation in groundwater and limit leaching to groundwater (U.S. EPA, 2025k). Because of its high hydrophobicity and high affinity for soil sorption, it is unlikely that DCHP will migrate from landfills via groundwater infiltration or surface runoff. As such, EPA did not model DCHP leaching from landfills to groundwater or surface water systems.

Although persistence in landfills has not been directly measured, DCHP can undergo abiotic degradation via carboxylic acid ester hydrolysis to form monocyclohexyl phthalate and cyclohexanol (U.S. EPA, 2017). Hydrolysis is likely to be slow and is not considered a significant abiotic degradation pathway with a half-life of 11.66 years at a pH of 7 at 25 °C (U.S. EPA, 2017). In both the upper and lower landfill zones, DCHP is shielded from light and photolysis is not considered a significant abiotic degradation pathway. DCHP can degrade biologically in the upper landfill. In the lower landfill, high temperatures and low water content may partially or completely inhibit biological degradation. DCHP will readily degrade in aerobic, moist soils representative of upper landfills with aerobic half-life of 8.1 to 16.8 days (NCBI, 2020; EC/HC, 2015). DCHP is more persistent under anaerobic conditions such as

those that would exist in lower landfills with an anaerobic half-life reported at 26.4 days (Yuan et al., 2002).

There is limited information available related to the uptake and bioavailability of DCHP in soils. DCHP's solubility and sorption coefficients suggest that bioaccumulation and biomagnification will not be of significant concern for soil-dwelling organisms adjacent to landfills. Similarly, no studies were identified evaluating the bioaccumulation potential of DCHP. Based on solubility (1.48 mg/L) and hydrophobicity (log K_{OW} = 4.82; log K_{OC} = 4.47), DCHP is not expected to have potential for significant bioaccumulation, biomagnification, or bioconcentration in exposed organisms. BAF and BCF was modeled using the BCFBAF™ Model in EPI Suite™ with an estimated BCF of 708 and BAF of 67 (log BCF = 2.85 and log BAF = 1.83) (U.S. EPA, 2017).

3.2.1 Weight of Scientific Evidence Conclusions

EPA did not identify data describing, or evidence of DCHP leaching from landfills. Based on the biodegradation and hydrolysis data available for DCHP under conditions relevant to landfills, DCHP is unlikely to persist in landfills. Because of this—in combination with DCHP's low solubility and high affinity for particulate and organic media—EPA has robust confidence that DCHP is unlikely to be present in large quantities in landfill leachate and is therefore unlikely to migrate from landfills. Furthermore, the limited bioavailability data suggests that while soil dwelling organisms may be exposed in landfills, they are not expected to substantially bioaccumulate DCHP.

4 SURFACE WATER CONCENTRATION

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data to obtain concentrations of DCHP in ambient surface water and aquatic sediments. Although the available monitoring data were limited, DCHP has been detected in surface water and in aquatic sediments. However, EPA cannot correlate monitoring levels with any releases associated with DCHP TSCA COUs. That is, the Agency does not have any facility-specific DCHP release data since facilities do not report releases of DCHP to surface waters from TSCA COUs to EPA programs. Therefore, EPA estimated the releases to surface water as described in *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g). Using these release estimates, EPA conducted modeling of surface water 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.

Federal effluent limitation guidelines (ELGs) regulate the maximum allowable levels of concentrations achievable with treatment for certain chemicals across various industry sectors and processes. While ELGs have been established for diethylhexyl phthalate (DEHP), butyl benzyl phthalate (BBP), and dibutyl phthalate (DBP) for some processes, no chemical-specific ELGs have been established for DCHP. Similarly, EPA has established ambient water quality criteria (AWQC) for DEHP, BBP, and DBP, which inform limits that states and authorized Tribes set for point source discharges regulated under the National Pollutant Discharge Elimination System (NPDES) to protect the designated uses of waters under the Clean Water Act. EPA has not established any AWQC for DCHP.

4.1 Modeling Approach for Estimating Concentrations in Surface Water

EPA conducted modeling using the EPA's Variable Volume Water Model (VVWM) in Point Source Calculator tool (PSC) (U.S. EPA, 2019c) to estimate surface water and sediment concentrations of DCHP. PSC inputs include physical and chemical properties of DCHP (*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and estimated DCHP releases to water (U.S. EPA, 2025g), which are used to predict receiving water column concentrations. PSC was also used to estimate DCHP 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 in environmental media. DCHP has a log K_{OC} of 4.5, indicating a high potential to sorb to suspended particles in the water column and to settled sediment in the benthic environment (U.S. EPA, 2017).

Physical and chemical, and fate properties selected by EPA for this assessment were applied as inputs to the PSC tool (see Table 4-1). Selected values are described in detail in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k). A half-life based on anaerobic sediment was selected for the benthic half-life input as a more protective value than the aerobic sediment value, and in consideration of the potential for lower levels of oxygen in benthic sediments impacted by industrial releases. In addition to the values described in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k), the PSC 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}	29,512 mL/g
Water Column Half-Life	16.8 days at 25 °C
Photolysis Half-Life	0.44 days at 30N
Hydrolysis Half-Life	4,270.5 days at 25 °C
Benthic Half-Life	26.4 days at 25 °C
Molecular Weight	330.43 g/mol
Vapor Pressure (torr)	0.000000869
Solubility	1.48 mg/L
Heat of Henry	45,727 J/mol
Reference Temp	25 °C
[a] Selected values for these parameters are described in *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k).	

A common setup for the model environment and media parameters was applied consistently across all PSC runs. The standard EPA "farm pond" water body characteristics were used to parameterize the water column and sediment parameters (Table 4-2). Standardized water body model cell geometry was also applied consistently across runs—with a standardized width of 5m, length of 40 m, and depth of 1 m—representing a small section of the receiving stream. 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" Water Body Characteristics for PSC Model Inputs

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

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 DCHP to water for all OESs because there were no reported data from available sources (*e.g.*, TRI and Discharge Monitoring Reports [DMR]) (U.S. EPA, 2025g). Without TRI and DMR data, EPA cannot identify the receiving water bodies and their location-specific hydrological flow data. The Agency 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 DCHP-releasing facility. Databases that were queried to develop the distribution include EPA's Enforcement and Compliance History Online (ECHO) that contains facilities with a NPDES permit, National Hydrography Dataset Plus (NHDPlus), and NHDPlus V2.1 Flowline Network Enhanced Runoff Method (EROM) Flow database. 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 (*i.e.*, the lowest 30-day average flow rate that occurs in a 5-year period) are used to estimate acute, incidental human exposure through swimming or recreational contact. The annual average flow represents long-term flow rates, but a harmonic mean provides a more conservative estimate and is preferred for assessing potential chronic human exposure via drinking water. The harmonic mean is also used for estimating human exposure through fish ingestion because it takes time for chemical concentrations to accumulate in fish. Lastly, for aquatic or ecological exposure, a 7Q10 flow (*i.e.*, the lowest 7-day average flow that occurs in a 10-year period) is used to estimate exceedances of concentrations of concerns for aquatic life (U.S. EPA, 2007). The regression equations for deriving the harmonic mean and 7Q10 flows are provided in Appendix B. Hydrologic flows in the receiving water bodies 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 water bodies.

Application of paints and coatings OES was chosen as an appropriate OES for a screening level assessment based on it resulting in a conservatively high surface water concentration based on high volumes of releases paired with an assumption of a low flow (P50) in the receiving water body, with environmental concentrations exceeding those estimated in all other OESs. Additionally, the generic release scenario for the Application of paints and coatings OES estimates a combined release to fugitive air, water, incineration, or landfill. Because the proportion of the release from Application of paints and coating 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 surface water) were incorporated into the screening analysis for reference (Table 4-3). EPA's process for selecting the Application of paints and coating 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 PVC plastics compounding OES was applied as

continuous days of release per year as a conservative approach (*e.g.*, a scenario with 250 days of release per year was modeled as 250 consecutive days of release, followed by 115 days of no release, per year). The highest water column concentration averaged over the number of release days (*i.e.*, 250) was used to estimate general population and aquatic exposure. Appendix B describes the methods to calculate the rolling averages.

Releases were evaluated for resulting environmental media concentrations at the point of discharge (*i.e.*, in the immediate receiving water body receiving the effluent). Due to uncertainty about the prevalence of wastewater treatment from DCHP-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 DCHP from the water column prior to discharge. Modeling results are shown in Table 4-3. This analysis resulted in high estimated concentrations in the receiving water body 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.

Notably, many of the surface water concentrations estimated exceed the water solubility range reported and detailed in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L. Phthalate esters commonly exhibit a tendency to form colloidal suspensions in aquatic environments due to their hydrophobic nature and slight water solubility. Many phthalate esters exhibit large, nonpolar alkyl chains that resist dissolution in water, promoting aggregation into small droplets or particles. These colloidal formations can remain suspended in water, allowing phthalates to be present at concentrations exceeding their nominal solubility limits. Colloidal suspensions of phthalate esters are more likely to occur in environments with high levels of organic matter or suspended particulate matter, which can adsorb phthalates and stabilize these suspensions. Colloidal suspensions are also more likely to occur with the higher molecular weight, larger chain length, and lower solubility phthalates such as DCHP.

These high-end values are carried through to the ecological risk assessment for further evaluation as a conservative high-end approach to screen for ecological risk discussed in Section 5 of the *Risk Evaluation for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025l) and in the *Environmental Hazard Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025f)

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

OES	Number of Operating Days Per Year[a]	Daily Release (kg/day) [a]	Median 7Q10 Total Water Column Concentration (µg/L)	Median 7Q10 Benthic Pore Water Concentration (µg/L)	Median 7Q10 Benthic Sediment Concentration (µg/kg)
Application of paints and coatings *Without wastewater treatment* (P50 flow rate with high-end release)	287	41.81	33,700 [b]	19,900 [b]	23,500,000
PVC plastics compounding *Without wastewater treatment* (P50 flow rate with	254	5.20	218	126	148,000

OES	Number of Operating Days Per Year[a]	Daily Release (kg/day) [a]	Median 7Q10 Total Water Column Concentration (µg/L)	Median 7Q10 Benthic Pore Water Concentration (µg/L)	Median 7Q10 Benthic Sediment Concentration (µg/kg)
high-end release)					

[a] Details on operating days and daily releases are provided in *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g).
[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L

The OES with the highest total water column concentrations (Application of paints and coatings 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 DCHP concentrations (as in the modeling shown previously in this section), and another with a wastewater treatment removal efficiency of 68.6 percent applied, substantially reducing the modeled concentrations in the receiving water body. The DCHP surface water concentration after application of the removal efficiency is more likely to represent human exposure to DCHP in drinking water, where additional dilution and 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]	Median Harmonic Mean Flow (m^3/d)	Median 30Q5 Flow (m^3/d)	Removal Efficiency Applied (%)	Harmonic Mean Concentration (µg/L)	30Q5 Concentration (µg/L)
Application of Paints and Coating *Without wastewater treatment* (P50 flow rate with high-end release)	41.8	2,033	3,530	0.00	11600 [b]	19990 [b]
Application of Paints and Coating *With wastewater treatment* (P50 flow rate with high-end release)	41.8	2,033	3,530	71.2	3341 [b]	5757 [b]
PVC plastics compounding *Without Wastewater Treatment* (P50 flow rate with high-end release)	6.13	22,882	13,753	0.00	70.5	124

[a] Details on modeling release estimates are provided in *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g).
[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L

4.2 Measured Concentrations

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. Measured concentrations of DCHP in surface water and sediment are presented in Section 4.2.1 and 4.2.2, respectively.

4.2.1 Measured Concentrations in Surface Water

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 and Canada that examined DCHP in surface water (WA DOE, 2022; Keil et al., 2011) (Table 4-5). In 2021, the Washington State Department of Ecology conducted a statewide survey of phthalate concentrations in surface waters and sediments of eight rivers and eight lakes across Washington state, and in marine water sediments. In general, near-surface water column samples (≈1 m below the water surface) and lower-surface water column samples (1 m above the sediment surface) were collected from each water body in the spring and fall of 2021, with a few exceptions associated with poor weather, shallow conditions, and high river flow rates. No samples reported DCHP above detection limits.

One study conducted in the United States and Canada reported concentrations of DCHP in surface water (Keil et al., 2011) (Table 4-5). Marine waters from 66 sampling locations were collected from Puget Sound, Washington, a highly urbanized watershed with more than three million residents. Twenty-two marine water samples were collected from Barkley Sound, British Columbia, Canada, a watershed with less human influence and a lower population density. The marine waters were analyzed for 37 compounds commonly found in homes, 3 of which were phthalates (DEHP, DBP, and DCHP). As illustrated in Figure 2 of that study, DCHP was detected a higher fraction of the time in Barkley Sound (≈50% of the time) vs. Puget Sound (≈10% of the time). Based on Figure 3 of that study, DCHP concentrations in Barkley Sound were detected at a wider range of concentrations (mean: approximately 2 ng/L; max: approximately 14 ng/L) compared with Puget Sound (approximately <1–3 ng/L).

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

Reference	Sampling Location	DCHP Concentration	Sampling Notes	Study Quality Rating
WA DOE (2022)	Washington, United States	ND (<0.5 µg/L)	Freshwater samples from 16 lakes and rivers across Washington and marine samples from the Puget Sound	NA
Keil et al. (2011)	Puget Sound, Washington, United States Barkley Sound, British Columbia, Canada	Barkley Sound FOD: ≈50% Mean (range) of detections: ≈2 (ND–14) ng/L Puget Sound FOD: ≈10% ND to 3 ng/L *Detection limits NR*	Marine waters at 66 samples in Puget Sound, Washington, and 22 samples in Barkley Sound, British Columbia, Canadas (2010)	Medium
FOD = frequency of detection; ND = not detected; NR = not reported				

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. Two studies were identified from the United States and Canada that examined DCHP in sediment (WA DOE, 2022; Lin et al., 2003) (Table 4-6). During the Washington State Department of Ecology survey, 27 freshwater sediment samples were collected in the spring and fall of 2021, and 31 marine water sediment samples (21 from Puget Sound and 10 from Elliott Bay) were sampled in the spring of 2021. Overall, very few detections of phthalates were found in freshwater sediment samples, and DCHP was not found in any of the freshwater sediment samples. Seven of the 31 marine sediment samples contained one or more phthalates; 6 of these 7 samples were from Elliott Bay. DCHP was detected in one sample from Elliott Bay (marine sediment) near the downtown waterfront at 66.5 µg/kg dw.

No studies from Canada reported detectable concentrations of DCHP in sediment. Lin et al. (2003) described a new method for quantifying individual phthalate ester isomers and phthalate ester isomeric mixtures in sediments and fish. This new method as well as an established gas chromatography method were used to quantify concentrations of phthalate ester congeners in surficial sediments and striped seaperch in False Creek, Vancouver, British Columbia, Canada, an urbanized marine inlet. However, of 18 individual phthalate ester congeners targeted, only eight were detected (DMP, DEP, DiBP, DnBP, BBP, DEHP, DnOP, and DNP).

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

Reference	Sampling Location	DCHP Concentration	Sampling Notes	Study Quality Rating
WA DOE (2022)	United States	Freshwater: ND (dw) µg/kg Marine: FOD: 1/31 Range: ND–66.5 (dw) µg/kg *Detection limits varied across sites*	27 freshwater sediment samples from lakes and rivers across WA, 2021 31 marine sediment samples from Puget Sound and Elliott Bay, WA, 2021	NA
Lin et al. (2003)	Canada	ND *Detection limits NR*	16 surficial sediments from False Creek, Vancouver, BC, date NR	High

dw = dry weight; FOD = frequency of detection; ND = not detected; NA = not assessed; NR = not reported

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 DCHP in surface water and sediment using estimated release amounts and estimated receiving water body flow rates from a distribution of known releasing facilities. PSC considers model inputs of physical and chemical properties of DCHP

(*i.e.*, K_{OW}, K_{OC}, water column half-life, photolysis half-life, hydrolysis half-life, and benthic half-life) and allows EPA to model predicted sediment concentrations in addition to water column concentrations. The use of physical and chemical properties of DCHP refined through the systematic review process and supplemented by EPA models increases confidence in the application of the PSC tool. A standard EPA water body was used to represent a consistent and conservative receiving water body scenario. Uncertainty associated with location-specific model inputs (*e.g.*, flow parameters and meteorological data) is present as no facility locations were identified for DCHP releases and modeled values for DCHP release to surface water were used. EPA has moderate confidence in the estimated releases from facilities to surface water that 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 DCHP to surface water, while the reported measured concentrations represent sampled ambient water concentrations of DCHP. Because the release of DCHP 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 spatially or temporally associated with releases of DCHP. In addition, when modeling with PSC, EPA considered the generic scenario releases directly discharged to surface waters both with and without prior treatment, applying a generic removal efficiency. EPA recognizes that the untreated scenario is a conservative assumption that results in no removal of DCHP prior to release to surface water.

Concentrations of DCHP within the sediment were estimated using the high-end release estimates from generic scenarios and estimates of 7Q10 hydrologic flow data for the receiving water body that were derived from NHD modeled EROM flow data. The 7Q10 flow represents the lowest 7-day flow in a 10-year period and is a conservative approach for examining a condition where a potential contaminant may be predicted to be elevated due to periodic low flow conditions. Surrogate flow data collected via ECHO API and the NHDPlus V2.1 EROM flow database include self-reported hydrologic reach codes on NPDES permits and the best available flow estimations from the EROM flow data. The confidence in the flow values used, with respect to the universe of facilities for which data were pulled, should be considered moderate-to-robust. However, there is uncertainty in how representative the median flow rates are as applied to the facilities and COUs represented in the DCHP release modeling. Additionally, a regression-based calculation was applied to estimate flow statistics from NHD-acquired flow data, which introduces some additional uncertainty. EPA assumes that the results presented in this section include a bias toward overestimation of resulting environmental concentrations due to conservative assumptions considering the uncertainties.

4.4 Weight of Scientific Evidence Conclusions

Modeled inputs were derived from reasonably available literature collected and evaluated through EPA's systematic review process for this TSCA risk evaluation. All monitoring and experimental data included in this analysis were from articles rated "medium" or "high" quality from this process. Monitoring data demonstrates that DCHP can be detected in various types of water and sediment around the country. Although monitoring data are limited and may not specifically target peak concentrations in the environment resulting from facility effluent, environmental monitoring data show generally low concentrations within the water column.

Due to the lack of reported release data for facilities discharging DCHP to surface waters, releases were modeled, and the high-end estimate for each COU was applied for surface water modeling. EPA had slight to moderate confidence in modeled releases for OES that did not have reported releases as

described in the *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g). To estimate surface water concentration, modeled releases were paired with a distribution of generic flows that best represented the OES assessed (Table 4-3). Although a specific flow value could not be selected based on reasonably available data, EPA has slight to moderate confidence that when using the flow distribution, the surface water concentrations estimated represent possible environmental concentrations.

For OESs that had modeled releases that were not specific to water, EPA assumed that all of the release would be directly discharged to surface water, to represent an upper bound of surface water concentrations. The Agency has slight confidence in the estimated value of the surface water concentrations when making such an assumption. However, using a conservative assumption of releases all going to water alongside the assumptions of a low flow receiving water body and no wastewater treatment, EPA has robust confidence that the surface water concentrations estimated are appropriate for use in a screening evaluation. The high-end modeled concentrations, based on modeled releases, for surface water and sediment exceeded the highest values available from monitoring studies by one to two orders of magnitude, with some values exceeding water solubility. This supports EPA's approach in conducting a screening evaluation using the highest modeled DBP concentrations.

The high-end modeled concentrations in the surface water and sediment exceeded the highest values available from monitoring studies by more than three orders of magnitude. Additionally, surface water concentrations estimated using P50 flow exceeded the high-end of the range of the water solubility (1.48 mg/L) reported in *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2025k)for the Application of paints and coatings. Surface water concentrations estimated using P50 flow for the PVC plastics compound OES exceeded the low-end range of the water solubility (0.03 mg/L). This confirms EPA's expectation that modeled concentrations presented here are biased toward overestimation.

Overall, EPA has robust confidence that the high-end estimated surface water concentration modeled using the Application of paints and coating 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.

5 SURFACE WATER EXPOSURE TO GENERAL POPULATION

Concentrations of DCHP 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, EPA used two surface water concentrations in its assessment: (1) modeled concentrations from the Application of paints and coatings OES, and (2) modeled concentrations from the PVC plastics compounding OES as estimated in Section 4.1 (Table 4-4). For the modeled concentrations, Application of paints and coatings was the highest among OESs that discharge to multiple media type and PVC plastics compounding was the highest among OESs discharging to water only. For both OESs, the concentrations correspond highest modeled 95th percentile release. EPA also estimated exposure using the highest monitored surface water concentrations for comparison.

5.1 Modeling Approach

5.1.1 Dermal Exposures

The general population may swim in surface waters (streams and lakes) that are affected by DCHP 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 used for these exposure estimates are provided in Appendix A.1. EPA used the dermal permeability coefficient (K_p) of 0.012 cm/h (U.S. EPA, 2025b). EPA used the Consumer Exposure Model (CEM), Version 3.2 (U.S. EPA and ICF Consulting, 2022) to estimate the steady-state aqueous permeability coefficient of DCHP.

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 Application of paints and coatings and PVC Plastics compounding OESs (Table 4-5) as the surface water concentration. In addition to these modeled concentrations, the monitored concentrations from NWQMC (2021) were included for comparison. The monitored water column concentration are roughly four to six orders of magnitude less than the high-end modeled counterparts. Doses calculated using the surface water monitoring data are four to six orders of magnitude lower than corresponding doses modeled using the high-end Application of paints and coatings and PVC Plastics compounding OESs.

Table 5-1. Dermal (Swimming) Doses Across Lifestages[2]

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)
Application of Paints and Coating *Without wastewater treatment* (P50 flow rate)	19,990 [b]	11,600 [b]	1.75E−01	2.79E−04	1.34E−01	2.14E−04	8.15E−02	1.30E−04
PVC Plastics Compounding *With wastewater treatment* (P50 flow rate)	204	197	1.79E−03	4.74E−06	1.37E−03	3.63E−06	8.31E−04	2.20E−06
Highest Monitored Surface Water [a]	0.014	0.014	1.2E–07	3.4E–10	9.4E–08	2.6E–10	5.7E–08	1.6E–10

30Q5 = lowest 30-day average flow in a 5-year period; POT = potential
[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.
[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L

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

5.1.2 Oral Ingestion Exposures

The general population may swim in surfaces waters (streams and lakes) that are affected by DCHP 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 Application of paints and coatings 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)

Equation 5-4. Average Daily Incidental Calculation

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

Where:

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

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

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

Scenario	Water Column Concentrations		Adult (21+ years)		Youth (11–15 years)		Child (6–10 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Application of Paints and Coating *Without Wastewater Treatment*	19,990 [b]	11,600 [b]	6.90E−02	1.10E−04	1.07E−01	1.70E−04	6.03E−02	9.59E−05
PVC plastics compounding[a] *Without Wastewater Treatment*	204	197	7.04E−04	1.86E−06	1.09E−03	2.89E−06	6.16E−04	1.63E−06
Highest monitored surface water[a]	0.014	0.014	4.8E–08	1.3E–10	7.5E–08	2.1E–10	4.2E–08	1.2E–10

30Q5 = lowest 30-day average flow in a 5-year period; POT = potential
[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.
[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L

5.2 Weight of Scientific Evidence Conclusions

No facility- or site-specific information was reasonably available when estimating release of DCHP to the environment. Environmental releases to water were estimated using generic scenarios (U.S. EPA, 2025g). 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. For OES that did not have facility release data, EPA modeled releases. The Agency has slight to moderate confidence for modeled releases and slight to moderate confidence in the estimated surface water concentrations based on the modeled releases that were modeled only to water. For the modeled releases that were not specific to water, EPA only has slight confidence in the estimated surface water concentration because the proportion of the release from just surface water could not be determined from reasonably available information. However, the high-end of those resulting concentrations and exposure estimates are presented in this document and EPA has robust confidence that the estimates presented are appropriate for screening based on the conservative assumptions including low flow scenarios, no wastewater treatment assumptions, and swimming directly at the point of discharge immediately following discharge. Monitoring studies reporting highest monitored surface water concentrations of 0.014 µg/L support EPA's conclusion that the modeled surface water concentrations are appropriately conservative.

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 for reasonable exposure scenarios. The screening approach applied for modeling, in conjunction with the available monitoring data showing lower concentrations than those modeled, provide robust confidence that releases to surface water will not exceed the release concentrations presented in this assessment, which do not appear to pose risk to human health.

Swimming Ingestion/Dermal Estimates

Two scenarios (youth being exposed dermally and through incidental ingestion while swimming in

surface water) were assessed as high-end potential exposures to DCHP in surface waters. EPA's *Exposure Factors Handbook* provided detailed information on the youth skin surface areas and event per day of the various scenarios (U.S. EPA, 2021a). Non-diluted surface water concentrations were used when estimating dermal exposures to youth swimming in streams and lakes. DCHP concentrations will dilute when released to surface waters, but it is unclear what level of dilution will occur when the general population swims in waters with DCHP releases.

6 DRINKING WATER EXPOSURE TO GENERAL POPULATION

Drinking water in the United States typically comes from surface water (*i.e.*, lakes, rivers, and reservoirs) and groundwater. The source water then flows to a treatment plant where it undergoes a series of water treatment steps before being dispersed to homes and communities. The National Primary Drinking Water Regulations under the Safe Drinking Water Act identify maximum contaminant levels (MCLs) or treatment techniques generally on a contaminant-by-contaminant basis. Currently, an MCL has not been developed for DCHP. As described in 3.2, because of its high hydrophobicity and high affinity for soil sorption, it is unlikely that DCHP will migrate from landfills via groundwater infiltration. Therefore, drinking water exposure in this assessment is focused on drinking water sourced from surface water.

Very limited information is available on the removal of DCHP in drinking water treatment plants, as stated in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2025k). Based on the low water solubility and log K_{OW}, DCHP 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 DCHP 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 DCHP via drinking water. No monitoring data were identified by the EPA that measured DCHP in drinking water in the United States.

EPA used two surface water concentrations in its assessment: (1) modeled concentrations from the Application of paints and coatings OES, and (2) modeled concentrations from the PVC plastics compounding OES. For the modeled concentrations, Application of paints and coatings was the highest among OESs that discharge to multiple media type and PVC plastics compounding was the highest among OESs discharging to water only. For both OESs, the concentrations correspond highest modeled 95th percentile release. EPA also estimated exposure using the highest monitored surface water concentrations for comparison.

6.1 Modeling Approach for Estimating DCHP General Population Exposures from 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 analysis, EPA assessed the highest modeled surface water concentration using facility releases from Application of paints and coatings OES and PVC plastics compounding. , and 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 water body. When applied, a wastewater treatment efficiency of 71.2 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, lifetime average daily dose [LADD], lifetime average daily concentration [LADC])
DWT	=	Removal during drinking water treatment (assume 0% for DCHP)
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:

$ADRPOT$	=	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 DCHP)
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)

The ADR and ADD from drinking water 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, infants, 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. Drinking Water Doses Across Lifestages

Scenario	Surface Water Concentrations		Adult (21+ years)		Infant (Birth to < 1 year)		Toddler (1–5 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
PVC Plastics Compounding *Without Wastewater Treatment*	204	197	8.21E−03	1.51E−03	2.88E−02	3.85E−03	1.02E−02	1.65E−03
Application of Paints and Coating *Without Wastewater Treatment*	19,990 [b]	11,600 [b]	8.04E−01	1.00E−01	2.82E00	2.56E−01	1.00E00	1.10E−01
Application of Paints and Coating *with Wastewater Treatment*	5,757 [b]	3,341 [b]	2.32E−01	2.89E−02	8.13E−01	7.38E−02	2.89E−01	3.16E−02
Highest Monitored Surface Water [a]	0.014	0.014	5.6E–07	1.21E−07	2.0E–06	3.09E−07	7.0E–07	1.33E−07

30Q5 = lowest 30-day average flow in a 5-year period; POT = potential
[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.
[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L.

6.2 Evidence Integration for Drinking Water

Based on modeling of the estimated releases, EPA estimates little to no potential exposure to DCHP via drinking water—even under conservative high-end release scenarios. These exposure estimates also assume that the drinking water intake location is very close (within a few km) to the point of discharge and do not incorporate any dilution beyond the point of discharge. Actual concentrations in raw and finished water are likely to be lower than these conservative estimates as applying dilution factors will decrease the exposure for all scenarios, and traveling additional distances downstream would allow further partitioning and degradation. Monitoring data also present evidence for generally low concentrations in ambient waters beyond direct points of release. Screening level risk estimates derived from the exposures discussed in this section are presented in Appendix C and suggest no expected risk below the benchmark MOE at the upper bound of exposure for reasonable scenarios.

6.3 Weight of Scientific Evidence Conclusions

EPA has moderate to robust confidence in the surface water as drinking water exposure scenario due to the site-specific uncertainty presented in this section, as well as robust evidence of presenting an upper bound of exposure showing screening level risk estimates above the benchmarks described in Section

3.2, EPA did not assess drinking water estimates as a result of leaching from landfills to groundwater and subsequent migration to drinking water wells.

7 FISH INGESTION EXPOSURE TO GENERAL POPULATION

To estimate exposure to humans from fish ingestion, EPA used multiple surface water concentrations in its assessment: (1) the upper and lower limit of the water solubility, (2) modeled concentrations from the Plastics compounding OES at various flow rates, and (3) modeled concentrations from Use of paints and coatings OES. EPA selected 0.03 to 1.48 mg/L for the applicable range of DCHP's solubility in water, where the minimum value is an observed empirical value and the maximum is modeled by EPI Suite™ (see *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k)). The true solubility of DCHP may be lower than 1.48 mg/L, with concentrations in the environment being lower based on environmental monitoring data. For the modeled concentrations, Application of paints and coatings was the highest among OESs that discharge to multiple media type, and PVC plastics compounding was the highest among OESs discharging to water only. For both OESs, the concentrations correspond to the harmonic mean based on the highest modeled 95th percentile release (unless noted otherwise) without consideration of wastewater treatment.

Another important parameter in estimating human exposure to a chemical through fish ingestion is the BAF. BAF is preferred over the BCF because it considers the animal's uptake of a chemical from both diet and the water column. For DCHP, a BAF of 67 L/kg was estimated using the Arnot-Gobas method for upper trophic organisms (see *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k)). Table 7-1 compares the fish tissue concentration calculated using a BAF with the measured fish tissue concentrations obtained from literature. Calculated DCHP concentrations in fish tissue are up to three orders of magnitude higher than empirical levels reported within published literature.

In addition, EPA calculated fish tissue concentrations using the highest measured DCHP 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 3 ng/L (3.0×10^{-6} mg/L) from Puget Sound, Washington (Keil et al., 2011). Fish tissue concentrations calculated with the monitored surface water concentrations are six orders of magnitude lower than those reported within published literature (Table 7-1).

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

Data Description and Source	Surface Water Concentration	Fish Tissue Concentration
Water solubility limit	Upper bound 1.48 mg/L (U.S. EPA, 2025k; Howard et al., 1985)	1.08E02 mg/kg ww
	Lower bound 0.03 mg/L (U.S. EPA, 2025k; Howard et al., 1985)	
Modeled surface water concentrations	Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	1.32E01, 2.58, 2.0E–02 mg/kg ww for P50, P75, and P90 flow
	Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L	7.77E02, 1.22E2, 4.72 mg/kg ww for P50, P75, and P90 flow

Data Description and Source	Surface Water Concentration	Fish Tissue Concentration
	for P50, P75, P90 flow	
Highest measured concentration from a U.S. study (Keil et al., 2011)	3.00E–06 mg/L	2.01E–07 mg/kg ww
Fish tissue monitoring data (wild-caught) [a] One U.S. study collected 21 fish samples across 11 urban lakes and ponds (Lucas and Polidoro, 2019)	N/A	ND to 1.0E–01 mg/kg ww
Fish tissue monitoring data (wild-caught) [a] One Chinese study collected 207 fish samples across 17 different species (Hu et al., 2020)		<LOD to 2.91E–01 mg/kg
ND = non-detect; HE = high-end modeled 95th percentile releases; ww = wet weight [a] These studies identified through systematic review that reported measured DCHP 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 *Data Extraction Information for General Population, Consumer, and Environmental Exposure for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025d) (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 kg of body weight for the general population, as shown in Table_Apx A-2. A young toddler between 1 and 2 years old has the highest 90th percentile fish IR per kilogram of body weight. This section estimates exposure and risks for these two lifestages with the highest fish IR per kilogram of body weight as a screening level approach.

The ADR and ADD for non-cancer exposure estimates were calculated using the 90th percentile and central tendency IR, respectively. Cancer exposure (LADD) and risks were not characterized because there is insufficient evidence of DCHP's carcinogenicity (U.S. EPA, 2025j). Estimated exposure to DCHP from fish ingestion were calculated with 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 ww)
IR	=	Fish ingestion rate (g/kg-day)
$CF1$	=	Conversion factor for mg/µg (0.001 mg/µg)
$CF2$	=	Conversion factor for kg/g (0.001 kg/g)
ED	=	Exposure duration (year)

AT = Averaging time (year)

The inputs to this equation can be found in the *Fish Ingestion Risk Calculator for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025h). 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 limits and modeled data are presented in Table 7-2. Corresponding screening level risk estimates are shown in Appendix E.1.

Risk estimates (Appendix E.1) are above the benchmark of 30 at the upper- and lower-bound of the water solubility limit and modeled surface water concentrations for the Plastics compounding OES. For the OESs with water-specific releases, the fish ingestion pathway is not expected to a concern for the general population.

For Application of paints and coatings, acute non-cancer MOEs are 7 or 11 depending on the lifestage at the P50 flow rate. The benchmark is 30. MOEs are above benchmark of 30 at the P75 and P90 flow rate. However, EPA has only slight confidence in these results. The modeled concentrations at P50 flow rate exceed the upper limit of water solubility by one order of magnitude and the lower limit by three orders of magnitude. The generic scenario used to estimate the environmental releases for this OES are directed to a combination of fugitive air, stack air, incineration, landfill, or water (U.S. EPA, 2025l). Information is not available to determine what proportion of the total release, if any, is directed to water. In the screening level assessment, EPA assumed all is discharged to water. However, without further information, the Agency is unable to refine its analysis because of the resultant slight confidence and high uncertainty in assuming fraction may be released to water. All OESs discharging to multiple media types are therefore not further considered.

Table 7-2. General Population Fish Ingestion Doses

Surface Water Concentration	Adult ADR (mg/kg-day)	Young Toddler ADR (mg/kg-day)	Adult ADD (mg/kg-day)
Water solubility limit 1.48 and 0.03 mg/L for upper and lower bound	2.75E–02 (upper) 5.58E–04 (lower)	4.09E–02 (upper) 8.28E–04 (lower)	6.25E–03 (upper) 1.27E–04 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	3.66E–03 (P50 flow) 7.16E–04 (P75 flow) 6.41E–06 (P90 flow)	5.44E–03 (P50 flow) 1.06E–03 (P75 flow) 9.52E–06 (P90 flow)	8.32E–04 (P50 flow) 1.63E–04 (P75 flow) 1.46E–06 (P90 flow)
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	2.16E–01 (P50 flow) 3.38E–02 (P75 flow) 1.31E–03 (P90 flow)	3.20E–01 (P50 flow) 5.02E–02 (P75 flow) 1.95E–03 (P90 flow)	4.90E–02 (P50 flow) 7.68E–03 (P75 flow) 2.98E–04 (P90 flow)
ADD = average daily dose; ADR = acute dose rate; HE = high-end, 95th percentile release			

7.2 Subsistence Fish Ingestion Exposure

Subsistence fishers represent a potentially exposed or susceptible subpopulation(s) (PESS) group due to their greatly increased consumption of fish (average of 142.4 g/day compared to a 90th percentile of 22.2 g/day for the general population) (U.S. EPA, 2000). 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, using the same inputs as the general population with the exception of ingestion rate. EPA is unable to determine subsistence fishers' exposure estimates specific to younger lifestages based on lack of reasonably available information on fish ingestion rates for the younger lifestages. Furthermore, unlike the general population fish ingestion rates, there is no high-end (*e.g.*, 90th or 95th percentile) ingestion rate for subsistence fishers. The same value was used to estimate both the ADD and ADR.

Risk estimates (Appendix E.2) are above the benchmark of 30 at the lower bound of the water solubility limit and modeled surface water concentrations for Plastics compounding. This OES discharges to water-only. For all OESs with water-specific releases, the fish ingestion pathway is not expected to a concern for the subsistence fisher.

For Application of paints and coatings, acute and chronic non-cancer MOEs are 2 and 11 at the P50 and P75 flow rates, respectively. The benchmark is 30. MOEs are above benchmark of 30 at the P90 flow rate. However, EPA has only slight confidence in these results. The modeled concentrations at P50 and P75 flow rate exceed the upper limit of water solubility by up to one order of magnitude and the lower limit by up to three orders of magnitude. The generic scenario used to estimate the environmental releases for this OES are directed to a combination of fugitive air, stack air, incineration, landfill, or water (U.S. EPA, 2025l). Information is not available to determine what proportion of the total release, if any, is directed to water. In the screening level assessment, EPA assumed all is discharged to water. However, without further information, the Agency is unable to refine its analysis because of the resultant slight confidence and high uncertainty in assuming what fraction may be released to water. All OESs discharging to multiple media types are therefore not considered further.

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

Surface Water Concentration	ADR/ADD (mg/kg-day)
Water solubility limit 1.48 and 0.03 mg/L for upper and lower bound	1.77E–01 (upper) 3.58E–03 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	2.35E–02 (P50 flow) 4.59E–03 (P75 flow) 4.11E–05 (P90 flow)
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	1.38E00 (P50 flow) 2.17E–01 (P75 flow) 8.41E–03 (P90 flow)
ADD = average daily dose; ADR = acute dose rate; HE = high-end, 95th percentile release	

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 as well as 63 state recognized tribes. Tribal cultures are inextricably linked to their lands, which provide all their needs from hunting, fishing, food gathering, and grazing horses to commerce, art, education, health care, and social systems. These services flow among natural resources in continuous interlocking cycles, creating a multi-dimensional relationship with the natural environment and forming the basis of *Tamanwit* (natural law) (Harper et al., 2012). Such an intricate connection to the land and the distinctive lifeways and cultures between individual tribes create many unique exposure scenarios that can expose tribal members to higher doses

of contaminants in the environment. EPA used the reasonably available information to quantitatively evaluate the tribal fish ingestion pathway for DCHP but lacks reasonably available data to assess other unique exposure scenarios unique to tribal populations.

U.S. EPA (2011a) (Chapter 10, Table 10-6 of that document) 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. For the heritage rates, the corresponding screening level exposure and risk estimates are presented alongside other ingestion rates but not considered further in this assessment because no available information can substantiate if heritage rates reflect current consumption patterns. Similar to subsistence fishers, EPA used the same ingestion rate to estimate both the ADD and ADR. For current ingestion rates, U.S. EPA (2011a) provides values specific to younger lifestages, but adults still consume higher amounts of fish per kilogram of body weight. An exception is for the Squaxin Island Tribe in Washington that reported an ingestion rate of 2.9 g/kg-day for children under 5 years of age. That ingestion rate for children is nearly the same as the adult ingestion rate of 2.7 g/kg-day for the Suquamish Tribe. As a result, exposure estimates based on current ingestion rate focused on adults (Table 7-4).

Table 7-4 presents multiple exposure estimates for the tribal populations. Risk estimates (Table_Apx E-3) are above benchmark of 30 at the lower bound of the water solubility limit and modeled surface water concentrations for Plastics compounding, except for one scenario. At the P50 flow rate and 95th percentile ingestion rate, the MOE was 17. EPA does not consider the exposure and subsequent risk estimates to be realistic for that scenario. The scenario compounds multiple conservative assumptions, most notably the use of a high-end, 95th percentile release volume occuring to water bodies with low flow rates (*i.e.*, P50). It also exceeds the lower bound of the water solubility limit by an order of magnitude. At the central tendency release and same P50 flow for the receiving water body, estimated water concentrations (0.04 µg/L) are only slightly higher than the lower bound of the water solubility limit of 0.03 mg/L, and risk estimates are at 50 compared to a benchmark of 30. Moreover, DCHP has not been readily measured or monitored in aquatic organisms and has low potential for bioaccumulation, biomagnification, and uptake based on a BCF of 708 and a BAF of 67 L/kg (U.S. EPA, 2025f). For these reasons, EPA has only slight confidence in the risk estimates for the Plastics compounding OES at the P50 flow rate and 95th percentile ingestion rate for tribal populations. Plastics compounding, which resulted in the highest modeled surface water concentration among all OESs with water-specific releases, did not result in risk estimates below benchmark where confidence is above slight. EPA therefore concludes that fish ingestion is not expected to be a pathway of concern for tribal populations for all OESs with water-only releases.

For the Application of paints and coatings OES, acute and chronic non-cancer MOEs are 0 or 1 and 2 or 7 at the P50 and P75 flow rates, respectively. MOEs are above the benchmark at the P90 flow rate. However, EPA has only slight confidence in these results. The modeled concentrations at P50 and P75 flow rate exceed the upper bound of water solubility by up to one order of magnitude and the lower bound by up to three orders of magnitude. The generic scenario used to estimate the environmental releases for this OES are directed to a combination of fugitive air, stack air, incineration, landfill, or water (U.S. EPA, 2025l). Information is not available to determine what proportion of the total release, if any, is directed to water. In the screening level assessment, EPA assumed all is discharged to water. However, without further information, the Agency is unable to refine its analysis because of the resultant slight confidence and high uncertainty in assuming fraction may be released to water. All OESs discharging to multiple media types are therefore not further considered.

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 1.48 and 0.03 mg/L for upper and lower bound	2.68E–01 (upper) 5.43E–03 (lower)	1.08 (upper) 2.19E–02 (lower)	2.04 (upper) 4.14E–02 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	3.56E–02 (P50 flow) 6.96E–03 (P75 flow) 6.24E–05 (P90 flow)	1.44E–01 (P50 flow) 2.81E–02 (P75 flow) 2.52E–04 (P90 flow)	2.72E–01 (P50 flow) 5.31E–02 (P75 flow) 4.76E–04 (P90 flow)
Plastics compounding (generic scenario for water-only release, CT, without wastewater treatment) 4.10E01 for P50 flow	7.40E–03	2.99E–02	5.64E–02
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	2.10 (P50 flow) 3.29E–01 (P75 flow) 1.28E–02 (P90 flow)	8.47 (P50 flow) 1.33 (P75 flow) 5.15E–02 (P90 flow)	16.0 (P50 flow) 2.51 (P75 flow) 9.72E–02 (P90 flow)
ADD = average daily dose; ADR = acute dose rate; CT = central tendency; HE = high-end; IR = ingestion rate			

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. A conservative screening analysis using the water solubility limit and modeled concentrations for two OESs resulted in risk estimates below the benchmark in several scenarios. EPA has slight confidence in all of those scenarios with MOEs below benchmark. For the Plastics compounding OES, where environmental releases are estimated to water only, confidence is slight because of compounding conservatism, modeled surface water concentration exceeding the lower bound of the water solubility limit, and DCHP's low bioaccumulation and bioconcentration potential. At a central tendency release estimate and higher flow rates, risks are not expected. For the Application of paints and coatings OES, where environmental releases are estimated to multiple media types, confidence is slight because of unavailable information to proportion what fraction of the discharge is directed to water.

Lastly, it is important to emphasize that DCHP is expected to have low potential for bioaccumulation, biomagnification, and uptake by aquatic organisms because of its low water solubility and high hydrophobicity. This is supported by the estimated BCF and BAF values of 703 and 67 L/kg, respectively, which does not meet the criteria to be considered bioaccumulative (BCF/BAF > 1,000). DCHP in water is expected to partition to suspended organic material present in the water column and to not be persistent in surface water because of its rapid degradation. Furthermore, EPA did not find reasonably available data sources that report the aquatic bioconcentration, bioaccumulation, and trophic

transfer of DCHP through food webs.

As modeled surface water concentrations are biased toward overestimation and bioconcentration, bioaccumulation, and trophic transfer of DCHP are 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 ambient air exposure assessment for DCHP. The Agency's modeling estimates both short- and long-term concentrations in ambient air. EPA considers monitoring data from published literature for additional insight into ambient air concentrations of DCHP.

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 DCHP in the ambient air at three distances (*e.g.,* 100; 100–1,000, and 1,000 m) from the releasing facility. IIOAC also estimates dry, wet, and total deposition rates of DCHP 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 DCHP 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 DCHP as described in the *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g).

The maximum EPA estimated daily release value for DCHP was 46.8 kg/site−day and categorized under the Use of paints and coatings – without engineering controls OES with an unknown media of release (*i.e.,* could comprise releases to air, land, water, or incineration, or any combination and could be either fugitive, stack, or any combination). Because 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 to both at the same time, which may overestimate ambient concentrations of DCHP.

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 m, 100–1,000 m, and 1,000 m
- Meteorological Stations:
    - South (Coastal): Surface and Upper Air Stations at Lake Charles, Louisiana
- Operating Scenario: 365 and 287 days per year; 24 hours/day
- Topography: Urban and Rural
- Particle Size:
    - Coarse (PM_{10}): Particulate matter with an aerodynamic diameter of 10 microns
    - Fine ($PM_{2.5}$): Particulate matter with an aerodynamic diameter of 2.5 microns

EPA used default 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 Default 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 (°)	0
Release height (m)	3.05

8.1.2 IIOAC Model Output Values

The IIOAC model provides multiple output values (see *Ambient Air Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (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 m distance from a releasing facility.
- **High-end, daily-average:** represents the 95th percentile daily average of all modeled hourly concentrations across the entire distribution of modeled concentrations at 100 m.
- **High-end, annual-average:** 95th percentile annual-average concentration across the entire distribution of modeled concentrations at 100 m.
- **High-end, total annual-average deposition**: 95th percentile annual-average total deposition rate across the entire distribution of modeled total deposition rates at 100 m.

8.1.3 Modeled Results from IIOAC

All results for each scenario described in Section 8.1.1 are included in the *Ambient Air Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (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 provides source apportioned concentrations and deposition rates (fugitive and stack) based on the respective releases. To evaluate exposures and total deposition rates for this ambient air assessment, EPA assumes the fugitive and stack releases occur simultaneously throughout the day and year. Therefore, the total concentration and deposition rate used to evaluate exposures and derive risk estimates in this ambient air assessment is the sum of the separately modeled fugitive and stack concentrations and total deposition rates at 100 m from a releasing facility. The source apportioned concentrations and the total concentrations for the scenario used are provided in Table 8-2.

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

Source Type	Daily-Average Concentration ($\mu g/m^3$)	Annual-Average Concentration ($\mu g/m^3$)
Fugitive	86.24	83.87
Stack	4.00	3.42
Total	90.25	87.29

The source apportioned wet and dry deposition rates and the total deposition rates for the scenario used in the *Environmental Hazard Assessment for Dicyclohexyl Phthalate (DCHP)* (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 m from Releasing Facility

Source Type	Total Annual Deposition Rate (g/m^2)		
	Total	Wet	Dry
Fugitive	1.41E−03	1.39E−03	2.02E−05
Stack	2.74E−04	2.64E−04	1.63E−05
Total	1.68E−03	1.66E−03	3.66E−05

8.2 Measured Concentrations in Ambient Air

EPA reviewed published literature as part of its Systematic Review process, as described in the *Systematic Review Protocol for Dicyclohexyl Phthalate* (U.S. EPA, 2025n) to identify studies where ambient air concentrations of DCHP 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 three foreign studies which measured concentrations of DCHP and no U.S. studies. The three foreign studies (Spain, South Korea, and China) are summarized in Appendix F. Two studies (Spain and China) looked at ambient air while the remaining study (South Korea) looked at water concentrations.

Measured concentrations of DCHP in these studies were low, generally in the ng/m^3 or pg/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 (AMTIC) archive but did not locate any monitored DCHP 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 DCHP ambient air concentrations were estimated using the maximum EPA estimated daily ambient air release, conservative meteorological data, and a distance of 100 m from a releasing facility. The modeled concentrations are higher than measured concentrations (Sections 8.1 and 8.2 respectively). Caution is needed when interpreting such a comparison; however, because modeled concentrations are near a releasing facility (100 m away), and it is unknown if the sampling sites are located at a similar distance from a site. Additionally, measured concentrations represent all sources (TSCA and other sources) contributing DCHP to the ambient air, while modeled concentrations are specific to TSCA sources.

8.3.1 Strengths, Limitations, and Sources of Uncertainty for Modeled Air 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.

DCHP did not have any reported releases in databases EPA typically relies upon for facility reported release data (*e.g.*, TRI or NEI). Therefore, DCHP releases were estimated and used as direct inputs to the IIOAC Model. Any limitations and uncertainties of these estimated releases, as described in the *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g), are carried over to this ambient air exposure assessment.

IIOAC 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 DCHP. This is in addition to the data gap EPA has on certain parameters, like building dimensions, stack heights, and release elevation, because such information has not been provided by industry to EPA for consideration. This absence creates additional limitations on using other models to their full potential. Furthermore, IIOAC does not consider the presence or location of residential areas relative to the 100 m distance from releasing facilities, the size of the facility, and the release point within a facility. For larger facilities, 100 m from a release point may still fall within the facility property where individuals within the general population are unlikely to live or frequent. In contrast, for smaller facilities, there may be individuals within the general population living 100 m away from the release point and therefore could be exposed continuously. However, most individuals may not stay within their residences 24 hours per day, 7 days per week throughout the year.

The use of estimated annual release data and number of operating days to calculate daily average releases assumes operations are continuous and releases are the same for each day of operation. This can underestimate short-term or daily exposure 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 46.8 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, because EPA

assumes the entire release is either fugitive or stack, modeled concentrations and deposition rates for fugitive and stack releases are not additive as they cannot happen at the same time. Nonetheless, EPA still provides a total exposure and deposition rate from both release types as if they occurred at the same time for this screening level assessment. This provides slight 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 robust confidence that exposure to and deposition rates of DCHP 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, the Agency would have slight 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). These estimated releases have limitations and uncertainties as described in the *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g), which carried over to this assessment.

8.4 Weight of Scientific Evidence Conclusions

EPA has slight confidence in the exposure scenario modeled for this assessment since emissions are assumed to be either all fugitive or all stack and are not additive (exposure to fugitive or stack releases cannot occur at the same time under the assumptions modeled) and EPA still adds results together as if they occur at the same time. The Agency has moderate confidence in the IIOAC-modeled results used to characterize exposures and deposition rates because 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 robust confidence the exposure estimates are health-protective. Based on the results presented here and risk estimates described in the *Risk Evaluation for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025l) EPA has robust confidence the ambient air pathway is not a pathway of concern for either exposure to or deposition rates of DCHP.

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 *Ambient Air Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025a).

9.2 Overall Conclusions

Based on the results from the analysis of the maximum estimated release and high-end exposure concentrations presented in this document and the *Non-Cancer Human Health Hazard Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025j), EPA does not expect an inhalation risk from ambient air nor ingestion risk from air to soil deposition to result from exposures to DCHP from industrial releases. Because no exposures of concern were identified at the maximum release scenario, EPA does not expect a different finding for smaller releases and therefore additional or more detailed analyses for exposure to DCHP through inhalation of ambient air or ingestion from air to soil deposition are not necessary.

10 HUMAN MILK EXPOSURE

Infants are a potentially susceptible subpopulation 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 DCHP is a developmental toxicant (U.S. EPA, 2025j). 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 DCHP from human milk ingestion. The Agency concluded that the most appropriate approach is to use human health hazard values that are based on gestational exposure as the subsequent sections will explain in more detail.

10.1 Biomonitoring Information

DCHP has the potential to accumulate in human milk because of its small mass (330.4 Daltons or g/mol) and lipophilicity (log K_{OW} = 4.82). EPA identified two biomonitoring studies from reasonably available information that investigated if DCHP or its metabolites were present in human milk. In a study that collected 30 samples from 30 German mothers, DCHP was detected in 8 of the samples with a maximum concentration of 4.6 ng/g (Zimmermann et al., 2012). Another German study detected DCHP in 17 percent of its samples (n = 78). Of the samples with measured concentrations above the limit of detection (LOD = 4 ng/g), the maximum concentration is 9.1 ng/g (Fromme et al., 2011). Neither of the studies characterized the possibility of occupational exposure to DCHP. No U.S. biomonitoring studies were identified.

It is important to note that biomonitoring data does not distinguish between exposure routes or pathways and does not allow for source apportionment. 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.

10.2 Hazard Information

EPA considered developmental and reproductive toxicity studies of rats that evaluated the effects of oral exposures to DCHP 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, perinatal, or pre-pubertal oral exposures to DCHP (see *Non-Cancer Human Health Hazard Assessment for Dicyclohexyl Phthalate* (U.S. EPA, 2025j)). No studies have evaluated only lactational exposure from quantified levels of DCHP in milk. However, the hazard values are based on developmental and reproductive toxicity following maternal exposure during gestation and are therefore expected to protect nursing infants.

10.3 Modeling Information

EPA formed an interdisciplinary workgroup in 2021 to investigate if and how to evaluate risks from infant exposure to chemicals through ingestion of human milk. One of the workgroup's goals was to identify peer-reviewed models that can quantify chemical concentrations in human milk and are applicable to a range of chemical classes (*i.e.*, chemical agnostic models) and data availability scenarios to best support TSCA risk evaluations. The workgroup 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, no half-life data were identified for either DCHP or its primary monoester

metabolite, mono-cyclohexyl phthalate (MCHP). No additional secondary metabolites of DCHP were identified (U.S. EPA, 2025j). Without half-life data, the Kapraun Model cannot be used to quantify lactational transfer and exposure for TSCA COUs.

Instead, exposure estimates for workers, consumers, and the general population were compared against the hazard value based on developmental toxicity following maternal exposure during gestation.

10.4 Weight of Scientific Evidence Conclusions

The lack of studies evaluating lactational exposure to DCHP and the lack of sensitive and specific half-life data precluded EPA from modeling human milk concentrations by COU. However, the Agency has robust confidence that a qualitative evaluation of exposure due to DCHP in human milk is protective for a nursing infant because multigenerational studies were evaluated to derive the hazard values. The multigenerational studies observed the effects on offspring across at least three generations resulting from maternal exposure during lactation, gestation, and other exposure periods. The hazard values are thus expected to protect a nursing infant's greater susceptibility during this unique lifestage whether due to sensitivity or greater exposure per body weight.

11 URINARY BIOMONITORING

The use of human biomonitoring data is an important tool for determining total exposure to a chemical for real world populations. Reverse dosimetry using human biomonitoring data can provide an estimate of the total dose (or aggregate exposure) responsible for the measured biomarker. Source-specific contributions to intake doses are not able to be estimated using reverse dosimetry; therefore, these intake doses are not directly comparable to the calculated doses presented throughout this document associated with specific COUs. However, the total intake dose estimated from reverse dosimetry can provide context for the exposure estimates based on only TSCA COUs. This section discusses urinary biomonitoring data, which represent total exposure from all sources for different life stages.

11.1 DCHP Metabolite Concentrations in Urinary Biomonitoring Studies

Phthalates have elimination half-lives on the order of several hours and are quickly excreted from the body in urine and to some extent in feces (ATSDR, 2022; EC/HC, 2015). Therefore, the presence of phthalate metabolites in the Centers for Disease Control and Prevention's (CDC) National Health and Nutrition Examination Survey (NHANES) urinary biomonitoring data indicates recent phthalate exposure.

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 one metabolite of DCHP, MCHP, measured in the 1999 to 2010 NHANES cycles. Sampling details can be found in Appendix G. Urinary concentrations of DCHP metabolites were quantified for different life stages. The life stages assessed included women of reproductive age (16–49 years), adults (16+ years), adolescents (11 to <16 years), and children (6 to <11 years).

CDC stopped collecting urinary MCHP data after the 2009 to 2010 NHANES cycle, likely due to low detection rates and limited variability in the data. For example, in the 2009 to 2010 survey year (the last survey in which MCHP was monitored), MCHP was above the LOD in 4.3 percent of samples for all adults 16 years and older and 7.9 percent of samples for all children aged 3 to less than 16 years. Meaningful statistical analyses, including temporal trend analyses, could not be performed due to low variance in the urinary DCHP data.

Given the lack of recent urinary biomonitoring data for DCHP, EPA did not conduct reverse dosimetry to calculate daily intake values for DCHP.

11.2 Summary of DCHP Biomonitoring Studies

EPA reviewed DCHP studies identified in the systematic review process (U.S. EPA, 2025n) for this risk evaluation to determine if a source of nationally representative data beyond NHANES, and collected after 2010, was available for analysis. A total of 12 studies were identified as that evaluated urinary MCHP levels, 2 of which analyzed data from NHANES and were therefore excluded (see Table 11-1). The remaining 10 publications represented 8 different studies (2 publications each from the Plastics and Personal-care Products use in Pregnancy (P4) study and the Canadian Health Measures Survey [CHMS]). Although each of these eight studies used urinary biomonitoring data for MCHP, the frequency of detection of MCHP in the samples was very low (<30%) and not suitable for a nationally representative chemical risk assessment. Additionally, the study populations in these studies were outside the target populations for this risk evaluation as they were either too specific (*e.g.*, a cohort examining specific health concerns) or not measured in the United States.

Based on these findings, EPA has concluded that there is no additional suitable source of DCHP biomonitoring data fit for use in this risk evaluation.

Table 11-1. Summary of Urinary Biomonitoring Studies of DCHP Since 2010

Reference	Study Name	Sample Size	LOD/LOQ for MCHP in Urine (µg/L or ng/mL)	Percentage of Samples with Levels of MCHP above the LOD/LOQ in Urine	Study Quality Rating
Pollack et al. (2014)	Plastics and Personal-care Products use in Pregnancy (P4) study	473	0.2	5%	Medium
Arbuckle et al. (2016)	Endometriosis, Natural Fstory, Diagnosis, and Outcomes (ENDO) Study	80	0.2	0%	High
Fisher et al. (2015)		80	0.2	Not reported	Medium
Bae et al. (2015)	Longitudinal Investigation of Fertility and the Environment (LIFE) Study	95	0.2–1.0[a]	5%	Medium
Shapiro et al. (2015)	Maternal–Infant Research on Environmental Chemicals (MIREC) Study	1,152	Not reported	<25%	Medium
Haines et al. (2016)	Canadian Health Measures Survey (CHMS)	3,237	0.2[b]	13%[b]	N/A
		3,235	0.09[c]	28%[c]	N/A
Health Canada (2013)	N/A	40	0.98–1.57[a]	3%	N/A
Buckley et al. (2012)	Right From the Start (RFTS) study	50	0.28	2%	Medium
Philips et al. (2020)	Generation R Study	1,192	0.008–0.3[a]	19%	Medium

[a] Range for all study metabolites
[b] CHMS Cycle 1 (2007–2009), ages 6–49 years
[c] CHMS Cycle 2 (2009–2011), ages 3–79 years

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 related to the biomonitoring of DCHP and collected in accordance with the *Systematic Review Protocol Supporting TSCA Risk Evaluations for Chemical Substances, Version 1.0: A Generic TSCA Systematic Review Protocol with Chemical-Specific Methodologies* (U.S. EPA, 2021b) and the *Systematic Review Protocol for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025n). 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 DCHP 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

Studies on DCHP concentration in aquatic species within the pool of reasonably available information were primarily coupled with larger investigations on multiple phthalate esters. Concentrations of DCHP within several different aquatic species originate from two previously published studies.

Lucas et al. (2019) reported DCHP concentrations of 0.11 µg/g wet weight (ww) in green sunfish (*Lepomis cyanellus*) tissue found in a recreational fishery in metro-Phoenix, Arizona. Twenty-one different species of fish were sampled from 11 sites within metro-Phoenix. Although phthalates were found in all the sampled fishes, only the green sunfish from one of the fisheries was found to have measured concentrations of DCHP. Green sunfish was noted to be a resident fish, which means that the measured concentrations can be safely assumed to be due to exposure within this recreational fishery (Lucas and Polidoro, 2019).

From marine animals collected near the coast of China, DCHP concentrations were detected in muscle tissues of crustaceans, molluscs, and fish (Hu et al., 2020). Eight different phthalates, including DCHP, were sampled from 28 different marine species. DCHP concentrations were detected in only seven species and ranged from the level of detection to 0.045 µg/g ww, with this highest amount sampled from the fish *Collichthyes niveatus*. DCHP was also found in crustaceans, with the gazami crab (*Portunus trituberculatus*) containing up to 0.017 µg/g ww DCHP. No DCHP was detected in marine molluscs in that study.

12.2 Trophic Transfer

EPA does not expect DCHP to persist in surface water, groundwater, or air (see Section 4.4 in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k). DCHP may persist in sediment, soil, biosolids, or landfills after release to these environments, but its bioavailability is expected to be limited (U.S. EPA, 2025k). Additionally, based on uncertainty around the range of estimated values for the limit of water solubility (ranging from 0.041–1.48 mg/L) and high hydrophobicity (log K_{OW} = 4.82; log K_{OC} = 4.47), DCHP is expected to have low bioaccumulation potential, low biomagnification potential, and low potential for physiological uptake (U.S. EPA, 2025k). The BCFs and BAFs of most phthalate esters, including DCHP, are below the Canadian Environmental Protection Act bioaccumulation criterion of 5,000 (Government of Canada, 2000). Specifically, results from the BCFBAF module in EPI Suite™ predicts a BCF of 708 and BAF of

67 for DCHP (U.S. EPA, 2017). The estimated BCF/BAF suggest that DCHP does not meet the criteria to be considered bioaccumulative, and bioaccumulation and bioconcentration in aquatic and terrestrial organisms are not expected to be important environmental processes for DCHP. Despite DCHP's relatively high octanol-water partition coefficient (log K_{OW} = 4.82), metabolic transformation after dietary uptake but before absorption (*i.e.*, pH enhanced hydrolysis in the gastrointestinal tract) and metabolic transformation after absorption may account for low overall bioaccumulation potential (Gobas et al., 2003). This conclusion is consistent with the observations made for other phthalates with measured BCF/BAFs such as DIDP, DINP, BBP and DEHP (Mackintosh et al., 2004). EPA also did not find reasonably available evidence that report the aquatic bioconcentration, aquatic bioaccumulation, aquatic food web magnification, terrestrial biota-sediment accumulation, or terrestrial bioconcentration of DCHP.

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

12.3 Weight of Scientific Evidence Conclusions

Given the reasonably available data, EPA has robust confidence that DCHP (1) is not readily found or if found is in relatively low concentrations in organism tissues, (2) has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms, and thus (3) low potential for trophic transfer through food webs.

The conclusion that DCHP is not readily detected in organism tissue is supported by the lack of studies reporting biomonitoring data and the low prevalence of DCHP data compared to other phthalates. This conclusion is weakened because only one of these studies was conducted in the United States. The conclusion that DCHP has low bioaccumulation and biomagnification potential is supported by the estimated BCF/BAF values, the relatively low concentrations detected in fish species, and the lack of reasonably available studies that report DCHP bioconcentration or biomagnification. This conclusion is weakened by the use of estimated/modeled values for BCF and BAF. Similar values from laboratory bioassays or field measurements would strengthen the EPA's confidence in these BCF/BAF estimates.

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

13.1 Environmental Exposure Conclusion

The EPA assessed environmental concentrations of DCHP in air, water, and land (soil, biosolids, and groundwater) for use in environmental exposure. DCHP will preferentially sorb into sediments, soils, particulate matter in air, and in wastewater solids during wastewater treatment. High-quality studies of DCHP biodegradation rates and physical and chemical properties indicate that DCHP will have limited persistence and mobility in soils receiving biosolids (U.S. EPA, 2025k).

Surface water, pore water, and sediment concentrations of DCHP were modeled using VVWM-PSC (Section 4.1). The PVC plastics compounding OES resulted in the highest estimated release to water, followed by recycling. DCHP concentrations in receiving waters were estimated for these COUs and ranged from 0.057 µg/L to 165 µg/L DCHP in the water column in low flow (7Q10) conditions. In one study, DCHP concentrations measured in the water column did not exceed 0.014 µg/L (Keil et al., 2011). Monitoring by the Washington State Department of Ecology resulted in no DCHP detection above the detection limit (0.05 µg/L) (WA DOE, 2022). No information is available on the potential continuous or persistent nature of DCHP in the water column of natural systems or from specific release sites.

For the land pathways, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered. No U.S. data were available reporting or estimating the DCHP concentrations in biosolids or biosolid-applied soils (Section 3.1). A conservative estimate of 0.71 mg/kg dw was calculated from the 95th percentile[3] of the highest reported average concentration of DCHP in biosolids (the mean and SD 0.31 ± 0.20 mg/kg dw reported by Wu et al. (2019)). DCHP is readily biodegradable in soil with an aerobic half-life of 8.1 to 16.8 days in shallow, moist soils (NCBI, 2020; EC/HC, 2015). Based on high-quality physical and chemical property data, EPA determined that DCHP will have low persistence potential and mobility in soils. Limited measured data were reasonably available from the scientific literature on DCHP concentrations in soils, biosolids, soils receiving biosolids, and landfills. EPA has robust confidence that DCHP is unlikely to be present in large quantities in landfill leachate and is therefore unlikely to migrate from landfills.

Limited reasonably available information was available related to the uptake and bioavailability of DCHP soils. Based on the range of estimates of water solubility (30–1,480 µg/L) and hydrophobicity (log K_{OW} = 4.82, log K_{OC} = 4.47), DCHP is expected to have low bioavailability in soil. DCHP has not readily measured or monitored in aquatic or terrestrial organisms and has low bioaccumulation and biomagnification potential. Therefore, DCHP has low potential for trophic transfer through food webs. DCHP is expected to have minimal air to soil deposition. Given the reasonably available data, EPA has robust confidence that DCHP (1) is not readily found, or if found, is in relatively low concentrations in organism tissues; and (2) has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms. Therefore, there is low potential for trophic transfer through food webs.

[3] The 95th percentile may be calculated by the following equation, assuming normal distribution:
$95\text{th percentile} = \text{mean} + 1.96 \times \text{SD}$

13.2 Weight of Scientific Evidence Conclusions for Environmental Exposure

The weight of scientific evidence supporting the exposure estimate is decided based on the strengths, limitations, and uncertainties associated with the exposure estimates, which are discussed in detail for biosolids (Section 3.1.1), landfills (Section 3.2.1), surface water (Section 4.4), ambient air (Section 8.4), and environmental biomonitoring and trophic transfer (Section 12). EPA summarized its weight of scientific evidence using confidence descriptors: robust, moderate, slight, or indeterminate. The Agency used general considerations (*i.e.*, relevance, data quality, representativeness, consistency, variability, uncertainties) as well as chemical-specific considerations for its weight of scientific evidence conclusions.

13.3 General Population Screening Conclusion

The general population can be exposed to DCHP 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 whereas exposures via the land pathway (biosolids and landfills) were qualitatively assessed. Using the high-end estimates of environmental media concentrations summarized in Table 13-1, general population exposures were estimated for the lifestage that would be most exposed based on intake rate and body weight.

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

OES[a]	Release Media	Environmental Media	DCHP Concentration
Application of paints and coatings *Without Wastewater Treatment*	Water	Surface water (30Q5, median flow)	19,990 µg/L
		Surface water (harmonic mean, median flow)	11,600 µg/L
Application of paints and coatings *With Wastewater Treatment*	Water	Surface water (30Q5, median flow)	6,277 µg/L
		Surface water (harmonic mean, median flow)	3,624 µg/L
Application of paint and coatings	Fugitive air	Daily-averaged total (fugitive and stack, 100m)	90.25 µg/m^3
		Annual-averaged total (fugitive and stack, 100m)	87.29 µg/m^3
[a] Table 1-1 provides the crosswalk of OESs to COUs			

Table 13-2 summarizes the conclusions for the exposure pathways and lifestages that were assessed for the general population. EPA conducted a quantitative evaluation for the following, incidental dermal exposure and incidental ingestion from swimming in surface water, drinking water ingestion, fish ingestion, and exposure from ambient air inhalation. Biosolids and landfills were assessed qualitatively in Sections 3.1 and 3.2, respectively. Results indicate that no pathways were of concern for DCHP for the highest exposed populations.

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

OES(s) [a]	Exposure Pathway	Exposure Route	Exposure Scenario (Section)	Lifestage	Major Pathway[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
Application of paints and coatings; PVC plastics compounding	Surface water	Dermal	Dermal exposure to DCHP in surface water during swimming (Section 5.1.1)	Adult (21+ years)	No
		Oral	Incidental ingestion of DCHP in surface water during swimming (Section 5.1.2)	Youth (11–15 years)	No
Application of paints and coatings; PVC plastics compounding	Drinking water	Oral	Ingestion of drinking water sourced from surface water (Section 6)	Infant (<1 year)	No
Plastics compounding; Application of paints and coatings	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	Adult (21+ years)	No
			Ingestion of fish for subsistence fishers (Section 7.2)	Adult (21+ years)	No
			Ingestion of fish for tribal populations (Section 7.3)	Adult (21+ years)	No
Application of paint and coatings	Ambient air	Inhalation	Inhalation of DCHP in ambient air resulting from industrial releases (Section 9.1)	All	No

[a] Table 1-1 provides a crosswalk of industrial and commercial COUs to OESs
[b] Using the MOE approach as a risk screening tool, an exposure pathway was determined to not be a major pathway of concern if the MOE ≥ 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 determined based on the strengths, limitations, and uncertainties associated with the exposure estimates. These are discussed in detail for biosolids (Section 3.1.1), landfills (Section 3.2.1), surface water (Section 4.3.1), drinking water (Section 6.3), fish ingestion (Section 7.4.1), ambient air (Section 8.3.1), and human milk (Section 10.4), respectively. EPA did not conduct reverse dosimetry to calculate daily intake values for DCHP given the lack of recent urinary biomonitoring data from NHANEs and the lack of additional data sources fit for use in this risk evaluation. The Agency summarized its weight of scientific evidence using the following 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.

The Agency determined robust confidence in its qualitative assessment of 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. For its quantitative exposure assessment of surface water (Section 5.2), drinking water (Section 6.3), fish ingestion (Section 7.4), ambient air (Section 8.4), and human milk (Section 10.4), 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 that 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.

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APPENDICES

Appendix A EXPOSURE FACTORS

Table_Apx A-1. Body Weight by Age Group

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

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

Table_Apx A-2. Fish Ingestion Rates by Age Group

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

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

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 (hours/day)	8	24	
EF	Exposure frequency (days/year)	250	365	
EY	Exposure years (years)	40	33 Adult 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, up to the 95th percentile residential occupancy period. See Table 16-5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a). Note: These age bins may vary for different measurements and sources
AT	Averaging time non-cancer	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	Equal to total exposure duration or 365 days/yr × EY; whichever is greater	See pg. 6–23 of Risk assessment guidance for superfund, volume I: Human health evaluation manual (Part A). (U.S. EPA, 1989)
	Averaging time cancer	78 years (28,470 days)	78 years (28,470 days)	See Table 18-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)
BW	Body weight (kg)	80	80 Adult 7.83 Infant (birth to <1 year) 16.2 Toddler (1–5 years) 31.8 Child (6–10 years) 56.8 Youth (11–15 years) 71.6 Youth (16–20 years) 65.9 Adolescent woman of childbearing age (16 to <21) – apply to all developmental exposure scenarios	See Table 8-1 of the *Exposure Factors Handbook* (U.S. EPA, 2011a) (Refer to Figure 31 for age-specific BW) Note: These age bins may vary for different measurements and sources See Table 8-5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a)
$IR_{dw\text{-}acute}$	Drinking water ingestion rate (L/day) – acute	3.219 adult	3.219 Adult 1.106 Infant (birth to <1 year) 0.813 Toddler (1–5 years) 1.258 Child (6–10 years) 1.761 Youth (11–15 years) 2.214 Youth (16–20 years)	See Tables 3-15 and 3-33; weighted average of 90th percentile consumer-only ingestion of drinking water (birth to <6 years) (U.S. EPA, 2011a)
$IR_{dw\text{-}chronic}$	Drinking water ingestion rate (L/day) – chronic	0.880 Adult	0.880 Adult 0.220 Infant (birth to <1 year) 0.195 Toddler (1–5 years) 0.294 Child (6–10 years) 0.315 Youth (11–15 years) 0.436 Youth (16–20 years)	Chapter 3 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 3-9 per capita mean values; weighted averages for adults (years 21– 49 and 50+), for toddlers (years 1–2, 2–3, and 3 to <6).
IR_{inc}	Incidental water ingestion rate (L/h)		0.025 Adult 0.05 Child (6 to < 16 years)	Evaluation of Swimmer Exposures Using the SWIMODEL Algorithms and Assumptions (U.S. EPA, 2015a)
IR_{fish}	Fish ingestion rate (g/day)		22 Adult	Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (U.S. EPA, 2014) This represents the 90th percentile consumption rate of fish and

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
				shellfish from inland and nearshore waters for the U.S. adult population 21 years of age and older, based on NHANES data from 2003–2010
IR_{soil}	Soil ingestion rate (mg/day)	50 Indoor workers 100 Outdoor workers	100 Infant (<6 months) 200 Infant to Youth (6 months to <12 years) 100 Youth to Adult (12+ years) 1,000 Soil Pica Infant to Youth (1 to <12 years) 50,000 Geophagy (all ages)	U.S. EPA Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (1991) Chapter 5 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 5-1, Upper percentile daily soil and dust ingestion
SA_{water}	Skin surface area exposed (cm^2) used for incidental water dermal contact		19,500 Adult 7,600 Child (3 to < 6 years) 10,800 Child (6 to < 11 years) 15,900 Youth (11 to < 16 years)	Chapter 7 of the *Exposure Factors Handbook* (U.S. EPA, 2011a), Table 7-1, Recommended Mean Values for Total Body Surface Area, for Children (sexes combined) and Adults by Sex
Kp	Permeability constant (cm/hr) used for incidental water dermal contact		0.001 Or calculated using K_p equation with chemical specific K_{OW} and MW (see exposure formulas)	EPA Dermal Exposure Assessment: Principles and Applications (U.S. EPA, 1992), Table 5-7, "Predicted K_p Estimates for Common Pollutants"
SA_{soil}	Skin surface area exposed (cm^2) used for soil dermal contact	3,300 Adult	5,800 Adult 2,700 Child	EPA Risk Assessment Guidance for Superfund RAGS Part E for Dermal Exposure (U.S. EPA, 2004)
AF_{soil}	Adherence factor (mg/cm^2) used for soil dermal contact	0.2 Adult	0.07 Adult 0.2 Child	EPA Risk Assessment Guidance for Superfund RAGS Part E for Dermal Exposure (U.S. EPA, 2004)

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

Age Group	Lipid Intake through Human Milk (g/kg day)[a]	
	Mean	Upper (95th percentile)
Birth to <1 month	6.2	9.0
1 to <3 month	5.7	8.2
3 to <6 month	4.3	6.3
6 to <12 month	3.4	5.4
Birth to <1 year	4.2	6.4

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

A.1 Surface Water Exposure Activity Parameters

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

Input	Description (Units)	Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)	Notes	Reference
BW	Body weight (kg)	80	56.8	31.8	Mean body weight. Chapter 8 of the *Exposure Factors Handbook*, Table 8-1	U.S. EPA (2021a)
SA	Skin surface area exposed (cm^2)	19,500	15,900	10,800	U.S. EPA Swimmer Exposure Assessment Model (SWIMODEL)	U.S. EPA (2015a)
ET	Exposure time (hr/day)	3	2	1	High-end default short-term duration from U.S. EPA Swimmer Exposure Assessment Model (SWIMODEL)	U.S. EPA (2015a)
ED	Exposure duration (years for ADD)	57	5	5	Number of years in age group, up to the 95th percentile residential occupancy period. Chapter 16 of the *Exposure Factors Handbook*, Table 16-5.	U.S. EPA (2021a)
AT	Averaging time (years for ADD)	57	5	5	Number of years in age group, up to the 95th percentile residential occupancy period. Chapter 16 of the *Exposure Factors Handbook*, Table 16-5.	U.S. EPA (2021a)
K_p	Permeability coefficient (cm/hr)	0.012 cm/hour			CEM estimate aqueous K_p	(U.S. EPA and 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)
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)
$IR_{inc-daily}$	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)	33	5	5	Number of years in age group, up to the 95th percentile residential occupancy period. Chapter 16 of the *Exposure Factors Handbook*, Table 16-5	U.S. EPA (2021a)

Input	Description (Units)	Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)	Notes	Reference
AT	Averaging time (years for ADD)	33	5	5	Number of years in age group, up to the 95th percentile residential occupancy period. Chapter 16 of the *Exposure Factors Handbook*, Table 16-5.	U.S. EPA (2021a)
CF1	Conversion factor (mg/µg)	1.00E−03				
CF2	Conversion factor (days/year)	365				

Appendix B ESTIMATING HYDROLOGICAL FLOW DATA FOR SURFACE WATER MODELING

Due to a lack of available data about facilities releasing DCHP to surface water under some OES, generic release scenarios were modeled for those OES. To develop relevant receiving water body flow distributions to pair with the estimated releases, for each OES relying on generic scenarios, a distribution of flow metrics was generated by collecting flow data for facilities across aligning with relevant NAICS codes associated with the respective OES. An example of relevant NAICS codes assigned to the Use of automotive care products OES is provided in Table_Apx B-1. The full table of NAICS codes assigned to OESs is included in *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025g).

Table_Apx B-1. Example of NAICS Codes Selected to Identify Relevant Facilities with Discharges to Surface Water and Derive OES-Specific Receiving Water Body Flow Distributions

OES	NAICS
Application of paints and coatings	332431 – Metal Can Manufacturing 335931 – Current-Carrying Wiring Device Manufacturing 337110 – Wood Kitchen Cabinet and Countertop Manufacturing 337122 – Nonupholstered Wood Household Furniture Manufacturing 337124 – Metal Household Furniture Manufacturing 337127 – Institutional Furniture Manufacturing 337211 – Wood Office Furniture Manufacturing 337214 – Office Furniture (except Wood) Manufacturing 337215 – Showcase, Partition, Shelving, and Locker Manufacturing 811120 – Automotive Body, Paint, Interior, and Glass Repair

EPA's ECHO database was accessed via the API (https://echo.epa.gov/tools/web-services; accessed December 17, 2025) and queried for facilities regulated under the Clean Water Act within the relevant NAICS codes for each OES. All available NPDES permit IDs were retrieved from the facilities returned by the query. It is important to note that while these NAICS codes cover the relevant sectors of industry within which this particular use of BBP can be found, the pool of facilities from which receiving water body data are collected are not necessarily all discharging BBP.

The Discharge Monitoring Report (DMR) REST service was then queried via the ECHO API (https://echo.epa.gov/tools/web-services/facility-search-water; accessed December 17, 2025) to return the NHDPlus reach code associated with the receiving water body for each available facility's NPDES permit. Modeled flow metrics were then extracted for the retrieved reach codes from the NHDPlus V2.1 Flowline Network EROM flow database (U.S. EPA and USGS, 2016). For each OES, all the receiving water body and flow information for each unique facility was pooled together from each respective NAICS code. After the further processing described below to derive the flow statistics for each receiving water body in the OES-specific distribution, selected percentiles (P50, P75, and P90) were used to model potential ranges of receiving water body concentrations. For example, the P50 7Q10 flow for the Use of automotive care products OES represents the P50 value from all 7Q10 flows derived from facility permit and NHDPlus data for that OES. It can also be thought of as the 7Q10 flow for the median water body receiving effluent within those NAICS codes.

The EROM database (U.S. EPA and USGS, 2016) 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 EPA's Exposure and Fate Assessment Screening Tool (EFAST) (U.S. EPA, 2007) to convert between these flow metrics and solved for the 7Q10 using Equation_Apx B-1. In previous assessments, the EPA has selected the 7Q10 flow as a representative low-flow scenario for biological impacts due to effluent in streams, while the harmonic mean represents a more average flow for assessing chronic drinking water exposure.

Equation_Apx B-1. Calculating the 7Q10 Flow

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

Where:

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

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

Equation_Apx B-2. Calculating the Harmonic Mean Flow

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

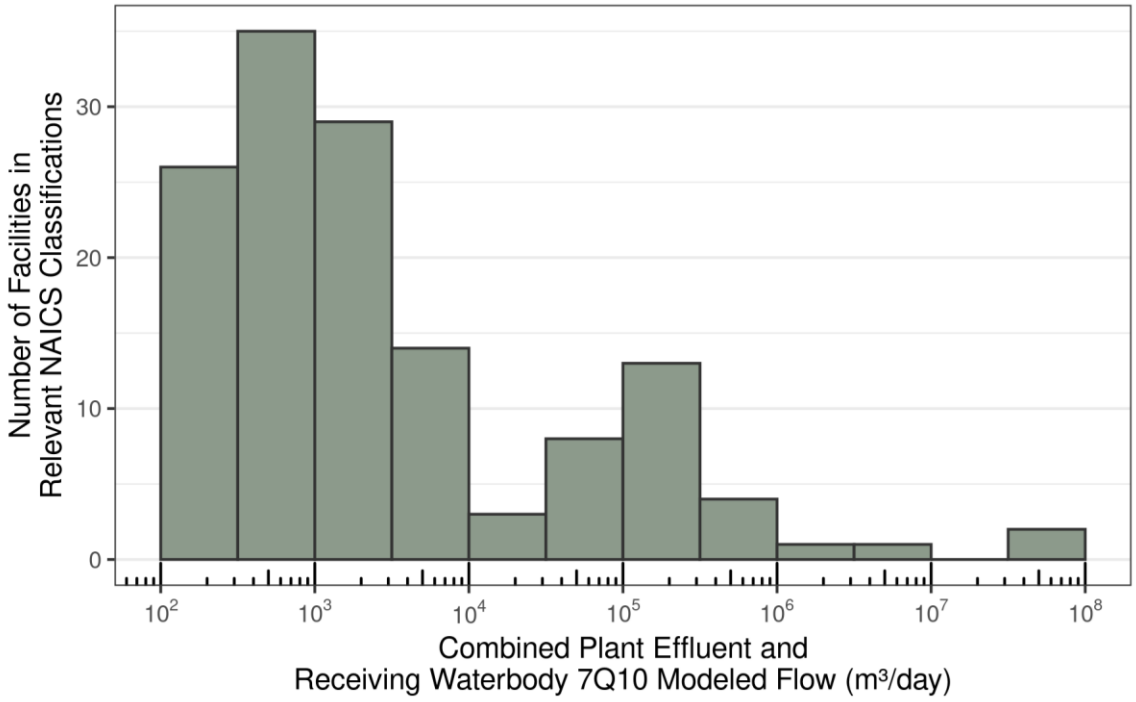
Where:

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

In addition to the individual releasing facilities that report to TRI and DMR that were queried for permit and flow data, a generic flow distribution was developed to apply to the generic scenarios for OES without programmatic data. A distribution of flow metrics was generated by collecting flow data for facilities across one NAICS code associated with DCHP-releasing facilities (Figure_Apx B-1). The ECHO database was similarly queried for all available permit and receiving water body information within the NAICS code, then processed in the same way to retrieve and generate flow metrics.

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 0.07 cubic feet per second, derived from the average reported effluent flow rate across facilities, was applied. The receiving water body 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 water body 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 to represent a more complete range of potential flow rates. (Table_Apx B-2). When comparing generic scenario releases and flow percentiles to known releases from facilities within relevant phthalate COUs and their respective receiving waterbodies, EPA was unable to constrain the analysis to a single flow percentile, as the P50, P75, and P90 flows are derived from relevant facilities, and each condition is plausible.



Figure_Apx B-1. Distribution of Receiving Water Body 7Q10-Modeled Flow for Facilities with Relevant NAICS Classifications for the Application of Paints and Coatings OES

Table_Apx B-2. Flow Statistics Applied for Generic Release to Surface Water Scenarios

OES	Number of Facilities	Number of NAICS Codes	Flow Statistic	Percentile Flows (m^3/day)		
				P50	P75	P90
Application of Paints and Coatings	136	10	HM	3,548	25,229	593,285
			7Q10	1,191	8,212	220,055
			30Q5	2,051	13,938	336,425

For other OES that did not rely on generic scenarios, individual facilities reported their releases to EPA's TRI and DMR systems. For such OESs, the actual releasing facilities and their respective receiving water body details were looked up using the ECHO API and NHDPlus V2.1 approach described above. The specific flow statistics (7Q10, 30Q5, HM) for those site-specific receiving water bodies were applied, rather than generic distributions, and therefore selecting of percentiles was not a necessary step for these facilities.

Quantified release estimates to surface water were evaluated with PSC modeling, applying the receiving water body flows retrieved from the NHDPlus. For each COU with surface water releases of wastewater effluent, the highest estimated release to surface water was modeled. The total days of release associated with the highest OES surface water releases was applied as continuous days of release per year (*e.g.*, a scenario with 250 days of release per year was modeled as 250 consecutive days of release, followed by 115 days of no release, per year). Estimates from PSC were evaluated for the highest resulting concentrations in an averaging window equal to the total days of release (*e.g.*, a scenario with 250 days of release was evaluated for the highest 250-day average concentration), using the averaging calculations within PSC.

Appendix C GENERAL POPULATION SURFACE WATER RISK SCREENING RESULTS

C.1 Incidental Dermal Exposures (Swimming)

Based on the estimated dermal doses in Table 5-1, EPA screened for risk to adults, youth, and children. Table_Apx C-1 summarizes the acute MOEs based on the dermal doses. All MOEs for various scenarios and lifestages can be found in *Surface Water Human Exposure Risk Calculator for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025m). Using the total acute dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 for all scenarios associated with the PVC plastics compounding OES. For Application of paints and coatings OES, scenarios where the modeled releases are paired with the median and 75th percentile flow are below the benchmark of 30. However, EPA had slight confidence in the exposure estimates for the Application of paints and coatings OES because releases were modeled as discharging to multiple environment media where there is insufficient information to determine the fraction going to each of the media types. Additionally, surface water concentrations for Application of paints and coatings estimated using P50 and P75 flow exceeded the high end of the range of the water solubility (1.48 mg/L) reported in *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2025k) by 100 to 1,000 percent, which is an unlikely scenario. Surface water concentrations estimated using P50 flow for the PVC plastics compound OES exceeded the low-end range of the water solubility (0.03 mg/L). Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects for dermal absorption through swimming is not expected.

Table_Apx C-1. Risk Screen for Incidental Dermal (Swimming) Doses for Adults, Youths, and Children for the High-End Release Estimate from Modeling and Monitoring Results (Benchmark MOE = 30)

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[a] *Without Wastewater Treatment*	204	197	1,341	1,751	2,887
Application of Paints and Coating (P50) *Without wastewater treatment*	19,990 [b]	11,600 [b]	14	18	29
Application of Paints and Coating (P75) *Without wastewater treatment*	3,030 [b]	1,820 [b]	90	118	194
Highest monitored surface water[a] *Without Wastewater Treatment*	0.014	0.014	20,000,000	26,000,000	42,000,000

30Q5 = Lowest 30-day average flow in a 5-year period

[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L

C.2 Incidental Ingestion

Based on the estimated incidental ingestion doses in Table 5-2, EPA screened for risk to adults, youth, and children. Table_Apx C-1 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 for all scenarios associated with the PVC plastics compounding OES. For Application of paints and coatings OES, scenarios where the modeled releases are paired with the median and 75th percentile flow are below the benchmark of 30. However, EPA had slight confidence in the exposure estimates for the Application of paints and coatings OES because releases were modeled as discharging to multiple environment media where there is insufficient information to determine the fraction going to each of the media types. Additionally, surface water concentrations for Application of paints and coatings estimated using P50 and P75 flow exceeded the high end of the range of the water solubility (1.48 mg/L) reported in *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2025k) by 100 to 1,000 percent, which is an unlikely scenario. Surface water concentrations estimated using P50 flow for the PVC plastics compound OES exceeded the low-end range of the water solubility (0.03 mg/L). All modeled surface water concentrations exceeded the highest monitored value of 14 ng/L. Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects for incidental ingestion through swimming is not expected.

Table_Apx C-2. Risk Screen for Incidental Ingestion Doses for Adults, Youths, and Children, for the High-End Release Estimate from Modeling and Monitoring Results (Benchmark MOE = 30)

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[a] *Without Wastewater Treatment*	204	197	3,410	2,198	3,897
Application of Paints and Coating (P50) *Without wastewater treatment*	19,990[b]	11,600[b]	35	22	40
Application of Paints and Coating (P75) *Without wastewater treatment*	3,030[b]	1,820[b]	230	148	262
Highest Monitored Surface Water[a]	0.014	0.014	50,000,000	32,000,000	57,000,000

30Q5 = Lowest 30-day average flow in a 5-year period

[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L.

Appendix D GENERAL POPULATION DRINKING WATER RISK SCREENING RESULTS

Based on the estimated drinking water doses in Table 6-1, EPA screened for risk to adults (21+ years), infants (birth to <1 year), and toddlers (1–5 years). Table_Apx D-1 summarizes the acute and chronic MOEs based on the drinking water doses. All MOEs for various scenarios and lifestages can be found in *Surface Water Human Exposure Risk Calculator for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025m). Using the total acute and chronic dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30 (U.S. EPA, 2025i) except for the Application of paints and coatings OES, which is based on a high-end release estimate to multiple environmental media. This protective screening scenario, with the entirety of the estimated environmental release assumed to be released directly to surface water, results in an MOE less than the benchmark for scenarios where the modeled releases are paired with the median and 75th percentile flow. However, EPA had slight confidence in the exposure estimates for the Application of paints and coatings OES because releases were modeled as discharging to multiple environment media where there is insufficient information to determine the fraction going to each of the media types. Additionally, surface water concentrations for Application of paints and coatings estimated using P50 and P75 flow exceeded the high-end of the range of the water solubility (1.48 mg/L) reported in *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2025k).

EPA had greater confidence in surface water concentrations estimated using the PVC plastics compounding OES as releases were reported to only water. Concentrations estimated using P50 flow for the PVC plastics compounding OES still exceeded the low-end range of the water solubility (0.03 mg/L) but still resulted in MOEs above the benchmark for all scenarios. Based on the conservative modeling parameters for drinking water concentration and exposure factors parameters, risk for non-cancer health effects for drinking water ingestion is not expected.

This assessment assumes that concentrations at the point of intake for the drinking water system are equal to the concentrations in the receiving water body at the point of release, where treated effluent is being discharged from a facility. In reality, some distance between the point of release and a drinking water intake would be expected, providing space and time for additional reductions in water column concentrations via degradation, partitioning, and dilution. Some form of additional treatment would typically be expected for surface water at a drinking water treatment plant, including coagulation, flocculation, and sedimentation, and/or filtration. This treatment would likely result in even greater reductions in DCHP 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 (Benchmark MOE = 30)

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
PVC Plastics Compounding *Without Wastewater Treatment*	204	197	292	1,592	83	623	234	1,454
Application of Paints	19,990[a]	11,600[a]	3	24	1	9	2	22

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
and Coating (P50) *Without Wastewater Treatment*								
Application of Paints and Coating (P50) *with Wastewater Treatment*	5757[a]	3340[a]	10	83	3	33	8	76
Highest Monitored Surface Water[a]	0.014	0.014	4,300,000	20,000,000	1,200,000	7,800,000	3,400,000	18,000,000

30Q5 = Lowest 30-day average flow in a 5-year period

[a] Keil et al. (2011) reported the highest monitored surface water concentration, as described further in Section 4.2.1. This is a single maximum value from the study and does not correspond to either the 30Q5 or harmonic mean concentrations. However, it was used in both instances to compare exposure estimates based on modeled and monitored surface water concentrations.

[b] Exceeds the water solubility range reported in the *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2025k) of 0.030–1.48 mg/L.

Appendix E FISH INGESTION RISK SCREENING RESULTS

E.1 General Population

Table_Apx E-1. Risk Estimates for Fish Ingestion Exposure for General Population (Benchmark MOE = 30)

Surface Water Concentration and Scenario	Acute Non-Cancer MOE UFs = 30		Adult Chronic Non-Cancer MOE
	Adult	Young Toddler	
Water solubility limit 1.48 and 0.03 mg/L for upper and lower bound	87 (upper) 4,303 (lower)	59 (upper) 2,898 (lower)	384 (upper) 18,953 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	655 (P50 flow) 3,353 (P75 flow) 374,157 (P90 flow)	441 (P50 flow) 2,258 (P75 flow) 252,011 (P90 flow)	2,886 (P50 flow) 13,902 (P75 flow) 1,648,074 (P90 flow)
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	11 (P50 flow) 71 (P75 flow) 1,831 (P90 flow)	7 (P50 flow) 48 (P75 flow) 1,233 (P90 flow)	49 (P50 flow) 312 (P75 flow) 8,065 (P90 flow)
HE = high-end, 95th percentile release; MOE = margin of exposure; UF = uncertainty factor			

E.2 Subsistence Fishers

Table_Apx E-2. Risk Estimates for Fish Ingestion Exposure for Subsistence Fishers (Benchmark MOE = 30)

Surface Water Concentration and Scenario	Acute and Chronic Non-Cancer MOE
Water solubility limit 1.48 and 0.03 mg/L for upper and lower bound	14 (upper) 671 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	102 (P50 flow) 523 (P75 flow) 58,331 (P90 flow)
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	2 (P50 flow) 11 (P75 flow) 285 (P90 flow)
HE = high-end, 95th percentile release; MOE = margin of exposure; UF = uncertainty factor 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

Table_Apx E-3. Risk Estimates for Fish Ingestion Exposure for Tribal Populations (Benchmark MOE = 30)

Surface Water Concentration and Scenario	Acute and Chronic Non-Cancer MOE[a]		
	Current IR, Mean	Current IR, 95th Percentile	Heritage IR
Water solubility limit 1.48 and 0.03 mg/L for upper and lower bound	9 (upper) 442 (lower)	2 (upper) 110 (lower)	1 (upper) 58 (lower)
Plastics compounding (generic scenario for water-only release, HE, without wastewater treatment) 1.97E02, 3.85E01, 3.45E–01 mg/L for P50, P75, P90 flow	67 (P50 flow) 345 (P75 flow) 38,455 (P90 flow)	17 (P50 flow) 85 (P75 flow) 9,526 (P90 flow)	9 (P50 flow) 45 (P75 flow) 5,045 (P90 flow)
Plastics compounding (generic scenario for water-only release, CT, without wastewater treatment) 4.10E01 for P50 flow	324	80	43
Application of paints and coatings (generic scenario for multimedia releases, HE, without wastewater treatment) 1.16E04, 1.82E03, 7.10E01 mg/L for P50, P75, P90 flow	1 (P50 flow) 7 (P75 flow) 188 (P90 flow)	0 (P50 flow) 2 (P75 flow) 47 (P90 flow)	0 (P50 flow) 1 (P75 flow) 25 (P90 flow)

CT = central tendency; HE = high-end; IR = ingestion rate; MOE = margin of exposure

[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.

van Drooge et al. (2020) sampled indoor classrooms and outdoor playgrounds of primary schools in Barcelona, Spain. DCHP concentrations were higher in indoor samples (95–110 ng/m^3) vs. outdoor samples (9–12 ng/m^3). The study suggested that the higher indoor concentrations likely reflect the use of plastics in classroom material. This study received an overall data quality rating of medium under EPA's systematic review.

A second study by Lee et al. (2019) detected DCHP in particulates with a mean concentration of 0.01 ng/m^3 and median of 0.03 ng/m^3 across four samples. Sampling was conducted in two streams leading to an artificial lake, as well as in the lake itself, in South Korea. This study received an overall data quality rating of high under EPA's systematic review.

A third study conducted in Lake Chaohu, China, (He et al., 2019) measured atmospheric particles at a lakeshore site and found a maximum concentration of 3.66 pg/m^3. This study hypothesized the source of atmospheric phthalate esters was long-range transport from Guangdong Province in Southern China. Guangdong province is described as an intensive area of manufacturing industries and electronic waste dismantling industries.

Appendix G URINARY BIOMONITORING METHODS AND RESULTS

EPA analyzed urinary biomonitoring data from CDC's NHANES, which reports urinary concentrations for 15 phthalate metabolites specific to individual phthalate diesters. One metabolite of DCHP, MCHP, has been reported in the NHANES data. MCHP has been reported in NHANES beginning with the 1999 cycle and measured in 15,829 members of the general public, including 4,130 children aged 15 years and under and 11,699 adults aged 16 years and over. Urinary MCHP concentrations were quantified using high performance liquid chromatography-electrospray ionization-tandem mass spectrometry. LODs 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). See also Table_Apx G-2 and Table_Apx G-3.

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

NHANES Cycle	MCHP (ng/mL)
1999–2000	0.93
2001–2002	0.93
2003–2004	0.20
2005–2006	0.30
2007–2008	0.30
2009–2010	0.402

Table_Apx G-2. Summary of Urinary MCHP Concentrations (ng/mL) from all NHANES Cycles between 1999–2010[a]

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	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)
1999–2000	Adults	All adults	1,827	1,827 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.07 (0.98–1.17)	6.4 (5.12–7.52)
1999–2000	Adults	Females	964	964 (100%)	1.2792 (1.2792–1.2792)	1.809 (1.2792–5.427)	1.38 (1.21–1.58)	7.11 (6.7–8)
1999–2000	Adults	Males	863	863 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	0.96 (0.89–1.05)	4.57 (3.37–6.73)
1999–2000	Adults	At or above poverty level	412	412 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.05 (0.97–1.14)	6.09 (4.74–7.52)
1999–2000	Adults	Below poverty level	377	377 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.14 (0.93–1.56)	5.12 (3.65–7.05)
1999–2000	Adults	Unknown income	798	798 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.16 (0.99–1.36)	6.4 (2.97–12.95)
1999–2000	Adults	White non-Hispanic	738	738 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.14 (1.04–1.25)	6.6 (5.23–8)
1999–2000	Adults	Black non-Hispanic	363	363 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	0.83 (0.8–0.88)	3.05 (2.37–3.46)
1999–2000	Adults	Mexican American	550	550 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.608)	1.07 (1–1.13)	5.56 (3.28–8)
1999–2000	Adults	Other	176	176 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	0.98 (0.8–1.22)	6.7 (3.28–10.18)
1999–2000	WRA	All women of reproductive age	618	618 (100%)	1.2792 (1.2792–1.2792)	1.809 (1.2792–5.427)	1.07 (0.98–1.17)	6.4 (5.12–7.52)
1999–2000	WRA	At or above poverty level	118	118 (100%)	1.2792 (1.2792–1.2792)	2.01 (1.2792–8.643)	1.05 (0.97–1.14)	6.09 (4.74–7.52)
1999–2000	WRA	Below Poverty Level	146	146 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.14 (0.93–1.56)	5.12 (3.65–7.05)
1999–2000	WRA	Black non-Hispanic	126	126 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	0.83 (0.8–0.88)	3.05 (2.37–3.46)
1999–2000	WRA	Mexican American	208	208 (100%)	1.2792 (1.2792–1.2792)	1.809 (1.2792–5.427)	1.07 (1–1.13)	5.56 (3.28–8)
1999–2000	WRA	Other	71	71 (100%)	1.2792 (1.2792–1.2792)	2.412 (1.2792–11.658)	0.98 (0.8–1.22)	6.7 (3.28–10.18)
1999–2000	WRA	Unknown Income	275	275 (100%)	1.2792 (1.2792–1.2792)	1.2792 (1.2792–1.2792)	1.16 (0.99–1.36)	6.4 (2.97–12.95)
1999–2000	WRA	White non-Hispanic	213	213 (100%)	1.2792 (1.2792–1.2792)	1.9095 (1.2792–8.643)	1.14 (1.04–1.25)	6.6 (5.23–8)
1999–2000	Children	All children	714	714 (100%)	1.2792 (1.2792–1.2792)	3.417 (2.01–4.824)	1.1 (0.95–1.24)	4 (3.28–6.7)
1999–2000	Children	Females	362	362 (100%)	1.2792 (1.2792–1.2792)	2.01 (1.2792–4.02)	1.11 (0.92–1.36)	4.69 (2.97–8)
1999–2000	Children	Males	352	352 (100%)	1.2792 (1.2792–1.2792)	3.819 (2.01–7.638)	1.1 (0.93–1.23)	3.6 (2.91–12.95)
1999–2000	Children	Children (6 to <11 years)	276	276 (100%)	1.2792 (1.2792–1.2792)	4.02 (2.01–7.839)	1.31 (1.12–1.6)	6.4 (3.28–12.95)
1999–2000	Children	Adolescents (11 to <16 years)	438	438 (100%)	1.2792 (1.2792–1.2792)	3.216 (2.01–4.02)	0.91 (0.79–1.09)	3.12 (2.51–4.26)
1999–2000	Children	At or above poverty level	191	191 (100%)	1.2792 (1.2792–1.2792)	2.211 (2.01–2.814)	1.07 (0.93–1.23)	3.37 (2.78–4.69)
1999–2000	Children	Below poverty level	215	215 (100%)	1.2792 (1.2792–1.2792)	3.015 (1.2792–4.824)	1.22 (0.91–1.5)	4.98 (3.12–9.14)
1999–2000	Children	Unknown income	220	220 (100%)	1.2792 (1.2792–1.2792)	7.638 (1.2792–16.683)	1.06 (0.72–1.35)	7.13 (1.66–31.98)
1999–2000	Children	White non-Hispanic	158	158 (100%)	1.2792 (1.2792–1.2792)	2.01 (1.2792–3.417)	1.12 (0.91–1.32)	3.37 (2.72–8)
1999–2000	Children	Black non-Hispanic	229	229 (100%)	1.2792 (1.2792–1.2792)	3.216 (2.412–4.221)	0.95 (0.83–1.03)	3.5 (2.46–4.84)
1999–2000	Children	Mexican American	264	264 (100%)	1.2792 (1.2792–1.2792)	1.809 (1.2792–2.814)	1.27 (1.15–1.42)	4.98 (4–6.4)
1999–2000	Children	Other	63	63 (100%)	1.2792 (1.2792–1.2792)	5.829 (1.2792–7.638)	1.1 (0.8–1.38)	6.7 (2.06–12.95)
2001–2002	Adults	All adults	2,004	2,004 (6.39%)	0.4264 (0.4264–0.4264)	1.005 (0.804–1.206)	0.4 (0.37–0.43)	1.94 (1.64–2.2)
2001–2002	Adults	Females	1,019	1,019 (5.59%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.53 (0.48–0.58)	2.03 (1.68–2.84)

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	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)
2001–2002	Adults	Males	985	985 (7.21%)	0.4264 (0.4264–0.4264)	1.005 (0.603–1.206)	0.35 (0.32–0.37)	1.77 (1.42–2.15)
2001–2002	Adults	At or above poverty level	463	463 (6.05%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.4 (0.36–0.44)	1.94 (1.64–2.21)
2001–2002	Adults	Below poverty level	361	361 (6.37%)	0.4264 (0.4264–0.4264)	0.603 (0.4264–1.206)	0.38 (0.34–0.46)	2.24 (1.64–3.22)
2001–2002	Adults	Black non-Hispanic	414	414 (7.25%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–1.407)	0.31 (0.27–0.35)	1.33 (1.09–1.64)
2001–2002	Adults	Mexican American	445	445 (4.72%)	0.4264 (0.4264–0.4264)	0.603 (0.4264–0.804)	0.39 (0.36–0.45)	2.13 (1.58–2.51)
2001–2002	Adults	Other	162	162 (12.35%)	0.4264 (0.4264–0.4264)	1.407 (0.804–3.417)	0.42 (0.34–0.51)	2.51 (1.3–4.26)
2001–2002	Adults	Unknown income	1,052	1,052 (6.56%)	0.4264 (0.4264–0.4264)	1.206 (0.4264–1.206)	0.38 (0.29–0.55)	1.33 (0.99–1.42)
2001–2002	Adults	White non-Hispanic	983	983 (5.8%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.41 (0.37–0.45)	1.91 (1.64–2.24)
2001–2002	WRA	All women of reproductive age	659	659 (5.92%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.4 (0.37–0.43)	1.94 (1.64–2.2)
2001–2002	WRA	At or above poverty level	154	154 (5.19%)	0.4264 (0.4264–0.4264)	1.005 (0.603–1.206)	0.4 (0.36–0.44)	1.94 (1.64–2.21)
2001–2002	WRA	Below poverty level	136	136 (5.15%)	0.4264 (0.4264–0.4264)	1.206 (0.4264–1.206)	0.38 (0.34–0.46)	2.24 (1.64–3.22)
2001–2002	WRA	Black non-Hispanic	144	144 (6.94%)	0.4264 (0.4264–0.4264)	1.005 (0.4264–1.407)	0.31 (0.27–0.35)	1.33 (1.09–1.64)
2001–2002	WRA	Mexican American	172	172 (6.4%)	0.4264 (0.4264–0.4264)	1.206 (0.4264–4.623)	0.39 (0.36–0.45)	2.13 (1.58–2.51)
2001–2002	WRA	Other	57	57 (5.26%)	0.4264 (0.4264–0.4264)	1.407 (0.4264–3.819)	0.42 (0.34–0.51)	2.51 (1.3–4.26)
2001–2002	WRA	Unknown income	331	331 (6.34%)	0.4264 (0.4264–0.4264)	1.005 (0.4264–1.206)	0.38 (0.29–0.55)	1.33 (0.99–1.42)
2001–2002	WRA	White non-Hispanic	286	286 (5.24%)	0.4264 (0.4264–0.4264)	0.603 (0.4264–1.005)	0.41 (0.37–0.45)	1.91 (1.64–2.24)
2001–2002	Children	All children (3 to <16)	778	778 (9.13%)	0.4264 (0.4264–0.4264)	0.804 (0.804–1.005)	0.43 (0.38–0.5)	1.9 (1.46–2.37)
2001–2002	Children	Females	392	392 (7.14%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.005)	0.43 (0.38–0.5)	1.55 (1.38–2.09)
2001–2002	Children	Males	386	386 (11.14%)	0.4264 (0.4264–0.4264)	1.206 (1.005–1.407)	0.45 (0.37–0.51)	2.17 (1.15–4.26)
2001–2002	Children	Adolescents (11 to <16 years)	456	456 (9.65%)	0.4264 (0.4264–0.4264)	1.005 (0.603–1.005)	0.37 (0.33–0.43)	1.78 (1.01–3.05)
2001–2002	Children	At or above poverty level	192	192 (8.33%)	0.4264 (0.4264–0.4264)	0.804 (0.804–1.005)	0.43 (0.37–0.48)	1.78 (1.29–2.17)
2001–2002	Children	Below poverty level	237	237 (10.97%)	0.4264 (0.4264–0.4264)	1.005 (0.603–3.015)	0.47 (0.39–0.58)	2.46 (1.33–4.26)
2001–2002	Children	Black non-Hispanic	275	275 (9.09%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.005)	0.38 (0.34–0.41)	1.47 (1–1.71)
2001–2002	Children	Children (6 to <11 years)	322	322 (8.39%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.53 (0.47–0.62)	2.17 (1.52–2.56)
2001–2002	Children	Mexican American	232	232 (10.34%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.206)	0.47 (0.41–0.55)	2.37 (1.38–3.22)
2001–2002	Children	Other	49	49 (8.16%)	0.4264 (0.4264–0.4264)	0.804 (0.603–1.005)	0.42 (0.28–0.71)	1.59 (0.97–4.26)
2001–2002	Children	Unknown income	313	313 (8.63%)	0.4264 (0.4264–0.4264)	0.603 (0.603–1.005)	0.44 (0.26–0.64)	0.99 (0.82–1.94)
2001–2002	Children	White non-Hispanic	222	222 (8.11%)	0.4264 (0.4264–0.4264)	1.005 (0.804–1.407)	0.45 (0.37–0.52)	2.04 (1.29–3.05)
2003–2004	Adults	All adults	1,889	1,889 (8.73%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.603)	0.25 (0.23–0.26)	1.24 (1.09–1.35)
2003–2004	Adults	Females	980	980 (9.08%)	0.2843 (0.2843–0.2843)	0.402 (0.2843–0.804)	0.32 (0.29–0.36)	1.58 (1.29–1.9)
2003–2004	Adults	Males	909	909 (8.36%)	0.2843 (0.2843–0.2843)	0.402 (0.2843–0.603)	0.22 (0.2–0.23)	0.94 (0.75–1.26)
2003–2004	Adults	At or above poverty level	474	474 (8.44%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.402)	0.24 (0.23–0.26)	1.24 (1.05–1.35)
2003–2004	Adults	Below poverty level	393	393 (9.41%)	0.2843 (0.2843–0.2843)	0.402 (0.2843–0.603)	0.24 (0.2–0.29)	1.02 (0.77–1.42)

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	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	Adults	Black non-Hispanic	423	423 (13.71%)	0.2843 (0.2843–0.2843)	0.402 (0.2843–0.603)	0.19 (0.18–0.2)	0.75 (0.68–0.95)
2003–2004	Adults	Mexican American	423	423 (9.69%)	0.2843 (0.2843–0.2843)	0.603 (0.2843–1.005)	0.25 (0.22–0.27)	1.09 (0.92–1.5)
2003–2004	Adults	Other	142	142 (2.82%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.2843)	0.25 (0.22–0.33)	1.24 (0.77–1.9)
2003–2004	Adults	Unknown income	904	904 (8.08%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–1.005)	0.27 (0.22–0.33)	1.76 (0.51–2.37)
2003–2004	Adults	White non-Hispanic	901	901 (6.88%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.2843)	0.25 (0.23–0.27)	1.29 (1.09–1.42)
2003–2004	WRA	All women of reproductive age	606	606 (8.75%)	0.2843 (0.2843–0.2843)	0.402 (0.2843–0.804)	0.25 (0.23–0.26)	1.24 (1.09–1.35)
2003–2004	WRA	At or above poverty level	137	137 (8.03%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.2843)	0.24 (0.23–0.26)	1.24 (1.05–1.35)
2003–2004	WRA	Below poverty level	169	169 (10.65%)	0.2843 (0.2843–0.2843)	0.603 (0.2843–1.206)	0.24 (0.2–0.29)	1.02 (0.77–1.42)
2003–2004	WRA	Black non-Hispanic	157	157 (11.46%)	0.2843 (0.2843–0.2843)	0.804 (0.2843–1.206)	0.19 (0.18–0.2)	0.75 (0.68–0.95)
2003–2004	WRA	Mexican American	146	146 (12.33%)	0.2843 (0.2843–0.2843)	0.603 (0.2843–2.412)	0.25 (0.22–0.27)	1.09 (0.92–1.5)
2003–2004	WRA	Other	49	49 (4.08%)	0.2843 (0.2843–0.2843)	0.603 (0.2843–0.603)	0.25 (0.22–0.33)	1.24 (0.77–1.9)
2003–2004	WRA	Unknown income	262	262 (7.63%)	0.2843 (0.2843–0.2843)	2.412 (0.2843–2.412)	0.27 (0.22–0.33)	1.76 (0.51–2.37)
2003–2004	WRA	White non-Hispanic	254	254 (5.91%)	0.2843 (0.2843–0.2843)	0.2843 (0.2843–0.2843)	0.25 (0.23–0.27)	1.29 (1.09–1.42)
2003–2004	Children	All children	716	716 (16.2%)	0.2843 (0.2843–0.2843)	0.804 (0.804–1.005)	0.27 (0.23–0.31)	1.33 (0.84–1.83)
2003–2004	Children	Females	375	375 (13.6%)	0.2843 (0.2843–0.2843)	1.005 (0.402–1.005)	0.29 (0.23–0.33)	1.24 (0.98–1.9)
2003–2004	Children	Males	341	341 (19.06%)	0.2843 (0.2843–0.2843)	1.005 (0.804–1.809)	0.26 (0.23–0.29)	1.31 (0.57–2.62)
2003–2004	Children	Adolescents (11 to <16 years)	430	430 (16.28%)	0.2843 (0.2843–0.2843)	0.804 (0.603–1.206)	0.23 (0.2–0.27)	1.09 (0.69–1.78)
2003–2004	Children	At or above poverty level	183	183 (14.75%)	0.2843 (0.2843–0.2843)	0.804 (0.603–1.005)	0.27 (0.23–0.32)	1.42 (0.81–1.98)
2003–2004	Children	Below poverty level	237	237 (14.77%)	0.2843 (0.2843–0.2843)	0.804 (0.402–1.005)	0.27 (0.22–0.32)	0.81 (0.6–0.98)
2003–2004	Children	Black non-Hispanic	258	258 (15.89%)	0.2843 (0.2843–0.2843)	0.804 (0.804–1.005)	0.23 (0.2–0.27)	0.81 (0.71–0.94)
2003–2004	Children	Children (6 to <11 years)	286	286 (16.08%)	0.2843 (0.2843–0.2843)	0.804 (0.603–1.005)	0.32 (0.29–0.38)	1.58 (0.92–2.62)
2003–2004	Children	Mexican American	229	229 (16.59%)	0.2843 (0.2843–0.2843)	1.005 (0.402–1.005)	0.3 (0.26–0.34)	1.59 (0.71–2.37)
2003–2004	Children	Other	52	52 (23.08%)	0.2843 (0.2843–0.2843)	0.603 (0.402–3.819)	0.31 (0.21–0.41)	0.81 (0.43–2.62)
2003–2004	Children	Unknown income	267	267 (18.73%)	0.2843 (0.2843–0.2843)	2.412 (0.2843–2.814)	0.32 (0.16–0.62)	2.25 (0.38–2.25)
2003–2004	Children	White non-Hispanic	177	177 (14.12%)	0.2843 (0.2843–0.2843)	1.005 (0.603–1.206)	0.27 (0.23–0.33)	1.14 (0.89–1.9)
2005–2006	Adults	All adults	1,831	1,831 (2.13%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.36 (0.35–0.38)	1.65 (1.42–1.85)
2005–2006	Adults	Females	935	935 (1.5%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.49 (0.45–0.53)	2.24 (1.85–2.51)
2005–2006	Adults	Males	896	896 (2.79%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.31 (0.3–0.33)	1.18 (0.97–1.33)
2005–2006	Adults	At or above poverty level	436	436 (3.67%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.37 (0.35–0.39)	1.71 (1.42–1.94)
2005–2006	Adults	Below poverty level	340	340 (1.47%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.34 (0.3–0.39)	1.15 (0.91–2.03)
2005–2006	Adults	Black non-Hispanic	464	464 (2.16%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.27 (0.26–0.29)	0.97 (0.74–1.18)
2005–2006	Adults	Mexican American	390	390 (2.31%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.35 (0.33–0.37)	1.38 (0.99–1.9)
2005–2006	Adults	Other	131	131 (4.58%)	0.4264 (0.4264–0.4264)	0.603 (0.4264–10.653)	0.33 (0.27–0.4)	1.33 (0.93–1.71)

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	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)
2005–2006	Adults	Unknown income	955	955 (1.57%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–0.804)	0.42 (0.32–0.71)	1.71 (1.15–3.28)
2005–2006	Adults	White non-Hispanic	846	846 (1.65%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.37–0.41)	1.71 (1.52–2.03)
2005–2006	WRA	All women of reproductive age	616	616 (1.62%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.36 (0.35–0.38)	1.65 (1.42–1.85)
2005–2006	WRA	At or above poverty level	143	143 (2.8%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.37 (0.35–0.39)	1.71 (1.42–1.94)
2005–2006	WRA	Below poverty level	146	146 (1.37%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.34 (0.3–0.39)	1.15 (0.91–2.03)
2005–2006	WRA	Black non-Hispanic	162	162 (1.23%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.27 (0.26–0.29)	0.97 (0.74–1.18)
2005–2006	WRA	Mexican American	158	158 (1.27%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.35 (0.33–0.37)	1.38 (0.99–1.9)
2005–2006	WRA	Other	62	62 (4.84%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–1.005)	0.33 (0.27–0.4)	1.33 (0.93–1.71)
2005–2006	WRA	Unknown income	299	299 (1%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.42 (0.32–0.71)	1.71 (1.15–3.28)
2005–2006	WRA	White non-Hispanic	234	234 (1.28%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.37–0.41)	1.71 (1.52–2.03)
2005–2006	Children	All children	717	717 (2.09%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.38 (0.35–0.39)	1.15 (0.97–1.47)
2005–2006	Children	Females	343	343 (1.75%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.41 (0.38–0.45)	1.42 (1.18–2.51)
2005–2006	Children	Males	374	374 (2.41%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.35 (0.32–0.38)	0.82 (0.75–1.09)
2005–2006	Children	Adolescents (11 to <16 years)	412	412 (0.97%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.33 (0.28–0.35)	1.22 (0.8–1.71)
2005–2006	Children	At or above poverty level	185	185 (2.16%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.37 (0.35–0.39)	1.22 (1.07–1.71)
2005–2006	Children	Below poverty level	195	195 (3.08%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.37 (0.32–0.43)	0.97 (0.78–1.22)
2005–2006	Children	Black non-Hispanic	214	214 (1.87%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.33 (0.25–0.36)	0.89 (0.68–1.18)
2005–2006	Children	Children (6 to <11 years)	305	305 (3.61%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.44 (0.41–0.46)	1.18 (1.09–2.03)
2005–2006	Children	Mexican American	247	247 (3.24%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.34–0.45)	1.47 (1.07–2.36)
2005–2006	Children	Other	64	64 (0%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.38 (0.28–0.51)	1.33 (0.8–1.71)
2005–2006	Children	Unknown income	319	319 (1.57%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.69 (0.24–0.8)	1.15 (0.75–1.71)
2005–2006	Children	White non-Hispanic	192	192 (1.56%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.35–0.41)	1.15 (0.8–1.94)
2007–2008	Adults	All adults	2,021	2,021 (3.61%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.37–0.41)	1.86 (1.64–2.24)
2007–2008	Adults	Females	1,030	1,030 (3.88%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.55 (0.48–0.6)	2.84 (2.03–3.05)
2007–2008	Adults	Males	991	991 (3.33%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.34 (0.32–0.35)	1.33 (1.09–1.58)
2007–2008	Adults	At or above poverty level	505	505 (3.37%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.38 (0.35–0.41)	1.8 (1.45–2.03)
2007–2008	Adults	Below poverty level	392	392 (3.57%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.36 (0.33–0.41)	1.85 (1.48–2.24)
2007–2008	Adults	Black non-Hispanic	434	434 (4.84%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.33 (0.3–0.35)	1.25 (1.02–1.52)
2007–2008	Adults	Mexican American	371	371 (4.31%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.37 (0.34–0.41)	1.38 (1.22–1.47)
2007–2008	Adults	Other	294	294 (5.78%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–1.206)	0.4 (0.36–0.53)	2.84 (1.48–3.88)
2007–2008	Adults	Unknown income	948	948 (3.48%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–2.01)	0.44 (0.38–0.58)	2.84 (1.42–8.39)
2007–2008	Adults	White non-Hispanic	922	922 (2.06%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.4 (0.37–0.42)	1.94 (1.58–2.84)
2007–2008	WRA	All women of reproductive age	571	571 (3.85%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.39 (0.37–0.41)	1.86 (1.64–2.24)

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	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	WRA	At or above poverty level	132	132 (3.03%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.38 (0.35–0.41)	1.8 (1.45–2.03)
2007–2008	WRA	Below poverty level	143	143 (3.5%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–1.608)	0.36 (0.33–0.41)	1.85 (1.48–2.24)
2007–2008	WRA	Black non-Hispanic	129	129 (5.43%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–1.608)	0.33 (0.3–0.35)	1.25 (1.02–1.52)
2007–2008	WRA	Mexican American	125	125 (4%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–2.211)	0.37 (0.34–0.41)	1.38 (1.22–1.47)
2007–2008	WRA	Other	95	95 (3.16%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.4 (0.36–0.53)	2.84 (1.48–3.88)
2007–2008	WRA	Unknown income	250	250 (4.8%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.44 (0.38–0.58)	2.84 (1.42–8.39)
2007–2008	WRA	White non-Hispanic	222	222 (3.15%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.4 (0.37–0.42)	1.94 (1.58–2.84)
2007–2008	Children	All children	583	583 (6.69%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–1.206)	0.4 (0.38–0.43)	1.58 (1.22–2.03)
2007–2008	Children	Females	280	280 (5.71%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.43 (0.37–0.51)	2.03 (1.33–3.28)
2007–2008	Children	Males	303	303 (7.59%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–2.01)	0.38 (0.33–0.42)	1.45 (1.09–1.64)
2007–2008	Children	Adolescents (11 to <16 years)	265	265 (5.66%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–2.01)	0.34 (0.32–0.38)	1.58 (1.08–2.37)
2007–2008	Children	At or above poverty level	162	162 (7.41%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–1.608)	0.39 (0.36–0.46)	1.64 (1.15–1.86)
2007–2008	Children	Below poverty level	186	186 (6.99%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.41 (0.36–0.46)	1.48 (1.18–2.37)
2007–2008	Children	Black non-Hispanic	163	163 (7.36%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–1.206)	0.37 (0.34–0.42)	1.52 (1.25–2.13)
2007–2008	Children	Children (6 to <11 years)	318	318 (7.55%)	0.4264 (0.4264–0.4264)	1.005 (0.4264–1.608)	0.53 (0.43–0.61)	1.64 (1.22–2.84)
2007–2008	Children	Mexican American	160	160 (6.25%)	0.4264 (0.4264–0.4264)	0.804 (0.4264–1.206)	0.41 (0.36–0.48)	1.39 (1.18–1.78)
2007–2008	Children	Other	105	105 (9.52%)	0.4264 (0.4264–0.4264)	1.206 (0.4264–2.211)	0.4 (0.3–0.62)	2.03 (0.99–3.55)
2007–2008	Children	Unknown income	196	196 (5.1%)	0.4264 (0.4264–0.4264)	1.005 (0.4264–2.412)	0.47 (0.3–1.38)	3.55 (0.99–8.53)
2007–2008	Children	White non-Hispanic	155	155 (4.52%)	0.4264 (0.4264–0.4264)	0.4264 (0.4264–0.4264)	0.4 (0.36–0.46)	1.58 (1.09–2.03)
2009–2010	Adults	All adults	2,127	2,127 (4.28%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.26 (0.25–0.28)	1.22 (1.04–1.33)
2009–2010	Adults	Females	1,040	1,040 (3.75%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.35 (0.32–0.37)	1.36 (1.22–1.65)
2009–2010	Adults	Males	1,087	1,087 (4.78%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.23 (0.22–0.24)	0.93 (0.85–1.04)
2009–2010	Adults	At or above poverty level	550	550 (3.82%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.26 (0.25–0.29)	1.12 (1–1.27)
2009–2010	Adults	Below poverty level	469	469 (4.69%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.25 (0.22–0.28)	1.27 (0.9–2)
2009–2010	Adults	Black non-Hispanic	400	400 (6.75%)	0.28 (0.28–0.28)	0.52 (0.28–0.76)	0.2 (0.18–0.22)	0.88 (0.65–1.12)
2009–2010	Adults	Mexican American	393	393 (6.11%)	0.28 (0.28–0.28)	0.42 (0.28–0.54)	0.25 (0.23–0.27)	1.12 (0.8–1.47)
2009–2010	Adults	Other	336	336 (3.87%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.28 (0.24–0.31)	1.33 (0.97–2.55)
2009–2010	Adults	Unknown income	905	905 (4.31%)	0.28 (0.28–0.28)	0.42 (0.28–4.12)	0.28 (0.22–0.33)	1.47 (1–2.33)
2009–2010	Adults	White non-Hispanic	998	998 (2.71%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.28 (0.25–0.3)	1.22 (0.97–1.34)
2009–2010	WRA	All women of reproductive age	608	608 (3.78%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.26 (0.25–0.28)	1.22 (1.04–1.33)
2009–2010	WRA	At or above poverty level	162	162 (3.09%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.26 (0.25–0.29)	1.12 (1–1.27)
2009–2010	WRA	Below poverty level	186	186 (3.23%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.25 (0.22–0.28)	1.27 (0.9–2)
2009–2010	WRA	Black non-Hispanic	113	113 (6.19%)	0.28 (0.28–0.28)	0.56 (0.28–0.92)	0.2 (0.18–0.22)	0.88 (0.65–1.12)

NHANES Cycle	Age Group	Subset	Sample Size	Detection Frequency[a]	50th Percentile (95%CI) (ng/mL)	95th Percentile (95% CI) (ng/mL)	Creatinine Corrected 50th Percentile (95%CI) (ng/mL)	Creatinine Corrected 95th Percentile (95% CI) (ng/mL)
2009–2010	WRA	Mexican American	102	102 (4.9%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.25 (0.23–0.27)	1.12 (0.8–1.47)
2009–2010	WRA	Other	116	116 (5.17%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.28 (0.24–0.31)	1.33 (0.97–2.55)
2009–2010	WRA	Unknown income	211	211 (5.21%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.28 (0.22–0.33)	1.47 (1–2.33)
2009–2010	WRA	White non-Hispanic	277	277 (1.81%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.28 (0.25–0.3)	1.22 (0.97–1.34)
2009–2010	Children	All children (3 to < 16 years)	622	622 (7.88%)	0.28 (0.28–0.28)	0.64 (0.46–0.86)	0.29 (0.25–0.32)	1.34 (1.04–1.87)
2009–2010	Children	Females	310	310 (7.42%)	0.28 (0.28–0.28)	0.52 (0.28–0.76)	0.34 (0.27–0.38)	1.47 (1.12–2.15)
2009–2010	Children	Males	312	312 (8.33%)	0.28 (0.28–0.28)	0.68 (0.28–0.86)	0.26 (0.23–0.29)	1.34 (0.78–2.22)
2009–2010	Children	Adolescents (11 to <16 years)	281	281 (6.41%)	0.28 (0.28–0.28)	0.42 (0.28–0.68)	0.23 (0.2–0.25)	1 (0.74–1.12)
2009–2010	Children	At or above poverty level	167	167 (7.78%)	0.28 (0.28–0.28)	0.68 (0.4–0.86)	0.27 (0.25–0.32)	1.34 (1–2.22)
2009–2010	Children	Below poverty level	186	186 (7.53%)	0.28 (0.28–0.28)	0.52 (0.28–0.88)	0.3 (0.24–0.39)	1.12 (0.76–2.15)
2009–2010	Children	Black non-Hispanic	116	116 (9.48%)	0.28 (0.28–0.28)	0.62 (0.28–1.33)	0.25 (0.19–0.31)	0.94 (0.65–2.04)
2009–2010	Children	Children (6 to <11 years)	341	341 (9.09%)	0.28 (0.28–0.28)	0.96 (0.62–1.91)	0.37 (0.34–0.43)	1.8 (1.4–3.11)
2009–2010	Children	Mexican American	173	173 (6.36%)	0.28 (0.28–0.28)	0.48 (0.28–0.64)	0.32 (0.28–0.36)	1.12 (0.78–1.87)
2009–2010	Children	Other	125	125 (8%)	0.28 (0.28–0.28)	0.64 (0.28–5.37)	0.31 (0.26–0.39)	2.55 (0.78–5.26)
2009–2010	Children	Unknown income	214	214 (8.88%)	0.28 (0.28–0.28)	0.28 (0.28–0.28)	0.25 (0.21–0.32)	1.87 (0.61–3.11)
2009–2010	Children	White non-Hispanic	208	208 (8.17%)	0.28 (0.28–0.28)	0.68 (0.28–0.96)	0.26 (0.24–0.34)	1.34 (0.98–2.22)

[a] After publication of data from the 1999–2000 and 2001–2002 NHANES cycles, CDC determined that the analytical standards used for MCHP were of insufficient purity and subsequently applied a correction factor to this data. As a result, the data for these years appears to be higher than the initial laboratory-derived values and are all above the detection limit of 0.93 ng/mL. The bulk of the MCHP values for these 2 cycles are 1.2792, which is likely the imputed value of non-detects after the application of the correction factor.

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

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th percentile	P-Value, 50th Percentile	Regression Coefficient, 95th Percentile	P-Value, 95th Percentile
1999–2010	MCHP	Adults	All adults	Age	sex race income	–	<0.001	–	<0.001
1999–2010	MCHP	Adults	All adults	Income	age sex race	–	0.0064	–	<0.001
1999–2010	MCHP	Adults	All adults	Race	age sex income	–	<0.001	–	<0.001
1999–2010	MCHP	Adults	All adults	Sex	age race income	–	0.2028	–	<0.001
1999–2010	MCHP	Adults	All adults	Years	age sex race income	–	<0.001	–0.0635	<0.001
1999–2010	MCHP	Adults	At or above poverty level	Years	age sex race	–	<0.001	–0.0378	<0.001
1999–2010	MCHP	Adults	Below poverty level	Years	age sex race	–	<0.001	–0.0378	<0.001
1999–2010	MCHP	Adults	Black non-Hispanic	Years	age sex income	–	<0.001	–0.0102	<0.001
1999–2010	MCHP	Adults	Females	Years	age race income	–	<0.001	–0.005	<0.001
1999–2010	MCHP	Adults	Males	Years	age race income	–	<0.001	–0.0920	<0.001
1999–2010	MCHP	Adults	Mexican-American	Years	age sex income	–	<0.001	–0.0568	<0.001
1999–2010	MCHP	Adults	Other	Years	age sex income	–	<0.001	–0.082	<0.001
1999–2010	MCHP	Adults	Unknown income	Years	age sex race	–	<0.001	–	<0.001
1999–2010	MCHP	Adults	White non-Hispanic	Years	age sex income	–	<0.001	–0.0840	<0.001
1999–2010	MCHP	Children	All children (<16 years)	Age	sex race income	–	0.0253	–	0.0041
1999–2010	MCHP	Children	All children (<16 years)	Income	age sex race	–	0.0021	–	0.6628
1999–2010	MCHP	Children	All children (<16 years)	Race	age sex income	–	<0.001	–	0.9094
1999–2010	MCHP	Children	All children (<16 years)	Sex	age race income	–	<0.001	–	<0.001
1999–2010	MCHP	Children	Adolescents (11 to <16 years)	Years	sex race income	–	<0.001	–0.0590	<0.001
1999–2010	MCHP	Children	Toddlers (3 to <5 years old)	Years	sex race income	–	<0.001	–0.0539	<0.001
1999–2010	MCHP	Children	Children (6 to <10 years old)	Years	sex race income	–	<0.001	–0.0012	0.6275
1999–2010	MCHP	Children	All children (<16 years old)	Years	age sex race income	–	<0.001	–0.0396	<0.001
1999–2010	MCHP	Children	At or above poverty level	Years	age sex race	–	<0.001	–0.0295	<0.001
1999–2010	MCHP	Children	Below poverty level	Years	age sex race	–	<0.001	–0.0939	<0.001
1999–2010	MCHP	Children	Black non-Hispanic	Years	age sex income	–	<0.001	–0.0921	<0.001
1999–2010	MCHP	Children	Females	Years	age race income	–	<0.001	–0.0511	<0.001
1999–2010	MCHP	Children	Males	Years	age race income	–	<0.001	–0.027	<0.001
1999–2010	MCHP	Children	Mexican-American	Years	age sex income	–	<0.001	–0.0986	<0.001
1999–2010	MCHP	Children	Other	Years	age sex income	–	<0.001	–0.024	<0.001

Years	Metabolite	Group	Subset	Regression Variable	Covariates	Regression Coefficient, 50th percentile	P-Value, 50th Percentile	Regression Coefficient, 95th Percentile	P-Value, 95th Percentile
1999–2010	MCHP	Children	Unknown income	Years	age sex race	–	<0.001	0.19326	<0.001
1999–2010	MCHP	Children	White non-Hispanic	Years	age sex income	–	<0.001	–0.0489	<0.001
1999–2010	MCHP	Women	All women of reproductive age	Age	sex race income	–	<0.001	–	<0.001
1999–2010	MCHP	Women	All women of reproductive age	Income	age sex race	–	1	–	<0.001
1999–2010	MCHP	Women	All women of reproductive age	Race	age sex income	–	0.0027	–	<0.001
1999–2010	MCHP	Women	All women of reproductive age	Sex	age race income	–	<0.001	–	<0.001
1999–2010	MCHP	Women	All women of reproductive age	Years	age sex race income	–	<0.001	0.0951	<0.001
1999–2010	MCHP	Women	At or above poverty level	Years	age sex race	–	<0.001	–0.0549	0.0146
1999–2010	MCHP	Women	Below poverty level	Years	age sex race	–	<0.001	0.04062	0.1413
1999–2010	MCHP	Women	Black non-Hispanic	Years	age sex income	–	<0.001	0.04821	0.0286
1999–2010	MCHP	Women	Females	Years	age race income	–	<0.001	0.0951	<0.001
1999–2010	MCHP	Women	Mexican-American	Years	age sex income	–	<0.001	0.28976	<0.001
1999–2010	MCHP	Women	Other	Years	age sex income	–	<0.001	−0.0832	0.1766
1999–2010	MCHP	Women	Unknown income	Years	age sex race	–	<0.001	0.84182	<0.001
1999–2010	MCHP	Women	White non-Hispanic	Years	age sex income	–	<0.001	–1	<0.001