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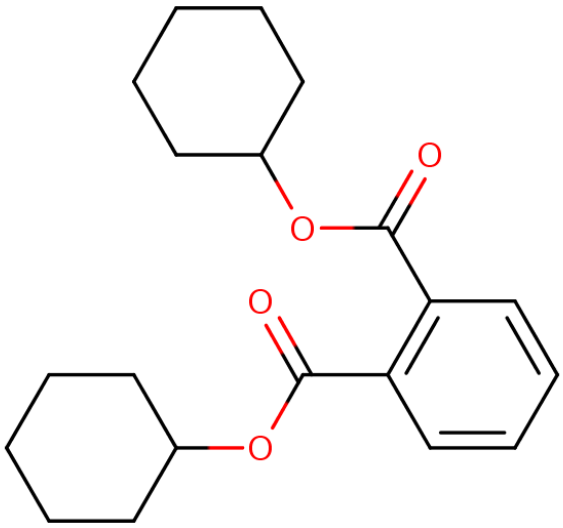
Draft Environmental Media and General Population and Environmental Exposure Assessment for Dicyclohexyl Phthalate (DCHP)

Technical Support Document for the Draft Risk Evaluation

CASRN 84-61-7



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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
BCF	Bioconcentration factor
CDC	Centers for Disease Control and Prevention (U.S.)
CEM	Consumer Exposure Model
COU	Condition of use
DAD	Dermal absorbed dose
DI	Daily intake
DCHP	Dicyclohexyl phthalate
ECHO	EPA's Enforcement and Compliance History Online database
F_{ue}	Fractional urinary excretion
IIOAC	Integrated Indoor-Outdoor Air Calculator Model
EPA	Environmental Protection Agency (U.S.) (or the Agency)
HEC	Human equivalent concentration
HED	Human equivalent dose
HM	Harmonic mean
IR	Ingestion rate
Koa	Octanol:air coefficient
Koc	Organic carbon:water partition coefficient
Kp	Dermal permeability coefficient
LADD	Lifetime average daily dose
MCNP	Mono-(carboxynonyl) phthalate
MOE	Margin of exposure
NAICS	North American Industry Classification System
NHANES	National Health and Nutrition Examination Survey
NPDES	National Pollutant Discharge Elimination System

199	OCSPP	Office of Chemical Safety and Pollution Prevention
200	OES	Occupational exposure scenario
201	OPPT	Office of Pollution Prevention and Toxics
202	PESS	Potentially exposed or susceptible subpopulation(s)
203	POD	Point of departure
204	PSC	Point Source Calculator tool
205	SD	Standard deviation
206	TRI	Toxics Release Inventory
207	TSCA	Toxic Substances Control Act
208	WWTP	Wastewater treatment plant

SUMMARY

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

EPA evaluated the reasonably available information for various environmental media concentrations and estimated exposure using a 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 are summarized below:

- EPA assessed environmental concentrations of DCHP in air, water, and land (soil, biosolids, and groundwater) for use in environmental exposure and general population exposure assessment.
  - For the land pathway, there are uncertainties in the relevance of limited monitoring data for biosolids and landfill leachate to the COUs considered. However, based on high-quality physical and chemical property data, EPA determined that 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 water pathway, DCHP released into water is expected to predominantly partition into sediment. The high-end modeled total water column concentration of DCHP for the acute human exposure scenarios was 126 µg/L, which 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.
  - While DCHP may persist in sediment, soil, biosolids, or landfills after release to these environments, DCHP's bioavailability is expected to be limited.
- Screening-level risk estimates using high-end modeled 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 high-end modeled water concentration were conducted because screening-level risk estimates indicated potential risks. In the refined scenarios, which are expected to be more representative of exposures than the high-end screening analysis, no risk was identified.
- EPA concluded that there are no exposure 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 document supports the *Draft Risk Evaluation for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024g). DCHP is a common chemical name for the category of chemical substances under one 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 document 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 *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c). Table 1-1 provides a crosswalk between COUs and occupational exposure scenarios (OESs). Table 1-2 shows the types of releases to the environment by OES.

Table 1-1. Crosswalk of Conditions of Use to 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
Disposal	Disposal	Disposal	Waste handling, treatment, and

Life Cycle Stage	Category	Subcategory	OES(s)
			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

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

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
Manufacturing	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
Non-PVC material compounding	Fugitive or stack air
	Fugitive air, water, incineration, or landfill
	Water, incineration, or landfill
	Water
	Incineration or landfill

OES	Type of Discharge,[a] Air Emission,[b] or Transfer for Disposal[c]
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 disposal	Releases to all media are possible but non-quantifiable due to a lack of identified process- and product-specific data

[a] Direct discharge to surface water; indirect discharge to non-POTW; indirect discharge to POTW
[b] Emissions via fugitive air or stack air, or treatment via incineration
[c] 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 *Draft Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f). Briefly, based on DCHP's fate parameters (*e.g.*, Henry's Law constant, log KOC, water solubility, fugacity modeling), EPA anticipated DCHP to be predominantly in 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.

Environmental exposures using the predicted concentrations of DCHP are presented in Section 12. 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. The Agency assumed that if there is no risk for an individual identified as having the potential for the highest exposure associated with a COU for a given pathway of exposure, then that pathway was determined not to be a major pathway of general population exposure and not pursued further. If any pathways were identified as a potential exposure pathway for the general population, further exposure assessments for that pathway would be conducted to include higher tiers of modeling when available, refinement of exposure estimates, and exposure estimates for additional subpopulations and COUs/OESs.

Table 1-3 summarizes the exposure pathways assessed for the general population. For DCHP, exposures to the general population via surface water, drinking water, fish ingestion, and ambient air were quantified, and modeled concentrations were compared to environmental monitoring data when possible. Exposures via the land pathway (*i.e.*, biosolids and landfills) were qualitatively assessed because 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 Table 1-3, biosolids, landfills, surface water, drinking water, fish ingestion, and ambient air are not pathways of concern for DCHP for highly exposed populations based on the OES leading to the highest concentrations of DCHP in environmental media.

272 **Table 1-3. Exposure Pathways Assessed for General Population Screening Level Assessment**
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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
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
PVC plastics compounding	Drinking water	Oral	Ingestion of drinking water (Section 6.1.1)	No
PVC plastics compounding	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	No
All			Ingestion of fish for subsistence fishers (Section 7.2)	No
All			Ingestion of fish for tribal populations (Section 7.3)	No
Application of paints and coatings	Ambient air	Inhalation	Inhalation of DCHP in ambient air resulting from industrial releases (Section 439.1)	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 *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c) and summarized in Table 1-2 of this assessment, 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.

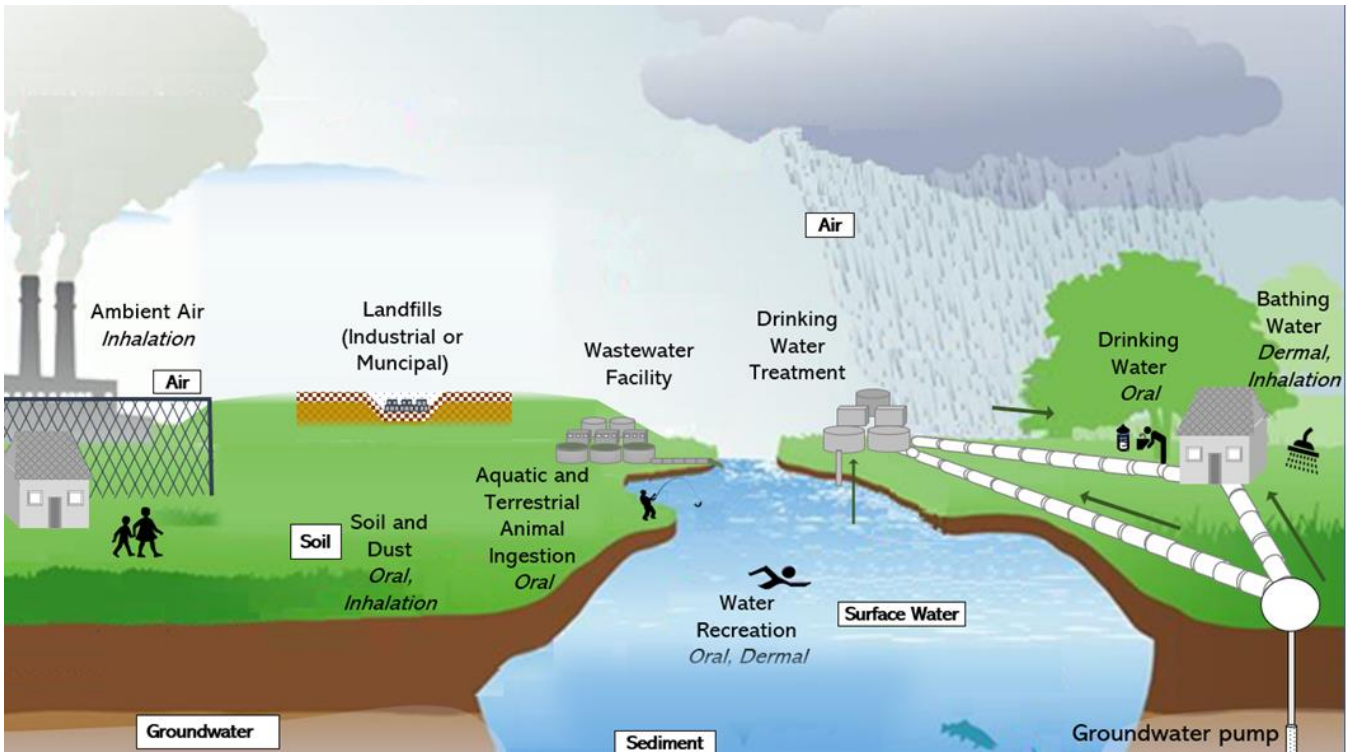

Figure 2-1. Potential Human Exposure Pathways for the General Population
The diagram presents the media (white text boxes) and routes of exposure (italics for oral, inhalation, or dermal) for the general population. Sources of drinking water from surface or water pipes is depicted with grey arrows.

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)
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	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	Drinking water	Oral	Ingestion of drinking water	Adults, youths, and children	Quantitative, Section 6.1.1
All	Fish ingestion	Oral	Ingestion of fish for General Population	Adults and children	Quantitative, Section 7.1
PVC plastics compounding			Ingestion of fish for subsistence fishers	Adults	Quantitative, Section 7.2
PVC plastics compounding			Ingestion of fish for tribal populations	Adults	Quantitative, Section 7.3
Application of paints and coatings	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, 2022, 10555664}. 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 and exposure estimates for additional subpopulations and COUs.

2.2 Margin of Exposure Approach

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

Equation 2-1. Margin of Exposure Calculation

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

Where:

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

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

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

Table 2-2. Non-cancer 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; MOE = margin of exposure; NOAEL = no-observed-adverse-effect level; LOAEL = lowest-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.

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

3 LAND PATHWAY

EPA searched peer-reviewed literature, gray literature, and databases of environmental monitoring data 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 relevant compartments including leachate, activated sludge, and biosolids. 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 since 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 was 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 will be discussed in further depth 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. Three Chinese studies, however, 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). 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 to 0.0836 mg/kg), comprising less than 1 percent of the total phthalate concentration in biosolids (Zhu et al., 2019). A 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 to 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 to 1350 mg/kg) (Meng et al., 2014).

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). 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). However, like 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 (the 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, will 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 pre-treatment, 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 (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, 2024f). 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 and 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.

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

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

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 was 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, 2024f). 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 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. 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 Conclusion

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. Further, 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 was detected in surface water and in aquatic sediments. However, 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 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 *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c). 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.

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, 2024c), 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 model (see Table 4-1). Selected values are described in detail in the *Draft Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f). A half-life based on anaerobic sediment was selected for the benthic half-life input as a more protective value than the aerobic sediment value, and in consideration of the potential for lower levels of oxygen in benthic sediments impacted by industrial releases. In addition to the values described in the *Draft Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f), the PSC model relies on the Heat of Henry parameter, which was estimated from temperature variation of the Henry's Law constant calculated by HENRYWIN™ in EPI Suite™ (U.S. EPA, 2015b).

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

Parameter	Value[a]
K_{OC}	29,512 mL/g
Water Colum 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

Parameter	Value[a]
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 *Draft Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)*(U.S. EPA, 2024f).	

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

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

Parameter	Value
DFAC (represents the ratio of vertical path lengths to depth as defined in EPA's exposure analysis modeling system [EXAMS]) (U.S. EPA, 2019c))	1.19
Water column suspended sediment	30 mg/L
Chlorophyll	0.005 mg/L
Water column f_{oc} (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, 2024c). Without TRI and DMR data, EPA cannot identify the receiving water bodies and their location-specific hydrological flow data. EPA instead generated a distribution of flow metrics by collecting flow data for facilities across a North American Industry Classification System (NAICS) code associated with each COU for a 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 National

Pollutant Discharge Elimination System (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.

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

For each COU with surface water releases of wastewater effluent, surface water release values from the PVC plastics compounding OES (the OES with the highest estimated release to surface water) were used as a conservative screening analysis (Table 4-3). 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 waterbody 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, with treated effluent showing up to a 68.6 percent reduction in one study (Wu et al., 2019). Modeling results are shown in Table 4-3. This analysis resulted in high estimated concentrations in the receiving waterbody and sediment because of a high-end release amount combined with lower hydrologic flow and without consideration of wastewater treatment. These values are carried through to the ecological risk assessment for further evaluation as a conservative high-end approach to screen for ecological risk discussed in Section 12.

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

OES	Number of Operating Days Per Year[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)
PVC plastics compounding	254	6.13	165	95.3	112,000

[a] Details on operating days and daily releases are provided in Draft *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c).

The OES with the highest total water column concentrations (PVC plastics compounding) was additionally run under the 50th percentiles of 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 waterbody.

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)
PVC plastics compounding *Without Wastewater Treatment*	6.13	69,800	48,600	0.00	87.7	126
PVC plastics compounding *With Wastewater Treatment*	6.13	69,800	48,600	68.6	27.5	39.6

[a] Details on modeling release estimates are provided in Draft *Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c).

4.2 Measured Concentrations

4.2.1 Measured Concentrations in Surface Water

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
WA DOE (2022)	Washington, United States	ND (<0.5 µg/L)	Freshwater samples from 16 lakes and rivers across WA and marine samples from the Puget Sound
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–3 ng/L *Detection limits NR*	Marine waters at 66 samples in Puget Sound, WA and 22 samples in Barkley Sound, BC, 2010
FOD = frequency of detection; ND = not-detect; NR = not reported			

4.2.2 Measured Concentrations in Sediment

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
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
Lin et al. (2003)	Canada	ND *Detection limits NR*	16 surficial sediments from False Creek, Vancouver, BC, date NR
dw = dry weight; FOD = frequency of detection; ND = not-detect; 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 waterbody 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 model. A standard EPA waterbody was used to represent a consistent and conservative receiving waterbody scenario. Uncertainty associated with location-specific model inputs (*e.g.*, flow parameters and meteorological data) is present as no facility locations were identified for 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 which were applied as inputs to the surface water modeling conducted in this assessment.

The modeled data represent estimated surface water (water column, benthic porewater, and sediment) concentrations near facilities that are actively releasing 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 over-estimation of resulting environmental concentrations due to conservative assumptions that remain protective where there are uncertainties in release details.

4.4 Weight of Scientific Evidence Conclusions

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

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

5 SURFACE WATER EXPOSURE

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 0) and fish ingestion exposure (Section 7).

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

5.1 Modeling Approach

5.1.1 Dermal 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 0. EPA used the dermal permeability coefficient (K_p) (0.012 cm/hr) (U.S. EPA, 2024b). EPA used the Consumer Exposure Model (CEM), Version 3.2 (U.S. EPA, 2022d) 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 PVC Plastics compounding OES (Table 4-5) as the surface water concentration. Dose values are presented both with and without a wastewater treatment removal efficiency of 68.6 percent applied. Dermal doses were also calculated using the highest values from ambient surface water monitoring data (Section 4.2.1) as the surface water concentration. Doses calculated using the surface water monitoring data are three to four orders of magnitude lower than corresponding doses modeled using the high-end PVC Plastics compounding OES.

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)
PVC Plastics compounding[a] *Without Wastewater Treatment*	126	87.7	1.1E–03	2.1E–06	8.5E–04	1.6E–06	5.1E–04	9.8E–07
PVC Plastics compounding[a] *With Wastewater Treatment*	39.6	27.5	3.5E–04	6.6E–07	2.7E–04	5.1E–07	1.6E–04	3.1E–07
Highest monitored surface water[b]	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] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] 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.

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.

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

The following equations were used to calculate incidental oral (swimming) doses for adults, youth, and children using the 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 0. 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)
PVC plastics compounding[a] *Without Wastewater Treatment*	126	87.7	4.3E–04	8.3E–07	6.7E–04	1.3E–06	3.8E–04	7.3E–07
PVC plastics compounding[a] *With Wastewater Treatment*	39.6	27.5	1.4E–04	2.6E–07	2.1E–04	4.0E–07	1.2E–04	2.3E–07
Highest monitored surface water[b]	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] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] 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.

5.2 Weight of the 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, 2024c). Due to uncertainties inherent in this approach, conservative assumptions and methods were utilized to evaluate an upper bounding limit to be applied as a protective screening assessment. As stated in Section 4.4 there is moderate confidence in the modeled concentrations as being representative of actual releases, with a bias toward over-estimation. Screening-level risk estimates derived from the exposures modeled in this section are discussed in Appendix C and demonstrate no risk estimates to the general population below the benchmark. The screening approach applied for modeling, in conjunction with the available monitoring data showing lower concentrations than those modeled, provide multiple lines of evidence and robust confidence that releases to surface water will not exceed the release concentrations presented in this assessment, which do not appear to pose risk to human health.

Swimming Ingestion/Dermal Estimates

Two scenarios (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

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.

Very limited information is available on the removal of DCHP in drinking water treatment plants, as stated in the *Draft Chemistry, Fate, and Transport Assessment for Dicyclohexyl Phthalate (DCHP* (U.S. EPA, 2024f). 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.

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, only the highest modeled facility release was included in the drinking water exposure assessment, alongside the highest monitored surface water concentration. Drinking water doses were calculated using the following equations:

Equation 6-1. Acute Drinking Water Ingestion Calculation

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

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. The lifetime average daily dose (LADD) was not estimated because available data are insufficient to determine the carcinogenicity of DCHP (see *Draft Non-cancer Human Health Hazard Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024e)). Therefore, EPA is not evaluating DCHP for carcinogenic risk. Table 6-1 summarizes the drinking water doses for adults, infants, and toddlers for a scenario applying no wastewater treatment and for a scenario applying only wastewater treatment. Exposure estimates are low for all lifestages and scenarios, including for infants with the highest drinking water intake per body weight and assuming no wastewater treatment is applied.

Table 6-1. Drinking Water Doses across Lifestages

Scenario	Surface Water Concentrations		Adult (21+ years)		Infant (birth to < 1 year)		Toddler (1–5 years)	
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)	ADR_{POT} (mg/kg-day)	ADD (mg/kg-day)
Plastics compounding[a] *Without Wastewater Treatment*	126	87.7	5.1E–03	2.6E–06	1.8E–02	6.7E–06	6.3E–03	2.9E–06
Plastics compounding[a] *With Wastewater Treatment*	39.6	27.5	1.6E–03	8.3E–07	5.6E–03	2.1E–06	2.0E–03	9.1E–07
Highest monitored surface water[b]	0.014	0.014	5.6E–07	4.2E–10	2.0E–06	1.1E–09	7.0E–07	4.6E–10

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

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 D, and suggest no expected risk below the benchmark MOE at the upper bound of exposure.

6.3 Weight of the Scientific Evidence Conclusions

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

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

7 FISH INGESTION EXPOSURE

To estimate exposure to humans from fish ingestion, EPA used the upper limit of DCHP's solubility in water (1.48 mg/L) modeled using EPI Suite™ (see *Draft Chemistry, Fate, and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f)) as a screening approach. 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. The BAF is another important parameter when estimating human exposure to a chemical from fish ingestion. A BAF is preferred over a 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 *Draft Chemistry, Fate, and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f)). Table 7-1 compares the fish tissue concentration calculated using a BAF with the measured fish tissue concentrations obtained from literature. For comparison, Table 7-1 also includes fish tissue concentrations that were derived from a BCF of 708 L/kg using a regression-based method (U.S. EPA, 2024f). Fish tissue concentration calculated with a predicted BAF and upper-bound water solubility limit was lower than the concentration calculated with a predicted BCF, but up to three orders of magnitude higher than empirical levels reported within published literature.

Surface water concentrations for DCHP associated with PVC plastics compounding (the OES with the highest value for DCHP release to water) were modeled using VVWM-PSC as described in Section 4.1, to represent the upper-bound of DCHP concentration in receiving waters. Table 7-1 compares DCHP concentrations in fish tissue using the harmonic mean of the modeled surface water concentrations based on the highest modeled 95th percentile release and 50th percentile flow metric distribution (P50). Modeled DCHP concentrations in fish tissue are up to two orders of magnitude greater than the highest DCHP concentrations reported within aquatic biota (Table 7-1).

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

Data Approach	Data Description	Surface Water Concentration	Fish Tissue Concentration
Water solubility limit	Predicted BCF (regression-based) of 708 L/kg (U.S. EPA, 2017)	1.48 mg/L (U.S. EPA, 2017)	1.04E03 mg/kg
	Predicted BAF (Arnot-Gobas method) of 67 L/kg (U.S. EPA, 2017)	1.48 mg/L (U.S. EPA, 2017)	9.92E01 mg/kg
Modeled surface water concentration	Predicted BCF (regression-based) of 708 L/kg (U.S. EPA, 2017)	0.087 mg/L (harmonic mean, P50 flow distribution)	6.17E01 mg/kg
	Predicted BAF (Arnot-Gobas method) of 67 L/kg (U.S. EPA, 2017)	0.087 mg/L (harmonic mean, P50 flow distribution)	5.88 mg/kg
Monitored surface water concentration	Highest measured concentration from a U.S. study (Keil et al., 2011) and predicted BAF (Arnot-Gobas method) of 67 L/kg (U.S. EPA, 2017)	1.00E–05 mg/L	9.38E–04 mg/kg
Fish tissue monitoring data (wild-caught)	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

Data Approach	Data Description	Surface Water Concentration	Fish Tissue Concentration
	One Chinese study collected 207 fish samples across 17 different species (Hu et al., 2020)		<LOD to 2.91E–01 mg/kg
ww = wet weight			

7.1 Exposure Due to Fish Ingestion for General Population

EPA estimated exposure from fish consumption using age-specific ingestion rates (Table_Apx A-2). Adults have the highest 50th percentile fish ingestion rate (IR) per kilogram of body weight for the general population, as shown in Table_Apx A-2. A young toddler between 1 and 2 years 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, lifetime average daily dose) and risks were not characterized because there is insufficient evidence of DCHP's carcinogenicity (U.S. EPA, 2024e). 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 *Draft Fish Ingestion Risk Calculator for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024d). The number of years within an age group (*i.e.,* 62 years for adults) was used for the exposure duration and averaging time to estimate non-cancer exposure. The exposures calculated using the water solubility limit and BAF are presented in Table 7-2. Corresponding screening-level risk estimates are shown in Appendix E.1. Fish ingestion is not expected to be a pathway of concern for the general population based on the conservative screening-level risk estimates using an upper-bound of exposure.

Table 7-2. General Population Fish Ingestion Doses

	Adult ADR (mg/kg-day)	Young Toddler ADR (mg/kg-day)	Adult ADD (mg/kg-day)
Water solubility limit (1.48 mg/L)	2.75E–02	4.09E–02	6.25E–03

7.2 Exposure due to Fish Ingestion for Subsistence Fishers

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.

Conservative exposure estimates based on the water solubility limit resulted in screening-level risk estimates below the benchmark as described in Appendix E.2. Therefore, EPA subsequently refined its evaluation by using modeled surface water concentrations based on the highest estimated 95th percentile release for the PVC plastics compounding OES as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c) and the 50th percentile flow. This refined analysis did not result in screening-level risk estimates below the benchmark. Therefore, ingestion of fish potentially contaminated with DCHP is not expected to be a pathway of concern for the subsistence fisher.

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

	ADR/ADD (mg/kg-day)
Water solubility limit (1.48 mg/L)	1.77E–01
Modeled surface water concentration for PVC plastics compounding, P50 flow, Untreated (0.087 mg/L)	1.05E–02

7.3 Exposure due to Fish Ingestion for Tribal Populations

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

U.S. EPA (2011a) (see Chapter 10, Table 10-6) summarizes relevant studies on current tribal-specific fish ingestion rates that covered 11 tribes and 94 Alaskan communities. The daily ingestion rates for the

94 Alaskan communities are reported as a minimum, median, and maximum. However, those values were not considered because the study did not report the sampled age group, which precludes calculation of an ingestion rate per kilogram of body. The median value is also lower than the mean ingestion rate per kilogram of body weight 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. This value is also the highest among all central tendency values in the *Exposure Factors Handbook* (U.S. EPA, 2011a). In comparison, the ingestion rates for the adult subsistence fisher and 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).

Because current fish consumption rates are suppressed by contamination, degradation, or loss of access, EPA reviewed existing literature for ingestion rates that reflect heritage rates. Heritage rates refer to those that existed prior to non-indigenous settlement on tribal fisheries resources, as well as changes in culture and lifeways (U.S. EPA, 2016). Heritage ingestion rates were identified for four tribes, all located in the Pacific Northwest region. The highest heritage ingestion rate was reported for the Kootenai Tribe in Idaho at 1,646 g/day (RIDOLFI, 2016) (that study was funded through an EPA contract). The authors 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 and using the average caloric value for fish. Notably, the authors acknowledged that assuming 75 percent of caloric intake comes from fish may overestimate fish intake.

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

Table 7-4 presents multiple exposure estimates for the tribal populations. Conservative exposure estimates based on the water solubility limit resulted in screening-level risk estimates below the benchmark, as described in Appendix E.3. Therefore, EPA refined its evaluation by using modeled surface water concentrations based on the (1) highest estimated 95th percentile release for the PVC plastics compounding OES as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c); (2) treated wastewater; and (3) untreated wastewater using the P50, P75, and P90 flow metrics from the distribution. The higher flow metrics are expected to be more representative of the flow conditions associated with high-end releases. The more refined exposure estimates did not result in risk estimates below the benchmarks (see Appendix E.3). In addition, exposure estimates using modeled surface water concentrations are at least

one order of magnitude higher than those using the maximum monitored surface water concentration. This indicates that modeled concentrations are conservative. Overall, ingestion of fish potentially contaminated with DCHP is not expected to be a pathway of concern for tribal populations.

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

	ADR/ADD (mg/kg-day)	
	Current IR	**Heritage IR**
Water solubility limit (1.48 mg/L)	2.68E–01	2.04
Modeled surface water concentration for PVC plastics compounding, P50 flow (0.087 mg/L)	1.59E–02	1.21E–01
Modeled surface water concentration for PVC plastics compounding, P75 flow (3.48E–03 mg/L)	6.30E–04	4.80E–03
Modeled surface water concentration for PVC plastics compounding, P90 flow (2.4E–04 mg/L)	4.40E–05	3.35E–04
Modeled surface water concentration for PVC plastics compounding, P50 flow, Treated (2.7E–02 mg/L)	4.97E–03	3.79E–02
Highest monitored surface water concentration (1.0E–05 mg/L)	2.53E–06	1.93E–05

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 either the water solubility limit or modeled concentrations based on the P50 flow resulted in risk estimates below the benchmark only for tribal populations (see Appendix E). EPA refined its analysis for tribal populations by incorporating higher flow rates and treatment efficiency because of large differences between modeled and measured surface water and fish tissue concentration data. As shown in Equation 7-1, surface water concentration of DCHP is a key input for calculating exposure through fish ingestion. When modeling with PSC, EPA assumed all releases were directly discharged to surface waters without prior treatment on-site or routed through publicly owned treatment works prior to release. This assumption, coupled with high-end (95th percentile) release scenarios and P50 flow distribution, likely overestimates modeled concentrations. EPA expects high-end releases to discharge to surface waters with higher flow conditions like P75 or P90.

Lastly, it is critical to note that DCHP is expected to have low potential for bioaccumulation, biomagnification, and uptake by aquatic organisms because of its low water solubility and high hydrophobicity. Additional details are provided in Section 12. 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 over-estimation and bioconcentration,

bioaccumulation, and trophic transfer of DCHP is not expected, EPA has robust confidence that fish ingestion is not a pathway of concern for all populations.

8 AMBIENT AIR CONCENTRATION

EPA considers both modeled and monitored concentrations in the ambient air for this draft ambient air exposure assessment for 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 the Integrated Indoor/Outdoor Air Calculator (IIOAC) Model to estimate daily-average and annual-average concentrations of DCHP in the ambient air. IIOAC is a spreadsheet-based tool that estimates outdoor air chemical concentrations using pre-run results from a suite of dispersion scenarios in a variety of meteorological and land-use settings within EPA's American Meteorological Society/EPA Regulatory Model (AERMOD). Additional information on IIOAC can be found in the user guide (U.S. EPA, 2019d).

In line with previously peer-reviewed methodology for fenceline communities (U.S. EPA, 2022b), EPA's analysis with IIOAC estimates ambient concentrations of DCHP at three distances (*e.g.*, 100; 100 to 1,000, and 1,000 meters) from the releasing facility. EPA uses the maximum estimated release across all COUs from the *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c) as a screening-level assessment for inhalation exposure via the ambient air pathway.

8.1.1 Release and Exposure Scenarios Evaluated

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

- Release: Maximum Release (kg/site-day)
- Release Dataset: Estimated releases (no TRI or National Emissions Inventory [NEI] release data reported) as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c)
- Release Type: Stack and Fugitive
- Distances Evaluated: 100, 100–1,000, and 1,000 m
- Meteorological Stations (selected to represent high-end and central tendency meteorologic data based on a sensitivity analysis of the 14 meteorological stations included within the IIOAC model which tended to result in high-end (more conservative) and central tendency modeled concentrations):
    - South (Coastal):
        - Surface and Upper Air Stations at Lake Charles, Louisiana
    - West North Central:
        - Surface Station at Sioux Falls, South Dakota
        - Upper Air Station, Omaha, Nebraska
- Operating Scenario: 250 days per year; 24 hr/day and 8 hr per day to identify the scenario resulting in the maximum ambient air concentration
- Topography: Urban and Rural
- Particle Size:
    - Coarse (PM_{10}): Particulate matter with an aerodynamic diameter of 10 µm
    - Fine ($PM_{2.5}$): Particulate matter with an aerodynamic diameter of 2.5 µm

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 (degrees)	0
Release height (m)	3.05

8.1.2 IIOAC Model Output Values

The IIOAC model provides multiple output values (see *Draft Ambient Air Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024a)). A description of select outputs relied upon in this assessment are provided below. These outputs were relied upon because they represent a more conservative exposure scenario where modeled concentrations are expected to be higher; thus, more protective of exposed populations and ensuring potential high-end exposures are not missed during screening for the ambient air pathway.

Fenceline Average: represents the daily-averaged and annual-averaged 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 meters.

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

8.1.3 Modeled Results from IIOAC

All results for each scenario described in Section 8.1.1 are included in the *Draft Ambient Air Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024a). EPA used the highest estimated concentrations across all modeled scenarios to evaluate exposures near a releasing facility. This conservative exposure scenario represents a national level exposure estimate inclusive of sensitive and locally impacted populations who live next to a releasing facility.

The IIOAC Model provides source apportioned concentrations (fugitive and stack) based on the respective releases. To evaluate exposures for this ambient air assessment, EPA assumes the fugitive and stack releases occur simultaneously throughout the day and year. Therefore, the total concentration used to evaluate exposures and derive risk estimates in this ambient air assessment is the sum of the separately modeled fugitive and stack concentrations 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 below.

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	3.01	2.06
Stack	64.56	44.22
Total	67.57	46.28

8.2 Measured Concentrations in Ambient Air

EPA reviewed published literature as part of its Systematic Review process, as described in the *Draft Systematic Review Protocol for Dicyclohexyl Phthalate* (U.S. EPA, 2024h) to identify studies where ambient air concentrations of DCHP were measured. The available data found was limited to three foreign studies (Spain, South Korea, and China) that are summarized in Appendix F. Two studies looked at ambient air while one looked at water concentrations. Although EPA looked for U.S. studies that may be associated with TSCA COUs, in this case there were no U.S. studies identified in systematic review. As such, EPA considered references to foreign monitoring studies that received a medium or high-quality rating. Measured concentrations of DCHP in ambient air in these foreign studies were low; concentrations were in the ng/m^3 range or lower, suggesting DCHP in outdoor air is not a major exposure pathway. However, it is important to acknowledge that the relevance of these foreign studies to reflect sources and ambient air concentrations in the United States is unknown, limiting the utility of these data to this assessment.

Specifically, the information needed to link the monitoring data to foreign industrial processes and crosswalk those to processes occurring in the United States is not available. Furthermore, regulations of emissions standards often vary between the United States and foreign countries, which is also an uncertainty in considering foreign monitoring data. Information, on the proximity of the monitoring site to a releasing facility is also unknown. The measured data also cannot be tied to TSCA COUs.

EPA also reviewed EPA's Ambient Monitoring Technology Information Center archive but did not locate any monitored DCHP concentrations (U.S. EPA, 2022a).

8.3 Evidence Integration

Modeled DCHP ambient air concentrations are higher than measured concentrations outside the United States (Section 8.2). Measured concentration values can be found in Appendix F. This is an expected outcome since EPA's modeling uses high-end releases, and conservative meteorological data. Furthermore, the Agency considers high-end modeled concentration near a releasing facility (100 m). The distances of the sampling sites from the monitoring studies to releasing facilities is unknown.

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

The approach and methodology used and presented in this ambient air exposure assessment replicates previously peer reviewed approaches and methods and incorporates feedback received. EPA has robust confidence in the IIOAC modeling and use of the screening approach and its associated results to characterize exposure to DCHP from nearby releasing facilities. The approach and methodology have undergone peer review. EPA relies upon results from a more conservative, high-end exposure scenario consisting of high-end and mean meteorological scenario, maximum release scenario across all reported releases and TSCA COUs, high-end (95th percentile) modeled concentrations at 100 m from the releasing facility. These conditions ensure results from EPA's screening approach represent, and do not

miss potential high-end exposures.

Limitations of the approaches and methods used for modeling are generally associated with overall limitations of IIOAC. For example, IIOAC modeling is based on pre-run scenarios within AERMOD. As such, default input parameters for IIOAC are confined to those input parameters utilized for those pre-run AERMOD scenarios and cannot be changed. The default inputs include default stack parameters, 2011 to 2015 meteorological data, and the lack of site-specific information like building dimensions, stack heights, elevation, and land use.

Another limitation is the use of estimated releases as direct inputs to the IIOAC Model. DCHP did not have any reported releases in the databases EPA typically relies upon for facility reported release data (*e.g.,* TRI or NEI). Therefore, releases of DCHP from facilities estimated using generic scenarios were used. These estimated releases have limitations and uncertainties as described in the *Draft Environmental Release and Occupational Exposure Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024c), which carried over to this draft assessment.

8.4 Weight of Scientific Evidence Conclusions

Although certain assumptions were used and uncertainties exist, EPA has an overall robust confidence that the modeled results used for screening the ambient air pathway do not miss potential high-end exposures and are protective of both environmental and general population exposures. Additionally, the Agency has robust confidence in the modeled results because they were obtained through peer reviewed models and methodologies, and the results represent potential exposures at a distance where populations have been observed to live or reside for many years.

9 AMBIENT AIR EXPOSURE

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 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).

9.2 Overall Conclusions

Based on the results from the analysis of the maximum estimated release and high-end modeled exposure concentrations presented in this document, the derived risk estimates for this high-end, conservative, and protective scenario did not indicate concern that adverse effects would result from exposures to DCHP from industrial releases via the ambient air pathway (See the *Draft Ambient Air IIOAC Exposure Results and Risk Calcs* supplemental file (U.S. EPA, 2024a)). Based on this finding, EPA determined more refined modeling for estimated exposures (under less conservative/protective exposure scenarios) was not warranted and therefore did not pursue more refined modeling for estimated exposures for DCHP via the ambient air pathway, inhalation route.

10 HUMAN MILK EXPOSURES

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, 2024e). 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 *Draft Non-cancer Human Health Hazard Assessment for Dicyclohexyl Phthalate* (U.S. EPA, 2024e)). 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, 2024e). 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

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 NHANES urinary biomonitoring data indicates recent phthalate exposure.

EPA analyzed urinary biomonitoring data from the U.S. Centers for Disease Control and Prevention (CDC) 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 old), adults (16+ years old), adolescents (11 to <16 years old), and children (6 to <11 years old).

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 (see Appendix G for further details). 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, 2024h) 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, two 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.

1414 **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
Pollack et al. (2014)	Plastics and Personal-care Products use in Pregnancy (P4) study	473	0.2	5%
Arbuckle et al. (2016)	Endometriosis, Natural history, Diagnosis, and Outcomes (ENDO) Study	80	0.2	0%
Fisher et al. (2015)		80	0.2	Not reported
Bae et al. (2015)	Longitudinal Investigation of Fertility and the Environment (LIFE) Study	95	0.2–1.0[a]	5%
Shapiro et al. (2015)	Maternal–Infant Research on Environmental Chemicals (MIREC) Study	1,152	Not reported	<25%
Haines et al. (2016)	Canadian Health Measures Survey (CHMS)	3,237	0.2 [b]	13% [b]
		3,235	0.09 [c]	28% [c]
Health Canada (2013)	N/A	40	0.98–1.57 [a]	3%
Buckley et al. (2012)	Right From The Start (RFTS) study	50	0.28	2%
Philips et al. (2020)	Generation R Study	1,192	0.008–0.3 [a]	19%

[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

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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 *Draft 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 *Draft Systematic Review Protocol for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024h). 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 this study.

12.2 Trophic Transfer

EPA does not expect DCHP to persist in surface water, groundwater, or air (see Section 4.4 in the *Draft Chemistry, Fate, and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024f). 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, 2024f). 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, 2024f). The bioconcentration and bioaccumulation factors 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 modelling analysis of the trophic transfer of DCHP through food webs.

12.3 Weight of Scientific Evidence

Given the reasonably available data, EPA has robust confidence that (1) DCHP is not readily found or if found is in relatively low concentrations in organism tissues, (2) DCHP 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/modelled 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 GENERAL POPULATION AND ENVIRONMENTAL EXPOSURE

13.1 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 while 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
PVC plastics compounding *Without Wastewater Treatment*	Water	Surface water (30Q5, median flow)	126 µg/L
		Surface water (harmonic mean, median flow)	87.7 µg/L
PVC plastics compounding *With Wastewater Treatment*	Water	Surface water (30Q5, median flow)	39.6 µg/L
		Surface water (harmonic mean, median flow)	27.5 µg/L
Application of paint and coatings	Fugitive air	Daily-averaged total (fugitive and stack, 100m)	67.57 µg/m^3
		Annual-averaged total (fugitive and stack, 100m)	46.28 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[a]	Exposure Pathway	Exposure Route	Exposure Scenario	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
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
PVC plastics compounding	Drinking water	Oral	Ingestion of drinking water (Section 0)	Infant (<1 year)	No
All	Fish ingestion	Oral	Ingestion of fish for general population (Section 7.1)	Adult (21+ years)	No
PVC plastics compounding			Ingestion of fish for subsistence fishers (Section 7.2)	Adult (21+ years)	No
PVC plastics compounding			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 was equal to or exceeded the benchmark MOE of 30.

13.2 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.

13.3 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, 2024, 11799641}.

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 available study, DCHP concentrations measured in the water column did not exceed 0.014 µg/L {Kiel, 2011, 788135}. Monitoring by the Washington State Department of Ecology resulted in no DCHP detection above the detection limit (0.05 µg/L) {Washington State Department of Ecology, 2022, 11784545}. 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, 2019, 5442818@@author-year}). DCHP is readily biodegradable in soil with an aerobic half-life of 8.1 to 16.8 days in shallow, moist soils {EC/HC, 2015, 3688160; NLM, 2020, 6629414}. 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 to 1480 µg/L) and hydrophobicity (log Kow = 4.82, log Koc = 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 is not readily found, or if found, is in relatively low concentrations in

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

organism tissues, and that DCHP has low bioaccumulation and biomagnification potential in aquatic and terrestrial organisms. Therefore, there is low potential for trophic transfer through food webs.

13.4 Weight of the 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 **Error! Reference source not found.**). EPA summarized its weight of scientific evidence using confidence descriptors: robust, moderate, slight, or indeterminate confidence descriptors. EPA used general considerations (i.*e*., relevance, data quality, representativeness, consistency, variability, uncertainties) as well as chemical-specific considerations for its weight of scientific evidence conclusions.

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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)

1806 **Table_Apx A-3. Recommended Default Values for Common Exposure Factors**

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
ED	Exposure duration (hrs/day)	8	24	
EF	Exposure frequency (days/year)	250	365	
EY	Exposure years (years)	40	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)

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
$IR_{dw\text{-}chronic}$	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/hr)		0.025 Adult 0.05 Child (6 to < 16 years)	Evaluation of Swimmer Exposures Using the SWIMODEL Algorithms and Assumptions (U.S. EPA, 2015a)
IR_{fish}	Fish ingestion rate (g/day)		22 Adult	Estimated Fish Consumption Rates for the U.S. Population and Selected Subpopulations (U.S. EPA, 2014) This represents the 90th percentile consumption rate of fish and shellfish from inland and nearshore waters for the U.S. adult population 21 years of age and older, based on NHANES data from 2003–2010
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

Symbol	Definition	Recommended Default Value	Recommended Default Value	Source
		Occupational	Residential	
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/hr			CEM estimate aqueous K_p	(U.S. EPA, 2022d)

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

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

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Appendix B ESTIMATING HYDROLOGICAL FLOW DATA FOR SURFACE WATER MODELING

A distribution of flow metrics was generated by collecting flow data for facilities across a NAICS code associated with conditions of use for DCHP-releasing facilities (Table 4-3). EPA's ECHO database was accessed via the Application Programming Interface (API) and queried for facilities regulated under the Clean Water Act within the one relevant NAICS code (U.S. EPA, 2022c). All available NPDES permit IDs were retrieved from the facilities returned by the query. An additional query of the DMR REST service was conducted via the ECHO API to return the National Hydrography Dataset Plus (NHDPlus) reach code associated with the receiving waterbody for each available facility.

Modeled flow metrics were then extracted for the retrieved reach codes from the NHDPlus V2.1 Flowline Network's EROM Flow database. The EROM database provides modeled monthly average flows for each month of the year. While the EROM flow database represents averages across a 30-year time period, the lowest of the monthly average flows was selected as a substitute for the 30Q5 flow used in modeling, as both approximate the lowest observed monthly flow at a given location. The substitute 30Q5 flow was then added into the regression equation used by the EPA's Exposure and Fate Assessment Tool, Version 2014 (E-FAST) (U.S. EPA, 2007) to convert between these flow metrics and solved for the 7Q10 using Equation_Apx B-1. E-FAST is a dilution-based model that estimates chemical concentrations in surface water concentrations for use in general population and aquatic exposure assessments. In previous assessments, the EPA has selected the 7Q10 flow as a representative low flow scenario for biological impacts due to effluent releases into streams, while the harmonic mean represents long-term flow for assessing chronic drinking water exposure.

Equation_Apx B-1. Calculating the 7Q10 Flow

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

Where:

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

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

Equation_Apx B-2. Calculating the Harmonic Mean Flow

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

Where:

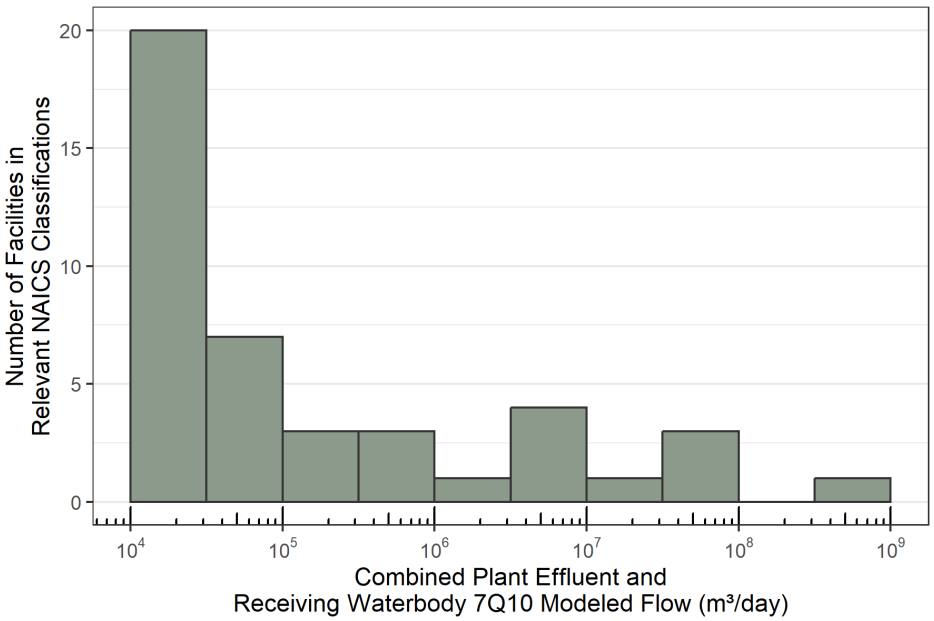
HM = Modeled harmonic mean flow, in MLD
AM = Annual average flow from NHD, in MLD

$7Q10$= Modeled 7Q10 flow from the previous equation, in MLD

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

NAICS Code	NAICS Name
325199	All Other Basic Organic Chemical Manufacturing

In addition to the hydrologic flow data retrieved from the NHDPlus database, information about the facility effluent rate was collected, as available, from the ECHO API. A minimum effluent flow rate of six cubic feet per second, derived from the average reported effluent flow rate across facilities, was applied. The receiving waterbody 7Q10 flow was then calculated as the sum of the hydrologic 7Q10 flow estimated from regression and the facility effluent flow. From the distribution of resulting receiving waterbody flow rates across the pooled flow data of all relevant NAICS code, the median 7Q10 flow rate was selected to be applied as a conservative low flow condition across the modeled releases (Figure_Apx B-1). Additional refined analyses were conducted for the scenarios resulting in the greatest environmental concentrations by applying the 75th and 90th percentile (P75 and P90, respectively) flow metrics from the distribution, which were expected to be more representative of the flow conditions associated with high-end releases.



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

Quantified release estimates to surface water were evaluated with PSC modeling, applying the receiving waterbody flows estimated from the developed distribution. For each COU with surface water releases of wastewater effluent, the highest estimated release to surface water was modeled. The total days of release associated with the highest OES surface water release was applied as continuous days of release per year (for example, a scenario with 250 days of release per year was modeled as 250 consecutive days of release, followed by 115 days of no release, per year). Raw daily water column concentration estimates from PSC were manually evaluated for the highest resulting concentrations in an averaging window equal to the total days of release (for example, a scenario with 250 days of release was evaluated for the highest 250-day average concentration). The frollmean function in the data.table package in R was used to calculate the rolling averages. The function takes in the concentration values

to be averaged (extracted from the PSC Daily Output File) and the number of values to include in the averaging window which was total days of release (extracted from the PSC Summary Output File). The function outputs a list of averages from consecutive averaging windows (for example, the first average will be for values 1 – total days of release and the second average will be for values 2 – total days of release +1).

Appendix C GENERAL POPULATION SURFACE WATER RISK SCREENING RESULTS

C.1 Incidental Dermal 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. Using the total acute dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30. *Based on the conservative modeling parameters for surface water concentration and exposure factors parameters, risk for non-cancer health effects for dermal absorption through swimming is not expected.*

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

Scenario	Water Column Concentrations		Adult (21+ years)	Youth (11–15 years)	Child (6–10 years)
	30Q5 Conc. (µg/L)	Harmonic Mean Conc. (µg/L)	Acute MOE	Acute MOE	Acute MOE
PVC plastics compounding[a] *Without Wastewater Treatment*	126	87.7	2,171	2,835	4,674
PVC plastics compounding[a] *With Wastewater Treatment*	39.6	27.5	6,913	9,029	15,000
Highest monitored surface water[b] *Without Wastewater Treatment*	0.014	0.014	20,000,000	26,000,000	42,000,000
Highest monitored surface water[b] *With Wastewater Treatment*	0.0044	0.0044	62,000,000	81,000,000	130,000,000

30Q5 = Lowest 30-day average flow in a 5-year period
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] 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.

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. *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

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*	126	87.7	5,521	3,559	6,310
PVC plastics compounding[a] *With Wastewater Treatment*	39.6	27.5	18,000	11,000	20,000
Highest monitored surface water[b]	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] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] 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.

Appendix D GENERAL POPULATION DRINKING WATER RISK SCREENING RESULTS

Based on the estimated drinking water doses in Table 6-1, EPA screened for risk to adults, infants, and toddlers. Table_Apx D-1 summarizes the acute and chronic MOEs based on the drinking water doses. Using the total acute and chronic dose based on the highest modeled 95th percentile, the MOEs are greater than the benchmark of 30. Based on the conservative modeling parameters for drinking water concentration and exposure factors parameters, risk below the benchmark MOE for non-cancer health effects for drinking water ingestion is not expected.

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

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[a] *With Wastewater Treatment*	126	87.7	473	910,000	135	360,000	379	830,000
PVC plastics compounding[a] *With Wastewater Treatment and Drinking Water Treatment*	39.6	27.5	1,507	2,900,000	430	1,100,000	1,208	2,600,000
Highest monitored surface water[b]	0.014	0.014	4,300,000	5,700,000,000	1,200,000	2,200,000,000	3,400,000	5,200,000,000

30Q5 = Lowest 30-day average flow in a 5-year period
[a] Only this OES was used in the screening assessment because it resulted in the highest surface water concentrations.
[b] 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.

Appendix E FISH INGESTION RISK SCREENING RESULTS

E.1 General Population

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

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

	Acute Non-cancer MOE UFs = 30		Adult Chronic Non-cancer MOE UFs = 30
	Adult	Young Toddler	
Water solubility limit (1.48 mg/L)	87	59	384

E.2 Subsistence Fishers

Acute and chronic non-cancer risk estimates were below their benchmarks using the water solubility limit as the surface water concentration. EPA then refined its evaluation of this exposure pathway by modeling surface water concentrations based on the highest modeled 95th percentile release for the PVC plastics compounding OES and the 50th percentile flow. The acute and chronic non-cancer risk estimates are one order of magnitude above their corresponding benchmarks using release data (Table_Apx E-2). Based on the conservative modeling parameters for surface water concentration, ingestion of fish potentially contaminated with DCHP is not expected to be a major pathway of concern for subsistence fishers.

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

	Acute and Chronic Non-cancer MOE UFs = 30
Water solubility limit (1.48 mg/L)	14
Modeled surface water concentration for PVC plastics Compounding, P50 flow, Untreated (0.087 mg/L)	229
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

Acute and chronic non-cancer risk estimates were below their benchmarks using the water solubility limit as the surface water concentration (Table_Apx E-3). EPA then refined its analysis by modeling surface water concentrations based on the highest modeled 95th percentile release for the PVC plastics compounding OES, the 50th percentile flow, and untreated releases. The acute and chronic non-cancer risk estimates are still below the benchmark of 30 based on the heritage fish consumption rate. EPA further refined its analysis applying the modeled surface water concentrations based on (1) treated wastewater, and (2) untreated wastewater using the P75 and P90 flow metrics from the distribution. The higher flow metrics are expected to be more representative of the flow conditions associated with high-end releases. EPA also included the highest monitored surface water concentrations from Kiel et al. (2011). Kiel et al. (2011) detected DCHP in the Barkley Sound of Washington State at a maximum

concentration of 0.01 µg/L. Non-cancer risk estimates based on the tribal heritage fish consumption rate using modeled concentrations and higher flow distributions are above the corresponding benchmark by one to two orders of magnitudes. In comparison, the risk estimates using the highest monitored surface water concentration exceed the benchmark by four orders of magnitude. These results indicate that the modeled concentrations are conservative, as discussed in Section 4.3.

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

	Acute and Chronic Non-cancer MOE UFs = 30	
	Current IR	**Heritage IR**
Water solubility limit (1.48 mg/L)	9	1
Modeled surface water concentration for PVC plastics compounding, P50 flow, Untreated (8.7E–02 mg/L)	151	20
Modeled surface water concentration for PVC plastics compounding, P75 flow, Untreated (3.48E–03 mg/L)	3,812	500
Modeled surface water concentration for PVC plastics compounding, P90 flow, Untreated (2.4E–04 mg/L)	54,597	7,163
Modeled surface water concentration for PVC plastics compounding, P50 flow, Treated (2.7E–02 mg/L)	482	63
Highest monitored surface water concentration (1.0E–05 mg/L)	947,643	124,326
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.		

Appendix F AMBIENT AIR MONITORING STUDY SUMMARY

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.

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.

A third study conducted in Lake Chaohu, China, (HEW, 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 and under and 11,699 adults aged 16 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

2006 **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–<11 years old)	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–<16 years old)	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–<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–<16 years old)	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–<11 years old)	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–<16 years old)	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–<11 years old)	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–<16 years old)	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–<11 years old)	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–<16 years old)	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–<11 years old)	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–<16)	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–<16 years old)	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–<11 years old)	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 two cycles are 1.2792, which is likely the imputed value of non-detects after the application of the correction factor.

2007
2008

2009 **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 old)	Age	sex race income	–	0.0253	–	0.0041
1999–2010	MCHP	Children	All children (<16 years old)	Income	age sex race	–	0.0021	–	0.6628
1999–2010	MCHP	Children	All children (<16 years old)	Race	age sex income	–	<0.001	–	0.9094
1999–2010	MCHP	Children	All children (<16 years old)	Sex	age race income	–	<0.001	–	<0.001
1999–2010	MCHP	Children	Adolescents (11–<16 years old)	Years	sex race income	–	<0.001	–0.0590	<0.001
1999–2010	MCHP	Children	Toddlers (3–<5 years old)	Years	sex race income	–	<0.001	–0.0539	<0.001
1999–2010	MCHP	Children	Children (6–<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

2010