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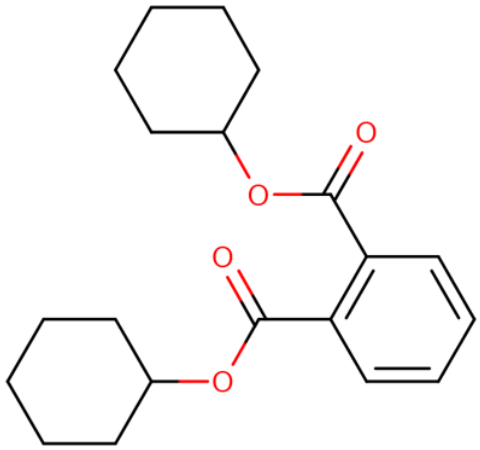
Environmental Hazard Assessment for Dicyclohexyl Phthalate (DCHP)

Technical Support Document for the Risk Evaluation

CASRN 84-61-7



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KEY ABBREVIATIONS AND ACRONYMS

AF	Assessment factor
CASRN	Chemical Abstracts Service Registry Number
COC	Concentration(s) of concern
DCHP	Dicyclohexyl phthalate
DMSO	Dimethylsulfoxide
ECHA	European Chemicals Agency
JME	Japanese Ministry of the Environment
LC50	Lethal concentration at which 50 percent of test organisms die
LOEC	Lowest-observed-effect-concentration
LOAEL	Lowest-observed-adverse-effect level
MOA	Mode of action
NITE	National Institute of Technology and Evaluation

NOEC	No-observed-effect-concentration
NOAEL	No-observed-adverse-effect level
OECD	Organisation for Economic Co-operation and Development
PNEC	Predicted no effect concentration
TSCA	Toxic Substances Control Act
TSD	Technical Support Document
U.S.	United States

SUMMARY

This technical support document (TSD) accompanies the Toxic Substances Control Act (TSCA) *Risk Evaluation for Dicyclohexyl Phthalate (DCHP)* (also called "the risk evaluation") (U.S. EPA, 2025). DCHP is a common chemical name for the chemical substance dicyclohexyl phthalate (CASRN 84-61-7). See Appendix C of the risk evaluation for a complete list of all the TSDs and supplemental files for the DCHP risk evaluation.

The U.S. Environmental Protection Agency (EPA or the Agency) found limited definitive environmental hazard data for DCHP. The reasonably available studies found aquatic acute exposure hazards above the water solubility limit of 1.48 mg/L for DCHP. However, DCHP caused chronic reproductive effects to an aquatic invertebrate and a fish species following chronic exposure at concentrations below the water solubility limit. EPA derived a concentration of concern (COC) for reproductive effects of chronic DCHP water exposure of 32 µg/L (Table S-1).

In terrestrial habitats, the available data suggest that DCHP may cause hazard to land mammals through dietary exposures. A hazard effects threshold was estimated based on laboratory rodent experiments because wild organism hazard studies were not reasonably available. EPA derived a terrestrial mammal hazard threshold for reduced body weight over two generations of dietary exposure of 179.3 mg/kg bw/day DCHP.

No hazard data were reasonably available for birds, reptiles, terrestrial invertebrates, and plants. Additional studies received from public comments were considered between the public release of the draft and final versions of this TSD, but none reported DCHP hazards.

Table S-1. Environmental Hazard Thresholds for DCHP

Receptor Group	Exposure Duration	Hazard Threshold (COC or HV)	Citation
Aquatic invertebrates	Chronic	32 µg/L	(NITE, 2000c)
Terrestrial vertebrates	Chronic	179.3 mg/kg bw/day	(Hoshino et al., 2005)
COC = concentration of concern; HV = hazard value			

1 INTRODUCTION

Dicyclohexyl phthalate is a granular, solid, medium-chain phthalate ester used as a plasticizer in manufacturing. Like most phthalates, DCHP is expected to cause acute adverse effects on organisms through a non-specific, narcotic mode of toxic action (Parkerton and Konkel, 2000), but is considered to have an anti-androgenic mode of action (MOA) leading to endocrine disruption under chronic exposures. EPA reviewed studies of the potential toxicity of DCHP to aquatic and terrestrial organisms.

High variability of experimental and estimated values of DCHP water solubility are reported in the surveyed literature and noted here for the limited studies of hazard effects on aquatic organisms. Reports of the water solubility of DCHP range from 0.2 to 4.0 mg/L (U.S. EPA, 2024). EPA calculated a water solubility value of 1.48 mg/L to represent the upper limit for the solubility of DCHP in water as estimated by an Epi Suite™ model (U.S. EPA, 2024). Multiple aquatic toxicity studies described in this TSD note difficulties with keeping DCHP in aqueous solution for the even short duration (*e.g.,* 72-hour) studies (Mathieu-Denoncourt et al., 2016). In addition, the Swedish Chemical Agency indicates that the maximum realized DCHP water concentrations may be as low as 30 µg/L (KemI, 2023), which suggests a potentially lower water solubility value than used in this risk evaluation.

In a sexual development test (OECD TG 234) for zebrafish, stable test concentrations close to the nominal value were only achieve for the two lowest test concentrations of 10.4 and 28.2 µg/L with the use of dimethylsulfoxide (DMSO) as a solvent. The authors observed a precipitate in all testing chambers and found evidence of DCHP colloids in treatments greater than 30 µg/L (KemI, 2023). Thus, EPA uses the water solubility of 1.48 mg/L in this assessment/TSD, but notes that the functional water solubility for some studies may have varied due to environmental or experimental conditions. These factors were considered in EPA's evaluation of DCHP.

2 APPROACH AND METHODOLOGY

EPA reviewed the potential environmental hazards associated with DCHP and identified 10 references (see Table 3-1 and Table 4-1) that were identified from the *Final Scope of the Risk Evaluation for Dicyclohexyl Phthalate; CASRN 84-61-7* (also called "the scope document") (U.S. EPA, 2020). This review included three laboratory rodent studies (Table 4-1) as well as studies identified through documents produced by the European Chemicals Agency (ECHA) (ECHA, 2014) and Environment Canada and Health Canada (Health Canada, 2020; EC/HC, 2015) using the data quality evaluation metrics and criteria described in EPA's *Draft Systematic Review Protocol Supporting TSCA Risk Evaluations for Chemical Substances, Version 1.0* (also called the "2021 Draft Systematic Review Protocol") (U.S. EPA, 2021a). Two studies were assigned an overall quality determination of high while all other studies were assigned medium ratings. The results of one additional study that was reported by the Swedish Chemical Agency (KemI, 2023) were reviewed and described in this TSD; however, the source data were not available for EPA review. Therefore, a data quality evaluation rating was not assigned, nor was a hazard threshold assigned based on these data.

The environmental effects of DCHP have also been reviewed by several regulatory agencies, including separate assessments by Health Canada and Environment Canada (Health Canada, 2020; EC/HC, 2015), and ECHA (ECHA, 2014). EPA reviewed the information in these environmental assessments. The reports from Canada and ECHA rely on ECHA study summaries of DCHP toxicity bioassays that used standard Organisation for Economic Co-operation and Development (OECD) test guidelines. These ECHA summaries as well as a Substance Evaluation Conclusion (KemI, 2023) describe the results of studies originally conducted by the Japanese Ministry of the Environment (JME). EPA reviewed the ECHA summaries and the JME source data. The JME reports contain relevant raw and summary data in tables and figures presented in English, but the narratives are in Japanese. EPA bundled the ECHA summaries to the Japanese reports and together, these packets of information were assigned data quality evaluations of medium in EPA's systematic review process (NITE, 2000a, b, c, d). The Agency had confidence in the conclusions drawn by other international regulatory authorities based on the study results and summaries. EPA summarized the critical concentration thresholds from these reports and used the reported values to add to the weight of scientific evidence supporting the hazard effects characterization.

3 AQUATIC SPECIES HAZARD

Seven references documenting DCHP water exposure hazard effects on aquatic organisms were reasonably available for EPA to review. One study was assigned an overall data quality level of high and the other references were assigned medium (Table 3-1). No studies of dietary exposure to aquatic organisms were reasonably available to assess the potential hazards from DCHP ingestion.

Mathieu-Denoncourt et al. (2016) exposed gastrula-stage (Nieuwkoop and Faber [NF] development stages 11 and 12) embryos of western clawed frogs (*Silurana tropicalis)* to nominal DCHP concentrations of 0.6, 6, 23, 60, and 600 mg/L and observed embryo mortality and malformations over 72 hours. DMSO (0.82%) was used as a solvent to dissolve the solid DCHP into solution and was used in a solvent control. The authors reported measured concentrations at 24 hours of exposure as 0.3, 1.5, 4.1, 19.0, and 99.3 mg DCHP/L, indicating substantial DCHP "degrading" from solution over the course of the experiment. Compared to control treatments, 4 percent higher embryo mortality occurred after 72 hours of the reported 4.1 mg/L exposure, while 95 and 100 percent mortality occurred at 19.0 and 99.3 mg/L treatments, respectively. The study authors of reported a no-observed-effect-concentration (NOEC) and lowest-observed-effect-concentration (LOEC) of 1.5 mg/L and 4.1 mg/L, respectively. Although no lethal concentration at which 50 percent of test organisms died (*i.e.*, LC50) was reported, an LC50 of frog embryo mortality was reported as 5.5 mg/L by the same first author in a thesis based upon the same experiment as the peer-reviewed publication (Mathieu-Denoncourt et al., 2014). Effective DCHP concentrations in the water were reported as the average between the starting concentration and the concentration at 24 hours. Thus, the study reports considerable uncertainty about the realized water solubility of DCHP in their bioassays and the DCHP concentrations in solution vs. the DCHP that degraded or precipitated as a solid after 24 hours. Because 4.1 mg/L was reported as an average concentration between the start of the bioassay (7.0 mg/L) and 24 hours later (1.2 mg/L) for this treatment, EPA has slight confidence that the reported hazard effect concentration of 4.1 mg/L was maintained throughout the study duration.

Hemocyte cultures from the freshwater prawn (*Macrobrachium rosenbergii*) were exposed to 0.1 mg/L DCHP (Sung et al., 2003). Hemocyte necrosis was approximately 80 percent greater after 20 minutes of 0.1 mg/L exposure compared to control treatments, while hemocyte adhesion, pseudopodia stretching, phenoloxidase activity, superoxide production, and apoptosis were not affected. Although this study was rated medium-quality and provides some evidence for the acute effects of DCHP on an invertebrate's respiratory and immune systems, the relationship between the measured endpoints and survival or reproduction at the population-level was not established. Moreover, most measured endpoints were not affected by DCHP, and DCHP concentrations were not analytically verified. Thus, EPA did not use the results of this study for a hazard threshold.

The ECHA dossier (*Committee for Risk Assessment RAC Opinion proposing harmonised classification and labelling at EU level of Dicyclohexyl phthalate, EC number: 201-545-9, CAS number: 84-61-7* (ECHA, 2014)) reported summaries of studies of DCHP toxicity to fish, algae, and *Daphnia* (water fleas). Notably, these ECHA dossier reports summarized studies by JME (NITE, 2000a, b, c, d). The fish (*Oryzias latipes*) acute study was conducted at concentrations above the limit of solubility that EPA has calculated to be 1.48 mg/L in EPA's *Physical Chemistry and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* (U.S. EPA, 2024) and did not find hazard effects (NOEC > 2.0 mg/L)—despite the use of dimethylformamide as a solvent to achieve and maintain those concentrations in solution (NITE, 2000b). A similar study conducted at high concentrations found no effects of 72-hour DCHP exposures on the green algae *Raphidocelis subcapitata* at concentrations exceeding 2 mg/L (NITE, 2000d). A 48-hour *Daphnia* immobilization study also did not find hazard effects of acute DCHP exposure to *Daphnia magna* up to and above 2 mg/L (NITE, 2000a). All three of these studies

failed to observe acute DCHP exposure effects in treatments with 2.0 mg/L DCHP, which is above the water solubility reported in the *Physical Chemical and Fate and Transport Assessment for Dicyclohexyl Phthalate (DCHP)* TSD (U.S. EPA, 2024) limit of 1.48 mg/L DCHP.

Another report of a study conducted by JME and subsequently summarized in an ECHA dossier describes the results of a 21-day *Daphnia* reproduction test (OECD No.211 semi-static; dimethylformamide solvent) (NITE, 2000c). In that study, the mean number of offspring produced by parent *Daphnia* ranged from 135 to 139 neonates over 21 days across 0, 0.018, 0.058, and 0.181 mg/L DCHP treatments. Only 121 neonates were produced in the next highest concentration (0.572 mg/L), which was a 12.9 percent reduction in *Daphnia* reproduction. Thus, the authors reported an NOEC of 0.181 mg/L and the LOEC of 0.572 mg/L DCHP (NITE, 2000c). EPA calculated the geometric mean of this NOEC and LOEC to be 0.32 mg/L DCHP as the aquatic chronic exposure hazard threshold. In agreement with EPA, Environment Canada (EC/HC, 2017) relied on this study to derive a predicted no effect concentration (PNEC).

One chronic exposure study in fish was reported by the Swedish Chemicals Agency (KemI, 2023), but because EPA was not able to obtain the source data, a data quality evaluation rating could not be established. The summary reports shorter body lengths, lower body weights, and inconsistent male and female vitellogenin (egg precursor) effects after 28 days of 32.0 µg/L DCHP water exposures, indicating potential endocrine disrupting effects. The report also describes the difficulties in keeping DCHP in solution and concluded that the true water solubility of DCHP is closer to 30 µg/L than 1,000 µg/L (1 mg/L). Thus, due to the stated difficulties of maintaining these concentrations in solution, there is a large amount of uncertainty in the accuracy and precision of the hazard value (*i.e.*, the actual DCHP concentration to which the fish were exposed).

Environment Canada: Environment and Climate Change Canada and Health Canada assessed DCHP environmental hazard in 2015 (EC/HC, 2015) and later published a regulatory update in 2020 (Health Canada, 2020). Environment Canada assessed the DCHP environmental risks using the available data in (ECHA, 2014) and the *Daphnia* 21-day reproduction effect from an ECHA summary (ECHA, 2014) to develop a critical toxicity value of 0.181 mg/L. Environment Canada set a PNEC at 0.06 mg/L by applying a standard assessment factor of three (AF = 3) to account for unstated uncertainties. The European Union is currently evaluating DCHP for endocrine disrupting properties in the environment based on the 21-day *Daphnia* study (NITE, 2000c) and fish early life stage tests (KemI, 2023).

Table 3-1. DCHP Hazard to Aquatic Organisms

Test Organism (Species)	Hazard Values	Study Duration	Endpoint	Citation (Data Evaluation Rating)
Western clawed frog (*Silurana tropicalis*)	5.5 mg/L LC50 1.5/4.1 mg/L[a] (NOEC/LOEC)	72-hour (acute duration)	Embryo (gastrula) mortality	(Mathieu-Denoncourt et al., 2016), (Mathieu-Denoncourt et al., 2014) (High)
Freshwater prawn, (*Macrobrachium rosenbergii*)	>0.1 mg/L (NOEC) [b]	40-minute (acute duration)	Hemocyte necrosis	(Sung et al., 2003) (Medium)
Japanese medaka (*Oryzias latipes*)	>2 mg/L (LC50)	96-hour (acute duration; static renewal)	Mortality	(NITE, 2000b) (Medium) OECD TG 203 [c]

Test Organism (Species)	Hazard Values	Study Duration	Endpoint	Citation (Data Evaluation Rating)
Water flea (*Daphnia magna*)	>2 mg/L (LC50)	48-hour (acute duration; static)	Mortality	(NITE, 2000a) (Medium) OECD TG 203 [c]
Freshwater alga (*Raphidocelis subcapitata*)	>2 mg/L (NOEC)	24/72-hour (acute duration; static)	Growth inhibition	(NITE, 2000d) (Medium) OECD TG 203 [c]
Water flea (*Daphnia magna*)	**0.32 mg/L** (geometric mean of 0.181 mg/L; NOEC; and 0.572 mg/L LOEC)	21-day (chronic duration; static renewal)	Reproduction	(NITE, 2000c) (Medium) OECD TG 211 [c]
Zebrafish (*Danio rerio*)	10.4/32.0 µg/L NOEC/LOEC; geometric mean = 18.24	60-day partial lifecycle test; (chronic duration; flow through)	Vitellogenin; reduced length; reduced weight	(KemI, 2023) (Medium) OECD TG 234 [c]

Bolded number indicates the hazard value used for calculating the aquatic concentration of concern (COC).
[a] Average measured concentration after 24 hours. Starting/nominal concentrations were 2.7/7 mg DCHP/L (NOEC/LOEC). DMSO solvent. The LC50 and the LOEC are above the limit of solubility (1.48 mg/L).
[b] One nominal concentration tested using acetone as a solvent. No effects on hemocyte adhesion, pseudopodia stretching, phenoloxidase activity, superoxide production, or apoptosis.
[c] Results also reported in ECHA summary.

Aquatic Organism Hazard Conclusions

The European Union and Environment Canada determined hazard thresholds for aquatic organisms using the same ECHA statement from a 21-day *Daphnia* reproduction study at the NOEC of 0.181 mg/L DCHP conducted using a standard OECD TG 211 protocol. EPA reviewed this ECHA summary and the source data in the JME report on which the ECHA summary was based (NITE, 2000c). The Agency also used the geometric mean of the NOEC (0.181 mg/L) and LOEC (0.572 mg/L). In addition, EPA reviewed one available acute exposure study that determined that 50 percent amphibian embryo mortality occurred at 5.5 mg/L DCHP and significant decreases in survival compared to controls began at 4.1 mg/L; however, the authors reported that DCHP rapidly "degraded" and was no longer dissolved solution—making conclusions about hazard effects of DCHP in solution uncertain (Mathieu-Denoncourt et al., 2016). Furthermore, ECHA reports of a fish early life stage bioassay indicate that the maximum realized DCHP water concentrations may be closer to 30 µg/L DCHP, and reported non-apical and inconsistent endocrine-disruptive effects at DCHP concentrations at 32.0 µg/L. Therefore, EPA notes the potential endocrine effects of DCHP to early fish life stages stated in the ECHA summary but did not set a threshold hazard value based on that study. No reasonably available studies of the dietary effects of DCHP on aquatic organisms were reviewed.

4 TERRESTRIAL SPECIES HAZARD

No studies on terrestrial wildlife involving mammals, birds, invertebrates, or plant species were identified. In lieu of terrestrial wildlife studies, three references for rat studies as human health model organisms were used to determine the best available DCHP concentration that affected apical endpoints (survival, reproduction, growth) in rodents and that could serve as an indication of hazard effects in wild mammal populations. These dietary DCHP concentrations were expressed as doses in units of mg/kg-bw/day. Because body weight was normalized, EPA used it as a screening surrogate for effects on ecologically relevant wildlife species to evaluate chronic dietary exposure to DCHP.

EPA reviewed and rated three laboratory rodent studies as surrogates for hazards of DCHP to wild mammal populations (Table 4-1). Ahbab and Barlas (2015) and Li et al. (2016) found testicular pathologies in *in-utero* rat pups from mothers gavaged 10 and 20 mg/kg bw/day respectively. Hoshino et al. (2005) found reduced body weights in F1 parental rats fed diet exposures of 402 mg DCHP/kg bw/d (lowest-observed-adverse-effect level or LOAEL) but not in parental or F2 rats. The no-observed-adverse-effect-level (NOAEL) dose in this study was 80 mg/kg bw/day. EPA calculated the geometric mean to arrive at a hazard effect of approximately 9 percent lower body weight in this F1 generations of 179.3 mg/kg bw/day dietary DCHP exposure. Hoshino et al. (2005) also found testicular atrophy as well as reduced weight gain in F1 prepubescent male pups at 85 mg/kg bw/day (LOAEL).

The following studies highlight the potential long-term effects of DCHP dietary exposure: through lower body weights (Hoshino et al., 2005); a 12 to 47 percent decrease in testosterone (Li et al., 2016; Ahbab and Barlas, 2015); an increase in male testicular malformations such as one occurrence of testis dysgenesis (Li et al., 2016); and a 300 percent increase in large Leydig cell clusters compared to control rats (Ahbab and Barlas, 2015). However, considerable uncertainties surround whether or how these effects on individual growth and reproductive development translate into effects on wild mammal fitness and population parameters. Thus, EPA has moderate confidence that these hazard doses can affect the range of wildlife mammal species at these magnitudes and dietary levels and in the same manner.

Table 4-1. DCHP Hazard to Laboratory Rats

Test Organism	Hazard Values	Duration; Endpoints	Citation (Data Evaluation Rating)
Norway rat; Sprague Dawley	80/402 mg/kg bw/day (NOAEL/LOAEL) Geometric mean = **179.3 mg/kg bw/day**	2-generation reproduction; 9% reduction in body weight	(Hoshino et al., 2005) (Medium)
Norway rat; Wistar	20 mg/kg bw/day (LOAEL)	*In-utero*; 12% decline in testosterone; histopathology in testes	(Ahbab and Barlas, 2015) (High)
Norway rat; Sprague Dawley	100 mg/kg bw/day (LOAEL)	*In-utero*; 47% decreased testicular testosterone and one instance of testis dysgenesis	(Li et al., 2016) (Medium)
Bolded number indicates the hazard value used for calculating the terrestrial concentration of concern (COC).			

Terrestrial Organism Hazard Conclusions

No studies of terrestrial wildlife were reasonably available to review. In lieu of this absence of information, EPA investigated the published effects of dietary DCHP exposure to laboratory rodents that may translate to survival, growth, or reproduction effects on wild mammal populations. Of the three studies reviewed, only Hoshino et al. (2005) reported an apical endpoint of reduced growth (body weight) beginning after 70 days of dosing. Thus, EPA has highlighted the reproductive effects of DCHP on terrestrial mammal dietary exposure of 179.3 mg/kg bw/day based on Hoshino et al. (2005).

5 WEIGHT OF SCIENTIFIC EVIDENCE CONCLUSIONS FOR ENVIRONMENTAL HAZARD

EPA concluded that no acute hazard effects were identified from exposures to DCHP below the limits of water solubility (1.48 mg/L (U.S. EPA, 2024)). DCHP poses potential chronic hazard effects to aquatic organisms based on two studies, one described in JME reports (NITE, 2000c) and the other by the Swedish Chemicals Agency (KemI, 2023). The reviewed studies highlight the difficulties of keeping DCHP in solution—even in acute water exposure studies. These studies suggest that DCHP may precipitate to its solid state at environmental concentrations above approximately 30 to 50 µg/L (KemI, 2023; Mathieu-Denoncourt et al., 2016). Thus, the weight of evidence for DCHP hazards to aquatic organisms indicate that no acute exposure hazards occur below the limit of solubility of less than 1.48 mg/L (Table 3-1) (U.S. EPA, 2024), but potential reproductive and endocrine disrupting effects may occur at lower concentrations (KemI, 2023; NITE, 2000c).

DCHP poses a hazard to terrestrial mammals at a dietary dose of 179.3 mg/kg bw/d and is supported by evidence extrapolated from laboratory rodent studies. This conclusion is limited by uncertainties surrounding the lack of available studies of wild animal or plant populations. Additionally, because laboratory rodent results were used to represent potential DCHP growth and reproductive effects across the spectrum of mammal species, EPA acknowledges the potential uncertainties in extrapolating this threshold dose to mammals with different sizes, metabolic rates, diets, and physiologies. Finally, relatively small differences in growth rate or reproductive development might translate into relatively small population-scale effects on wild mammals compared to the myriad challenges facing wild mammals. Thus, EPA contends that this hazard threshold value is conservatively protective across potentially DCHP wild mammal populations.

5.1 Strengths, Limitations, Assumptions, and Key Sources of Uncertainty for Environmental Hazard

EPA has robust confidence that DCHP poses little to no acute exposure hazard to aquatic organisms in the environment but moderate confidence that DCHP poses reproductive hazards at lower DCHP concentrations in water. These confidences are supported by specific lines of evidence outlined below. The general approach to EPA's consideration of the strengths, limitations, assumptions, and key sources of uncertainty for environmental hazard is outlined in Appendix A.

The study evaluated by EPA that had a high data quality rating (Mathieu-Denoncourt et al., 2016) found acute exposure effects of DCHP on survival only at concentrations (4.1 mg/L) above estimated water solubility limits. This study (1) received a high-data quality evaluation, (2) included analytically-sound water concentration measurements, (3) used a sensitive life stage of a sensitive organism (amphibian embryos), and (4) used a standard and relevant biological gradient/dose response experimental design. Thus, EPA has robust confidence in the quality of this study. These results are supported by acute duration studies on fish, *Daphnia*, and algae that did not find DCHP effects on organism survival up to 2 mg/L, which exceeds the water solubility limit of 1.48 mg/L. Thus, all four of these studies indicate no hazard effects up and above the limit of solubility, providing corroborating evidence and confidence in this conclusion, and are consistent with other EPA-reviewed, non-U.S. regulatory documents. These additional studies summarized in the ECHA dossier are well supported by standard methods that use OECD testing guidelines (NITE, 2000a, b, c, d). EPA has robust confidence in the quality of the database, the consistency, strength, and precision of the hazard effects study results, the dose-response experimental designs of the studies, and the relevancy of the studies (see Table 5-1).

EPA has robust confidence in the threshold for chronic exposures to aquatic organisms based on the studies that were summarized in the JME reports (NITE, 2000c) as well as ECHA's DCHP dossier. These studies used standard OECD test guidelines and were described in detail. The 21-day *Daphnia* reproduction test in the summary was also used by Environmental Canada to derive an aquatic threshold concentration (EC/HC, 2017). That threshold relies on one medium quality study that was able to maintain consistent DCHP concentrations with the use of a solvent through daily renewal over 21 days (NITE, 2000c). Other studies document difficulties in being able to maintain consistent and similar DCHP concentrations over time (KemI, 2023; Mathieu-Denoncourt et al., 2016).

Considerable uncertainties about DCHP environmental water solubility limits documented by EPA (U.S. EPA, 2024) and other studies (KemI, 2023; Mathieu-Denoncourt et al., 2016) suggest that DCHP may leave solution (*e.g.*, as precipitate and/or sorption to organic matter) within minutes, making water exposure to DCHP transient and thus expected to have minimal hazard effects. DCHP concentrations used in studies of chronic exposure to invertebrates and fish conducted above 0.035 mg/L might have been transient in nature and not maintained in solution without frequent renewal, thus exceeding the realistic upper bound of environmental DCHP concentrations using a solvent. EPA therefore has slight confidence that DCHP can be maintained in solution without solvent at exposure concentrations high enough to cause hazard effects.

Also, DCHP is a solid at room temperature and has limited solubility in water, pointing to limited realistic exposures to aquatic organisms.

EPA acknowledges two limitations and uncertainties to the conclusions. First, only a limited number of studies have been published or reviewed to assess either acute or chronic exposure hazard. Second, the Agency also lacked reasonably available studies of chronic aquatic exposure in the sediment and dietary effects on aquatic organisms.

The conclusion that DCHP poses a hazard to terrestrial mammals at a dietary dose of 179.3 mg/kg bw/d is supported by evidence extrapolated from laboratory rodent studies. This hazard threshold is limited by uncertainties surrounding (1) the lack of available studies of wild animal or plant populations, and (2) whether laboratory rodent results represent effects that translate to wild populations. Specifically, because laboratory rodent results were used to represent potential DCHP growth and reproductive effects across the spectrum of mammal species, EPA acknowledges the potential uncertainties in extrapolating this threshold dose to mammals with different sizes, metabolic rates, diets, and physiologies. Relatively small differences in growth rate or reproductive development might translate into relatively small population-scale effects on wild mammals compared to the myriad challenges facing wild mammals. Thus, the Agency contends that this threshold value is conservatively protective across wild mammal populations. EPA has robust confidence in the terrestrial mammal quality of the database, the consistency, strength, and precision of the hazard effects study results, and the dose-response experimental designs of the studies. The Agency has moderate confidence in and the relevancy of the studies because of the uncertainties of extrapolating from laboratory rodent studies to wildlife populations. Overall, EPA has robust confidence in the terrestrial mammal hazard threshold value (Table 5-1).

No hazard data for terrestrial invertebrates, plants, and birds were reasonably available. The lack of studies on the hazard effects of DCHP on these organisms leaves some uncertainty about apical and subapical endpoints to these organisms.

Table 5-1. DCHP Evidence Table Summarizing the Overall Confidence Derived from Hazard Thresholds

Types of Evidence	Quality of the Database	Consistency	Strength and Precision	Biological Gradient/ Dose-Response	Relevance[a]	Hazard Confidence
Aquatic						
Acute aquatic	+++	+++	+++	+++	+++	Robust
Chronic aquatic invertebrates	+++	+++	+++	+++	+++	Robust
Aquatic plants and algae	+++	+++	+++	+++	+++	Robust
Terrestrial						
Terrestrial vertebrates	+++	+++	+++	+++	++	Robust

[a] Relevance includes biological, physical-chemical, and environmental relevance.
+++ Robust confidence suggests thorough understanding of the scientific evidence and uncertainties. The supporting weight of the scientific evidence outweighs the uncertainties to the point where it is unlikely that the uncertainties could have a significant effect on the hazard estimate.
++ Moderate confidence suggests some understanding of the scientific evidence and uncertainties. The supporting scientific evidence weighed against the uncertainties is reasonably adequate to characterize hazard estimates.
+ Slight confidence is assigned when the weight of the scientific evidence may not be adequate to characterize the scenario, and when the assessor is making the best scientific assessment possible in the absence of complete information. There are additional uncertainties that may need to be considered.

6 ENVIRONMENTAL HAZARD THRESHOLDS

Aquatic Organism Threshold

EPA calculates hazard thresholds to identify potential concerns to aquatic and terrestrial species. After weighing the scientific evidence, the Agency selects the appropriate toxicity value from the integrated data to use for hazard thresholds.

EPA derived a COC for aquatic organisms based on the weight of scientific evidence that chronic exposure to DCHP on aquatic organisms has been demonstrated at concentrations between the functional solubility estimated to be approximately 30 µg/L by the Swedish Chemicals Agency (KemI, 2023) and the water solubility limit determined to be 1.48 mg/L by EPA (U.S. EPA, 2024). COCs for aquatic organisms are calculated using a deterministic method, by dividing a toxicity value by an AF to account for inter- and intra-species variability, short- to long-term effects, as well as the extent of species covered by the dataset according to EPA methods (U.S. EPA, 2016). The Agency calculated a chronic water exposure COC based on the following equation:

$$\text{COC} = \text{toxicity value} \div \text{AF}$$

Where the 0.32 mg/L was the chronic toxicity value from the 21-day *Daphnia* reproduction test (NITE, 2000c) and an AF of 10. Thus, EPA derived a COC of reproductive hazard effects of 0.032 mg/L (32 µg/L) of chronic exposure of aquatic animals to DCHP.

Terrestrial Organism Threshold

Terrestrial mammal data were insufficient to find a toxicity reference value as outlined in EPA's *Guidance for Developing Ecological Soil Screening Levels (Eco-SSLs)* (U.S. EPA, 2005). Thus, the Agency derived a hazard value as the geometric mean of the NOAEL and LOAEL of reproductive hazards from a single study. Reduced weight gain was reported in a two-generation study by Hoshino et al. (2005). Potential endocrine disrupting effects of reduced testosterone and testes pathology was also documented in other studies. Based on the uncertainty of using surrogate laboratory rodent effects and the endpoints (growth) most likely to directly affect wild mammal populations, EPA has determined a terrestrial mammal hazard threshold of 179.3 mg/kg bw/d with the effect of approximately 9 percent reduced mammal body weight over two generations of dietary exposure.

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APPENDICES

Appendix A ENVIRONMENTAL HAZARD WEIGHT OF SCIENTIFIC EVIDENCE

A.1 Evidence Integration

Data integration includes analysis, synthesis, and integration of information for the risk evaluation. During data integration, EPA considers quality, consistency, relevancy, coherence, and biological plausibility to make final conclusions regarding the weight of the scientific evidence. As stated in the 2021 Draft Systematic Review Protocol, data integration involves transparently discussing the significant issues, strengths, and limitations as well as the uncertainties of the reasonably available information and the major points of interpretation. The general analytical approaches for integrating evidence for environmental hazard is discussed in Section 7.4 of the Draft Systematic Review Protocol.

The organization and approach to integrating hazard evidence is determined by the reasonably available evidence regarding routes of exposure, exposure media, duration of exposure, taxa, metabolism and distribution, effects evaluated, the number of studies pertaining to each effect, as well as the results of the data quality evaluation.

The environmental hazard integration is organized around effects to aquatic and terrestrial organisms as well as the respective environmental compartments (*e.g.*, pelagic, benthic, soil). Environmental hazard assessment may be complex based on the considerations of the quantity, relevance, and quality of the available evidence.

For DCHP, environmental hazard data from toxicology studies identified during systematic review have used evidence that characterizes apical endpoints; that is, endpoints that could have population-level effects such as reproduction, growth, or mortality. Additionally, mechanistic data that can be linked to apical endpoints will add to the weight of scientific evidence supporting hazard thresholds.

A.2 Weight of Scientific Evidence

After calculating the hazard thresholds that were carried forward, a narrative describing the weight of scientific evidence and uncertainties was completed to support EPA's decisions. The weight of scientific evidence fundamentally means that the evidence is both weighed (*i.e.*, ranked) and weighted (*i.e.*, a piece or set of evidence or uncertainty may have more importance or influence in the result than another). Based on the weight of scientific evidence and uncertainties, a confidence statement was developed that qualitatively ranks (robust, moderate, slight, or indeterminate) the confidence in the hazard threshold. The qualitative confidence levels are described below.

The evidence considerations and criteria detailed within the 2021 Draft Systematic Review Protocol (U.S. EPA, 2021a, b) guides the application of strength-of-evidence judgments for environmental hazard effect within a given evidence stream. These were adapted from Table 7-10 of the Protocol.

EPA used the strength-of-evidence and uncertainties from the 2021 Draft Systematic Review Protocol for the hazard assessment to qualitatively rank the overall confidence using for environmental hazard.

A.3 Rubric for Weight of Scientific Evidence

The weight of the scientific evidence fundamentally means that the evidence is weighed (*i.e.,* ranked) and weighted (*i.e.,* a piece or set of evidence or uncertainty may have more importance or influence in the result than another). Based on the weight of the scientific evidence and uncertainties, a confidence statement was developed that qualitatively ranks (robust, moderate, slight, or indeterminate) the confidence in the hazard threshold. The qualitative confidence levels are described below.

The evidence considerations and criteria detailed within (U.S. EPA, 2021b) guides the application of strength-of-evidence judgments for environmental hazard effect within a given evidence stream and were adapted from Table 7-10 of the 2021 Draft Systematic Review Protocol (U.S. EPA, 2021b).

EPA used the strength-of-evidence and uncertainties from (U.S. EPA, 2021b) for the hazard assessment to qualitatively rank the overall confidence rating for environmental hazard (Table_Apx A-1). Confidence levels of robust (+ + +), moderate (+ +), slight (+), or indeterminant are assigned for each evidence property that corresponds to the evidence considerations (U.S. EPA, 2021b). The rank of the *Quality of the Database* consideration is based on the systematic review overall quality determination (high, medium, or low) for studies used to calculate the hazard threshold, and whether there are data gaps in the toxicity dataset. Another consideration in the *Quality of the Database* is the risk of bias (*i.e.,* how representative is the study to ecologically relevant endpoints). Additionally, because of the importance of the studies used for deriving hazard thresholds, the *Quality of the Database* consideration may have greater weight than the other individual considerations. The high, medium, and low systematic review overall quality determinations correspond to the evidence table ranks of robust (+ + +), moderate (+ +), or slight (+), respectively. The evidence considerations are weighted based on professional judgment to obtain the overall confidence for each hazard threshold. In other words, the weights of each evidence property relative to the other properties are dependent on the specifics of the weight of the scientific evidence and uncertainties that are described in the narrative and may or may not be equal. Therefore, the overall score is not necessarily a mean or defaulted to the lowest score. The confidence levels and uncertainty type examples are described below.

1. **Confidence Levels**

   - Robust (+ + +) confidence suggests thorough understanding of the scientific evidence and uncertainties. The supporting weight of the scientific evidence outweighs the uncertainties to the point where it is unlikely that the uncertainties could have a significant effect on the exposure or hazard estimate.
   - Moderate (+ +) confidence suggests some understanding of the scientific evidence and uncertainties. The supporting scientific evidence weighed against the uncertainties is reasonably adequate to characterize exposure or hazard estimates.
   - Slight (+) confidence is assigned when the weight of the scientific evidence may not be adequate to characterize the scenario, and when the assessor is making the best scientific assessment possible in the absence of complete information. There are additional uncertainties that may need to be considered.

2. **Types of Uncertainties**

   The following uncertainties may be relevant to one or more of the weight of the scientific evidence considerations listed above and will be integrated into that property's rank in the evidence table:

   - *Scenario Uncertainty:* Uncertainty regarding missing or incomplete information needed to fully define the exposure and dose.

    - The sources of scenario uncertainty include descriptive errors, aggregation errors, errors in professional judgment, and incomplete analysis.
- *Parameter Uncertainty:* Uncertainty regarding some parameter.
    - Sources of parameter uncertainty include measurement errors, sampling errors, variability, and use of generic or surrogate data.
- *Model Uncertainty:* Uncertainty regarding gaps in scientific theory required to make predictions on the basis of causal inferences.
    - Modeling assumptions may be simplified representations of reality.

Table_Apx A-1 summarizes the weight of the scientific evidence and uncertainties while increasing transparency on how EPA arrived at the overall confidence level for each exposure hazard threshold. Symbols are used to provide a visual overview of the confidence in the body of evidence while de-emphasizing an individual ranking that may give the impression that ranks are cumulative (*e.g.*, ranks of different categories may have different weights).

Table_Apx A-1. Considerations That Inform Evaluations of the Strength of the Evidence Within an Evidence Stream (*i.e.*, Apical Endpoints, Mechanistic, or Field Studies)

Consideration	Increased Evidence Strength (of the Apical Endpoints, Mechanistic, or Field Studies Evidence)	Decreased Evidence Strength (of the Apical Endpoints, Mechanistic, or Field Studies Evidence)
The evidence considerations and criteria described below guide the application of strength-of-evidence judgments for an outcome or environmental hazard effect within a given evidence stream. Evidence integration or synthesis results that do not warrant an increase or decrease in evidence strength for a given consideration are considered "neutral" and are not described in this table (and, in general, are captured in the assessment-specific evidence profile tables).		
Quality of the database [a] (risk of bias)	• A large evidence base of high- or medium-quality studies increases strength. • Strength increases if relevant species are represented in a database.	• An evidence base of mostly low-quality studies decreases strength. • Strength also decreases if the database has data gaps for relevant species (*e.g.*, a trophic level that is not represented). • Decisions to increase strength for other considerations in this table should generally not be made if there are serious concerns for risk of bias; in other words, all the other considerations in this table are dependent upon the quality of the database.
Consistency	Similarity of findings for a given outcome (*e.g.*, of a similar magnitude, direction) across independent studies or experiments increases strength—particularly when consistency is observed across species, life stage, sex, wildlife populations, and across or within aquatic and terrestrial exposure pathways.	• Unexplained inconsistency (*i.e.*, conflicting evidence decreases strength.) • Strength should not be decreased if discrepant findings can be reasonably explained by study confidence conclusions; variation in population or species, sex, or life stage; frequency of exposure (*e.g.*, intermittent or continuous); exposure levels (low or high); or exposure duration.
Strength (effect magnitude) and precision	• Evidence of a large magnitude effect (considered either within or across studies) can increase strength. • Effects of a concerning rarity or severity can also increase strength, even if they are of a small magnitude. • Precise results from individual studies or across the set of studies increases strength, noting that biological significance is prioritized over statistical significance. • Use of probabilistic model (*e.g.*, Web-ICE, SSD) may increase strength.	Strength may be decreased if effect sizes that are small in magnitude are concluded not to be biologically significant, or if there are only a few studies with imprecise results.
Biological gradient/dose-response	• Evidence of dose-response increases strength. • Dose-response may be demonstrated across studies or within studies and it can be dose- or duration-dependent. • Dose response may not be a monotonic dose-response (monotonicity should not necessarily be expected (*e.g.*, different outcomes may be expected at low vs. high doses due to activation of different	• A lack of dose-response when expected based on biological understanding and having a wide range of doses/exposures evaluated in the evidence base can decrease strength. • In experimental studies, strength may be decreased when effects resolve under certain experimental conditions (*e.g.*, rapid reversibility after removal of exposure). • However, many reversible effects are of high concern. Deciding between these situations is informed by factors such as the toxicokinetics of the

	mechanistic pathways or induction of systemic toxicity at very high doses). • Decreases in a response after cessation of exposure (*e.g.*, return to baseline fecundity) also may increase strength by increasing certainty in a relationship between exposure and outcome (this is particularly applicable to field studies).	chemical and the conditions of exposure, endpoint severity, judgments regarding the potential for delayed or secondary effects, as well as the exposure context focus of the assessment (*e.g.*, addressing intermittent or short-term exposures). • In rare cases, and typically only in toxicology studies, the magnitude of effects at a given exposure level might decrease with longer exposures (*e.g.*, due to tolerance or acclimation). • Like the discussion of reversibility above, a decision about whether this decreases evidence strength depends on the exposure context focus of the assessment and other factors. • If the data are not adequate to evaluate a dose-response pattern, then strength is neither increased nor decreased.
Biological relevance	Effects observed in different populations or representative species suggesting that the effect is likely relevant to the population or representative species of interest (*e.g.*, correspondence among the taxa, life stages, and processes measured or observed and the assessment endpoint).	An effect observed only in a specific population or species without a clear analogy to the population or representative species of interest decreases strength.
Physical/chemical relevance	Correspondence between the substance tested and the substance constituting the stressor of concern.	The substance tested is an analog of the chemical of interest or a mixture of chemicals which include other chemicals besides the chemical of interest.
Environmental relevance	Correspondence between test conditions and conditions in the region of concern.	The test is conducted using conditions that would not occur in the environment.
[a] Database refers to the entire dataset of studies integrated in the environmental hazard assessment and used to inform the strength of the evidence. In this context, database does *not* refer to a computer database that stores aggregations of data records such as the ECOTOX Knowledgebase.		