



Australian Government

Department of Health and Aged Care

Australian Industrial Chemicals Introduction Scheme

1,4:7,10- Dimethanodibenzo[a,e]cyclooctene, 1,2,3,4,7,8,9,10,13,13,14,14-dodecachloro- 1,4,4a,5,6,6a,7,10,10a,11,12,12a- dodecahydro- (Dechlorane Plus)

Evaluation statement

26 June 2023



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AICIS evaluation statement

Subject of the evaluation

1,4:7,10-Dimethanodibenzo[a,e]cyclooctene, 1,2,3,4,7,8,9,10,13,13,14,14-dodecachloro-1,4,4a,5,6,6a,7,10,10a,11,12,12a-dodecahydro- (Dechlorane Plus)

Chemical in this evaluation

Name	CAS registry number
1,4:7,10-Dimethanodibenzo[a,e]cyclooctene, 1,2,3,4,7,8,9,10,13,13,14,14-dodecachloro-1,4,4a,5,6,6a,7,10,10a,11,12,12a-dodecahydro-	13560-89-9

Reason for the evaluation

Evaluation Selection Analysis indicated a potential human health and environmental risk.

Parameters of evaluation

The chemical is listed on the Australian Inventory of Industrial Chemicals (the Inventory). This evaluation is a human health and environmental risk assessment for all identified industrial uses of the chemical.

Summary of evaluation

Summary of introduction, use and end use

There is currently no specific information about the introduction, use and end use of the chemical in Australia. Dechlorane Plus (DP) is expected to be introduced into Australia in imported articles.

Based on international use information, DP is a chlorinated flame retardant which is primarily incorporated into:

- cables
- wires and wire connection products
- plastics and polymers
- specialist applications in aerospace and defence technologies.

The chemical is a member of a family of polychlorinated flame retardants introduced to replace Mirex (also known as Dechlorane). It is a widely marketed pesticide and flame retardant which was prohibited by the Stockholm Convention on Persistent Organic Pollutants in 2001 (Stockholm Convention n.d.a). It is also currently marketed as an alternative/replacement for commercial decabromodiphenyl ether (decaBDE) (POPRC 2022a).

The chemical is a high production volume commercial chemical in international markets, with global volumes around 750–6000 t/y based on production estimates and reports from the last two decades (POPRC 2022a). Recent estimates made in consultation with industry bodies suggest the contemporary production volume is in the range of 300–1000 tonnes per year, in:

- cables and wires for the motor vehicle sector (270–700 tonnes per year)
- aerospace and defence (7–22 tonnes per year)
- all other uses including electronics/electrical equipment, medical equipment
- marine applications (23–278 tonnes per year).

There is currently a global phase out on manufacture and use of the chemical, with potential time limited exemptions for use in motor vehicles, medical devices, aerospace and defence applications (POPRC 2022a).

Human health

Summary of health hazards

Based on the weight of evidence, the chemical has low acute toxicity via the oral, dermal, and inhalational routes. It is not an irritant to the skin, eye or respiratory system and is not a skin sensitiser. The chemical does not cause overt systemic health effects following repeated exposure, is not expected to be genotoxic or carcinogenic, and has not been shown to affect fertility or development. No data are available on neurodevelopmental effects in mammalian species.

However, given that the chemical is persistent and bioaccumulative, there is uncertainty about potential long term health effects. While available data indicate potential changes to thyroid hormone pathways, no consistent pattern of effects were evident. In a single study in mice, effects in adipose tissues and changes in biomarkers that may be associated with insulin resistance were reported. DP has been shown to inhibit insulin signalling in mammalian adipose cells and changes to gut microbiota were reported in a study in rats. From the available information it is not currently possible to determine with certainty whether the chemical causes adverse effects for human health.

Summary of health risk

Public

Based on international data there may be widespread use of articles containing the chemical in domestic settings, including in electronic appliances. The public may be directly or indirectly exposed to the chemical. Direct dermal exposure to the chemical from using polymer articles is considered to be negligible. However, the chemical can be released from articles present in indoor environments including homes, cars and schools. This may result in indirect exposure via inhalation of air as well as respiratory and oral exposure to dust containing the chemical. The chemical has been detected in indoor air and domestic dust samples in several countries. Humans may also be exposed to the chemical through food, water, soil, sediment and breast milk. Biological monitoring show that the chemical was detected in various tissues including serum, breast milk, cord blood, placental tissue, adipose tissue and hair.

Given the persistent and bioaccumulative properties of the chemical there is uncertainty regarding the long-term health effects from exposure to the chemical. Introduction by

manufacture or import, and the subsequent use of the chemical, including in articles, could increase potential risks to the public. The proposed means of managing risks to the environment would also minimise risk to the public (see **Proposed means for managing risks**).

Workers

During product formulation and packaging, dermal, ocular and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment.

Indirect exposure to the chemical in the workplace can occur due to presence of the chemical in indoor air and dust. Internationally, increased serum levels of the chemical were found in workers in e-waste recycling centres. Control measures to minimise inhalation exposure are needed to manage the risk to workers (see **Proposed means for managing risks** section). Workers in offices are also exposed to dust particles containing the chemical released from articles present in offices.

Given the persistent and bioaccumulative properties of the chemical there is uncertainty regarding the long term health effects from exposure to the chemical. Introduction by manufacture or import, and the subsequent use of the chemical, including in articles, could increase potential risks to workers. The proposed means of managing risks to the environment would also minimise risk to the workers (see **Proposed means for managing risks**).

Environment

Summary of environmental hazard characteristics

The chemical DP exhibits the characteristics of a Persistent Organic Pollutant (POP) as defined by the Stockholm Convention. The Stockholm Convention Technical Review Committee has agreed that the chemical meets the POP criteria in Annex D for:

- Persistence
- Bioaccumulation
- Potential for long range environmental transport (LRT)
- Adverse effects.

Environmental hazard classification

The chemical does not exhibit toxicity up to its solubility limit in standard aquatic ecotoxicity studies. However, the chemical is persistent and bioaccumulative, and there is other evidence indicating potential to cause adverse effects to environmental organisms. Therefore, according to the GHS guidance on classification of aquatic hazards (4.1.2.2), the chemical satisfies the criteria for classification according to the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) (UNECE 2017) as follows.

Environmental Hazard	Hazard Category	Hazard Statement
Chronic aquatic	Chronic aq. – Cat. 4	May cause long lasting effects to aquatic life

Summary of environmental risk

The chemical is used as a flame retardant in a wide range of products, including electrical and electronic equipment. A similar use pattern is assumed in Australia.

The chemical DP is a persistent and bioaccumulative chemical. This combination of hazard characteristics is of concern because it can increase the risk that environmental contaminants will become widely distributed in the environment and that they will contaminate food chains.

Given its high hydrophobicity and low water solubility, the chemical is expected to primarily accumulate in sediments. There are no measured data for DP concentrations in Australian sediments. However, international studies have identified high concentrations occurring around electronic waste recycling facilities. Sediments contaminated with the chemical may become a source of exposure for biota at the base of aquatic food-webs, such as benthic invertebrates and demersal fish and be transferred to organisms in higher trophic levels through bioaccumulation.

As persistent and bioaccumulative chemicals accumulate and remain in environmental biota over long time periods, it is difficult to predict their adverse effects. Available scientific literature suggests that there is a risk of adverse effects in aquatic species and other organisms due to oxidative stress, potential neurotoxicity and endocrine effects that may affect important biological processes. These studies demonstrate the potential for this persistent and bioaccumulative chemical to cause adverse effects under environmental exposure conditions.

Since the chemical has characteristics of a POP, there are potential significant long term risks to the environment from the manufacture, import and use of the chemical, including from introduction in articles. Given these characteristics, there is a risk to the environment that requires management (see **Proposed means for managing risks**).

Proposed means for managing risk

Workers

Information relating to safe introduction and use

The information in this statement, should be used by a person conducting a business or undertaking at a workplace (such as an employer) to determine the appropriate controls under the relevant jurisdiction Work Health and Safety laws.

Control measures that could be implemented to manage the risk arising from exposure to the chemical include, but are not limited to:

- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker
- minimising manual processes and work tasks through automating processes
- adopting work procedures that minimise splashes and spills
- cleaning equipment and work areas regularly
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Measures required to eliminate, or manage risk arising from storing, handling, and using a hazardous chemical depend on the physical form and the way the chemical is used.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Model codes of practice, available from the Safe Work Australia website, provide information on how to manage the risks of hazardous chemicals in the workplace, prepare an SDS and label containers of hazardous chemicals.

Environment

Recommendation to Department of Climate Change, Energy, the Environment and Water (DCCEEW)

It is recommended that the chemical be scheduled under the *Industrial Chemicals Environmental Management (Register) Act 2021* (ICEMR Act), with application of appropriate risk management measures to minimise further release to the environment from its introduction and use, including release from articles containing DP.

In making the scheduling decision consideration should be given to the following:

- DP has the characteristics of a POP as defined in Annex D of the Stockholm Convention.
- The chemical has known or potential use in products and end uses as listed in the 'Summary of introduction, use and end use' section. DP is expected to be introduced into Australia in imported articles.
- Since the chemical has the characteristics of a POP, there are potential significant long term risks to the environment from the manufacture, import and use of the chemical, including from introduction in articles.
- The Conference of Parties to the Stockholm Convention has accepted a recommendation to list the chemical on Annex A of the Convention with time limited exemptions.
- DP comprises two constituent stereoisomers. The proposed listing will also include its *syn*- (CAS No. 135821-03-3) and *anti*- (CAS No. 135821-74-8) isomers.
- DP has identified alternatives in most applications, and most global manufacturers have transitioned to alternative chemical technologies. However, based on information provided to the Stockholm Convention POPRC, time limited exemptions for use of the chemical may be permitted for some sectors with public safety constraints, particularly medical, automotive, aerospace and defence.

Conclusions

The conclusions of this evaluation are based on the information described in this statement.

Considering the proposed means of managing risks, the Executive Director is satisfied that the identified human health and environment risks can be managed.

Note: Obligations to report additional information about hazards under *Section 100* of the *Industrial Chemicals Act 2019* apply.

Supporting information

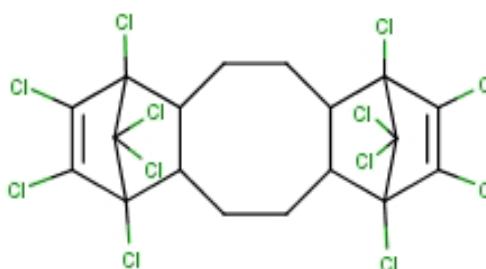
Chemical identity

The chemical DP is a reaction product that mainly comprises two constituent stereoisomers, *syn*-Dechlorane Plus (*syn*-DP, CAS No. 135821-03-3) and *anti*-DP (CAS No. 135821-74-8), at a ratio of approximately 1:3, or 25% *syn*-DP and 75% *anti*-DP (Sverko et al. 2011). These constituent stereoisomers are not listed on the Inventory.

Technical DP may also contain DP monoadducts (DPMA), 1,3-DPMA (CAS No. 70267-37-7) and 1,5-DPMA (CAS No. 10297-21-9) as impurities (Sverko et al. 2010), neither of which are listed on the Inventory:

Chemical name	1,4:7,10-Dimethanodibenzo[a,e]cyclooctene, 1,2,3,4,7,8,9,10,13,13,14,14-dodecachloro- 1,4,4a,5,6,6a,7,10,10a,11,12,12a-dodecahydro-
CAS No.	13560-89-9
Synonyms	Dechlorane Plus bis(hexachlorocyclopentadieno)cyclooctane; Dechlorane Plus 25 (Dech Plus); Dechlorane Plus 35 (Dech Plus-2); DP-515; Dechlorane 605
Molecular formula	C ₁₈ H ₁₂ Cl ₁₂
Molecular weight (g/mol)	653.72
SMILES	<chem>C1C2=C(/Cl)C3(Cl)C1CCC4C(CCC1C2(Cl)C3(Cl)Cl)C5(Cl)C(Cl)=C(Cl)C4(Cl)C5(Cl)Cl</chem>
Chemical description	The chemical is a reaction product that mainly comprises two stereoisomers

Structural formula



Relevant physical and chemical properties

Physical form	White crystalline powder
Melting point	Decomposition at 340–382°C
Vapour pressure	7.87 × 10 ⁻⁶ Pa at 25°C (calc.)

Water solubility	<1.67 ng/L (20–25°C) (exp.)
Henry's Law Constant	0.241 Pa·m ³ /mol at 25°C (calc.)
Ionisable in the Environment?	No
pKa	No ionisable groups
log Kow	9.3 (exp.)

The chemical DP is a colourless and odourless white solid. The chemical is poorly water soluble and is extremely lipophilic with a high octanol-water partition coefficient (log K_{ow} = 9.3) (Ghelli et al. 2021). The chemical can; therefore, be expected to accumulate in adipose tissues. DP is very slightly volatile. Based on the calculated Henry's Law constant it is moderately volatile from water.

Introduction and use

Australia

No specific information on Australian use, import, or manufacturing information have been identified.

The chemical DP is expected to be introduced into Australia in imported articles, based on international use patterns. DP is manufactured and marketed internationally at high volumes and is a component of many common articles (specifically, as a flame retardant in household items and electronic appliances, automobiles and wiring). Given the common and widespread use these items, emissions of DP are expected through the use and end-of-life disposal of the articles that contain it, such as e-waste from electronic products (Islam and Huda 2019).

International

According to compiled international data (POPRC 2022a), DP is a flame retardant mostly used in:

- insulation coating for cables and wires and wire connection products. The main sector of use is the automotive sector which is reported to account for majority of global use.
- a wide range of polymers and plastic mouldings. The amount of DP in these polymeric materials ranges from 8% to 40%
- two part resins for aerospace and defence applications, including epoxy adhesives, syntactic foams, potting compounds, 2-part epoxy void filler, connectors, wire/cables and other plastic components made from polypropylene
- motor vehicles, other motorised vehicles, trains and industrial and household machinery.

The chemical DP may also be used:

- in medical imaging and radiotherapy devices
- In fabrics, textiles and apparels, and plastic articles
- as an extreme pressure additive in greases

- as a colour intensifier in explosives in fireworks.

Global production of DP occurs in United States of America (USA) and China (POPRC 2022b). In the USA the manufacturer is OxyChem (Niagara Falls NY), which has produced DP for over 40 years (Betts 2006). Annual USA production was estimated to be 450–4500 tonnes since 1986 (Qiu et al. 2007). In China, annual production by Jiangsu Anpon Electrochemical Company (Huai'an) has been estimated at 300–1000 tonnes (Wang D-G et al. 2010). Therefore, global production is estimated at levels of up to 6000 tonnes per year, although manufacture in the USA may have ceased as of 2019 (POPRC 2022a). DP is considered a High Production Volume (HPV) chemical by the OECD, which indicates that more than 1000 tonnes of the chemical are used per year in at least one member country (OECD 2020).

The chemical DP is registered in the EU under REACH, for introductions between 100–1000 tonnes per annum (REACH n.d.). Sweden registered a use of 5 tonnes of DP in 2006 and 11 tonnes in 2005 (Kaj et al. 2010). In Canada, surveys indicate that between 1 and 10 tonnes of DP was imported by a few companies over the 2011–2016 period (Government of Canada 2019). In Japan, DP was listed as being imported and/or manufactured in the range 1–<1000 tonnes in 2015 (NITE 2020).

Existing Australian regulatory controls

AICIS

No specific controls are currently available for this chemical.

Public

No specific controls are currently available for this chemical.

Workers

The chemical is not listed on the Hazardous Chemical Information System (HCIS) and no specific exposure standards are available (SWA n.d.).

Environment

The use of the chemical is not subject to any specific national environmental regulations.

International regulatory status

Exposure Standards

No specific exposure standards for this chemical were identified.

United Nations

In 2019, the Stockholm Convention's Persistent Organic Pollutants Review Committee concluded DP (CAS No. 13560-89-9) and its *syn*-isomer (CAS No. 135821-03-3) and *anti*-isomer (CAS No. 135821-74-8) fulfilled the screening criteria in Annex D of the

Convention, accepting DP as exhibiting bioaccumulation, persistence, potential adverse environmental effects and its potential to undergo long range transport (decision POPRC-15/2) (POPRC 2019). Therefore, these chemicals are considered to have the characteristics of POPs. An Annex E has been prepared (POPRC 2022b) and the committee concluded that DP is likely, as a result of its long range environmental transport, to lead to significant adverse human health and/or environmental effects such that global action is warranted.

The review committee has completed an Annex F Risk Management Evaluation (RME) and made recommendations to the Conference of the Parties (COP) to the Stockholm Convention to list the chemical in Annex A of the Convention. This recommendation was accepted during the 11th COP in May 2023 (Stockholm Convention n.d.b). The recommendation included 5 year exemptions for use in motor vehicle, medical devices, aerospace and defence application and an exemption in the manufacture of replacement parts in these applications until end of life of the article or 2044 (POPRC 2022a).

Canada

The chemical DP is not listed under Schedule 1 (the Toxic Substances List) of the Canadian Environmental Protection Act 1999 (CEPA 1999) (Government of Canada 2020).

The chemical DP has been subject to assessment and risk management measures conducted under CEPA. In 2019, Environment and Climate Change Canada (ECCC) and Health Canada conducted a joint scientific assessment relevant to the evaluation of DP in Canada (Government of Canada 2019). This assessment concluded that DP is toxic under Section 64 of CEPA because it is entering the environment in a quantity or concentration or under conditions that have or may have an immediate or long term harmful effect on the environment or its biological diversity. It also concluded that DP meets the criteria for persistence and meets the criteria for bioaccumulation, as defined in the Persistence and Bioaccumulation Regulations made under CEPA.

Proposed amendments to the *Prohibition of Certain Toxic Substances Regulations, 2022* will prohibit the manufacture, import, use, sale and offer for sale of DP and all products containing the substance in Canada (Government of Canada 2022). Exemptions for the use of DP in specific aerospace and automobile parts will be in place for 5 years and an exemption on the use of articles containing DP will be in place for 20 years. It also involves pursuing the nomination of DP as a chemical of mutual concern under the *Canada – U.S. Great Lakes Water Quality Agreement*.

Asia

China's Ministry of Ecology and Environment (MEE) has included DP on their 2023 List of Key Controlled New Pollutants (MEE 2022). China intends to ban production, processing, use, import, and export of DP from January 2024.

European Union

A Regulatory Management Option Analysis (RMOA) was initiated for the chemical by ECHA based on bioaccumulation and persistence, which led to the chemical being identified and listed as a Substance of Very High Concern (SVHC) candidate (ECHA 2018a; ECHA 2018b). The chemical was further recommended for inclusion in Annex XIV of the REACH Regulation (List of Substances Subject to Authorisation). In 2022, an opinion of ECHA committees supporting a restriction under Annex XVII of REACH was published. This restriction proposes derogations of varying scope and length for several sector uses (ECHA 2022).

The chemical has been listed as a Network of Reference Laboratories, Research Centres and Related Organisations for Monitoring of Emerging Environmental Substances (NORMAN) Candidate Emerging Substance. Norway has added the chemical to the national list of priority substances from January 2019 with a national goal to phase out use by 2020 (POPRC 2020). The chemical is also listed in Sweden's KEMI PRIO Database as a phase-out substance for persistence, bioaccumulation and toxicity (PBT); and being a substance that is very persistent and bioaccumulative (vPvB).

United States

The chemical is listed under the Toxic Substances Control Act (TSCA) Inventory and is subject to the Chemical Data Reporting Rule, requiring notification of production, import and use volumes to the United States Environment Protection Agency (US EPA 2016). The chemical is registered as 'active' on the Chemical Substance Inventory, which indicates that it has recently been manufactured, imported or processed by industry in the USA (US EPA 2020b). DP is listed as a high production volume chemical (HPV) chemical in the United States (US EPA 2020a) with an estimated annual production of 450–4500 tonnes from 1986 (POPRC 2021).

Human exposure

Workers

There are direct and indirect sources of the chemical exposure at workplaces. Direct occupational exposure arises from industrial processes where the chemical is processed or handled. The possible routes of direct worker exposure are mainly via dermal contact. However, the degree of dermal absorption is likely to be very low based on the chemical's high molecular weight and log K_{ow} value (see **Relevant physical and chemical properties**). Inhalation of the chemical vapour is not expected due to its low vapour pressure (see **Relevant physical and chemical properties**).

After the chemical has been incorporated into the polymeric matrix, normal day-to-day dermal exposure from using polymer articles is considered to be negligible.

The main sources of indirect exposure in the workplace are exposure to dust and indoor air containing the chemical. Dermal exposure to dust or inhalation of air particles containing the chemical may occur at offices, industrial or recycling sites.

Occupational exposure studies

Workers in e-recycling facilities are exposed via inhalation of air particles containing the chemical released from e-waste. Treatment of e-waste consists of dismantling and crushing operations, which results in the release of the chemical from electronic equipment. There is limited information on the amount of electronic equipment containing the chemical that are recycled in Australia. There are no Australian measured data available for the chemical concentrations at recycling sites. The average (41 ng/m^3) total DP (sum of *anti*- and *syn*-DP) in personal air samples from a large-sized e-recycling facility ($n = 36$ workers) in Canada were higher compared with small-sized e-recycling facilities ($n = 22$ workers) which had an average of 6.5 ng/m^3 (flow rate: 2 L/min; mean sampling time: 456 minutes). Workers with the task of dismantling e-waste were exposed to concentrations of *anti*- and *syn*-DP 4–5 times higher than those with supervision tasks (Gravel et al. 2019).

Workers in offices are exposed to dust particles containing the chemical released from articles present in offices. The average concentrations of *anti*- and *syn*-DP detected in indoor floor dust sampled from 47 offices in the UK were 210 ng/g and 60 ng/g, respectively (POPRC 2021; Tao et al. 2016). In Australia, a mean value of 0.079 ng/g was reported from office dust samples (n = 4) collected in 2014 (POPRC 2021; Wong et al. 2017). Indoor air sampled from 20 offices in the UK had average concentrations of *anti*- and *syn*-DP of 1.8 pg/m³ and 1.3 pg/m³ (air sampling rate: 1.677 m³/day), respectively. The concentration of the chemical in dust from UK offices may be attributed to the wide variety of articles used in offices including furniture, electronic and electrical equipment. It may also reflect the recent replacement of office equipment and the local restrictions on other flame retardant chemicals (Tao et al. 2016).

Public

The public may be exposed to the chemical through direct or indirect means of exposure.

Public exposure to a chemical is not uniform across a population. Some groups or individuals may have higher potential exposures because, for example, they live in the vicinity of industrial sources.

Direct public exposure

The chemical is an additive flame retardant in many consumer articles. Due to the chemical's low vapour pressure, inhalation exposure is not expected.

Releases due to leaching or migration of the chemical resulting from normal public use are expected to be negligible due to the physical-chemical properties of the chemical. The negligible water solubility of the chemical also limits the potential for water, sweat or saliva-mediated transfer or migration. Therefore, normal day-to-day dermal exposure to the chemical from using polymer articles is considered to be negligible.

Although young children or toddlers may mouth articles containing the chemical there is no evidence that the chemical is present in products intended for children, such as toys. In a preliminary product testing conducted by Health Canada, the chemical was not detected in 39 subsamples collected from 23 children's manufactured items (e.g. foam chair, nursing pillows, toys, etc.) purchased in retail stores in Ottawa, Canada (Health Canada 2019).

Indirect public exposure

No specific Australian information is available.

Based on international data, indirect exposure of the public may occur by inhalation of air, and oral and dermal exposure to indoor dust containing the chemical. Flame retardants in articles can migrate into dust through abrasion or fragmentation processes (Cao et al. 2014).

Analysis on the distribution pattern of DP in dust showed there were more fine particles than coarse particles, making potential human exposure more likely (Cao et al. 2014).

In several countries, DP has been detected in dust samples from multiple sources, including offices, residential buildings, schools, childcare centres, cars and aircraft cabins (POPRC 2022a; POPRC 2021). While the maximum concentration of DP detected in dust was reported to be 124000 ng/g (Cao et al. 2014), typically lower values have been reported in most studies, ranging from 0.08 to 9900 ng/g (POPRC 2022a). No significant differences in

dust concentrations of various flame retardants, including DP, were found between the United Kingdom, Canada, Sweden and China (Wong et al. 2017).

Low levels of DP have been detected in indoor air samples from residential sources, in several countries (Canada, China, India, Japan, UK, USA). Urban air samples had higher levels of DP compared to rural air samples (POPRC 2022a).

Indirect exposure may also occur via food. DP has been detected in the following food groups: sugar and confectionary, legumes, fish, shellfish, meat and eggs, suggesting transition of DP into biota and food components. Overall, results suggested that contamination could be higher in fish and seafood compartment. Based on the measurements from various locations, levels of DP in food were around 1000 times higher in areas close to manufacturing or recycling facilities (POPRC 2021; POPRC 2022a).

Biological monitoring studies

Measured data from biological monitoring (such as blood) reflects actual exposure to the substance being detected. It indicates the occurrence of past exposure and the subsequent absorption into the body. It seldom specifies the actual route(s) of exposure, much less the primary route or the contribution of other routes.

The chemical DP has been detected in human blood and breast milk in various locations (POPRC 2021; POPRC 2022a). The highest DP levels have been detected in the blood (2958 ng/g) and hair (2159 ng/g) of workers and residents living near production facilities and e-waste recycling sites in China (ECHA 2021; POPRC 2021; POPRC 2022a). Median DP levels measured in breast milk ranged from 0.02 ng/g from Canadian residents to 4.46 ng/g in Chinese residents having spent more than 20 years in e-waste recycling region (POPRC 2021b). DP has also been detected in utero, in cord blood serum and placenta, indicating potential prenatal exposure (Ben et al. 2013; Ben et al. 2014; Chen et al. 2015; Yin et al. 2020; Zhang et al. 2013). According to the POPRC, 'maternal transfer of bioaccumulative substances in utero represents a potential risk to embryonic development and may represent the largest source of FRs input to offspring during the first few years of life' (POPRC 2022a).

Health hazard information

Toxicokinetics

The toxicokinetics of DP was studied in a non-guideline in vivo absorption, distribution and excretion study (REACH n.d.; ECHA 2021). Three groups of Sprague Dawley (SD) rats were administered a single dose of DP (in corn oil) by oral gavage at 1 mg/kg body weight (bw) (3 females and 2 males) or 113 mg/kg bw (2 females). Another group (2 females) was fed 1% DP in diet for 14 days prior to receiving the chemical at 1 mg/kg bw DP, via oral gavage. Minimal absorption of DP was observed after oral intake. In rats administered a single dose of 1 mg/kg bw, females and males excreted 83.5% and 92.7% of the dose in faeces, respectively, with the remaining administered dose absorbed. At 113 mg/kg bw, 96.5% was excreted in faeces. In the 1 mg/kg bw dose group pre-fed at 1% concentration, 102% of the dose was excreted in faeces indicating almost no absorption. In all animals, all organs and tissues besides the liver and residual carcass contained below 1 ppm. The liver of females at 113 mg/kg bw contained 1.66 ppm and the residual carcass contained 1.25 ppm. At 1 mg/kg bw, the livers of males and females contained 1.6% and 2.3% of the dose, respectively, and the residual carcass contained 5.1 and 5.1% of the dose, respectively. The residual carcass of the pre-fed females contained 4.4% of the dose. In almost all animals, the administered

dose was excreted unchanged in faeces. Less than 1% of the dose was excreted in urine and expired in air at 4 days in all dose groups.

In a non-guideline toxicokinetics study, 2 groups of SD rats (3/group; sex unspecified) were administered a single oral dose of 0.57 mg of radiolabelled DP (27.6 µCi in 0.5 mL) suspended in water (with 5% polysorbate 80 and 5 % gum Arabic) (REACH n.d.; ECHA 2021). One group was euthanised at 4 hours, while the other at 24 hours after administration. Within 4 hours after administration, <0.1% was excreted in urine, and at 24 hours, <1% was excreted. Within 24 hours after administration, mean faecal excretion of 94.6% of the administered dose was observed, with the remaining 5.4% being absorbed. The amounts of radioactivity detected in the blood (total blood volume), kidneys and liver were <2%, <1%, and <5% of the administered dose, respectively. One metabolite was found in the liver, but its identity and concentration were not reported.

Acute toxicity

Oral

Based on the available studies, the chemical has low acute oral toxicity. In a non-guideline acute oral toxicity study conducted similarly to the Organisation of Economic Co-operation and Development (OECD) Test Guideline (TG) 401, Sherman rats (3 males and 2 females/dose) were administered DP (in water, 0.5% polysorbate 80) by oral gavage at doses of 1500, 3000, 6000, 12 500 or 25000 mg/kg bw without controls. The median lethal dose (LD50) for acute oral toxicity was >25000 mg/kg bw (REACH n.d.).

In a non-guideline acute oral toxicity study conducted similarly to OECD TG 401, male SD rats (2/dose) were administered DP (in corn oil) by oral gavage at doses of 10, 31.6, 100, 316, 1000 or 3160 mg/kg bw without controls. The LD50 for acute oral toxicity was >3160 mg/kg bw (REACH n.d.).

Dermal

Based on the available study, the chemical has low acute dermal toxicity.

In a non-guideline acute dermal toxicity study (with limited study details), DP was applied dermally to rabbits (strain, sex unspecified; 2 with intact skin and 2 with abraded skin per dose) at doses of 500, 1000, 2000, 4000 or 8000 mg/kg bw without controls. The LD50 for acute dermal toxicity was >8000 mg/kg bw (REACH n.d.).

Inhalation

Based on the available studies, the chemical has low acute inhalation toxicity.

In a non-guideline acute inhalation toxicity study, rats (5/sex/dose; strain unspecified) were exposed to DP as a dust for 1 hour at concentrations of 105 or 300 mg/L without controls. The median lethal concentration (LC50) for inhalational toxicity was >300 mg/L (REACH n.d.).

In non-guideline acute inhalation toxicity study, Charles River rats (5/sex) were exposed to DP as a dust (particle size: 56.8% at 1–5 µm, 10.2% at 6–10 µm, 14.8% at 11–25 µm, 18.2% at >25 µm) for 4 hours at a concentration of 2.25 mg/L. The LC50 for inhalational toxicity was >2.25 mg/L (REACH n.d.).

Corrosion/Irritation

Skin irritation

No data are available.

Eye irritation

Based on the available study, the chemical is not considered to cause eye irritation.

In a non-guideline eye irritation study conducted similarly to OECD TG 405, DP (0.1 mL) was instilled into one eye of rabbits (strain, sex unspecified; 3/group). In 2 groups, the treated eyes were washed after 2–4 seconds, while the treated eyes in the third group were not washed. The eyes of each animal were observed for effects at 24, 48 and 72 hours after treatment. No irritation effects were observed in any animal (REACH n.d.).

Respiratory irritation

No signs of respiratory irritation were reported in acute and repeated dose inhalation toxicity studies (see **Acute inhalation toxicity** and **Repeated inhalation toxicity**).

Sensitisation

Skin sensitisation

Based on the available studies, the chemical is not considered to be a skin sensitiser.

In a non-guideline Buehler test conducted similarly to OECD TG 406, 10% DP (in propylene glycol) was applied topically (under occlusion) to the skin of guinea pigs (strain unspecified; 10 test, 5 control animals) for induction and challenge. For the induction phase, the chemical was applied for 5 hours per day (under occlusion). For challenge (2 weeks later), the chemical was applied to a naïve site for 5 hours (under occlusion), and reactions were recorded at 24 and 48 hours after application. No skin reaction in any animal was observed. (REACH n.d.).

Albino rabbits (n = 6) and healthy human volunteers (n = 50) were exposed topically to DP-impregnated or non-impregnated fabric (under occlusion). Rabbits were exposed to the fabric for 24 hours for 5 non-consecutive days. After a 2 week rest period, they were challenged for 24 hours. Human volunteers were exposed to the fabric for 24 hours for 10 non-consecutive days. After 11–13 days of rest period, they were challenged for 48 hours. No skin reactions were observed in any animal or volunteer (REACH n.d.).

Repeat dose toxicity

Based on the available studies, the chemical does not cause serious overt systemic health effects following repeated oral, dermal and inhalation exposure. Given the persistent and bioaccumulative properties of the chemical there is uncertainty regarding the long term health effects from exposure to the chemical. There is some evidence of potential effects on thyroid hormone and metabolic pathways (see **Endocrine effects**).

Oral

In a non-guideline repeated dose oral toxicity study conducted similarly to OECD TG 408, Charles River rats (15/sex/dose) were fed DP in the diet at doses of 0, 10000, 30000 or 100 000 ppm for 90 days. No treatment related mortalities occurred; however, 2 non-treatment related deaths occurred in the 100 000 ppm dose group. Slightly increased liver weights without histopathological findings were reported in the highest dose group. The reported no observed adverse effects level (NOAEL) was 100 000 ppm which corresponded to approximately 5870 mg/kg DP bw/day and 7670 mg/kg DP bw/day, for males and females, respectively (REACH n.d.).

In a non-guideline repeated dose oral toxicity study, SD rats (7 males/dose) were fed DP (in corn oil) at doses of 0, 1, 10, or 100 mg/kg bw/day in their feed, for 90 days. In a parallel study, SD rats (7 males/dose) were fed DP at 0 or 100 mg/kg bw/day for 45 days followed by 45 days in which rats were fed regular feed without DP. The main result of the study was that DP accumulated preferentially in the liver rather than in the muscles or serum. At the highest dose, the activities of alanine amino transferase (ALT), alkaline phosphatase (ALP) and total bile acids were significantly decreased, and glucose level had significantly increased. In the liver, gene-expression of several key enzymes were altered such as n-acetyltransferase, sulfotransferases and CYP2B1. However, no significant changes in absolute body and liver weights, or relative liver weights, were observed in any of the animals and no histopathological changes in the liver were reported. A non-significant increase in thyroid stimulating hormone (TSH) level was observed at the highest dose, but no other changes in thyroid hormones were reported (Li et al. 2013). The study did not report other parameters such as haematology, urinalysis, gross pathology or histopathology of many organs/tissues. The POPRC noted that 'background contaminations gave 100% detection of DP in the control tissues with increasing levels during the experiment which may have impacted the dose-responses on endpoints such as clinical serum parameters, serum thyroid hormones and liver mRNA expressions (POPRC 2022b).

In a combined repeated dose oral toxicity with reproduction/developmental toxicity study conducted according to OECD TG 422, CD-1 (CrI:CD(SD)) rats (10/sex/dose) were administered DP in olive oil by oral gavage at doses of 0, 750, 1500 or 5000 mg/kg bw/day for 28 days. No clinical or histopathological signs were observed. Accidental deaths due to misdosing were reported without any further details. A no observed effect level (NOEL) of >5000 mg/kg bw/day was reported (REACH n.d.).

In a non-guideline study on the effects of DP at the molecular level, male ICR mice (6/dose) were administered DP (in corn oil) by oral gavage at doses of 0, 500, 2000 or 5000 mg/kg bw/day for 10 days. No significant body weight changes were observed. No significant changes in the relative weights of kidneys and testicles were observed. At the 2000 mg/kg bw/day dose, relative liver weight was significantly increased. Analysis of gene expression of various metabolites in the liver and some markers of DNA oxidative damage showed that DP can induce oxidative stress in mouse livers (Wu et al. 2012). The study did not report other parameters such as haematology, urinalysis, gross pathology or histopathology.

Dermal

Based on the available study, the chemical does not cause serious overt systemic health effects following repeated dermal exposure.

In a non-guideline repeated dose dermal toxicity study similar to OECD TG 410, DP (50% in 3% aqueous methylcellulose) was applied topically (without occlusion) at 500 or 2000 mg/kg

bw/day, 5 days/week, for 28 days on the scarified skin of New Zealand White rabbits (5/sex/dose). Minimal local irritation was observed following repeated application for 18 days or longer, but no other clinical signs were observed. No systemic effects were reported. The reported NOAEL was >2000 mg/kg bw/day (REACH n.d.).

Inhalation

Based on the available study, the chemical does not cause serious overt systemic health effects following repeated inhalation exposure.

In a non-guideline repeated dose inhalation toxicity study similar to OECD TG 412, Charles River rats (5/sex/dose) were exposed by whole body inhalation to DP as a dust (particle size: 60% at $\leq 10 \mu\text{m}$) at target concentrations of 0, 0.5 or 1.0 mg/L (equivalent to 0, 0.64 and 1.524 mg/L analytical concentration) for 6 hours/day, 5 days/week, for 4 weeks. Increased liver weights with hepatocellular hypertrophy, increased lung weights without histopathological changes, and slight diuresis were observed in both dose groups. These changes were considered to be functional adaptations rather than treatment related adverse effects. The reported no observed adverse effect concentration (NOAEC) was 1.524 mg/L.

Genotoxicity

Based on the available data, the chemical is not expected to be genotoxic.

In a non-guideline in vitro bacterial reverse mutation assay similar to OECD TG 471, DP was reported to be not mutagenic in *Salmonella typhimurium* strains TA 98, TA 100, TA 1535, TA 1537 and TA 1538 with and without metabolic activation at concentrations up to 10 000 $\mu\text{g}/\text{plate}$ (REACH n.d.).

In an in vitro bacterial reverse mutation assay conducted according to OECD TG 471, DP was reported to be not mutagenic in *S. typhimurium* strains TA 98, TA 100, TA 102, TA 1535 and TA 1537 with and without metabolic activation at concentrations up to 5000 $\mu\text{g}/\text{plate}$ (REACH n.d.).

In an in vitro mammalian cell micronucleus test conducted according to OECD TG 487, DP was reported to be not clastogenic in human peripheral blood lymphocytes with and without metabolic activation at concentrations up to 750 $\mu\text{g}/\text{mL}$ (REACH n.d.).

In a non-guideline in vitro mammalian cell gene mutation test similar to OECD TG 476, DP was reported to be not mutagenic in mouse lymphoma L5178Y cells at concentration up to 20 $\mu\text{g}/\text{mL}$ without metabolic activation and up to 10 $\mu\text{g}/\text{mL}$ with metabolic activation. Higher concentrations could not be evaluated due to significant precipitation (REACH n.d.).

Carcinogenicity

No data are available to evaluate the carcinogenic potential of the chemical.

Reproductive and development toxicity

Based on the available study, the chemical is not expected to cause specific adverse effects on fertility and development following repeated oral exposure. No data are available on neurodevelopmental effects in mammalian species.

In a combined repeated dose oral toxicity with reproduction/developmental toxicity study, conducted according to OECD TG 422, CD-1 (Crj:CD(SD)) rats (20/sex/dose) were administered DP (in corn oil) by oral gavage at doses of 0, 750, 1500 or 5000 mg/kg bw/day. Males were treated for at least 63 days with 21 days pre-mating, 14 days of mating, and 28 days after mating. Females were treated for up to 64 days with 21 days pre-mating, 14 days of mating, and throughout gestation. Half of the pregnant females were euthanised on gestational day 20, the other pregnant females were further treated and euthanised on day 3 of lactation. No treatment related effects were reported in the parental animals. For the offspring (F1), no treatment related effects were observed with respect to viability, number of male and female pups, body weights, gross and visceral malformations or variations, and behaviour up to day 4 after birth. The reported NOEL for effects on parental animals and on embryofetal and postnatal development was >5000 mg/kg bw/day (REACH n.d.).

Endocrine effects

There is a concern that persistent polyhalogenated flame retardants such as DP have endocrine effects. While available data indicate potential changes to thyroid hormone pathways, no consistent pattern of effects were evident. In a single study in mice, effects in adipose tissues and changes in biomarkers that may be associated with insulin resistance were reported. DP has been shown to inhibit insulin signalling in vitro in mammalian adipose cells and changes to gut microbiota were reported in a study in rats. Sufficient data are not available to draw conclusions.

Effects on thyroid hormones

An association between serum thyroid hormones (TH) and DP levels was reported in human mother-infant pairs near an e-waste recycling area in China (Ben et al. 2014). The concentration in maternal sera from the 20-year residents' group was 2 to 3-fold higher than the 3-year residents' group. *Syn*- and *anti*-DP were detected in placenta and umbilical cord serum samples, indicating transfer from maternal to foetal tissues. Levels of thyroid stimulating hormone (TSH) were significantly lower in maternal serum in the group that had lived in the area for 20 years (n = 44), than for those whose mothers had been residents to the area for 3 years or less (n = 22), (p = 0.046). When concentrations of PBDEs were used as a control variable, the DP concentrations were positively associated with total triiodothyronine concentrations (T3) in sera from mothers who had lived in the area for over 20 years.

In a study of grade 5 students (n = 174, average age of 10 years) from schools near a petrochemical complex in China, linear regression analysis showed no significant association between serum *anti*- or *syn*-DP levels and levels of T3, T4 or TSH (Guo et al. 2018).

In another study comparing serum levels of adults living in an e-waste region (n = 54) and a control region (n = 58), lower levels of TSH, thyroid binding globulin and mRNA expression of thyroid receptor (TR α) and higher level of iodothyronine deiodinase 1 were observed in highly exposed individuals. The results were not statistically significant between the exposed and the control group. The *syn*- and *anti*-DP mean and range in serum was 57 (12–1000) and 58 (11–1450) ng/g lw in residents from the e-waste area, and 3.2 (0.36–12) and 5.9 (0.67–38) ng/g lw for the control group (Guo et al. 2019).

In a survey on 101 participants in rural Central Appalachia, a significant positive correlation was found between DP exposure for females (62% of the total sample) and thyroid stimulating hormone (TSH) level, but not with free thyroxine (FT4) and free triiodothyronine (FT3) levels (Wang et al. 2020).

In the 90 day SD rat study (see **Repeated dose toxicity – Oral**), a non-significant increase in thyroid stimulating hormone (TSH) level was observed at the highest dose, but no other changes in thyroid hormones were reported (Li et al. 2013).

In an in vitro study, the binding affinity of DP to thyroid receptors (TR) was measured by fluorescence competitive binding assay. DP was shown to directly bind to TRs (Zhu et al. 2022). Between the two TRs, dechloranes were more inclined to bind with TR β , suggesting that dechloranes may be selective in different tissues and organs, and act as TR antagonists (Zhu et al. 2022).

Effects on metabolism

In a 28 day feeding study, male C57BL/6 mice (n = 8/dose), fed either a high-fat diet (HFD) or regular diet (CD), were administered DP (in corn oil) at doses of 0, 10, 100 or 1000 $\mu\text{g}/\text{kg}$ bw/day by oral gavage daily except on days when a glucose tolerance test was performed (days 0, 7, 14, 21 and 28) (Peshdary et al. 2020).

The following effects on adipose tissues and changes in biomarkers that may be associated with insulin resistance were reported:

- Significantly impaired glucose tolerance was observed in the low and high dose groups (10 and 1000 $\mu\text{g}/\text{kg}$ bw/day) fed the high-fat diet with DP, but not in groups fed the regular diet with DP.
- Increased serum insulin in low and mid dose groups fed the regular diets.
- Increase in serum fructosamine, cholesterol and albumin in mice fed the high fat diet with DP.
- Increased serum levels of plasminogen activator inhibitor-1 (PAI-1) in the mid and high dose groups fed regular diet. DP induced the development of hypertrophied white adipose tissue (WAT), in the low and high dose HFD groups, and in the low dose CD group, as shown by histological examination of adipocyte hypertrophy.
- DP exposure induced “whitening” of brown adipose tissue (BAT), in the high dose HFD group and in the low and high dose CD groups. DP also reduced BAT uncoupling protein 1 expression, as shown by the decreased mRNA expression in a dose-dependent manner in the CD groups.

Both monotonic and non-monotonic dose responses for different biochemical parameters were observed. DP had no effect on total body weight, percent fat/lean mass and food consumption in all groups.

An in vitro insulin stimulation was conducted on murine adipocytes exposed to DP at 0, 1, 10, 100 or 1000 nM and human primary adipocytes exposed to DP at 0, 10, 100 or 1000 nM in dimethyl sulfoxide (Peshdary et al. 2020). DP directly inhibited insulin signalling in murine adipocytes and human primary subcutaneous adipocytes in vitro. Reduced insulin signalling levels (serine/threonine-specific kinase AKT) were observed in confluent 3T3-L1 murine preadipocytes treated with concentrations of DP (100 and 1000 nM), (by 36%, and 51%, respectively) as compared with control. In primary human adipocytes, insulin signalling levels were decreased in a dose-dependent manner (by 18%, 31%, and 37%, respectively) compared with control, reaching statistical significance with 1000 nM DP. However, DP had no effect on the expression levels of adipogenic markers.

A reduction in species diversity and richness of gut microbiota in both offspring and dams was reported in a study in rats fed 5 mg DP/kg/day through pregnancy and/or lactation (POPRC 2022b).

Environmental exposure

The chemical DP is released to the environment through diffuse emissions from imported articles as well as from point source emissions such as manufacturing plants, wastewater treatment plants (WWTP), e-waste recycling facilities and landfills.

Diffuse emissions from articles are expected to be the most common source of DP releases to the environment (OECD 2011). DP occurs in articles as an additive flame retardant. Sampling of wallpaper, latex paint, boards (e.g., floorboards), glue, sealant, PVC pipes and foam (e.g. sound absorbing foam) in China detected DP up to 5.2 ng/g (Hou et al. 2018). DP typically occurs in polymers at a concentration of 3% (ECHA 2021). Additive flame retardants are not chemically bound to the polymer matrix in which they are blended, so they can be released into the surrounding environment (Shaw et al. 2010).

Emissions to air are expected to be in the form of dust particles, which are commonly detected in indoor settings (Tao et al. 2016). As dust is quickly transported through the air, DP may contaminate regions distant from its source emissions. Globally, total DP atmospheric emissions are estimated to be 0.02 t/year and 3.2 t/year in a low and high emission scenario, respectively, with the latter modelling being closer to actual measurements in the Arctic (Hansen et al. 2020). Diffuse emissions of DP to the aquatic environment occur typically from laundering of treated fabrics (Saini et al. 2016), which are subsequently discharged into wastewaters (Schreder and La Guardia 2014) or directly into the environment.

Point emission sources of DP are manufacturing plants, wastewater treatment plants, e-waste recycling facilities, landfills and contaminated biosolids. The highest environmental concentrations have been observed in areas where DP is manufactured (Sverko et al. 2011). In Australia, emissions from this source are not expected because the chemical is not manufactured here.

Wastewaters from urbanised areas are usually collected in treatment plants before being released to the environment. Influent of Canadian wastewater treatment plants contained residues of DP in the range 17–247 ng/L (Shanmuganathan et al. 2018). Effluents still had measurable concentrations of the chemical (range 2–139 ng/L), while the bulk of residues was retained in the sludge and biosolids (de la Torre et al. 2011; Shanmuganathan et al. 2018). Thus, wastewater and sewage treatment plants act as point emission sources of DP into the aquatic environment.

An important source of point emissions is e-waste recycling facilities, where high levels of DP are frequently detected in soil (Ren et al. 2013; Yu et al. 2010) as well as in air and trees of the surrounding areas (Chen S-J et al. 2011). Although Australian recycling facilities have not yet reported the presence of DP, residues of other halogenated flame retardants found in urban soils of Melbourne are associated with e-waste facilities rather than manufacturing sites (McGrath et al. 2017). This suggests that DP is likely to be present in those areas as well. Since Australians generate among the highest per-capita rates of e-waste in the world (Islam and Huda 2020), with estimates of 485 kt in 2010 (Islam and Huda 2019), this source of emissions may be significant.

Articles containing DP are more likely to be disposed of to landfill, including a small proportion of electronic items. Some 6% of the landfill waste in South Australia is e-waste (Kiddee et al. 2014). Leachate from landfill usually contains halogenated flame retardants (Kiddee et al. 2014), and DP has been detected in wetland plants that receive leachate from a decommissioned landfill in Singapore (Wang Q and Kelly 2017).

The application of biosolids to agricultural land is a further route of exposure to DP, since this chemical is frequently detected in sludge and biosolids from wastewater treatment plants (POPRC 2021). Such contamination is eventually transferred to the aquatic environment via run-off from the land amended with biosolids (Navarro et al. 2018).

Environmental fate

Partitioning

The chemical partitions to soil when released into the environment.

Measurements of DP in air samples demonstrate that <1% of the chemical was present in the gas phase, indicating that almost all of the atmospheric DP is adsorbed onto particles (Hoh et al. 2006; Qiu et al. 2010; Reche et al. 2019). Strong sorption to aerosol particles is also expected based on a calculated log K_{OA} of 12.26 (Sverko et al. 2011). Sorption to particles may slow down reaction rates, increase the actual half-lives in air and facilitate the long range transport of the chemical (POPRC 2022b).

Given its low solubility in water (<1.67 ng/L) and high adsorption to organic matter (log K_{OW} 9.3), DP tends to partition to sediments in aquatic environments. A partition coefficient (log K_p) of 6.65 for sediment/water has been measured in the laboratory (OxyChem 2004).

Calculations with a standard multimedia partitioning model (Fugacity Level III) assuming equal emissions to air, water and soil compartments predict that this chemical will partition preferentially to the soil compartment (91.5%), with the remainder expected to go into water (5%) and sediment (3.4%) and negligible amounts into the air (gaseous) phase (0.056%) (US EPA 2017).

Degradation

The chemical DP is stable and undergoes minimal or no degradation in the environment by both abiotic and biological processes. There are no measured half-life data for degradation of this chemical in surface water, sediment or soil (POPRC 2022b; ECHA 2021). These characteristics demonstrate that DP meets the Persistence criterion in Annex D of the Stockholm Convention (UNEP 2001).

The chemical DP is considered to be relatively photo-stable in air under natural light conditions (>400 nm), although studies suggest that *anti*-DP might be more photo-degradable in air than *syn*-DP in the UV-C range (200–280 nm). Photo-reduction leading to dechlorination was the main photo-degradation process under xenon lamp UV irradiation (Wang S et al. 2013). The only study on photo-degradation in water estimated a half-life of >48 years (Chou et al. 1979), but this study is not representative of natural conditions because it used an aqueous solution of DP with 5% acetonitrile under a mercury lamp emitting wavelengths >290 nm for 168 hours (POPRC 2022b; ECHA 2021).

The chemical DP has no functional groups that are susceptible for hydrolysis and is not expected to hydrolyse in the environment.

Laboratory tests with sludge under aerobic conditions show minimal biodegradation of DP. A standard test with activated sludge (OECD 301C) reported 0.6% biodegradation in 2 weeks, indicating no biodegradability (POPRC 2022b). OxyChem (2004) reported DP as having 0% aerobic biodegradability based on the results of a 21 day standard (APHA and AWWA 1971) test. An experimental study with an agricultural soil incubated at 25°C showed that 4.2–8.2%

of the initial spiked DP had degraded after 260 days, and a soil half-life in the range of 1325–2948 days was estimated (Cheng et al. 2019).

The chemical DP resists degradation in natural environments. Monitoring data confirm that DP undergoes minimal degradation in sediments over time. In sediment core samples from Lake Ontario, Canada, DP was first detected in layers of the mid-1970s and its concentration increased rapidly to reach its peak (310 ng/g dry weight) in the mid-1990s (Qiu et al. 2007).

Bioaccumulation

The chemical DP bioaccumulates in organisms and is transferred through food webs. Based on the available monitoring data, the chemical meets the bioaccumulation criterion of Annex D of the Stockholm Convention, including section (c)(iii), which is relevant when monitoring data in biota indicates the potential of the chemical to bioaccumulate is sufficient to justify consideration within the scope of the Convention.

The DP's high log K_{OW} (9.3) indicates it will have a strong tendency to accumulate in fatty tissues of organisms. This property, in combination with its high calculated log K_{OA} (12.26) further indicates that the chemical is likely to bioaccumulate in air-breathing animals (Kelly et al. 2007).

The DP's tendency to bind to particles in aquatic environments means that ingestion of sediments by benthic invertebrates is a likely entry point for this chemical into food webs. Experimental evidence supports this pathway. Sediment-bound DP is bioavailable to benthic invertebrates, as demonstrated in the benthic worm *Lumbriculus variegatus* by Li et al. (2014), albeit at relatively low uptake rates (biota-sediment accumulation for DP stereoisomers ranging from 0.21–0.48 g organic carbon/g lipid). The chemical is also accumulated by soil invertebrates such as earthworms, which represent one of several entry points for DP into terrestrial food webs (Xiao et al. 2013).

In addition to bioaccumulation, maternal transfer of DP to eggs has been demonstrated. Eggs laid by hens (*Gallus domesticus*) that had been fed daily on food contaminated with this chemical (300 ng/day for 58 days), contained residues of DP in the range 100–230 ng/g lipid weight (lw) during the exposure period (Li Z-R et al. 2021). DP in eggs of birds is found exclusively in the yolk (Smythe et al. 2020). Field samples of water-snakes (*Enhydris chinensis*) and their eggs also showed DP residues of 150 and 99 ng/g lw, respectively, indicating maternal transfer of this chemical (Liu Y et al. 2018).

Bioconcentration factors (BCFs) of 5700 and 9300 L/kg for *syn*- and *anti*-DP, respectively, have been determined in common carp (*Cyprinus carpio*) exposed for 32 days to environmentally realistic concentrations of DP (~0.1 ng/L for each isomer) without use of solubilising agents (Wang et al. 2020). However, laboratory studies suggest that bioaccumulation of DP in fish and other aquatic organisms is more likely through ingestion of contaminated food (i.e. dietary exposure) than through passive bioconcentration from the water column. BMFs of 5.2 for *syn*-DP and 1.9 for *anti*-DP were measured in dietary exposures of rainbow trout (*Oncorhynchus mykiss*) to spiked food for 49 d followed by 112 d un-spiked food (Tomy et al. 2008). Dietary exposures in *Cyprinus carpio* yielded serum BMFs of 1.06 for *syn*-DP and 1.23 for *anti*-DP, although BMFs < 1 were determined for all other tested tissues (carcass, liver, gonad, gastrointestinal tract, gill) (Tang et al. 2018).

In several monitoring studies, biomagnification factors (BMFs) >1 have been observed for DP, indicating this chemical is bioaccumulative according to international criteria (Chen et al. 2021). Apple snails (*Pomacea canaliculata*) in rice paddies showed BMFs of 3.1 for *syn*-DP

and 2.3 for *anti*-DP isomers (She et al. 2013), and BMFs of 2.1 and 2.3 have been reported for insect predators such as frogs and toads near e-waste recycling facilities (Liu Y et al. 2018; Wu et al. 2018). Fish-eating birds such as the common kingfisher (*Alcedo atthis*) had estimated BMFs in the range 0.18 to 2.9, depending on fish species (Mo et al. 2013). Marine birds like the brown skua (*Stercorarius antarcticus*) that prey on Antarctic penguins showed an BMF of 18.9 (Kim et al. 2015), while in terrestrial food webs BMFs of 0.32 and 2.0 were estimated for predatory owls feeding on sparrows or rodents, respectively (Yu L et al. 2013).

There is also strong evidence indicating that DP biomagnifies up to high trophic levels, with numerous monitoring studies measuring trophic magnification factors (TMFs) >1 (POPRC 2021). TMFs of 11.3 and 6.6 for *syn*- and *anti*-DP, respectively were determined in an aquatic food web in an e-waste recycling region of China (Wu J-P et al. 2010). Another study from an e-waste recycling region of China (Huai'an) determined a TMF of >1 for DP (Wang D-G et al. 2015). Bioaccumulation of DP has also been determined in an Antarctic aquatic-terrestrial food web, with a TMF of 3.02 determined across nine species sampled from the Fildes Peninsula (Na et al. 2017).

Some data suggest differential uptake rates for *syn*-DP and *anti*-DP into different tissues and by different organisms at different exposure concentrations (Hang et al. 2013; Li Y et al. 2013a; Li Y et al. 2013b). There is also evidence to suggest that the dechlorination products of DP and DP-related compound, DPMA, are bioaccumulative (Wang D-G et al. 2015). Some field surveys have detected higher concentrations of DPMA than DP in tissues of lake trout (*Salvelinus namaycush*) (Sverko et al. 2010).

Environmental transport

The chemical DP undergoes atmospheric transport via aerosol particles and has potential for being transported away from the emission sources. These characteristics demonstrate that DP meets the criterion for long-range transport (LRT) in Annex D of the Stockholm Convention (UNEP 2001).

Modelling studies suggest that DP has transport and persistence properties similar to listed POPs (Sverko et al. 2011). Although DP has a relatively short half-life in air (<2 d), 99% of atmospheric DP is expected to be particle bound (Hoh et al. 2006), which is likely to give it a much greater persistence and hence potential for atmospheric transport (POPRC 2022b). The chemical has a modelled transfer efficiency [the percentage of emission flux to air that is deposited to the surface (water and soil) in a remote area] of 9.7%, which is above the threshold considered suitable for LRT potential (Government of Canada 2019).

Numerous monitoring studies demonstrate the LRT of DP to remote polar and mountainous regions via the atmosphere, ocean currents and migratory birds. DP has been detected in air, soil, sediment, water and biota of the Arctic, Antarctica and Tibetan plateau, which are far from production facilities and heavily populated areas (POPRC 2022b).

Air monitoring data from the Arctic (AMAP 2017; Salamova et al. 2014), Antarctica (Möller et al. 2010) and the Tibetan Plateau (Liu X et al. 2018; Ma et al. 2017) demonstrate that DP undergoes widespread atmospheric transport around the world. Air concentrations of DP up to 79 pg/m³ have been reported in the Southern Ocean around Tasmania (Möller et al. 2012). Ocean waters in the Arctic and Antarctic regions (Carlsson et al. 2018; Möller et al. 2010; Na et al. 2015) further support the LRT of this chemical. These studies confirm particle-bound transport as a major pathway for DP movement in both air and water matrices (Möller et al. 2010; Sverko et al. 2011).

Transport of DP via the atmosphere and ocean currents helps explain the presence of this chemical in soil, lichens and sediment samples taken from remote areas of Norway, Antarctica and Tibet (Gao et al. 2018; Liu X et al. 2018; Na et al. 2015; Yang et al. 2016).

DP has been detected in a wide range of Arctic biota (Carlsson et al. 2018; de Wit et al. 2020; Dreyer et al. 2019; Letcher et al. 2015; Simond et al. 2017; Vorkamp et al. 2015) including in:

- amphipods
- fish
- marine birds and raptors
- polar bears
- seals
- moss
- cetaceans.

The chemical DP has also been found in reindeer dung from Ny-Ålesund, Norway (Na et al. 2015). In Antarctica and the southern oceans, DP has been found in lichens, krill, molluscs, fish, birds, seals and found to bioaccumulate in both marine and terrestrial food webs (Gao et al. 2018; Kim et al. 2021; Na et al. 2017; Roscales et al. 2016).

The chemical DP has been detected in eggs, tissues and feathers of many bird species (Chen et al. 2021; Guerra et al. 2011; Huber et al. 2015; Mo et al. 2019), many of which are migratory. Transport of this chemical from sources to remote regions may occur via migratory birds such as gulls (Desjardins et al. 2019), the White stork (*Ciconia ciconia*) (Muñoz-Arnanz et al. 2011) and the Northern fulmar (*Fulmarus glacialis*) (Mortensen et al. 2022), and this provides further evidence of the LRT of DP.

The fraction of *anti*-DP in environmental samples decreases with increasing distance from the source as a result of more rapid degradation of the *anti*- isomer than the *syn*- isomer under UV-light (Möller et al. 2010). Some monitoring studies suggest that changes in isomer ratios with increasing distance from the sources provide evidence of LRT, but not all studies are conclusive in this regard (POPRC 2021).

Predicted environmental concentration (PEC)

The chemical DP is a dispersed pollutant detected in a wide range of environmental compartments worldwide. Australian environmental levels are assumed to be similar to the reported international monitoring data for soil, sediment, water and discharges from wastewater treatment plants (WWTP).

The chemical DP has been detected in soils at average 478 ng/g dw (dry weight), with highest levels of up to 3327 ng/g dw around e-waste recycling areas and 13400 ng/g dw near industrial electronic facilities in China (POPRC 2021; Wu et al. 2010; Yu Z et al. 2010), so it may occur at comparable levels in Australian industrial and e-waste recycling areas. Given its slow degradability, consecutive use of biosolids in one location will result in cumulative soil concentrations of this persistent chemical.

The chemical DP has been detected in riverine and coastal sediments at average loads of 110 ng/g dw, with highest residues of 8260 ng/g dw in e-waste recycling areas of China (POPRC 2021; Zhang et al. 2011). Similar loads can be expected to occur in Australian waterbodies, particularly those receiving discharges from e-waste recycling areas.

Concentrations of DP in freshwaters range from 0.2 ng/L in lakes (Lee et al. 2021) and 0.52 ng/L in stormwaters (Kaj et al. 2010) to an average 9.6 ng/L in rivers from industrial regions in China (Hong et al. 2018; Zhen et al. 2018). Seawaters have average levels of 0.42 ng/L, but can reach up to 5.1 ng/L in coastal waters from industrialised areas of China (Jia et al. 2011).

The chemical DP has been detected in influents of WWTPs at concentrations in the range 0.46 to 73 ng/L (Shanmuganathan et al. 2018; Xiang et al. 2014). After treatment, effluents still carry average residues of 9.3 ng/L, although concentrations as high as 139 ng/L have been measured in effluents of Canadian WWTPs (Shanmuganathan et al. 2018), most of it as particulate-bound residues since the dissolved fraction cannot contain more than 2 ng/L (He et al. 2014). Australian releases from STP facilities are expected to have similar levels of DP residues in the effluent.

Compounds related to DP have also been detected in environmental compartments and biota. These compounds may be impurities in the technical mixture (Li Y et al. 2013a; Sverko et al. 2010) and/or may be environmental transformation products of DP (POPRC 2021). These compounds are DP monoadduct (DPMA), decachloropentacyclooctadiene (DP-C110) and undecachloropentacyclooctadiene (DP-C111). They have been detected in aquatic sediment, fish and wild bird egg samples (Sverko et al. 2011; Wang D-G et al. 2015). There is some evidence to suggest these compounds as bioaccumulative (Wang D-G et al. 2015). Sverko et al. (2010) found DPMA occurring in fish tissues at higher concentrations than DP in Lake Ontario, Canada.

Environmental effects

The available aquatic and soil toxicity data are insufficient to determine DP toxicity according to domestic aquatic toxicity criteria. However, reported effects in available studies include:

- oxidative stress to most tested organisms
- changes to transcriptional responses of both thyroid and sex hormone related genes in the brain in zebrafish
- hypothyroidism in marine birds.

Potential neurotoxicity has been indicated in a study in zebrafish and a study in earthworms. However, the effects observed do not follow a dose-response pattern and these cannot be concluded as being due solely to the action of this chemical.

The available evidence indicates that DP has the potential to cause adverse effects in aquatic species and terrestrial organisms, and therefore meets the criterion of Annex D of the Stockholm Convention, specifically (e)(ii) which is satisfied if there is ecotoxicity data that indicate the potential for damage to the environment.

Effects on aquatic life

The very low water solubility of DP has presented challenges for the conduct of standard short and long duration aquatic toxicity tests. Very hydrophobic chemicals are difficult to test, particularly in aqueous exposures, as the chemical preferentially partitions out of the water column and adsorbs onto surfaces. This makes it difficult to accurately characterise exposure levels—particularly where nominal concentrations are used—and there is often a risk of overestimating the concentrations test organisms are exposed to (OECD 2019b). These concerns demonstrably apply to DP, which is known to be rapidly lost from the water column, even with the use of solubilising agents (Barón et al. 2016). To better characterise effects

due to chemical exposures some aquatic tests have measured DP concentrations in the tissues of exposed fish.

Acute and chronic toxicity

Ecotoxicity data from acute and chronic tests with DP are lacking. The difficulty DP' properties (in particular, high hydrophobicity) present for standard testing of this chemical mean that there is insufficient information from published tests to determine its aquatic toxicity according to domestic criteria. The weak evidence of potential neurotoxicity suggests the possibility of harm in the Australian environment (EPHC 2009).

Screening tests with embryonic zebrafish (*Danio rerio*) exposed to DP from 6 to 120h post fertilisation across concentrations spanning several orders of magnitude (up to 6.4µM = 4.2 mg/L) showed little or no effects on teratogenicity endpoints and locomotor behaviour (Noyes et al. 2015). A recent study that used a modified 96 h Fish Embryo Toxicity (FET) test (TG 236) also showed no acute toxicity or treatment related abnormalities in zebrafish embryos after exposing female fish to DP for 12 weeks. However, the validity of this study is questionable due to the high cumulative mortality rate (30–50%) at 24 hours post fertilisation in both the control and the exposed groups (Katsiadaki et al. 2022).

Nevertheless, adverse effects in aquatic organisms have been observed after exposure to DP and these include oxidative stress, potential neurotoxicity and endocrine effects. Several non-standard aquatic studies suggest potential effects in genetic, biochemical and histopathological assays. Oxidative stress has been observed in fish (Chen X et al. 2017; Hang et al. 2013; Kang et al. 2016; Liang et al. 2014), macroalgae (Gong et al. 2018) and marine bivalve exposure studies (Barón et al. 2016).

Neurotoxicity

Exposure of zebrafish embryos (*Danio rerio*) to a range of DP concentrations (15, 30 and 60 mg/L in solvent solution) significantly altered their movements and free-swimming speed in response to dark stimulation, inhibited axonal growth of primary motoneurons and induced apoptotic cell death and lesions in the muscle fibres of the fish (Chen X et al. 2017). Although the concentrations used by the authors are well above those found in the aquatic environment, the effects observed were linked to internal DP concentrations in the range 758 to 2148 ng/g dry weight, which overlap those found in fish of highly contaminated areas, thus overcoming some of the challenges in interpreting experiments based on aqueous (especially nominal) doses of highly hydrophobic chemicals. Axonal and behavioural alterations may indicate neurotoxic effects of a chemical (US EPA 1998), and the effects of DP correspond to this mode of action.

Effects on terrestrial life

Limited available data suggest DP as not inducing toxic effects in terrestrial organisms. Data indicate that earthworms do not suffer adverse effects in acute or chronic exposures to this chemical, although its reproductive effects have not been studied. Development of birds appears not to be affected by exposure to this chemical.

Acute and chronic toxicity

An acute NOEC value of 50 mg/kg soil dw for lethality and growth endpoints was determined for earthworms (*Eisenia fetida*) exposed to soil-spiked concentrations of DP for 14 days, in an experiment approximating OECD TG 207 (Zhang L et al. 2014). In chronic exposures of

E. fetida up to 28 days, also approximating OECD TG 207 methods, a NOEC of 12 mg/kg dry soil was determined for lethality and growth (Yang et al. 2016). In both cases, the endpoints referred to the highest nominal concentrations used, suggesting limited DP toxicity to these soil invertebrates.

Oxidative stress has been observed in earthworms (Zhang L et al. 2014), quails (Li Y et al. 2013b) and mice (Wu B et al. 2012), although these effects are usually reversible.

In vitro experiments with chicken embryonic hepatocytes did not show cytotoxic effects up to 3 μ M (1.9 mg/L), while chicken embryos following injection of DP (500 ng/g egg) into the air cell of eggs prior to incubation developed normally (Crump et al. 2011). Also, Japanese quails (*Coturnix japonica*) that developed under exposure of DP (~1 μ g/g egg) in the eggs did not have their liver and thyroid glands affected (Jacobsen et al. 2017).

Neurotoxicity

Long exposures (28 day) of earthworms (*Eisenia fetida*) to DP at 0.1, 0.5, 6.25 and 12.5 mg/kg soil dw resulted in inhibition of acetylcholinesterase (AChE), an enzyme involved in nerve signal conduction, but the reduction in enzyme activity did not follow a dose-dependent response (Yang et al. 2016). Similarly, shorter exposures to 0.1, 1, 10 and 50 mg/kg soil dw decreased AChE activity levels on day 14, although a hormetic effect was observed at 3 and 7 days. After 14 d exposure, changes in some transcriptomic profiles related to neurotoxicity were observed, further suggesting this mode of action (Zhang L et al. 2014). In both cases, the concentrations used are within the known range of residues of DP in soils and sediments in the environment.

Effects on sediment dwelling life

Sediment toxicity data is not available for DP (Government of Canada 2019).

This is a critical data gap in the characterisation of its toxicity, given its tendency to bind to sediments (OxyChem 2004)—in which it persists for very long timescales (Qiu et al. 2007)—and its subsequent bioavailability to sediment-dwelling organisms (Li et al. 2014).

Endocrine effects

Changes to transcriptional responses of both thyroid and sex hormone related genes in the brain were observed in a study in zebrafish (*Danio rerio*). Dietary exposures of adult zebrafish to this chemical (30 to 420 ng/g wet weight) prompted the upregulation of *cyp19b* sex hormone gene in the brain, whereas levels of androgen in the testes were not affected. Exposure to the highest concentration also upregulated the *crh* and *tsh β* thyroid genes in the brain and increased levels of T4 (thyroxine) in plasma by 31%. However, these effects were not observed in zebrafish embryos exposed to high aqueous concentrations of DP (267 ng/ml), suggesting that for aquatic organisms contaminated food rather than water is a more relevant route of exposure to this chemical (Kang et al. 2016).

A recent field study found that concentrations of DP up to 0.76 ng/g ww (average 0.28 ng/g ww) in liver of northern fulmars (*Fulmarus glacialis*) from the Faroe Islands in the North Atlantic, were positively correlated with imbalanced levels of T4 and T3 hormones in plasma and with mRNA levels of UDP-glucuronyltransferase-1 (UGT1) in the liver – an enzyme that regulates thyroid hormone metabolism (Mortensen et al. 2022). While it is difficult to draw conclusions from this study due to the small sample size (n = 9), these effects are consistent with similar effects observed in rats (Zhu et al. 2022) and zebrafish (Kang et al. 2016).

Predicted no-effect concentration (PNEC)

The chemical DP is highly bioaccumulative and environmentally persistent. These two hazard characteristics combined have the potential to result in a range of long term effects on aquatic life exposed to this chemical which cannot be readily identified through standard aqueous toxicity tests. For such chemicals, it is not currently possible to estimate a safe exposure concentration using standard extrapolation methods based on laboratory screening level tests. Therefore, PNECs have not been derived for this substance.

Categorisation of environmental hazard

The environmental hazards of DP were assessed by the international Review Committee of the Stockholm Convention on Persistent Organic Pollutants, which concluded the chemical has the characteristics of a POP (POPRC 2019).

Based on the information reviewed in this evaluation, the chemical has the characteristics of a POP according to the criteria in Annex D of the Convention:

Persistence

Based on measured data from degradation studies in air, water and sediment, DP meets the Annex D criteria for persistence.

Bioaccumulation

Based on high measured bioconcentration factors (BCF) in fish, a log K_{ow} value above the threshold of 5.0, and slow biotransformation in organisms, DP meets the Annex D criteria for bioaccumulation.

Adverse effects

Based on ecotoxicity data that indicate the potential for damage to the environment. DP meets the Annex D criteria for adverse effects.

Potential for long range environmental transport (LRT)

Based on evidence of monitoring data showing that long range atmospheric transport of the chemical to a receiving environment, DP meets the Annex D criteria for LRT.

Uncertainty

This evaluation was conducted based on information that may be incomplete or limited in scope. Some relatively common data limitations can be addressed through use of conservative assumptions (OECD 2019) or quantitative adjustments such as assessment factors (US EPA 1984). Others must be addressed qualitatively, or on a case-by-case basis (OECD 2019). The most consequential areas of uncertainty for this evaluation are discussed below:

- Introduction volumes and Australian environmental monitoring data were not available for DP. Therefore, Australian environmental concentrations are assumed to

be similar to those reported in international monitoring studies, apart from atmospheric concentrations, for which Australian data are available.

- The absence of suitable toxicity data (especially sediment tests), involving measured internal doses over chronic/multigenerational timeframes, poses difficulties for determining DP's toxicity. However, DP's global ubiquity, persistent and bioaccumulative properties, and reported effects in the scientific literature, indicate that adverse environmental effects cannot be ruled out.

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