Australian Government
Department of Health and Aged Care
Australian Industrial Chemicals Introduction Scheme

Heteropolycyclic-alkanol, carbomonocycle-alkanesulfonate

Assessment statement (CA09845)

10 April 2024

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AICIS assessment statement (CA09845)

Chemical in this assessment

AICIS Approved Chemical Name (AACN)
Heteropolycyclic-alkanol, carbomonocycle-alkanesulfonate

Reason for the assessment

An application for an assessment certificate under section 31 of the *Industrial Chemicals Act 2019* (the Act).

Certificate Application type

AICIS received the application in a Health Focus type.

Defined scope of assessment

The chemical has been assessed:

- as imported into Australia at up to 100 tonnes/year
- as imported at up to 30% concentration as a component of water treatment products for end use by industrial workers
- as a component of water treatment products, except for drinking water treatment

Summary of assessment

Summary of introduction, use and end use

The assessed chemical will not be manufactured or reformulated in Australia. It will be imported into Australia at up to 30% concentration as a component of water treatment products for end use by industrial workers. These products will not be used for treatment of drinking water.

There will be no consumer use of products containing the assessed chemical or public exposure to the treated water.

Human health

Summary of health hazards

The submitted toxicological data on an analogue chemical (see **Supporting information section**) indicate that the assessed chemical is:

- of low acute oral and dermal toxicity
- slightly irritating to skin and eyes
- a weak skin sensitiser

Based on the submitted combined repeated dose oral (gavage) toxicity study with reproduction/developmental toxicity screening test conducted in rats, the analogue chemical showed some systemic toxicity effects from 100 mg/kg bw/day (increased liver weights in females and statistically significantly increased T4 levels in males) or from 350 mg/kg bw/day (increased liver weights in males and increased thyroid weights in females), with decreased implantation sites in females at all test doses from 100 mg/kg bw/day (see **Supporting information** section).

The analogue chemical was positive in two *in vitro* assays (a bacterial reverse mutation assay and a Mouse Lymphoma Assay with metabolic activation). However, the analogue chemical was negative in an *in vitro* mammalian micronucleus test using TK6 cells and in an *in vivo* alkaline comet assay conducted on the jejunum, glandular stomach or liver tissues of rats. Considering the analogue data (including one negative rat alkaline comet assay), the assessed chemical is likely to be non genotoxic.

The submitted analogue data warrant hazard classification for skin sensitisation Category 1B for the assessed chemical (see section below).

No inhalation toxicity data were provided by the applicant.

Hazard classifications relevant for worker health and safety

The chemical satisfies the criteria for classification according to the *Globally Harmonized System of Classification and Labelling of Chemicals* (GHS) (UNECE 2017) for hazard classes relevant for worker health and safety as adopted for industrial chemicals in Australia as follows:

Health hazards	Hazard category	Hazard statement
Skin sensitisation	Skin Sens. 1B	H317: May cause an allergic skin reaction

Summary of health risk

Public

When introduced and used in the proposed manner, the public is not expected to be exposed to the assessed chemical. This assessment does not identify any risks to public health that require specific risk management measures.

Workers

Workers may be exposed to the assessed chemical at up to 30% concentration during connection and disconnection of the transfer lines to the water treatment system. While the exposure to the assessed chemical will be mainly dermal and ocular, inhalation exposure (if mists or aerosols are formed) may also occur. To mitigate the risks to workers from any skin sensitisation effects and repeated exposure, control measures would be required (see **Means for managing risk**) to minimise the exposure. It is anticipated by the applicant that engineering controls such as enclosed and automated processes and local ventilation will be implemented where possible. Workers are expected to wear appropriate personal protective equipment (PPE) such as impervious gloves, protective clothing and respiratory protection to reduce exposure.

Environment

Summary of environmental hazard characteristics

- Persistent
- Not Bioaccumulative
- Not Toxic

Environmental hazard classification

The chemical satisfies the criteria for classification according to the GHS (UNECE 2017) as Acute Category 3 (H402) and Chronic Category 3 (H412) based on the toxicity data for fish, invertebrates and algae. Considerations were also made for biodegradation of the assessed chemical.

Environmental Hazard	Hazard Category	Hazard Statement
Hazardous to the aquatic environment (acute / short-term)	Aquatic Acute 3	H402: Harmful to aquatic life
Hazardous to the aquatic environment (long-term)	Aquatic Chronic 3	H412: Harmful to aquatic life with long lasting effects

Summary of environmental risk

The assessed chemical will be introduced as a component of water treatment products. This use may result in the release of the assessed chemical to sewers, or surface waters. In these compartments, the assessed chemical is expected to mainly partition to the water compartment with small amounts partitioning to soil and sediment.

Although the assessed chemical is persistent, it does not meet all three PBT criteria. It is unlikely to have unpredictable long-term effects and its risk may be estimated by the risk quotient method (RQ = PEC ÷ PNEC). Based on calculated RQ values < 1 for the river and ocean compartments, the environmental risk from the introduction of the assessed chemical can be managed.

Means for managing risk

Workers

Recommendation to Safe Work Australia

- It is recommended that Safe Work Australia (SWA) update the *Hazardous Chemical Information System* (HCIS) to include the classification relevant to work health and safety (see **Hazard classifications relevant for worker health and safety**).

Information relating to safe introduction and use

The information in this statement, including recommended hazard classifications, 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.

The following control measures could be implemented to manage the risk arising from exposure to the assessed chemical during reformulation:

- Use of engineering controls such as
  - Automated and enclosed systems where possible
  - Adequate workplace ventilation to avoid accumulation of mists or aerosols
- Use of safe work practices to
  - Avoid contact with skin and eyes
  - Avoid inhalation of mists or aerosols
- Workers should wear the following personal protective equipment (PPE)
  - Impervious gloves
  - Protective clothing
  - Respiratory protection where local ventilation may be inadequate
- As the assessed chemical is a skin sensitiser, the control measures may need to be supplemented with health monitoring for any worker who is at significant risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health.
- A copy of the Safety Data Sheet (SDS) should be easily accessible to workers.

Conclusions

The Executive Director is satisfied that the risks to human health and the environment associated with the introduction and use of the industrial chemical can be managed.

Note:

1. Obligations to report additional information about hazards under s 100 of the *Industrial Chemicals Act 2019* apply.
2. You should be aware of your obligations under environmental, workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Supporting information

Chemical identity

AACN	Heteropolycyclic-alkanol, carbomonocycle-alkanesulfonate

Relevant physical and chemical properties

All measured values are based on the studies provided on the free base of the assessed chemical.

Physical form	Light brown powder
Melting point/Boiling point	Decomposes at 176 °C
Density	1240 kg/m^3 at 20 °C
Vapour pressure	0.0032 Pa at 25 °C
Water solubility	0.147 mg/L at 20°C
pK_a	Basic pK_a = 4.1 (calc.) Acid pK_a = 12.2 (calc.)
log K_{ow}	1.89
Log K_{oc}	2.08
Particle size	Inhalable fraction (< 100 µm): 60.5% Thoracic fraction (< 10 µm): 1.48% Respirable fraction (< 5.5 µm): 0.11%
Autoignition temperature	No self-ignition up to melting point
Flammability	Not highly flammable

Health hazard information

No toxicological data were submitted for the assessed chemical. The applicant has submitted toxicological data for a suitable analogue chemical, which were appropriate for read across to the assessed chemical.

Acute toxicity

Oral

In an acute oral toxicity study (OECD TG 425), 5 female Sprague Dawley (SD) rats were administered a single dose of the analogue chemical (in corn oil) at 2,000 mg/kg bw. No mortalities or macroscopic findings were observed in any treated animals. Abnormal clinical signs included piloerection (4/5), partially chewed food (2/5) and few faeces in cage (1/5), chromorhinorrhea (1/5) and localised hair loss on the side of the neck (1/5). Body weight gain appeared normal. The median lethal dose (LD50) was determined to be greater than 2,000 mg/kg bw indicating the analogue chemical is of low acute oral toxicity.

Dermal

In an acute dermal toxicity study (OECD TG 402), a single dose of the analogue chemical at 2,000 mg/kg bw was applied (semi-occlusive for 24 hours) on the intact skin of 10 Wistar rats (n = 5/sex). No mortalities or signs of systemic toxicity were observed. Blanching of the skin and/or light brown discolouration of the epidermis were observed in all treated animals at the 24-hour observation. All treated skin sites appeared normal by the 2-day observation in females and 6-day observation in males. Very slight erythema in a female (1/5) was observed at the 3-day observation only. There were no treatment-related macroscopic findings. Body weight gain was normal. The LD50 was determined to be greater than 2,000 mg/kg bw, indicating the analogue chemical is of low acute dermal toxicity.

Corrosion/Irritation

Skin irritation

The analogue chemical was tested for skin irritation using 2 male and 1 female albino New Zealand rabbits (OECD TG 404). A 1-hour or 4-hour, semi-occluded application of the undiluted test substance to the intact skin of the rabbits produced very slight erythema (maximum score of 1) in 3/3 animals and very slight oedema (maximum score of 1) in 2/3 animals at the 1-hour observation following 4-hour exposure, and very slight erythema (maximum score of 1) in 1/3 animal at the 24-hour observation following 1-hour exposure. Following the 4-hour exposure, the mean individual erythema or oedema scores were zero at 24, 48 and 72 hours. Under the conditions of this study, the analogue chemical was slightly irritating to skin but does not meet the GHS criteria for classification.

Eye irritation

The analogue chemical was tested for eye irritation using 2 male and 1 female albino New Zealand rabbits (OECD TG 405). A single application of the undiluted test substance to one eye of each rabbit produced no corneal or iridial effects. Very slight to slight conjunctival irritation (maximum score of 2) was observed in the treated eye of all animals at the 1-hour observation. The mean individual conjunctival redness scores at 24, 48 and 72 hours were 0.7, 0.3, 0.7, respectively. The mean individual conjunctival oedema scores at 24, 48 and 72 hours were 0.3, 0.0, 0.0, respectively. Under the conditions of this study, the analogue chemical was slightly irritating to the eyes but does not meet the GHS criteria for classification.

Sensitisation

Skin sensitisation

One in chemico and two in vitro cell based assays were conducted to evaluate the skin sensitisation potential of the analogue chemical. These tests are part of Integrated Approach to Testing and Assessment (IATA) which address specific key events of the Adverse Outcome Pathway (AOP) leading to development of skin sensitisation.

The direct peptide reactivity assay (DPRA) is an in chemico method and aims to address the first key event (KE) (molecular initiation) of the AOP by measuring the interaction of the test chemical with cysteine and lysine, small synthetic peptides representing the nucleophilic centres in skin proteins (OECD TG 442C). The ARE-Nrf2 luciferase assay aims to address the second KE (keratinocyte activation) of the AOP by measuring the expression of a reporter luciferase gene under the control of a promoter from the antioxidant response element (ARE), a responding gene known to be upregulated by contact sensitisers (OECD TG 442D). In the third KE assay, the Human Cell Line Activation test (h-CLAT) assay, the skin sensitization potential of the test substance is evaluated by measuring the changes in the expression of cell surface markers (CD54 and CD86) associated with the process of dendritic cell activation in the human leukemia cell line (THP-1) following exposure to a test substance (OECD TG 442E). The results of these assays are considered using the applicable Defined Approaches (DA) in the Defined Approaches for Skin Sensitisation (DASS) Guideline (OECD TG 497) for Classification and Labelling purposes. Based on the results of the AOP assays (negative DPRA, positive ARE-Nrf2 luciferase assay, positive h-CLAT) and using the ‘two out of 3’ DA in the DASS Guideline (OECD TG 497), the analogue chemical is predicted to be a skin sensitiser.

The skin sensitisation potency of the analogue chemical was evaluated using a local lymph node assay (LLNA) (OECD TG 429). Three groups of female mice (CBA/Ca) (5 animals/group) received topical applications (25 µL/ear) of the analogue chemical to the entire dorsum of each ear lobe at 10%, 25% and 50% concentrations in acetone/olive oil (4:1) for 3 consecutive days. On day 6, 250 µL of phosphate-buffered saline containing 20 µCi of ^{3}H-methyl thymidine (equivalent to 80.1 µCi/mL 3HTdR) were injected into each animal via the tail vein and the animals were euthanised approximately 5 hours afterward for further processing.

There were no mortalities or signs of systemic toxicity. Very slight to well-defined erythema was observed from days 2 to 6. The analogue chemical at 10%, 25% and 50% concentrations produced a Stimulation Index (SI) of 1.8, 4.0 and 4.5, respectively. The analogue chemical was characterised as a skin sensitiser and the concentration of the analogue chemical expected to cause a 3-fold increase in 3HTdR incorporation (extrapolated EC3 value) was calculated to be 18.2%.

The positive results of the analogue chemical in the LLNA test confirmed the assessed chemical as a skin sensitiser. Using the EC3 value of the analogue chemical (18.2%) against GHS criteria for classification, the assessed chemical is classified as a Category 1B skin sensitiser (H317: May cause an allergic skin reaction).

Repeat dose toxicity/Reproductive and development toxicity

In a range finding study, the analogue chemical was administered by oral gavage to Wistar rats (3 animals/sex/dose) for up to 14 consecutive days at 0, 500, 750, and 1,000 mg/kg bw/day (phase 1). Two high dose females were killed *in extremis* on day 9 due to adverse clinical signs including decreased respiratory rate, piloerection, lethargy, hunched posture, pallor of the extremities and/or dehydration. Necropsy revealed congested brown coloured contents in the stomach, small intestines and large intestines. These findings were considered by the study authors to be related to the viscosity of the test substance. Although no clinical signs or macroscopic findings were observed in the remaining high dose female, the animal was sacrificed due to the deaths of the other two animals. Based on these findings, two additional groups of female rats (3 animals/sex/dose) were administered the analogue chemical via oral gavage at doses of 750, and 1,000 mg/kg bw/day using a less viscous formulation of the test substance (phase 2).

Increased salivation was observed in phase 1 and phase 2 animals. Noisy respiration was observed in all groups and was considered by the study authors to reflect difficulties with dosing particular animals rather than treatment-related effects. Body weight loss or no body weight gain were observed in phase 1 and phase 2 high dose females, phase 1 high dose males, and phase 1 and phase 2 mid dose females. Lower food consumption was observed in phase 1 and phase 2 high dose females, phase 1 high dose males, and phase 1 and phase 2 mid dose females. Lower food conversion efficiency (the ratio of body weight change/dietary intake) was observed in phase 1 males and females, phase 2 high dose females, phase 1 and phase 2 mid dose females. Macroscopic observations included brown coloured contents in the stomach of phase 2 mid dose (2/3) and high dose (3/3) females. Based on the results of this study, dose levels of 100, 350 and 750 mg/kg bw/day were selected for further investigation.

In a combined repeated dose oral (gavage) toxicity study with reproduction/developmental toxicity screening test (OECD TG 422), the analogue chemical was administered daily in polyethylene glycol to Wistar rats (12 animals/sex/group) at dose levels of 0, 100, 350 and 750 mg/kg bw/day for up to 6 weeks for males (during pre-mating and mating) and 8 weeks for females (during pre-mating, mating, gestation and lactation).

One high dose female rat was found dead on Day 50. Clinical signs of toxicity observed on Day 49 included hunched posture, pallor of the extremities, apparent hypothermia and piloerection. This female also experienced a total litter loss on Day 1 *post partum*. Necropsy revealed an enlarged liver, spleen and right adrenal, a pale area on the liver, thin appearance of the non-glandular region of the stomach and raised limiting ridge and a pale mass in the right ventricle of the heart. Histopathology revealed abscessation in the lungs and marked inflammatory change in the heart with the presence of bacterial colonies, likely caused by sepsis that was complicated by pregnancy. The study authors stated that these effects were incidental and not treatment-related.

No treatment-related changes in body weight, body weight gain, water consumption, behavioural parameters, functional performance, sensory reactivity were observed in the surviving animals at up to 750 mg/kg bw/day.

In comparison to control, there was statistically significant increases in the absolute and relative mean liver weights of females in the low dose (8% and 10%, respectively), mid dose (22% and 22%, respectively) and high dose (33% and 34%, respectively) groups and males in the mid dose (13% and 14%, respectively) and high dose (29% and 33%, respectively) groups. There were also increases in the absolute and relative mean thyroid weight of females in the mid dose (43% and 44%, respectively) and high dose (31% and 33%, respectively) groups as well as the absolute and relative mean kidney weight (12% and 16%, respectively) and prostate

weight (26% and 24%, respectively) of males in the high dose group. These effects were considered adaptive or not treatment-related by the study authors as there was no supporting evidence of histopathology.

Macroscopic examinations revealed dark coloured contents in the stomach in males of the mid dose (2/12) and high dose (5/12) groups. Increased pelvic space in one kidney (2/12) and mottled appearance of the liver (2/5) were also observed in high dose males. In females, enlarged, mottled appearance or pale areas were observed in the liver of rats in the low dose (1/12), mid dose (2/12) and high dose (1/11) groups. However due to lack of dose-responses, the study authors considered these findings as incidental and not toxicologically relevant.

Microscopic observations included follicular hypertrophy in the thyroid glands in low dose (1/12), mid dose (9/12) and high dose (7/11) females. The study authors reported these effects as not toxicologically relevant due to lack of dose-responses.

In the high dose group, statistically significant increases in mean bile levels (180%) were noted in males. In high dose females, decreases in mean total protein levels (-16%, respectively) and albumin levels (-22%, respectively) were observed. In both sexes, increases in mean cholesterol levels were noted in the mid dose (27% for males and 86% for females) and high dose groups (72% for males and 76% for females). These changes were not considered by the study authors to be toxicologically relevant as there were no clear dose-related responses. There were also some haematology and clinical chemistry parameters with statistically significant differences but were within the historical control data for this strain of rats.

There was also a statistically significant increase in mean levels of thyroxine (T4) in low dose (20%), mid dose (24%) and high dose (30%) males in comparison to controls. The study authors considered these findings as incidental and not treatment-related.

The No Observed Adverse Effect Level (NOAEL) for systemic toxicity was reported by the study authors as 750 mg/kg bw/day, the highest dose tested.

No toxicologically significant changes were observed in any of the reproductive parameters investigated in this study such as mating and fertility indices, gestation index, birth indices, maternal care, post-implantation loss and post-natal survival.

A decrease in mean implantation sites were noted in the low dose (-12%), mid dose (-18%) and high dose groups (-24%) in comparison to control. As this effect was more pronounced in the high dose group, the NOAEL for reproductive toxicity was reported by the study authors to be 350 mg/kg bw/day.

In comparison to control, statistically significant decreases in mean litter size (-28%) at birth as well as mean litter weight per dam (-36%) and reduced mean body weight (-17% for males and -18% for females) by the termination of the study were reported for offspring of the high dose group. A statistically significant decrease in mean litter weight was also observed in the mid dose group (-18%). The study authors considered the reduced weight to be a secondary effect caused by the reduced litter size observed in the study.

A statistically significant increase in the mean actual and normalised anogenital distance was observed in mid dose (10% and 10%, respectively) and high dose (17% and 17%, respectively) females and high dose males (12% and 10%, respectively). However, as the individual values were within the historical controls for all animals (apart from the actual value of 1 male in the low dose group and 1 male in the high-dose group), the study authors considered these effects incidental and not treatment-related due to no dose-response.

There were no treatment-related effects on sex ratio, nipple count and T4 hormone level in pups. No abnormalities were noted during macroscopic external examination at necropsy and microscopic examination of the thyroid grands.

Although the NOAEL for systemic toxicity was reported by the study authors as 750 mg/kg bw/day (highest dose tested), there were increased mean liver weights (over 10% increase compared to control mean weights) in both sexes at mid and high dose levels, increased thyroid weights with follicular hypertrophy in females at mid and high dose groups and statistically significant increases in T4 levels in males of all treated groups. These effects could not be dismissed as non-adverse, without a recovery period in the study to determine the reversibility of organ weight changes and histopathological changes. Based on the reduction of body weights of offspring, the NOAEL for developmental toxicity was reported by the study authors as 350 mg/kg bw/day. However, decreased mean implantation sites were reported at all treatment groups, which may have influenced overall litter body weight.

Genotoxicity

In vitro

The analogue chemical was found to be mutagenic in an *in vitro* bacterial reverse mutation assay (Ames test) using *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and *Escherichia coli* strain WP2*uvr*A, with or without metabolic activation (OECD TG 471). Toxicity and precipitate were seen at ≥ 3,333 µg/plate. Dose-dependent increases in revertant colonies (9.1-fold, maximum increase) were observed with TA98, with metabolic activation. The increases were outside the historical control limit for this strain. No significant increases in the frequency of revertant colonies were recorded for the remaining bacterial strains at up to 5,000 µg/plate, with or without metabolic activation.

In an *in vitro* mammalian micronucleus test (OECD TG 487), the analogue chemical was found to be non-clastogenic or aneugenic in human lymphoblastoid (TK6) cells. The concentrations used in the main experiments (up to 450 µg/mL for the 4-hour treatment with and without metabolic activation, and up to 200 µg/mL for the 27-hour treatment without metabolic activation) were based on data from a preliminary test. Cytotoxicity was seen at ≥ 400 µg/mL in the 4-hour treatment and at ≥ 130 µg/mL in the 27-hour treatment. No significant or dose-dependent increases in micronuclei induction were recorded at any dose tested, in the presence or absence of metabolic activation.

In an *in vitro* mammalian cell gene mutation test (OECD TG 490), the analogue chemical was found to be mutagenic in mouse lymphoma L5178Y cells with metabolic activation. The concentrations used in the main experiments (up to 285 µg/mL and 500 µg/mL for the 4-hour treatment with and without metabolic activation, respectively, and up to 250 µg/mL for the 24-hour treatment without metabolic activation) were based on data from a preliminary toxicity test. A statistically significant increase in mutation frequency in small and large colonies was observed at the 285 µg/mL during the 4-hour treatment with metabolic activation. No biologically relevant increases in mutation frequency were observed after treatment with the analogue chemical at any tested concentrations without metabolic activation.

In vivo

In an *in vivo* alkaline comet assay (OECD TG 489) conducted as part of the combined repeat dose toxicity study with reproduction/developmental toxicity screening test (see **Repeat dose toxicity/Reproductive and developmental toxicity** section), male Wistar rats (5 animals/dose) were administered the analogue chemical in polyethylene glycol via oral gavage at 0, 100, 350 and 750 mg/kg bw/day for 45 consecutive days. No statistically significant

increases in the percentage tail intensity or median percentage tail intensity were observed in the jejunum, glandular stomach and liver in any of the treatment groups in comparison to the control. Under the conditions of the study, the chemical was considered to be non genotoxic.

It is noted that the analogue chemical was negative in an *in vitro* mammalian micronucleus test using TK6 cells and positive in inducing mutations in an *in vitro* Ames test and an *in vitro* mammalian cell gene mutation test with metabolic activation (L5178Y/TK+/- Mouse Lymphoma Assay). The analogue chemical was negative for clastogenicity in an *in vivo* alkaline comet assay conducted on the jejunum, glandular stomach or liver tissues of rats. Considering all the *in vitro* and *in vivo* results, the analogue chemical is likely to be non genotoxic.

Environmental exposure

The assessed chemical will not be manufactured or reformulated in Australia. Hence any environmental exposures from these activities are not expected.

The assessed chemical will be imported to Australia and transported to and packaged in customer industrial sites. The assessed chemical will be used as a component of water treatment products which will be dosed to the systems automatically using dedicated pumps and transfer lines. Hence minimal environmental exposure is expected during the dosing process.

Environmental exposures of the assessed chemical can occur during connection and disconnection of dedicated transfer lines to the system. Any accidental spills/drips occurring during this process, are expected to be minimal and collected and disposed of in accordance according to local government regulations.

Disposal of packaging containing residue of the assessed chemicals, will be sent to licensed companies or trained personnel for safe disposal and hence is not expected to have a significant release to the environment.

Environmental exposures of the assessed chemical are expected from the discharge of waste water to sewers. As a worst-case scenario, 100% of the annual import volume of the assessed chemical is assumed to be released to sewers.

Environmental fate

Partitioning

The assessed chemical is moderately water soluble (water solubility = 147 mg/L at 20°C), slightly volatile (vapour pressure < 0.0032 Pa at 20°C) and highly mobile in soils (log K_{OC} = 2.08). When the assessed chemical is released to water, a majority is expected to stay in water with very small amounts partitioning to soil and sediments.

Degradation

Based on its measured degradation in water, the assessed chemical is categorised as persistent.

A ready biodegradation screening test conducted using the OECD TG 301, DOC die away test showed 32.16% degradation for the assessed chemical. The assessed chemical did not meet the 10-day window and is considered to be not readily biodegradable.

A supplied hydrolysis study conducted according to OECD TG 111 test, for the assessed chemical shows half-life values > 1 year at pH 4, 7 and 9, suggesting the assessed chemical is hydrolytically stable in water under environmentally relevant conditions.

Bioaccumulation

The assessed chemical does not have the potential to bioaccumulate based on its measured log K_{OW} value.

No bioaccumulation information was provided for the assessed chemical. The measured partition coefficient of the assessed chemical is log K_{OW} = 1.89, which is below the domestic bioaccumulation threshold of log K_{OW} = 4.2 (EPHC, 2009).

Predicted environmental concentration (PEC)

A predicted environmental concentration (PEC) for Australian waters was calculated assuming 100% of the introduction volume is released into sewage treatment plants (STP) over 260 days per annum. The extent to which the assessed substance is removed from the effluent in STP processes is based on its physicochemical properties, modelled by SimpleTreat 3.0 (Struijs, 1996).

Based on the partitioning and biodegradability of the assessed chemical, a very small portion of the chemical is expected to partition to sludge (1%) while most of the chemical will remain in effluent (99 %). Total removal during STP treatment is estimated to be 1%. Therefore, 99% of the total introduction volume is estimated to be released to the aquatic environment.

This calculated value is conservative as not all uses of the assessed chemical are expected to result in release to STP.

The calculation of the PEC is detailed in the table below:

Total Annual Import Volume	100,000	kg/year
Proportion expected to be released to sewer	100%	
Annual quantity of chemical released to sewer	100,000	kg/year
Days per year where release occurs	260	days/year
Daily chemical release	384.62	kg/day
Water use	200.0	L/person/day
Population of Australia	25.423	Million
Removal within STP	1%	Mitigation
Daily effluent production	4,877	ML/day
Dilution Factor - River	1.0	
Dilution Factor - Ocean	10.0	

PEC - River	78.07	µg/L
PEC - Ocean	7.81	µg/L

Environmental effects

Effects on aquatic Life

Acute toxicity

The following measured median lethal concentration (LC50) and median effective concentration (EC50) values for model organisms were supplied for the assessed chemical:

Taxon	Endpoint	Method
Fish	96 hr LC50 > 47 mg/L	*Oncorhynchus mykiss* (rainbow trout) OECD TG 203 Semi-static conditions Measured concentration
Invertebrate	48hr EC50 = 41 mg/L	*Daphnia magna* (water flea) Immobility OECD TG 202 Static conditions Measured concentration
Algae	72 hr EC50 = 30 mg/L	*Pseudokirchneriella subcapitata* (green algae) Growth rate OECD TG 201 Static conditions Nominal concentration

Chronic toxicity

The following measured no-effect concentration (NOEC) values for model organisms were supplied for the assessed chemical:

Taxon	Endpoint	Method
Invertebrates	72 hr NOEC = 24 mg/L	*Daphnia magna* (water flea) Immobility OECD TG 202 Static/Semi-static/Flow-through conditions Measured concentration

Taxon	Endpoint	Method
Algae	72 hr NOEC = 5 mg/L	*Pseudokirchneriella subcapitata* (green algae) Growth rate OECD TG 201 Static conditions Nominal concentration

Predicted no-effect concentration (PNEC)

A predicted no-effect concentration (PNEC) of 100 µg/L was calculated for the assessed chemical in the aquatic environment. This value was derived using the endpoint value for chronic algae toxicity (5 mg/L). An assessment factor of 50 was applied to this endpoint as acute toxicity data were provided for all three trophic levels and chronic toxicity data were provided for two trophic levels (EPHC, 2009).

Categorisation of environmental hazard

The categorisation of the environmental hazards of the assessed chemical according to domestic environmental hazard thresholds is presented below:

Persistence

Persistent (P). Based on a measured degradation study, the assessed chemical is categorised as Persistent.

Bioaccumulation

Not Bioaccumulative (Not B). Based on the low measured log k_{ow} value, the assessed chemical is categorised as Not Bioaccumulative

Toxicity

Not Toxic (Not T). Based on available ecotoxicity values above 1 mg/L the assessed chemical is categorised as Not Toxic.

Environmental risk characterisation

Although the assessed chemical is persistent, it does not meet all three PBT criteria. It is hence unlikely to have unpredictable long-term effects (EPHC 2009). An estimate of risk may therefore be determined using the risk quotient method.

Based on the PEC and PNEC values determined above, Risk Quotients (RQ = PEC ÷ PNEC) have been calculated for release of the assessed chemical to water:

Compartment	PEC	PNEC	RQ
River	78.07 µg/L	100 µg/L	0.781
Ocean	7.81 µg/L	100 µg/L	0.078

For the river and ocean compartments, an RQ less than 1 indicates that introduction of the assessed chemical, in line with the terms outlined in this assessment certificate, is not expected to pose a significant risk to the environment. As such, the risk from the assessed chemical can be managed, based on consideration of the environmental hazard characteristics and estimated releases.

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