Peroxide, bis(1,1-dimethylethyl): Human health tier II assessment

26 October 2018

CAS Number: 110-05-4

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted

and published separately, using information available at the time, and may be undertaken at different tiers.

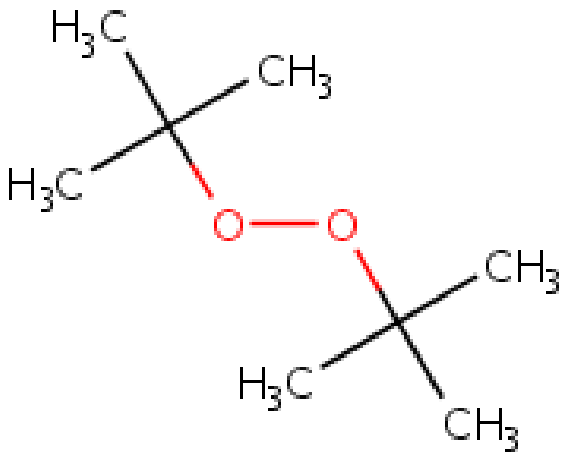
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	di-tert-butyl peroxide (DTBP) tert-butyl peroxide
Structural Formula	
Molecular Formula	C8H18O2
Molecular Weight (g/mol)	146.23
Appearance and Odour (where available)	Colourless liquid
SMILES	C(C)(C)(C)OOC(C)(C)C

Import, Manufacture and Use

Australian

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

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the United States (US) National Library of Medicine's Hazardous Substances Data Bank (HSDB); and the Organisation for Economic Co-operation and Development (OECD) Screening Information Data Set (SIDS) Initial Assessment Profile (SIAP) for di-tert-butyl peroxide (CAS No. 110-05-4).

In the USA, the production of the chemical in 2005 was 450–4500 tonnes. Worldwide production of the chemical was estimated to be approximately 10-50 kilotonnes in the year 2010 (OECD SIAP, 2012).

The chemical has reported domestic uses, including:

- in paints, lacquers and varnishes;
- in surface treatment products;
- in adhesives, binding agents; and
- as a corrosion inhibitor.

The chemical has reported commercial uses, including as a:

- solvent; and
- plastic hardener.

The chemical has reported site-limited uses, including:

- as a chemical intermediate;
- as a polymerisation initiator;
- in crosslinking/grafting polyethylene;
- crosslinking rubber;
- in the production of polyolefins;
- in acrylic resin manufacturing; and
- as a process regulator.

The chemical has reported non-industrial uses, including in:

- food contact products; and

- the manufacture of pharmaceuticals.

Restrictions

Australian

No known restrictions have been identified.

International

No known restrictions have been identified.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous, with the following hazard categories and hazard statements for human health in the Hazardous Chemical Information System (HCIS) (Safe Work Australia):

Germ cell mutagenicity – Category 2; H341 (Suspected of causing genetic defects)

Exposure Standards

Australian

No specific exposure standards are available.

International

An exposure limit of 100 mg/m^3 time weighted average (TWA) has been identified in Russia (Galleria Chemica).

Health Hazard Information

Bis(1,1-dimethylethyl) peroxide (CAS No. 110-05-4) is also referred to as di-tert-butyl peroxide (DTBP) and will be referred to as such, hereafter. The chemical is an organic peroxide and is widely used as a polymerisation initiator in a range of industrial processes.

Toxicokinetics

There are no conventional toxicokinetic studies available for DTBP; however, the physico-chemical properties of the compound, together with data from toxicological studies, provide some information on its expected absorption, distribution and excretion. The water solubility (170 mg/L), octanol-water partition coefficient (experimental log Kow 3.2) and vapour pressure (approximately 25.1 mmHg at 25°C) suggest some level of oral, dermal and inhalational absorption is expected. Results from a study conducted according to the OECD Test Guideline (TG) 422 (combined repeated dose toxicity study with a

reproduction/developmental toxicity screening test), wherein rats showed systemic effects (increased organ weights) following oral exposure, indicate that oral absorption likely occurred. Results observed in an OECD TG 413 (subchronic inhalation toxicity: 90-day study) study, wherein systemic effects (increased organ weights) were observed following repeated inhalational exposure, indicate that inhalation absorption occurs (OECD SIAP, 2012; REACH).

There are no data available on the metabolism of the compound.

Acute Toxicity

Oral

The chemical has low acute toxicity based on results from animal tests following oral exposure. The median lethal dose (LD50) in rats is >2000 mg/kg body weight (bw).

In a study conducted according to OECD TG 423 (acute oral toxicity – acute toxic class method), 6 female Wistar rats were orally administered DTBP via gavage at 2000 mg/kg bw. No mortalities occurred in this study. One animal showed slight shivering 3 hours after dosing. No other adverse effects were observed. The LD50 is >2000 mg/kg bw (OECD SIAP, 2012; REACH).

In a non-guideline study, DTBP was administered to 5 male ICR mice via oral gavage at 20 and 50 mL/kg (it is unclear whether the chemical was given to the animals as a formulation or whether this was the actual dose delivered). The initial study report indicated that lower doses may have been administered. One mortality occurred at 50 mL/kg. Sublethal effects of administration included agitation and slight limb paralysis. No LD50 was reported (REACH). Given the findings, an LD50 of >50 mL/kg can be assumed.

Dermal

The chemical has low acute toxicity based on results from animal tests following dermal exposure. The LD50 in rats is >2000 mg/kg bw.

In a study conducted according to OECD TG 402 (acute dermal toxicity), the clipped skin of 5 male and 5 female Wistar rats was topically treated with the test chemical at 2000 mg/kg bw. The material was left for 24 hours under semi occlusive conditions. No mortalities occurred during the study and no clinical signs were observed. The dermal LD50 was considered to be >2000 mg/kg bw (REACH).

A non-guideline study was conducted in which the test material was topically applied to the clipped skin of 5 male ICR mice at 10 mL/kg bw (it is unclear whether the chemical was given to the animals as a formulation or whether this was the actual dose delivered). Several experimental details have not been reported; however, no mortalities occurred in this study, no clinical signs were observed, and no local skin reactions were observed (REACH). No LD50 was reported. Given the findings, an LD50 of 10 mL/kg bw can be assumed.

Inhalation

The chemical has low acute toxicity based on results from animal tests following inhalation exposure. The median lethal concentration (LC50) in rats is >22 mg/L (equivalent to >22000 mg/m^3).

An acute inhalation toxicity study was conducted according to OECD TG 436 (acute inhalation toxicity – acute toxic class method). Wistar rats of both sexes (3/sex/dose) were exposed to the aerosolised chemical via nose only inhalation at 22 mg/L of air for a period of 4 hours. No mortalities occurred during this study. Shivering and tachypnoea were observed during exposure in all animals. Tachypnoea persisted up until 1 hour after the exposure period. Ruffled fur was observed in all animals from 1 hour after the exposure period, up until the end of day 2 of the observation period. There were no clinical signs from day 3 onwards. No gross pathological observations were identified at necropsy. On the basis of these findings, an LC50 of >22 mg/L was determined (REACH).

Corrosion / Irritation

Skin Irritation

The chemical produced no skin irritation in a study performed in accordance with OECD TG 404 (acute dermal irritation/corrosion).

In an OECD TG 404 study, the test chemical (0.5 mL) was applied to the shaved skin of 1 New Zealand White (NZW) rabbit for 3 minutes under semi-occlusive dressings. As no evidence of irritation was observed, the chemical was applied in the same manner to 3 additional animals, but for 4 hours. Animals were observed at 1, 24, 48 and 72 hours after removal of their dressings (or until adverse signs were reversed). After the 4 hour exposure, very slight erythema (grade 1) was observed in 1 animal at 1 day. Well defined erythema (grade 2) was observed in 1 animal between days 1 and 3. This was replaced by dryness of the skin from day 5 up to day 8. One animal showed no signs of adverse cutaneous reactions. Under these experimental conditions, the chemical was found to be a minimal skin irritant (REACH).

Eye Irritation

The chemical was reported to slightly irritate the eyes when tested according to OECD TG 405 (acute eye irritation/corrosion). The effects were reversible within 72 hours after application.

A study was conducted according to OECD TG 405 to assess the potential for DTBP to produce eye irritation. The chemical (0.1 mL) was instilled into 1 eye of each of 3 NZW rabbits. Animals were observed at 1, 24, 48, and 72 hours after instillation. There were no mortalities and no clinical signs were observed in any animal throughout the study. The chemical caused some slight, transient swelling and redness which was fully reversed within 72 hours. Mean scores across all time points for corneal opacity, iridial irritation, conjunctival irritation, and chemosis were 0, 0, 0.55 and 0, respectively. On the basis of these results, the chemical is not recommended for classification as an ocular irritant (REACH).

An ex vivo study was conducted according to OECD TG 437 (bovine corneal opacity and permeability test method for identifying 1) Chemicals inducing serious eye damage and 2) Chemicals not requiring classification for eye irritation or serious eye damage). Bovine corneas were harvested and incubated with DTBP (1 mL) for 10 minutes at 32 °C. Upon observation, no opacity was observed and no increase in corneal permeability was observed. Under these test conditions, the chemical is not considered to be irritating (REACH).

Sensitisation

Skin Sensitisation

The chemical was found not to induce dermal sensitisation when tested according to OECD TG 406 (skin sensitisation).

A Buehler test was conducted to assess the potential of DTBP according to OECD TG 406. Hartley guinea pigs (10/sex) were topically treated with the chemical at 100 %, once per week, for 3 consecutive weeks, for the induction phase of the assay. Following a 2 week rest period, animals were topically treated with the test chemical at 50 % (in mineral oil), for the challenge phase of the assay. Following induction, dermal scores of 1 (some with very slight oedema) were observed in 9/20 and 8/20 animals at 24 and 48 hours, respectively. Following inductions 2 and 3, dermal responses were comparable to those observed in induction 1 (Induction No. 2: dermal scores of 1 in 18/20 and 14/20 animals, at 24 and 48 hours, respectively; Induction No. 3: dermal scores of 1 in 14/20 at 24 and 48 hours). Following challenge at 50 %, dermal scores of 0 were observed in all animals. On the basis of these findings, the chemical was not considered to be a skin sensitiser (REACH).

Repeated Dose Toxicity

Oral

In an oral gavage study in rats, a no observed adverse effect level (NOAEL) of 300 mg/kg bw/day was reported.

The chemical was assessed for repeated dose oral toxicity as part of a study conducted according to OECD TG 422 (combined repeated dose toxicity study with a reproduction/developmental toxicity screen study). Wistar Han rats (10/dose/sex) were administered the chemical at 0, 100, 300 or 1000 mg/kg bw/day by oral gavage for 14 days prior to mating and during the mating period. Daily dosing of the females was continued throughout pregnancy, and up to day 4 of lactation. Dosing of males was continued up until the first dams had reached day 4 post-partum (42 days of administration). Treatment at 1000 mg/kg bw/day resulted in body weight effects in male animals. Decreased food consumption was also observed in these males. Animals demonstrated signs of discomfort in males and females in the 1000 mg/kg bw/day group (indicated by animals moving their heads through their bedding material). Some male and female animals in this group also showed ruffled fur. Increased liver and kidney weights were observed in animals in the 300 and 1000 mg/kg bw/day groups. Histopathological effects were observed in males and females in the 1000 mg/kg bw/day group, including in the livers (minimal centrilobular and diffuse hepatocellular hypertrophy with association of a consequent increase in diffuse follicular cell hypertrophy in thyroid glands) and kidneys (moderate diffuse tubular degeneration/regeneration with slight multifocal single cell necrosis and hyaline casts). Histopathological effects were observed in the forestomach (minimally increased incidence and severity of diffuse hyperkeratosis in males at 1000 mg/kg/day and females at 1000 mg/kg/day and 300 mg/kg/day). No adverse macroscopic findings were observed at necropsy. No reproductive effects were observed at any dose level. The investigators considered the effects observed at the 300 mg/kg bw/day dose level to be incidental and indistinguishable from observations made in control animals. Therefore, under the conditions of this study, the NOAEL is considered to be 300 mg/kg bw/day (OECD SIAP, 2012; REACH).

Dermal

No data are available

Inhalation

In a repeated dose inhalation toxicity study in male and female Wistar rats, the no observed adverse effect concentration (NOAEC) for the chemical was reported to be 993 mg/m^3.

A study was conducted to evaluate the potential for the chemical to produce repeated dose toxicity via inhalation according to OECD TG 413 (subchronic inhalation toxicity: 90-day study). Wistar rats of both sexes (10/sex/dose) were inhalationally exposed (nose-only) to the chemical at target concentrations of 0, 100, 300 or 1000 mg/m^3 (actual concentrations: 0, 101, 299 and 993 mg/m^3) 6 hours/day, 5 days/week for a total of 90 days. Animals in the highest dose group showed slight increases in relative kidney and liver weights. Animals in this group also showed slightly elevated cholesterol levels and slightly decreased creatinine levels. Although these changes were treatment-related, they were not considered to constitute adverse effects. On the basis of these findings, an NOAEC of 993 mg/m^3 was determined (REACH).

Genotoxicity

The chemical is classified as hazardous with hazard category ‘Germ cell mutagenicity – Category 2’ and hazard statement ‘Suspected of causing genetic defects’ (H341) in the Hazardous Chemical Information System (HCIS) (Safe Work Australia). Some in vivo studies support this classification.

In vitro

The chemical was assessed in 2 Ames bacterial reverse mutation assays according to OECD TG 471 (bacterial reverse mutation test) and in 2 non-guideline Ames assays, and was found to be non-genotoxic in (OECD SIAP, 2012; REACH):

- *Salmonella typhimurium* TA 1535, TA 1537, TA 98, TA 100 and TA 102 strains at concentrations up to 5000 µg/plate, both with and without S9 metabolic activation.
- *S. typhimurium* TA 1535, TA 1537, TA 97, TA 98 and TA 100 strains at concentrations up to 10000 µg/plate, both with and without S9 metabolic activation.
- *S. typhimurium* TA1535, TA1537, TA1538, TA98 and TA100 strains at concentrations up to 10000 µg/plate, both with and without S9 metabolic activation (investigators indicated that reporting issues regarding positive controls may have confounded this assay).
- *S. typhimurium* TA 98 and TA 100 (no concentration information reported) with S9 metabolic activation.

In a non-guideline SOS Chromotest genotoxicity assay, DTBP produced uncertain/borderline effects when tested in *Escherichia coli* strain PQ37. It was negative when tested in *E. coli* strains PM21 and GC4798. Few experimental details are available (REACH).

In a study conducted according to OECD TG 476 (in vitro mammalian cell gene mutation test), DTBP did not induce mutations in the mouse lymphoma thymidine kinase locus assay in the absence and presence of metabolic activation when tested up to 1480 µg/mL (REACH).

In vivo

A study was conducted according to OECD TG 489 (in vivo mammalian alkaline comet assay). Male SD rats (12/dose) were exposed to the chemical at 0, 7.5, 15 or 30 ppm via nose-only inhalational exposure for 6 hours/day for 3 consecutive days. The test substance gave a negative response (non-DNA damaging) in the comet assay in the nasal tissues of the male rats (REACH).

In a study conducted similarly to OECD TG 474 (mammalian erythrocyte micronucleus test), male Wistar rats (5/dose) were inhalationally exposed (nose-only) to the chemical at target concentrations of 0, 100, 300 or 1000 mg/m^3 (actual concentrations: 0, 101, 299 and 993 mg/m^3) 6 hours/day, 5 days/week 13 weeks. Microscopic examination of bone marrow smears harvested from the femurs of the animals showed evidence of chromosomal damage and/or damage to the mitotic apparatus of bone marrow erythrocytes. The chemical was not considered to be genotoxic under these test conditions (REACH).

In a study conducted according to OECD TG 474, Swiss Ico: OF1 mice of both sexes were administered the chemical at 0, 500, 1000 mg/kg bw (10 animals/dose) or 2000 mg/kg bw (16 animals) via 2 intraperitoneal injections delivered on 2 consecutive days. Animals were euthanised 24 hours after the second dose and bone marrow cells were harvested. The chemical produced a statistically significant increase in the number of micronucleated polychromatic erythrocytes (MPEs) at each dose tested. The chemical was positive for genotoxicity under these test conditions (REACH).

In a study conducted similarly to OECD TG 474, ICR mice of both sexes (5/sex/dose) were administered a single dose of DTBP via oral gavage at 0, 1250, 2500 or 5000 mg/kg bw. The micronucleus study was performed twice. In the first assay, there was a statistically significant increase in the frequency of MPEs in the 1250 mg/kg bw and 5000 mg/kg bw dose groups. In the second assay, there was a statistically significant increase in the frequency of MPEs in 2500 and 5000 mg/kg bw female mice, and in 5000 mg/kg bw in male mice; however, this result was considered equivocal as the number of MPE induced in both assays were within the historical solvent control range. The investigators determined that the chemical was weakly positive under the conditions of this study (REACH).

A chromosome aberration study was conducted according to OECD TG 483 (mammalian spermatogonial chromosome aberration test). Male ICR mice (5/dose) were administered DTBP at 0, 200, 1000 or 2000 mg/kg bw via 2 intraperitoneal injections delivered on 2 consecutive days. Following treatment, colchicine was given 4-5 hours prior to euthanasia to arrest cells in metaphase, and the testes were removed. No statistically significant increase in the percentage of spermatogonial cells with structural chromosome aberrations were identified in any dose group. Under these test conditions, the chemical was not considered to be genotoxic (REACH).

Carcinogenicity

No conventional carcinogenicity studies are available. However, the chemical has been shown to be negative in tumour promotion studies. In 2 studies in mice, the chemical was inactive in the promotion of papillomas or carcinomas in 'initiated skin'. No skin tumours developed following dermal application of DTBP for 81 weeks or for 80 weeks when applied prior to a promotor (REACH).

Reproductive and Developmental Toxicity

The chemical was assessed in a study conducted according to OECD TG 422 (combined repeated dose toxicity study with a reproduction/developmental toxicity screen study). HanRcc:Wistar rats of both sexes (10/sex/dose) were administered the test chemical by oral gavage at 0, 100, 300 or 1000 mg/kg bw/day, for 42 days (for males) and throughout premating, mating, gestation and lactation until day 4 post-partum (for females). Parental effects have previously been described (see **Repeated dose toxicity: Oral**). No histopathological abnormalities were observed in the reproductive organs of animals at any dose level. All females were mated (except 1 in the highest dose group). There were no treatment-related effects on pre-implantation loss, implantation rate, post-implantation loss, the number of living pups at first litter check, the post-natal loss, and the gestation period. Under the conditions of this study, the NOAEL for reproductive and developmental toxicity was 1000 mg/kg bw/day (OECD SIAP, 2012; REACH).

The chemical was assessed in a study conducted according to OECD TG 414 (prenatal developmental toxicity study). The chemical was administered to time-mated SD rats via oral gavage for 16 days (between days 3 and 19 of gestation) at 0, 100, 300 or 1000 mg/kg bw/day. There were no treatment-related adverse effects on any developmental parameters assessed. Under these test conditions the NOAEL for developmental toxicity was 1000 mg/kg bw/day (OECD SIAP, 2012; REACH).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (mutagenicity).

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, dermal, ocular, oral 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. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

NICNAS Recommendation

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory. No further assessment is required.

Regulatory Control

Public Health

Products containing the chemical should be labelled in accordance with state and territory legislation (SUSMP, 2018).

Work Health and Safety

The chemical is recommended for classification and labelling aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

From 1 January 2017, under the model Work Health and Safety Regulations, chemicals are no longer to be classified under the Approved Criteria for Classifying Hazardous Substances system.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Genotoxicity	Not Applicable	Suspected of causing genetic defects - Cat. 2 (H341)*

[a] Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

[b] Globally Harmonized System of Classification and Labelling of Chemicals (GHS) United Nations, 2009. Third Edition.

[*] Existing Hazard Classification. No change recommended to this classification

Advice for consumers

Products containing the chemical should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from dermal, ocular oral and inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures that could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- health monitoring for any worker who is at risk of exposure to the chemical, if valid techniques are available to monitor the effect on the worker's health;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

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.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

References

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