Cyclohexene, 4-ethenyl-: Human health tier II assessment

01 July 2016

CAS Number: 100-40-3

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.

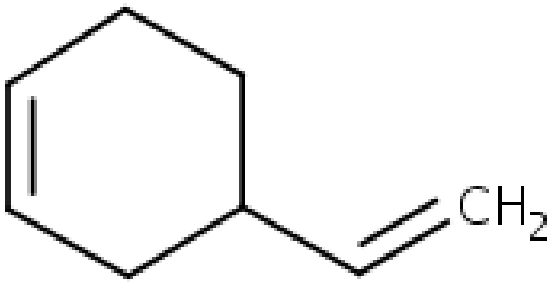
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	vinylcyclohexene cyclohexenylethylene 1-vinylcyclohex-3-ene 4-vinylcyclohex-1-ene 1,2,3,4-tetrahydrostyrene
Structural Formula	
Molecular Formula	C8H12
Molecular Weight (g/mol)	108.183
Appearance and Odour (where available)	Colourless liquid
SMILES	C(=C)C1CC=CCC1

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 US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB); and various international assessments including from the International Agency for Research on Cancer (IARC,1994), the National Toxicology Program (NTP, 1986).

The chemical has reported domestic uses in the SPIN database, including in:

- adhesives and binding agents;

- fillers; and
- paints; and
- lacquers and varnishes.

However, it should be noted that SPIN does not distinguish between direct use of the chemical, or use of the materials that are produced from chemical reactions involving the chemical. Based on the overall information, the chemical is more likely to be found in industrial processes involving manufacture of other chemicals than in finished domestic products.

The chemical has reported commercial use as a solvent.

The chemical mainly has reported site-limited use as an intermediate in the manufacture of other chemicals such as polyolefins, flame retardants and fragrances.

The chemical has reported non-industrial use as an intermediate in the manufacture of insecticides and flavouring compounds.

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 not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

The chemical has an exposure limit of 0.4 mg/m^3 (0.1 ppm) time weighted average (TWA) in different countries such as Canada, Denmark, Iceland, Indonesia, Malaysia, Singapore, Spain, Switzerland and the United States of America (Galleria Chemica).

The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value (TLV) of 0.1 ppm (0.4 mg/m^3) 8-hour TWA (IARC, 1994).

Health Hazard Information

The chemical, referred to as vinylcyclohexene (VCH) in this report, can lead to the formation of toxic metabolites, including 4-VCH diepoxide (VCD). This metabolite (CAS No. 106-87-6) was assessed separately under the IMAP framework (NICNAS). There is strong evidence that VCD is a key factor in the toxicity of the chemical (Smith et al., 1990b; TCEQ, 2011).

Toxicokinetics

The metabolism of the chemical has been extensively studied. The conversion of the chemical to epoxides and their subsequent destruction of oocytes are 'critical steps in VCH-induced ovarian tumours' (Smith et al., 1990b).

In rats and mice, the chemical is reported to be readily absorbed through the gastrointestinal (GI) tract and mostly excreted in the urine (Smith et al., 1990a). The chemical was more efficiently metabolised into mono- and di-epoxides (including VCD) in mice than in rats, leading to a higher accumulation of the chemical in rats (RAC, 2012a and 2012b).

In vitro studies showed that, in terms of metabolic pathway, rats rather than mice were a more comparable model for humans (Smith & Sipes, 1991; RAC, 2012a). The difference in epoxide formation between rats and mice is considered a plausible explanation for ovarian toxicity observed in mice only (Keller et al., 1997; Smith et al., 1990b; RAC, 2012a).

In a toxicokinetic study conducted in female B6C3F1 mice and Fischer 344 (F344) rats, the radiolabelled chemical was administered as a single oral dose of 400 mg/kg bw (^{14}C-vinylcyclohexene) or as a single intraperitoneal (i.p.) injection of 800 mg/kg bw. In both species, the chemical accumulated mostly in adipose tissue and was eliminated in urine and expired air (50–60 % and 30–40 %, respectively, based on the detected radioactivity). Twenty-four hours after the oral dosing, mice and rats excreted 97 and 88 %, respectively of the absorbed dose (RAC, 2012b; Smith et al., 1990a). Following i.p. dosing, the metabolite VCD was measured in the blood at peak levels of 41 nmol/mL in mice two hours post-injection and at less than 2.5 nmol/mL in rats at all times (Smith et al., 1990a).

Supporting these results, in vitro studies showed that the rate of epoxidation of the chemical to VCD was 6.5-fold greater in mouse liver microsomes than that in rat liver microsomes (Smith et al., 1990a).

Another in vitro study, conducted using microsomes from liver, lung and ovaries of female Crl:CD BR rats and female B6C3F1 mice, showed that mice were more efficient than rats in metabolising the chemical into epoxides, but less efficient than rats in hydrolysing the epoxides. Therefore, mice are more sensitive to the chemical bioactivation (Keller et al., 1997).

In an in vitro study conducted using human microsomes, the main metabolite formed from the chemical was VCD. The formation rate of this metabolite was measured as 0.71 nmol/mg microsomal protein/min, reportedly '13- and 2-fold lower than the rate of VCD formation by female mouse and rat hepatic microsomes, respectively'. In addition, there was some evidence of significant epoxide hydrolysis activity, as the use of an epoxide hydrolase inhibitor was required to detect levels of epoxides in the study. This study suggested that, based on epoxide metabolism, humans might be less susceptible than mice to VCH-induced ovarian toxicity (Smith & Sipes, 1991).

Acute Toxicity

Oral

Based on the available data, the chemical has low acute toxicity following oral exposure.

In an study compliant with the Organisation for Economic Cooperation and Development (OECD) Test Guideline (TG) 401, Wistar rats (n = 5/sex/dose) were orally administered single doses of the chemical at 5010, 6310 or 7970 mg/kg bw. The median lethal dose (LD50) was determined to be 6700 mg/kg bw. Observed sub-lethal effects included piloerection, sedation, ataxia, staggering, tremor, hypothermia, halting motion, blood in urine, diarrhoea and defence reaction when touched. Body weight gain was significantly inhibited and animals that died presented hyperaemia and swelling in the mucosa of stomach and small intestine, swelling and discolouration of kidneys and hyperaemia of peritoneum and mucosa of the bladder (REACH).

Values of LD50 of 2600 mg/kg bw in Carworth-Wistar rats (IARC, 1985; NTP, 1986) and 2563 mg/kg bw in rats (HSDB) were also reported.

Dermal

Based on the available data, the chemical has low acute toxicity following dermal exposure.

An LD50 of 16640 mg/kg bw in rabbits was reported for the chemical (HSDB; IARC, 1985).

Inhalation

Based on the available data, the chemical has low acute toxicity following inhalation exposure.

Median lethal concentrations (LC50) of 27000 mg/m^3 (6095 ppm) in rats and 47000 mg/m^3 (10610 ppm) in mice were reported (IARC, 1985; NTP, 1986). Exposure to the chemical vapour at 8000 ppm for four hours killed 4/6 rats (HSDB; REACH).

Observation in humans

Some Russian rubber workers who inhaled the chemical at mean concentrations of 271 to 542 ppm (with a peak concentration of 677 ppm) showed effects related to exposure that included keratitis and rhinitis (inflammation of the eye and mucous membrane of the nose), headaches, hypotonia (low

muscle tone), leukopaenia (decreased number of white blood cells), lymphocytosis (increased number of lymphocytes in the blood) and impairment of carbohydrate metabolism (HSDB).

Corrosion / Irritation

Skin Irritation

Based on the available data, the chemical is a skin irritant, warranting hazard classification.

In a study conducted according to the OECD TG 404, white rabbits (n = 3/sex/dose) were exposed to the undiluted chemical (0.5 mL on shaved skin), for four hours. The mean overall irritation score for time periods of 1, 24, 48 and 72 hours was 3.79 (out of 8). The mean erythema score for time periods of 24, 48 and 72 hours was 2.56 (out of 4). Effects were not reversible within 14 days. Animals showed induration (hardening) and dryness of treated skin on day 6 post-exposure, cracked skin and formation of eschar on days 8–10 post-exposure (REACH).

Eye Irritation

Based on the available data, the chemical is not an eye irritant.

In a study conducted according to the OECD TG 405, white rabbits (n = 3/sex/dose) were exposed to the undiluted chemical by instillation of 0.1 mL into the right eye. Mean scores for time periods of 24, 48 and 72 hours were 0 for corneal opacity and iris effects, 1.33 for conjunctival redness and 0.11 for chemosis. Effects were fully reversible within six days post-exposure (REACH).

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is not considered to cause severe effects following repeated oral exposure.

In a subchronic gavage study, F344/N rats (n = 10/sex/dose) were administered the chemical at 0, 50, 100, 200, 400 or 800 mg/kg bw/day in corn oil, five days per week, for 13 weeks. One male at 400 mg/kg bw/day and one female at 800 mg/kg bw/day died before the end of the treatment. In both sexes, a slight dose-related decrease in the final mean body weight was observed at doses ≥100 mg/kg bw/day (13 % and 6 % lower in male and female rats at the highest dose, respectively, compared with the controls). All treated males had kidney lesions (of minimal severity, except at the highest dose), consisting in hyaline droplet degeneration (accumulation of eosinophilic granules and globules in the cell cytoplasm) of the proximal tubule. At the highest dose, there was inflammation in the submucosa of the non-glandular portion of the stomach in one male and three females. No other effects were observed (NTP, 1986). The study did not report a no observed adverse effect level (NOAEL) in rats due to reported kidney lesions in all treated males.

In another subchronic gavage study, B6C3F1 mice (n = 10/sex/dose) received doses of the chemical at 0, 75, 150, 300, 600 or 1200 mg/kg bw/day in corn oil, five days per week, for 13 weeks. Nine males and five females in the highest dose group and two females at 300 mg/kg bw/day died before the end of the treatment. All other deaths that occurred (1/10 and 2/10 females at 150 and 600 mg/kg bw/day, respectively) were considered to be related to gavage administration. In both sexes, final mean body weights were slightly lower compared with controls, at 600 and 1200 mg/kg bw/day. At the highest dose, mild acute inflammation of the stomach was reported in three males and one female, and all females had a reduction in the number of primary follicles and mature Graafian follicles in the ovaries. No other effects were observed (NTP, 1986). No effects were reported at 150 mg/kg bw/day in mice, indicating this as the NOAEL in the study.

Dermal

No data are available.

Inhalation

Based on the available data, the chemical is not considered to cause severe effects following repeated inhalation exposure.

In a subchronic toxicity study, Sprague Dawley rats and B6C3F1 mice (n = 10/sex/dose) were exposed to the chemical by inhalation for six hours a days, five days per week, for 13 weeks. Rats were exposed to 0, 250, 1000 or 1500 ppm (0, 1, 4 and 6 mg/L respectively), and mice were exposed to 0, 50, 250 or 1000 ppm (0, 0.2, 1 and 4 mg/L respectively).

On days 11 and 12, all male mice and 5/10 female mice exposed to 1000 ppm died and three additional female mice at 1000 ppm died before the end of the treatment. Lethargy was observed in all mice at 1000 ppm and histopathological lesions included ovarian atrophy in females. No other effects were reported.

A no observed adverse effect concentration (NOAEC) of 250 ppm was determined for mice (Bevan et al., 1996).

In rats, mean body weight gain was significantly decreased in males and females at the highest dose compared with controls. At 1500 and 1000 ppm, male and female rats showed increased absolute and/or relative liver weights, and male rats also showed increased relative kidney weight. All treated male rats showed increased accumulation of hyaline droplets in the kidneys. Lethargy was observed in rats at 1500 ppm. No rat mortalities or other effects were observed.

A NOAEC of 1000 ppm was determined for rats (Bevan et al., 1996).

Genotoxicity

Based on the weight of evidence from available data, the chemical is not considered to be genotoxic.

In a bacterial reverse mutation assay (compliant with OECD TG 471), the chemical was found negative in *Salmonella typhimurium* strains TA98, TA100, TA1535 and TA1537 and *Escherichia coli* WP2 uvr A, at doses of 1.25–5000 µg/plate, with or without metabolic activation (IARC, 1994; REACH).

An in vitro chromosomal aberration assay and a sister chromatid exchange (SCE) assay, both using Chinese hamster ovary (CHO) cells, showed negative results (TCEQ, 2011). Another in vitro mutagenicity test in L5178Y mouse lymphoma cells showed equivocal results (TCEQ, 2011). Details of these assays are not available.

The chemical was investigated for induction of micronuclei in SD rats and B6C3F1 mice following inhalation exposure to the chemical for six hours per day, for two days (mice were exposed to the chemical at 0, 250, 500 or 1000 ppm and rats were exposed to the chemical at 0, 500, 1000 or 2000 ppm), or 13 weeks (mice were exposed to the chemical at 0, 50, 250 or 1000 ppm and rats were exposed to the chemical at 0, 250, 100 or 1500 ppm). In both experiments, no statistically significant increases in micronucleated polychromatic erythrocytes (PCEs) were observed, either in rats or mice (Bevan et al., 2001).

Carcinogenicity

Based on the available data, the chemical is considered to be carcinogenic, warranting hazard classification. A non-genotoxic mode of action (MOA) has been proposed for ovarian carcinogenicity (TCEQ, 2011) and is considered as relevant to humans (RAC, 2012a).

The IARC (1994) has classified the chemical as ‘Possibly carcinogenic to humans’ (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animal testing.

The two-year study in F344/N rats was considered 'inadequate' to identify carcinogenic effects because of high rat mortalities in the study (NTP, 1986). The two-year B6C3F1 mice study showed 'clear evidence of carcinogenicity' of the chemical (NTP, 1986).

In a carcinogenicity study, F344/N rats (n = 50/sex/dose) were administered the chemical by gavage at doses of 0, 200 or 400 mg/kg bw/day in corn oil, five days per week, for 103 weeks. Due to a marked decrease in survival of treated animals during the study (high dose: 5/50 males and 13/50 females; low dose: 13/50 males and 28/50 females), statistical analysis gave different interpretations. Neoplastic lesions that were considered as significant (NTP, 1986; IARC, 1994) included increased incidence of skin tumours (squamous cell papillomas and/or carcinomas), in male rats only (vehicle control: 0/50, low dose: 1/50, high dose: 4/50) and increased incidence of clitoral gland tumours (adenomas or squamous cell carcinomas) in females at low dose (5/50 compared with 1/50 in the control group). The other neoplastic lesions included: uncommon tumours in the urinary bladder of treated females only (transitional cell papilloma and carcinoma in one animal in each of the high- and low-dose groups); a higher incidence of anterior pituitary gland tumours in females of the low-dose group (24/48) compared with vehicle controls (19/50) and a positive, but not significant, trend on the incidence of preputial gland adenomas or carcinomas in males (vehicle control: 1/50, low dose: 1/50, high dose: 3/50). Decreased incidences of adrenal gland phaeochromocytomas (vehicle control: 17/50, low dose: 16/50, high dose: 8/50), interstitial cell tumours of the testes (vehicle control: 35/50, low dose: 30/50, high dose: 29/50) and mononuclear cell leukaemia (vehicle control: 14/50, low dose: 8/50, high dose: 1/50) were observed in males and associated with the 'shortened survival' of dosed animals in this study (NTP, 1986).

In another carcinogenicity study, B6C3F1 mice (n = 50/sex/dose) were administered the chemical by gavage at doses of 0, 200 or 400 mg/kg bw/day in corn oil, five days per week, for 103 weeks. Neoplastic lesions that were considered as significant (NTP, 1986; IARC, 1994) included increased incidences of uncommon ovarian tumours — mixed tumours (vehicle control: 0/49, low dose: 25/48, high dose: 11/47) and granulosa cell tumours and carcinomas (1/49, 10/48 and 13/47 at control, low and high dose, respectively); and adrenal capsular adenomas (0/50, 3/49 and 4/48, at control, low and high dose, respectively), in females only. Other neoplastic lesions were seen but not considered as statistically significant because of the marked mortality in treated male mice: alveolar/bronchiolar adenomas and carcinomas in the lungs of male rats at increased incidence, especially in the low-dose group (11/50) compared with the high-dose group (4/50) and vehicle controls (4/49); and lymphomas in males, at a higher incidence at 200 mg/kg bw/day (7/50) than in the vehicle controls (4/50) and the high-dose group (5/50) (NTP, 1986).

The MOA proposed for mice ovarian toxicity was: 'following exposure and uptake, VCH is metabolised, primarily in the liver, to VCH-1,2-epoxide or VCH-7,8-epoxide, which are further metabolised to VCH-diepoxide (VCD). VCH-diepoxide enters the blood and circulates through the body. Upon reaching the ovary, VCH-diepoxide selectively destroys the primordial and primary follicles through a mechanism involving programmed cell death or apoptosis. Repeated exposures to VCH ultimately result in premature ovarian failure, due to complete follicular loss. Since 17β-estradiol and inhibin are no longer produced from the primordial and primary follicles in the ovary, loss of the negative feedback inhibition of follicle stimulating hormone (FSH) release from the hypothalamus and pituitary occurs, leading to high plasma levels of FSH. Increased plasma levels of FSH results in the initiation and/or promotion of ovarian tumours' (TCEQ, 2011).

This non-genotoxic MOA was considered as 'relevant to humans', although quantitative information on the VCD detoxification potential in humans via epoxide hydrolase is lacking (RAC, 2012a). Consequently, the chemical was classified as a Carcinogen Category 2 (H351) in the Regulation (EC) No. 1272/2008 for Classification, Labelling and Packaging (CLP) (RAC, 2012a).

Reproductive and Developmental Toxicity

Based on the available limited data, the chemical is not considered to have reproductive toxicity, despite the ovarian toxicity displayed in mice studies considered in the carcinogenicity classification (see **Carcinogenicity** section).

In a reproductive toxicity study, Swiss mice (number not available) were administered oral doses of the chemical at 0, 100, 250 or 500 mg/kg bw/day in corn oil for 18 weeks. Animals were housed in same sex pairs for one week and then cohabited in breeding pairs for 14 weeks. At the highest dose, the chemical induced a significant reduction in the number of ovarian follicles in females of both generations (33 % to 55 % less than in control females) and spermatid count (17 % less than in control males) in F1 mice (offspring of the parental generation F0). However, it did not cause significant effects on reproductive abilities in either F0 or F1 generations, at a dose causing slight general toxicity. Slight general toxicity (decreased body weights) was reported at the highest dose in F0 and F1 animals. Teratogenic effects were not evaluated (Grizzle et al., 1994; TCEQ, 2011).

No data are available to evaluate the potential for developmental toxicity effects of the chemical.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity). The chemical can also cause skin irritation.

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

The level and route of worker exposure will vary depending on the method of application and work practices employed. Given the critical health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise oral and dermal exposure are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

The data available support a new hazard classification in the HSIS (Safe Work Australia) (refer to **Recommendation** section).

NICNAS Recommendation

Assessment of the chemical is considered to be sufficient, provided that the recommended classification is adopted, and labelling and all other requirements are met under workplace health and safety and poisons legislation as adopted by the relevant state or territory.

Regulatory Control

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Irritating to skin (Xi; R38)	Causes skin irritation - Cat. 2 (H315)
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)	Suspected of causing cancer - Cat. 2 (H351)

[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 industry

Control measures

Control measures to minimise the risk from oral and dermal 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.

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