Propane, 2-methoxy-2-methyl-: Human health tier II assessment

07 February 2014

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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.

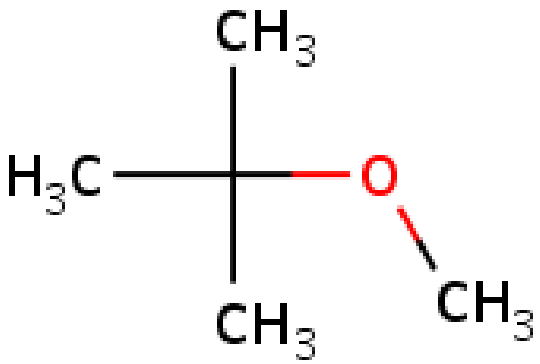
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	tert-butyl methyl ether methyl tert-butyl ether MTBE 2-methoxy-2-methylpropane methyl 1,1-dimethylethyl ether
Structural Formula	
Molecular Formula	C5H12O
Molecular Weight (g/mol)	88.15
Appearance and Odour (where available)	Colourless liquid with a terpene-like odour
SMILES	C(C)(C)(C)OC

Import, Manufacture and Use

Australian

The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was reported as less than 1 tonne.

No specific Australian use information has been identified.

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported commercial use including:

- as an octane enhancer in fuel;
- as a petrol additive in unleaded fuel to reduce unburnt hydrocarbon emissions; and
- as a process solvent and extraction agent in processes and products (not becoming part of articles).

The chemical has reported site-limited use including:

- in the manufacturing of bulk, large scale chemicals (including petroleum products)
- as an intermediate for manufacturing other substances; and
- as a laboratory solvent.

The following non-industrial uses have been identified internationally:

- clinical use to dissolve gallstones—cholelitholytic agent.

Restrictions

Australian

This chemical is listed in the *Fuel Quality Standards Act 2000* to be limited to 1 % by volume (max) in all grades for petrol and diesel fuel components.

According to the Department of the Enviroment, the chemical is 'effectively prohibited in petrol supplied in Western Australia, Queensland and South Australia through setting maximum content limits of 0.1% v/v, 0.5% v/v and 1% v/v, respectively' (Environment Australia, 2001).

International

In California and New York, the chemical was completely banned as a fuel additive from 31/12/2003 and 1/1/2004 respectively, and partially banned to no more than 0.5% (vol.) in gasoline sold or stored in Missouri or New Hampshire (EPA420-B-04-009,

2004). American (USA) state restrictions on the chemical are mainly due to the taste and odour problems arising from gasoline leaking into ground water.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous, with the following risk phrase for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

Xi; R38 (Irritating to skin)

Exposure Standards

Australian

The chemical has an exposure standard of 92 mg/m³ (25 ppm) time weighted average (TWA).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit of 110–183.5 mg/m³ (30-50 ppm) TWA in different countries such as Sweden, Denmark, Indonesia, Malta and the United Kingdom.

Health Hazard Information

Toxicokinetics

The chemical has a high vapour pressure. It is rapidly absorbed by oral and inhalation exposure in humans and rats. Absorption following dermal exposure is limited (Government of Canada, 1992; EU RAR, 2002). The chemical can be metabolised to formaldehyde and t-butanol. The metabolism of the chemical to formaldehyde is slow compared with the rate of formaldehyde oxidation, hence toxicity due to formaldehyde formulation is not expected (EU RAR, 2002).

Elimination of the metabolite t-butanol and the unchanged chemical can occur in expired air or they can be further metabolised, but at a lower rate, to 2-methyl-1,2-propanediol and a-hydroxyisobutyric acid, and excreted in urine. The elimination half-times for the different urinary metabolites varied between 2.9 and 5 hours in rats and between 7.8 and 17 hours in humans. Due to rapid elimination, the chemical or its metabolites do not significantly accumulate in the human body (EU RAR, 2002).

The accumulation of the chemical and metabolites following repeated exposure through inhalation is considered low. (Government of Canada, 1992).

Acute Toxicity

Oral

The chemical has low acute toxicity in animal tests following oral exposure.

The median lethal dose (LD50) in Sprague Dawley (SD) rats is greater than 2000 mg/kg bw. Observed sub-lethal effects included central nervous system depression, ataxia and laboured respiration (REACH).

Dermal

The chemical has low acute toxicity in animal tests following dermal exposure. The LD50 in rats is greater than 2000 mg/kg bw.

In one study SD rats (5/sex/dose) were exposed to 2000 mg/kg bw of the chemical over 24 hours with a 14-day observation period (LD50 >2000 mg/kg bw). No general clinical signs or behavioural alterations were seen; slight erythema was noted, but no macroscopic evidence was found at autopsy (REACH).

Inhalation

The chemical has low acute toxicity in animal tests following inhalation exposure (median lethal concentration (LC50) = 85 mg/L).

In an inhalation study, Charles River albino rats (5/sex/dose) were exposed to the chemical as vapour (whole body exposure) at 44, 65, 86, 99, 167 or 395 mg/L for four hours and observed for 14 days. Six animals died at or above a dose of 86 mg/L. Clinical signs observed were ataxia (loss of full control of bodily movements), tremors, lacrimation, unconsciousness and hyperactivity. Necropsy of animals revealed lung hyperaemia (excess of blood). An LC50 of 85 mg/L was reported (REACH).

Observation in humans

Data on human toxicity from the chemical was restricted to case reports of adverse effects following treatment for gallstones. Effects were mild and included breath odour, nausea, vomiting, drowsiness and mild inflammatory changes in the gallbladder following repeated treatment with low doses (Government of Canada, 1992).

In another study, 10 healthy males were exposed to 5, 25 or 50 ppm of the chemical in a chamber for two hours while exercising on a 50 Watt bicycle. There was no clear dose-effect relationship and no effects of the chemical vapour on short-term exposure to 50 ppm observed (REACH).

Corrosion / Irritation

Respiratory Irritation

Based on the information available, the chemical is not considered to be a respiratory irritant.

The chemical was slightly irritating to the respiratory system when tested in rats and mice (EU RAR, 2002). However, the OECD SIAR (2002) states that the chemical is 'a non-irritant to the respiratory system'.

Following four hours exposure of rats to >18,892 ppm of the commercial chemical (99.1 %) caused tachypnoea (abnormally rapid breathing) and nasal discharge, with respiration gradually slowing until the rats died (ATSDR, 1996; EU RAR, 2002).

When mice were exposed to 83, 277, 832, 2774 or 8321 ppm of the chemical for one hour, a mixed irritant response was observed with both sensory and pulmonary irritation at the highest dose. The RD50 (exposure concentration producing a 50% respiratory rate decrease) was determined to be 4604 ppm (EU RAR, 2002).

Skin Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to skin' (Xi; R38) in HSIS (Safe Work Australia). The available data support this classification.

The chemical, applied to unabraded rabbit skin, produced moderate erythema (mean score of 2.9 at 24, 48 and 72 hours) and moderate to severe oedema (mean score of 2.3 at 24, 48 and 72 hours) after one hour following the four-hour exposure period. The effects were reversible within eight days (REACH).

Eye Irritation

The chemical is a slight eye irritant.

The chemical was reported to slightly irritate the eyes of Chbb (SPF) strain rabbits when tested according to OECD Test Guideline (TG) 405. The average score for irritating the conjunctivae was reported as 1.3 with slight redness. The effects were reversible within six days (REACH).

Observation in humans

Two experimental studies where human subjects were exposed to the chemical vapours for one hour at 1.7 ppm or 1.39 ppm found no evidence of skin rash or eye irritation (EU RAR, 2002; ATSDR, 1996).

Sensitisation

Skin Sensitisation

Only limited data are available. Negative results are reported for skin sensitisation in animals up to a 1 % concentration of the chemical. In addition, the chemical does not include any functional groups associated with sensitisation, and human evidence has not been reported from the USA where the chemical has a history of use in gasoline.

The chemical 'was not sensitising in guinea pigs and there are no reports available concerning sensitisation in humans' (OECD, 2002).

Negative results were reported in a guinea pig maximisation test (equivalent to OECD TG 406) that used 1 % concentration of the chemical, and in a Landsteiner technique (non-guideline study) using 0.1 % concentration of the chemical. None of the animals reacted positively and the chemical did not elicit a response after the topical application challenge (REACH).

Repeated Dose Toxicity

Oral

Considering the no observed adverse effect levels (NOAEL) available from 13-week rat studies (209 mg/kg bw/day), and based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure.

In a repeated dose oral toxicity study (OECD Test Guideline (TG) 408) in Wister rats, the chemical was administered in drinking water to 110 animals at 0.5, 3.0, 7.5 or 15 mg/mL (males: 37, 209, 514, 972 mg/kg bw/d, females: 50, 272, 650, 1153 mg/kg bw/d) for 13 weeks. There were no mortalities, or changes to haematology or gross pathology, although there was an increase in water uptake and liver weight. The NOEAL was reported to be 209 mg/kg bw/day (REACH).

In another single study, the chemical was administered by oral gavage to SD rats (n=10/sex/dose) at 357, 714, 1071 or 1428 mg/kg bw/day for 14 days and at 100, 300, 900 or 1200 mg/kg bw/day for 90 days. The lowest observed adverse effect level (LOAEL) for the 14-day exposure was reported to be 357 mg/kg bw/day in females based on the decreased lung weight. For the 90-day exposure, although a statistically significant weight increase was observed in female kidneys at 300 mg/kg bw/day dose, the absence of findings in clinical chemistry or microscopy does not support the setting of the NOAEL at that level. A NOAEL of

300 mg/kg bw/day was determined for rats based on the statistically significant weight increase in both male and female livers at 900 mg/kg bw/day (REACH).

Dermal

No data are available.

Inhalation

Based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated inhalation exposure.

In a 90-day inhalation study (EPA Guideline 798.2450), Fischer 344 rats (n=25/sex/dose) were exposed (whole body) to the chemical at 797, 3920 or 8043 ppm for 13 weeks. There were no mortalities. There was mild toxicity at 3920 ppm with enlarged livers, kidneys and adrenal glands. At the reported highest concentration, male rats displayed mild lesions in the lymph nodes, spleen and kidney. The no observed adverse effect concentration (NOAEC) was reported to be 797 ppm (REACH).

In another inhalation toxicity study in SD rats (n=10/sex/dose), the chemical was administered (whole body) at 250, 500 or 1000 ppm for six hours a day, five days a week for 13 weeks. There was dose-related anaesthesia (insensitivity to pain) reported, but no treatment-related effects on haematology, clinical chemistry and urinalysis. There was a slight reduction in lung weights in females exposed to 1000 ppm, but no evidence of gross or histopathological effects (IUCLID 2000).

Observation in humans

There are reports of occupational exposure to the chemical as a component of motor fuels. Exposed workers had symptoms of headache, eye irritation, burning in the nose or throat, cough, nausea or vomiting, dizziness and disorientation. However, these reports were not conclusive given a number of confounding factors such as exposure to multiple volatile organic chemicals over the same period of time (REACH).

Genotoxicity

The chemical is not expected to be genotoxic.

The chemical produced negative results in several in vitro (Ames, mammalian cell gene mutation and mammalian chromosome aberration) and in vivo (mammalian bone marrow chromosomal aberration in rats, mammalian erythrocytes micronuclei and DNA repair from cultured primary hepatocytes in CD-1 mice) tests for gene mutation and clastogenicity (REACH; ATSDR, 1996).

One positive result has been reported in an Ames test (strain TA102) with S9 metabolic activation and in mouse lymphoma mutagenicity test with S9 activation. These results indicated that 'the formaldehyde formed in the presence of the exogenous S9 mix is probably responsible for the positive effect seen' (EU RAR, 2002). The metabolite of the chemical (formaldehyde) rapidly undergoes oxidation and would not have a significant genotoxic impact, but under the specific conditions of in vitro metabolic activation, a false positive result could occur (EU RAR, 2002). Based on the weight of evidence, the chemical is not considered to be genotoxic (OECD, 2002).

Carcinogenicity

The carcinogenic potential of the chemical is unclear and there is no epidemiological evidence to indicate that the chemical has been associated with cancer even with the widespread use in gasoline in the USA. Based on the data available, the chemical is considered to not require classification.

The International Agency for Research on Cancer (IARC) has classified the chemical as 'Not classifiable as to its carcinogenicity to humans' (Group 3), based on inadequate evidence for carcinogenicity in humans, and limited evidence for carcinogenicity in experimental animals (IARC, 1999). Based on indications of carcinogenicity in two animal species the European Union Risk Assessment Report (EU RAR) considers the chemical to be 'a borderline case between non-classification and Carc. Cat 3' (EU RAR, 2002). The National Toxicology Program considers the chemical should not be listed in the Report on Carcinogens (RoC) based on 'the uncertainty that the formation of the observed kidney tumors in rats and liver tumors in mice may be arising by mechanisms not relevant to humans, and the lack of any supporting human data' (NTP RoC, 1998).

There are several studies investigating the carcinogenic potential of the chemical and its metabolites, t-butanol and formaldehyde (EU RAR, 2002).

In a non-guideline study in rats, the chemical was administered by gavage at 250 or 1,000 mg/kg bw/day, four days a week for 104 weeks. A dose-related increased incidence of lymphomas and leukaemia was observed in female rats. There was also an increased incidence of testicular Leydig cell tumours (LCT) in males at the high dose (IARC, 1999). Analysing the LCT in rats, the EU RAR (2002) stated: 'it is unclear how the differences in physiology and anatomy between rat and human testis contribute to susceptibility to LCT tumours. Testicular cancer is a relatively uncommon cancer in humans. Most human testicular cancers originate either from germ or from Sertoli cells'. The tumours were observed in rats only at 'quite high doses' and the chemical 'lacks genotoxic properties'. It concludes that, 'no definitive conclusion can be drawn about the relevance of these tumors to man due to the lack of knowledge of the possible mode of action. However, considering all the available data, the relevance to man is probably not very significant'.

Inhalation studies in mice (CD-1) and rats (Fischer 344) exposed to 400, 3000 or 8000 ppm of the chemical in air for six hours a day, five days a week for 18 months (mice) or 24 months (rats) resulted in increased hepatocellular adenomas in female mice at the highest dose, but not in male mice. The liver tumours in mice were secondary to interaction with oestrogen (EU RAR, 2002). An increased incidence of renal tubular adenoma and carcinoma was observed in male rats at the two higher doses, but not in female rats (IARC, 1999). The chemical increased cell proliferation, which may have contributed to tumours in rats and mice at high doses (EU RAR, 2002).

Carcinogenicity of the metabolite t-butyl alcohol was investigated in B6CF1 mice by administering it at 5, 10 or 20 mg/mL in their drinking water for two years. The incidence of thyroid gland follicular cell hyperplasia was significantly increased (IARC, 1999). Tert-butyl alcohol also caused increased incidence of neoplastic lesions in the kidneys of rats (EU RAR, 2002).

The metabolite formaldehyde has been categorised as Carc. Cat 2: R49 (May cause cancer by inhalation) in the Hazardous Substance Information System (HSIS) (Safe Work Australia) due to animal studies where an increased incidence of nasal squamous cell carcinomas from inhalation studies in rats was observed (NICNAS, 2006). Based on: 1) rapid metabolism of formaldehyde to formic acid and 2)endogenously formed formaldehyde metabolised from the chemical in mouse liver cells does not lead to significant increase of DNA-crosslinking; the metabolite formaldehyde was not considered relevant in tumour formation observed with the chemical (EU RAR, 2001).

The relevance of renal tumours to humans is unlikely as these are considered likely to result from alpha-2μ-globulin mechanism, which is specific to male rats (EU RAR, 2002).

Epidemiological studies conducted during commercial use of the chemical for gasoline blending have not resulted in any association with human cancer (IARC, 1999).

Reproductive and Developmental Toxicity

The chemical is not considered to have reproductive or developmental toxicity.

In a one-generation reproductive toxicity study in rats exposed to the chemical vapour by inhalation (whole body) at doses 0, 250, 1000 or 2500 ppm prior to mating, during gestation and during lactation, the NOAEL for F1-animals was reported to be 250 ppm. The pup viability index was low at 1000 ppm (LOAEL) (EU RAR, 2002).

In a two-generation reproductive toxicity study in rats exposed to the chemical vapour by inhalation (whole body) at doses 0, 400, 3000 and 8000 ppm prior to mating, during gestation and during lactation until the day of sacrifice, the NOAEL for both the F1- and F2-animals was reported to be 400 ppm, due to lowered body weights. There were no changes in pregnancy rates, length of gestation, mating and fertility compared with the control. Increased relative liver weights were observed in F1-animals at the highest dose and in males (maybe F2 but not specifically stated) at 3000 ppm (EU RAR, 2002).

Developmental toxicity of the chemical through inhalation exposure has been tested in mice, rats and rabbits. No adverse effects on development were observed up to the highest doses tested in rats (2500 ppm) and rabbits (8000 ppm). Reduced

foetal body weight and skeletal abnormalities in pups were seen in the CD-1 mice at 4000 ppm, although, pup developmental effects were only observed secondary to maternal toxicity (EU RAR, 2002).

Other Health Effects

Neurotoxicity

The chemical is not considered to have neurotoxic potential.

In neurotoxicity studies (IARC, 1999) male and female Fischer 344 rats were exposed to 0, 800, 4000 or 8000 ppm of the chemical for six hours. The rats demonstrated sensory motor changes at 8000 ppm, such as ataxia, duck-walk, increased lacrimation, laboured respiration, decreased muscle tone, lowered body temperature and decreased hind-leg grip strength, indicative of central nervous system depression during the treatment/exposure. However, no changes were observed six hours after exposure.

In a longer-term study, animals were exposed to 0, 800, 4000 or 8000 ppm of the chemical for six hours a day for five days a week for 13 weeks. Any neurobehavioural changes were neither persistent nor cumulative. No histological changes were seen in the brain or peripheral nervous tissue.

Endocrine Disruption

Studies in female mice indicate the chemical to produce endocrine modulation suggestive of slight antioestrogen-like activity, but it did not show tumour-promoting activity (ATSDR, 1996; EU RAR, 2002).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- local effects—skin irritation.

Public Risk Characterisation

The general public may be exposed to the chemical through dermal and/or inhalation routes when refuelling petrol. However, based on the *Fuel Quality Standards Act 2000*, which limits the chemical to 1% by volume (maximum) in all grades for petrol and diesel fuel components, the concentration in these products is not considered to be sufficiently high to cause adverse toxicological effects. Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

Occupational exposure to the chemical may occur by inhalation and dermal absorption during its production, formulation, distribution, use and disposal, either as the pure chemical or blended into fuel. The health risks to workers from this chemical is considered to be low when control measures (such as use of protective clothing) are implemented. The concentration of the chemical in fuel products are low (maximum 1%) and therefore, no adverse effects are expected from the exposure.

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. Should changes in the Fuel Quality Standard allow more widespread use of the chemical in petrol and/or diesel, a Tier III assessment to clarify the carcinogenic potential of the chemical may be warranted.

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 hazards 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)

[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 dermal 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 which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- 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;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- 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 assist with meeting 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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