1H-1,2,4-Triazole: Human health tier II assessment

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CAS Number: 288-88-0

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.

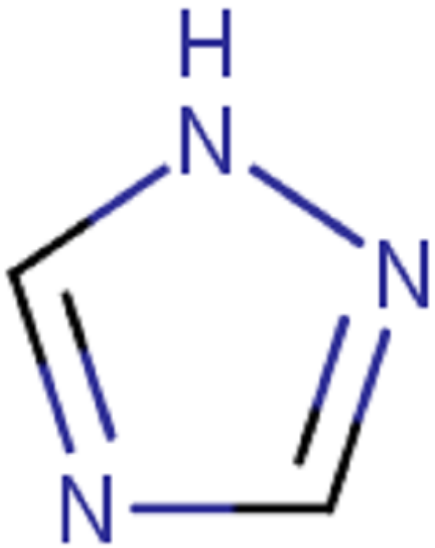
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	1,2,4-triazole s-triazole pyrrodiazole
Structural Formula	
Molecular Formula	C2H3N3
Molecular Weight (g/mol)	69.0667
Appearance and Odour (where available)	Colourless solid
SMILES	C1N=CNN=1

Import, Manufacture and Use

Australian

The chemical was reported under previous mandatory and/or voluntary calls for information, however, no specific use or volume data are available.

International

The following international uses have been identified through:

the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; the European Chemicals Agency (ECHA) substance information; Galleria Chemica; the Canadian Centre for Occupational Health and Safety, Registry of Toxic Effects of Chemical Substances (RTECS); the Substances and Preparations in Nordic countries (SPIN) database; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported commercial uses in washing and cleaning products. Whilst the reported use is generally considered to be evidence of potential domestic use, the source of the information did not identify any consumer use of the chemical (REACH, ECHAa).

The chemical has reported site limited uses, including:

- as a chemical intermediate in manufacturing; and
- as a photochemical.

The chemical has reported non-industrial uses in fertilisers and pharmaceuticals.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical is listed on the following (Galleria Chemica):

- European Union (EU) Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain;
- Chile list of substances which must not form part of the composition of cosmetic products;
- China list of banned substances for use in cosmetics; and
- Association of Southeast Asian Nations (ASEAN) Cosmetic Directive, Annex II Part 1 - List of substances which must not form part of the composition of cosmetic products.

Existing Work Health and Safety Controls

Hazard Classification

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

- Xn; R22 (acute toxicity)
- Xi; R36 (eye irritation)
- Repr. Cat. 3; R63 (developmental toxicity)

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standard is identified (Galleria Chemica).

An exposure limit of 5 mg/m^3 time weighted average (TWA) in Russia.

Health Hazard Information

Toxicokinetics

The chemical is a metabolite of triazole derived fungicides.

Studies investigating the toxicokinetics of the chemical in rats showed that the chemical is almost completely absorbed following oral dosing and is widely distributed throughout the tissues. The chemical is excreted mostly as the parent compound; in one study in rats, less than 2.8 % of the urinary radioactivity was attributable to other metabolites following administration of radiolabelled chemical as a single oral dose at 10 mg/kg body weight (bw). The chemical is excreted mainly via the urine. Up to 94 % of an oral or intravenous dose is excreted in the urine in the 48 hours after dosing, with 3-5 % excreted via the faeces in the same time period (JMPR, 2008).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in the HSIS (Safe Work Australia). The available data support this classification.

The median lethal dose (LD50) values in rats range from 1320-1750 mg/kg bw (RTECS, REACH). Observed signs of toxicity included sedation and breathing abnormalities (REACH).

Dermal

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

The dermal LD50 in rats was 3129 mg/kg bw (RTECS). Clinical signs of toxicity included ataxia, clear nasal discharge, iritis, scant and soft faeces, tremors, sedation and breathing abnormalities (REACH).

Inhalation

No data are available for the chemical.

Corrosion / Irritation

Skin Irritation

The chemical is reported to slightly irritate the skin in one animal study. The effects were not sufficient to warrant hazard classification.

In one skin irritation study using two rabbits, 0.5 g of the chemical (either applied undiluted or mixed in equal parts with saline) was applied to intact or abraded skin for 24 hours before being wiped clean. One intact site showed moderate erythema at 24 hours. Very slight erythema was observed at two abraded sites at 24 hours, and one site at 72 hours. The chemical caused slight skin irritation (JMPR, 2008). Another study in two rabbits showed no signs of skin irritation during a seven day observation period following application of 0.5 g of the chemical for 24 hours (JMPR, 2008).

Eye Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in the HSIS (Safe Work Australia). The available data support this classification.

In an eye irritation study in two rabbits, 50 mg of the chemical caused corneal opacity and slight iridial reddening and swelling which persisted for two days following ocular application in one rabbit. The conjunctivae were red and moderately swollen 24 hours after application in both rabbits. This effect persisted in one rabbit until day five, but the effects were absent on day seven. Mean scores for the 24, 48 and 72 hour observations were not available (REACH).

In an eye irritation study, two New Zealand White rabbits developed ocular effects following administration of 0.1 g of undiluted chemical. Corneal and conjunctival effects observed at the four hour observation had reversed by day seven of the study. Iridial effects were present until day 14. Investigators reported that the chemical was severely irritating under these test conditions (JMPR, 2008). Irritation scores were not reported.

Sensitisation

Skin Sensitisation

The chemical was negative for skin sensitisation in several animal studies, including a guinea pig maximisation test (GPMT) and a local lymph node assay (LLNA).

The chemical produced negative results in an LLNA conducted according to the Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 429 (skin sensitisation) in CBA/CaOlaHsd mice at concentrations of 0, 10, 25 and 34.6 % in dimethyl sulfoxide. The stimulation indices were reported as 1 (0 %), 1.2 (10 %), 1.9 (25 %) and 1.7 (34.6 %) (REACH).

The chemical was negative for skin sensitisation in two separate in vivo GPMT studies, both using methodology similar to OECD TG 406 (skin sensitisation). The chemical was tested on both occasions in Dunkin-Hartley guinea pigs. One study used an

intradermal induction concentration of 25 % (in water) and a topical induction concentration of 50 % in water following pre-treatment with 10 % sodium lauryl sulfate. A 50 % concentration in water vehicle was used for challenge. In the second test, the intradermal induction concentration was 10 % followed by topical induction at 75 % (in vaseline). The same concentration (75 %) was used for challenge (REACH).

Repeated Dose Toxicity

Oral

Based on the available animal data, repeated oral exposure to the chemical is not expected to cause severe adverse health effects, except at doses >200 mg/kg bw/day.

In a 90-day study, Wistar rats (n = 15/sex/dose) were administered the chemical in the diet at 0, 100, 500 or 2500 ppm (approximately equivalent to 0, 7.8/10.2, 37.9/54.2 or 212.3/266.7 mg/kg bw/day for males and females, respectively). Clinical signs of toxicity in the high dose group included reduced body weight gain and convulsions. High dose males showed changes to erythrocyte parameters consistent with microcytic hypochromic anaemia. Slight to moderate fat accumulation in liver parenchymal cells was observed in three high dose males. The no observed adverse effect level (NOAEL) was 500 ppm (approximately 37.9/54.2 mg/kg bw/day in males and females, respectively) (JMPR, 2008).

CD-1 mice (n = 20/sex/dose) were administered the chemical in the diet at 0, 500, 1000, 3000 or 6000 ppm (approximately equivalent to 0, 80/105, 161/215, 487/663 or 988/1346 mg/kg bw/day, for males and females respectively) for 90 days. Clinical signs of toxicity were observed in the two highest dose groups, and included tremors, yellow staining of the ventrum and rough coat. Decreased absolute brain weights were observed in males at concentrations of 3000 ppm and above and in high dose females. High dose animals of both sexes showed decreased numbers of Purkinje cells in the cerebellum, and in some cases axonal degeneration was also observed. Testes weights were decreased in males at doses of 1000 ppm and above, although this was significant at the highest dose only. Accompanying histopathology included increased numbers of apoptotic bodies, spermatid degeneration, spermatid depletion and spermatid asynchrony and tubular atrophy. Changes to the epididymides in this group (secondary to testicular effects) included increased germ cells and debris in the luminal duct or aspermia. The NOAEL was 1000 ppm for males (approximately 161 mg/kg bw/day) based on testicular effects and 3000 ppm for females (approximately 663 mg/kg bw/day) based on decreased brain weight and accompanying histopathology in the high dose group (JMPR, 2008).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

Based on the available in vitro and in vivo data, the chemical is not expected to be genotoxic.

The chemical was negative for genotoxicity in the following in vitro assays (REACH):

- a bacterial reverse mutation assay (similar to OECD TG 471) in *Salmonella typhimurium* strains TA 98, 100, 1535, 1537 and 1538 at concentrations up to 1500 µg/plate, with or without metabolic activation;
- a bacterial reverse mutation assay (similar to OECD TG 471) in *S. typhimurium* strains TA 98, 100, 1535, and 1537 at concentrations up to 5000 µg/plate (in the presence or absence of metabolic activation);

- a bacterial reverse mutation assay (US EPA guideline 870.5100) in *S. typhimurium* strains TA 98, 100, 1535, and 1537 at concentrations up to 7500 µg/plate (in the presence or absence of metabolic activation);
- a mammalian cell gene mutation test (OECD TG 476) in Chinese hamster ovary (CHO) cells at concentrations of 2.7, 5.4, 10.8, 21.6, 43.2, 86.4, 172.8, 345.5 and 691µg/plate (in the presence or absence of metabolic activation); and
- a mammalian chromosome aberration test (OECD TG 473) in rat lymphocytes at concentrations of 172.8, 345.5 and 691 µg/plate (in the presence or absence of metabolic activation).

An in vivo mammalian cell micronucleus assay in CD-1 mice was conducted similarly to OECD TG 474. Following a single oral gavage dose of the chemical at 1200 mg/kg bw, there was no observed increase in the frequency of micronucleated polychromatic erythrocytes, and no change in the ratio of polychromatic to normochromatic erythrocytes.

Carcinogenicity

No data are available for the chemical.

Reproductive and Developmental Toxicity

The chemical is classified as hazardous—Category 3 substance toxic to reproduction—with the risk phrase 'Possible risk of harm to the unborn child' (T; R63) in the HSIS (Safe Work Australia). The available data support this classification. Developmental effects were observed in rats in the absence of significant maternal toxicity in a two-generation study. The evidence of adverse effects on male fertility in rats and mice following repeated dosing was not considered to be sufficient to warrant hazard classification; significant effects occurred at only high doses and/or in the presence of other significant adverse health effects.

In a two-generation reproductive toxicity study, Wistar rats (n = 30/sex/dose) were administered the chemical daily at 0, 250, 500 or 3000 ppm in the diet (approximately 0, 15.4/17.5, 30.9/36.2 or 188.6/217.9 mg/kg bw/day, for parent generation males and females respectively). Fertility was reduced in the high dose dams (parent generation or F0), with only two dams delivering viable offspring. Adverse neuropathology (cerebellar degeneration/necrosis) was observed in high dose F0 animals. In the lower dose groups, clinical signs of toxicity were confined to reduced body weights in the first generation (F1) males receiving 250 ppm. The no observed adverse effect level (NOAEL) for reproductive effects was 250 ppm based on increases in sperm abnormalities in males from the F0 and F1 generations, and decreased corpora lutea in females from the F1 generation at 500 ppm. Effects on fertility at the high dose included reduced fertility index and reduced implantation sites. The reported NOAEL for developmental effects was 500 ppm (approximately 35.8 mg/kg bw/day). The NOAEL for parental toxicity was 500 ppm (approximately 30.9/36.2 mg/kg bw/day for males and females, respectively) based on adverse effects observed in the high dose group. A NOAEL for toxicity was not established for males in the F1 generation, due to effects on body weight at the lowest dose. For females in the F1 generation, the NOAEL was 500 pm (approximately 37.5 mg/kg bw/day) (REACH; JMPR, 2008).

Testicular effects were observed in CD-1 mice following 90 days of repeated dosing of the chemical in the diet. Effects included decreased testes weight, increased numbers of apoptotic bodies, spermatid degeneration, spermatid depletion and spermatid asynchrony and tubular atrophy. Testes weights were decreased in males at doses of 1000 ppm (approximately 161 mg/kg bw/day) and above, although this was significant at the highest dose of 6000 ppm (approximately 988 mg/kg bw/day) only. Changes to the epididymides (secondary to testicular effects) included increased germ cells and debris in the luminal duct or aspermia (JMPR, 2008). For additional details please refer to **Repeat Dose Toxicity**.

Developmental toxicity studies have been conducted in rats according to US EPA test guideline 83-3. In one study, female rats (n = 25/dose) were administered the chemical via gavage at doses of 0, 100 or 200 mg/kg bw/day. There was evidence of toxicity in high dose dams (significantly reduced weight gain). Foetal weight was reduced at both doses, and foetuses showed delayed bone development. Additional effects at the high dose included teratogenicity (hind leg malformation and cleft palate), and increased number of resorptions. The NOAEL for maternal toxicity was 100 mg/kg bw/day, and a no effect level was not established for foetal effects (REACH; JMPR, 2008).

In another developmental study, female rats (n = 25/dose) were administered the chemical via gavage at 0, 10, 30 or 100 mg/kg bw/day. Maternal toxicity was observed at 100 mg/kg bw/day (reduced weight gain). In the high dose group, foetal weight was

reduced and a high incidence of runts was observed. The NOAEL for maternal and foetal toxicity was 30 mg/kg bw/day (REACH).

In a developmental study, female rabbits (n = 25/dose) were administered the chemical via gavage at doses of 0, 5, 15, 30 or 45 mg/kg bw/day on gestation day (GD) six to 28. Five females were killed in the high dose group due to severe signs of toxicity, including body weight loss, decreased motor activity, ptosis, scant, soft or liquid faeces, clear perinasal substance, excessive salivation, hyperpnoea, lacrimation, head tilt and/or feeling cold to touch. One dam in the high dose group delivered early on day 29, after showing signs of intoxication. Gravid uterine weight was significantly reduced in the high dose group, and mean foetal weight was decreased in both sexes at this dose. Treatment-related urinary tract malformations occurred in four foetuses from two high dose litters. The NOAEL for maternal and foetal toxicity was 30 mg/kg bw/day (JMPR, 2008).

Other Health Effects

Neurotoxicity

Evidence of neurotoxicity (such as tremors, convulsions, reduced brain weights, decreased numbers of cerebellar Purkinje cells) has been observed in rats and mice following repeated oral dosing for 90 days (Refer to **Repeat Dose Toxicity**). Given the levels at which these effects were observed, hazard classification is not warranted for this effect.

In a 14-week combined toxicity and neurotoxicity study, Wistar rats (n = 20/sex/dose) were administered diets containing the chemical at 0, 250, 500, 3000 or 1000/4000 ppm (1000 ppm for first four weeks only, then 4000 ppm for the remainder of the study). The doses were approximately equivalent to 0, 16/19, 33/41, 183/234 or 210/275 mg/kg bw/day in males and females, respectively. The only clinical sign of toxicity was tremors observed in one high dose female. Body weight and body weight gain was significantly decreased in both sexes at doses of 183/234 mg/kg bw/day and above. Dose-related decreases in thyroid stimulating hormone (TSH) were observed in males; statistically significant at doses of 33 mg/kg bw/day and above. Absolute brain weight was significantly decreased in both sexes at 3000 ppm and males at 1000/4000 ppm. Brain weight was decreased in females at the high dose but this was not statistically significant. Increased incidence and severity of observed effects during the functional observation battery tests were observed in males and females at doses of 183/234 mg/kg bw/day and above. Effects included ungroomed appearance, red nasal and lacrimal stain, yellow stained urine, muscle fasciculations, tremors, gait incoordination, decreased activity in open field, decreased rearing, uncoordinated righting reflex and increased foot splay. Increased incidence and severity of nerve fibre degeneration was observed in peripheral nerves, Gasserian and dorsal root ganglia and spinal nerves of both sexes at doses of 183/234 mg/kg bw/day and above. Lesions were found in the anterior dorsal cerebellum of these treatment groups, consisting of degeneration/necrosis and nerve fibre degeneration (both sexes) and mineralisation and axonal degeneration (males). The NOAEL was determined to be 500 ppm (approximately 33/41 mg/kg bw/day in males/females) (JMPR 2008).

Endocrine Disruption

Based on the available data (JMPR, 2008), the chemical may have the potential to cause effects resulting from interaction with the thyroid hormone system.

In a 14-week dietary study in rats (refer to **Neurotoxicity** above), treatment with the chemical resulted in statistically significant decreases in TSH in males at doses of 500 ppm and above (equivalent to approximately 33 mg/kg bw/day). TSH levels were slightly decreased in females, although no obvious dose-response relationship was observed. In the absence of effects on the concentration of thyroxine (T4) and triiodothyronine (T3) in males, no findings of adverse thyroid histopathology, and noting the species sensitivity to thyroid effects, the decreased TSH was not considered to be toxicologically significant (JMPR, 2008).

The chemical was negative for effects on oestrogen biosynthesis (no reduction in aromatase activity and oestradiol and progesterone levels were unchanged) in vitro using immature rat granulosa cells (JMPR, 2008).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (developmental toxicity), systemic acute effects (acute toxicity from oral exposure) and local effects (eye irritation).

Public Risk Characterisation

It is not known whether the chemical is used in cosmetic/domestic products in Australia. Currently, there are no restrictions on using the chemical in cosmetics or domestic products in Australia. The use of the chemical in cosmetics is restricted overseas.

The reported use of the chemical overseas in cleaning/washing agents suggested the potential for domestic exposure; however, the source of information did not identify any specific consumer use of the chemical (REACH). Therefore, it was concluded that the chemical is used in cleaning/washing products for commercial use.

The chemical is listed on the EU Community Rolling Action Plan (CoRAP). The justification document lists the chemical as having wide dispersive use and notes that use of certain fungicides and use of the chemical in fertilisers may result in the presence of chemical residues in food, which may result in consumer exposure (ECHAb). However, as use of the chemical in consumer products could not be identified in Australia, and public exposure resulting from agricultural use is managed via other regulatory mechanisms, public risk is not considered to be unreasonable from industrial use and further risk mitigation via poisons scheduling is not recommended at this time.

Occupational Risk Characterisation

Based on the available data, the hazard classification in the Hazardous Substances Information System (HSIS) (Safe Work Australia) is considered appropriate.

During product formulation, dermal, ocular and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. 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. Given the critical health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise 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.

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).

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]
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Irritating to eyes (Xi; R36)*	Causes serious eye irritation - Cat. 2A (H319)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of harm to the unborn child (Xn; R63)*	Suspected of damaging the unborn child - Cat. 2 (H361d)

[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 oral and ocular 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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