Hydrazine, methyl-: Human health tier II assessment

27 November 2014

CAS Number: 60-34-4

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.

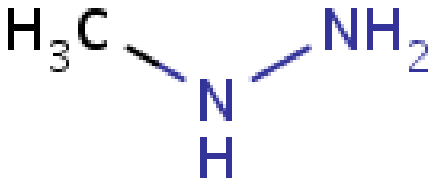
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	methylhydrazine hydrazomethane monomethylhydrazine (MMH) 1-methylhydrazine N-methylhydrazine
Structural Formula	
Molecular Formula	CH6N2
Molecular Weight (g/mol)	46.07
Appearance and Odour (where available)	Clear liquid with ammonia-like odor
SMILES	CNN

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 European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossiers; Galleria Chemica; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported site-limited uses:

- primarily as a component of jet fuels and altitude control fuel in missile propellants;
- in transfilling gas or liquids;
- as a chemical intermediate; and

- as a solvent.

The chemical has reported non-industrial use in pharmaceuticals.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical has no specific restrictions.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is listed in the Hazardous Substances Information System (HSIS) on the basis of an exposure standard, but there are no risk phrases assigned to the chemical (HSIS) (Safe Work Australia).

Exposure Standards

Australian

The chemical has a time weighted average (TWA) exposure standard of 0.019 mg/m^3 (0.01 ppm).

International

The chemical has the following exposure standards:

- 0.019–0.02 mg/m^3 (0.01 ppm) TWA in Canada, Denmark, Indonesia, Ireland, Malaysia, Mexico, Norway, Spain and the USA
- 0.35 mg/m^3 (0.2 ppm) TWA in countries including Egypt, France, Greece, Philippines, South Africa and Taiwan.

Health Hazard Information

The chemical is a volatile liquid and thus, inhalation is the major route of exposure. The chemical is mainly used as a component in rocket fuels and is a potent carcinogen, similar to unsymmetrical hydrazine (UDMH) (CAS No. 57-14-7) and hydrazine (CAS No. 302-01-2). Therefore, when toxicity data for the chemical are not available or insufficient to derive hazard properties, data on UDMH and/or hydrazine are used for the assessment of this chemical.

Toxicokinetics

The absorption and excretion patterns of the chemical are similar to those of UDMH (NICNAS). The radiolabelled chemical injected intraperitoneally (i.p.) to Sprague Dawley (SD) rats was excreted in the urine (22–41 %) and exhaled (23–34 %), with some retained in the tissues (25–55 %). In addition to CO_2, a greater amount of another gas, assumed to be methane, was also exhaled (Dost et al., 1964).

Groups of animals received i.p. injections of the radiolabelled chemical at doses of 22 mg/kg (20 mice), 15 mg/kg (20 rats) or 10 mg/kg (16 monkeys and 17 dogs), and within 24 hours, 25–40 % of the total dose was eliminated. Approximately 50 % of the total excretion was the chemical in its unchanged form. Mice, rats and monkeys excreted twice as much as dogs in the first two hours. The chemical accumulated in the liver, kidneys, bladder, pancreas and blood serum (Pinkerton et al., 1967).

In isolated hepatocytes and liver microsomes, the metabolism of the chemical was shown to produce free radicals via cytochromes P450. Cytocellular glutathione (GSH) was shown to scavenge the free radicals formed. Inhibition of the flavin adenine dinucleotide (FAD)-containing monoxygenase system did not alter the chemical's transformation. The study suggested that methyl radicals could be 'the alkylating species responsible for the toxic and/or

carcinogenic effect of methyl-hydrazines' (Albano et al., 1989, cited in HSDB). The related compound UDMH was similarly reported to produce reactive intermediates such as methyl radicals (NICNAS).

Acute Toxicity

Oral

The chemical has high acute oral toxicity in animals. Therefore, hazard classification is warranted.

Acute oral toxicity data on the chemical include the following median lethal doses (LD50): 32–33 mg/kg bw in rats, 29–33 mg/kg bw in mice and 22 mg/kg bw in hamsters (ChemIDPlus; HSDB; RTECS).

Dermal

The chemical has high acute dermal toxicity in animals and warrants hazard classification.

Both hydrazine and UDMH are classified for dermal toxicity and it is reported that the chemical is 'much more toxic than either hydrazine or UDMH by either (inhalation and skin) exposure route' (Keller, 1988).

Acute dermal toxicity data on the chemical include LD50 values of 183 mg/kg bw in rats, 95 mg/kg bw in rabbits, 239 mg/kg bw in hamsters and 48 mg/kg bw in guinea pigs (Kelly, 1988; ChemIDPlus; HSDB; RTECS).

Single applications of the chemical to dog skin (details not available) have been reported to induce opacity of the corneas, due to the chemical being translocated to the aqueous humor of the eyes (HSDB).

Inhalation

The chemical has high acute inhalation toxicity in animals and warrants hazard classification.

In a study (similar to the Organisation for Economic Co-operation and Development Test Guideline (OECD TG) 403) where SD rats were exposed (whole body) to the vapours of the chemical for up to four hours, the median lethal concentration (LC50) was determined to be 78 ppm (0.14 mg/L/4-hour) (Keller, 1988; REACH).

Acute toxicity data (study details not available) also include LC50 values of 34 ppm (0.06 mg/L/4-hour) in rats, 56 ppm (0.1 mg/L/4-hour) in mice, 143 ppm (0.27 mg/L/4-hour) in hamsters and 270 mg/m^3/4-hour (0.27 mg/L/4-hour) in guinea pigs (ChemIDPlus; RTECS).

Observation in humans

Three Apollo astronauts accidentally exposed to fumes containing a mix of the chemical (with dinitrogen tetroxide) during a descent from space for 8–10 minutes showed symptoms of tearing and burning of the eyes, skin burns, nausea, nasal irritation, retrosternal burning pain, chest tightness, airway obstruction, cough and mild pulmonary oedema. All effects cleared within five days. Although the toxic effects could be due to either the chemical or dinitrogen tetroxide, it was suggested that the toxicity of the chemical was probably similar to that of UDMH, which is known to induce pulmonary oedema and other toxic effects when inhaled (DeJournette, 1977).

Corrosion / Irritation

Respiratory Irritation

The chemical (liquid and vapour) was reported to be slightly irritating to the mucous membranes (MAK, 2012).

Skin Irritation

The limited available data suggest that the chemical is a slight skin irritant. Irritation scores are not available to consider classification of the chemical.

In a study on rabbits and guinea pigs, the chemical (dose not stated) applied to a clipped area of the back produced mild oedema, reversible within 24 hours (Rothberg & Cope, 1956, cited on TOXNET).

In a metabolism study, the chemical (dose not stated) applied to the shaved skin of dogs caused rapid discolouration persisting for six hours, and swelling of the treated area within 10 to 20 minutes that disappeared after six hours (Smith & Clark, 1969, cited on TOXNET).

The chemical is reported to produce 'severe skin and eye damage in animal models' (HSDB). However, no details were available to confirm these findings.

Eye Irritation

The limited available data suggest that the chemical is a slight eye irritant. Irritation scores are not available to consider classification of the chemical.

In a study on rabbits, 3 µL of the chemical applied to the left eye of the animals produced transient conjunctivitis and slight erythema (Rothberg & Cope, 1956, cited on TOXNET).

An in vitro study on rabbit cornea indicated that the application of the chemical caused almost immediate corneal swelling, probably due to a biochemical effect of the chemical on the endothelium (Takahashi & Dasher, 1969, cited on TOXNET).

Specific ocular effects include corneal damage as seen in dermal studies (see **Acute toxicity: Dermal**).

Observation in humans

The vapours of the chemical were reported to irritate the eyes, nose and throat. The chemical liquid was reported to be corrosive and 'burn skin and eyes' (HSBD). As details of these exposures were not available, this information was not considered suitable to classify the chemical.

Sensitisation

Skin Sensitisation

No data are available.

Repeated Dose Toxicity

Oral

No data are available.

Dermal

No data are available.

Inhalation

The chemical is reported to cause haemolytic anaemia in dogs, mice and, to a lesser extent, in rats, with no threshold for haemolysis. As these effects are considered relevant for humans, hazard classification is warranted.

In a six-month study, groups of Beagle dogs (n = 8), rhesus monkeys (n = 4), Wistar rats (n = 50) and ICR mice (n = 40) were intermittently exposed to the chemical at 0, 0.2, 1, 2, or 5 ppm, six hours/day for five days/week. An additional group (for all species) had continuous 0.2 ppm exposure for six months. Consistent dose-related effects were observed in all treated groups including decreased growth in rats from 1 ppm and in the continuous exposure group; decreased myeloid/erythroid elements ratio and increased erythropoietic activity in dogs from 1 ppm and in the continuous exposure group; increased methaemoglobin levels at 2 and 5 ppm in dogs; decreased red blood cells in dogs and monkeys from 0.2 ppm; reduced haematocrit and haemoglobin counts in dogs from the lowest dose. Deaths occurred in mice only (27 % and 15 % at 2 ppm and 5 ppm, respectively). The study concluded that the chemical produces 'dose-related haemolytic anaemia with Heinz body formation for which there is no threshold effect level' (MacEwen & Haun, 1971).

Histopathological effects from the above six-month inhalation study with various species (MacEwen & Haun, 1971) have been reported by Kroe (1971). The effects included hepatic haemosiderosis and cholestasis, and renal tubular haemosiderosis at 2 ppm and 5 ppm in dogs; hepatic centrilobular cholestasis, bile duct proliferation and centrilobular haemosiderosis, and splenic and renal tubular haemosiderosis at 5 ppm and to a lesser extent at 2 ppm in mice. Monkeys and rats did not exhibit any histopathological changes related to either intermittent or continuous exposure to the chemical. It is suggested that the observed differences are due to species susceptibility to the chemical-induced haemolysis (Kroe, 1971).

In a summarised study it was reported that rats, dogs and monkeys were exposed to vapours of the chemical at 0, 0.04, or 0.1 ppm, 24 hours/day, seven days/week for 90 days. Haematological effects were seen in rats and dogs at 0.1 ppm, along with discolouration of the liver at 0.1 ppm in dogs and elevated serum phosphorus in rats at 0.04 and 0.1 ppm (HSDB).

Human susceptibility is more similar to the dog than to the rat or to the monkey (MacEwen & Haun, 1971), suggesting that the six-month study results could be relevant for humans in terms of haematological effects. An in vitro study on human red blood cells concluded that the mechanism related to the haemolytic effects of the chemical was 'Heinz body formation with an increase in cell rigidity which probably causes an increased rate of cell sequestration and destruction in the spleen' (George, 1975).

Genotoxicity

Based on the results of the in vivo genotoxicity studies, the chemical is not considered to be genotoxic. However, some in vitro studies showed evidence of DNA damage.

There were several in vitro genotoxicity tests conducted, in which the chemical was found mostly negative, reporting:

- negative results in a preincubation assay in *Salmonella typhimurium* strains TA1535, TA1537, TA97, TA98 and TA100, up to 100 µg/plate, with or without metabolic activation (Mortelmans et al., 1986, cited in HSDB);
- negative results in a standard plate test with *S. typhimurium* strains G-46, TA1535, TA1537, TA1538, TA98 and TA100, up to 5 µL/plate, with or without metabolic activation (Brusick & Matheson, 1976);
- negative results in a bacterial gene mutation test with *S. typhimurium* strain TA 100 (Von Wright & Tikkanen, 1980, cited in HSDB);
- positive results in microbial assays on several strains of *Escherichia coli* (details not available), but less potent when compared with hydrazine (Von Wright & Tikkanen, 1980, cited in HSDB);
- positive findings in *S. typhimurium* strain TA102 without metabolic activation at concentrations of 0.5-2.0 µg/plate (DECOS, 2002);
- positive results for DNA lesions in the *E. coli* DNA repair-assay (DECOS, 2002);
- negative results in L5178Y mouse lymphoma cell assays at doses up to 0.1 µL/mL, with or without metabolic activation (Brusick & Matheson, 1976);
- negative results in an unscheduled DNA synthesis (UDS) test in human WI-38 cells, up to 1 µL/mL, with or without metabolic activation (Brusick & Matheson, 1976).
- negative findings for the induction of thymidine mutations (in contrast to UDMH and hydrazine) in a L5178Y mouse lymphoma cell assay (details not available) (Rogers & Back, 1981, cited in Keller, 1988);
- marginal activity in a host-mediated assay (details not available) in which UDMH produced negative results (Von Wright & Tikkanen, 1980, cited in HSDB).

DECOS (2002) stated that 'no increase in micronuclei was found in dogs exposed by inhalation' (details are not available).

In an in vivo dominant lethal test, the chemical injected (i.p.) at 0.26, 0.86 and 2.6 mg/kg bw to ICR mice, and at 0.215, 0.72 and 2.15 mg/kg bw to SD rats, did not induce any genetic damage to the germ cells leading to dead implants (Brusick & Matheson, 1976).

In a murine spermatogenesis test, B6C3F1 male mice were administered the chemical in one daily i.p. dose (time-dependent experiment) or five daily i.p. doses (dose-dependent experiment) of 3 mg/kg bw for seven weeks. Significantly decreased body weight, a clear increase in the percentage of abnormally shaped sperm and reduced sperm production were observed. The level of abnormalities reached its maximum three weeks after the exposure and returned to no response levels (NRL—average values of control results) within seven weeks. Similarly to UDMH (also tested in this study), the chemical was reported to be cytotoxic to spermatogenic cells in mice (Wyrobek & London, 1973).

Carcinogenicity

The available data indicate that the chemical is carcinogenic in animal studies and warrants hazard classification.

The International Agency for Research on Cancer (IARC) has not evaluated the chemical. As the chemical is a metabolite of gyromitrin (acetaldehyde formylmethylhydrazone), some relevant data were discussed when assessing gyromitrin (IARC,1983).

The Health Council of the Netherlands used these data to evaluate the chemical and concluded that the chemical should be considered as a carcinogen to humans based on reported studies (detailed below) where 'inhalation of N-methylhydrazine-induced benign and malignant tumours in mice and hamsters and oral (drinking water) exposure caused benign tumours in mice and malignant tumours in hamsters in one experiment' (DECOS, 2000).

The American Conference of Governmental Industrial Hygienists (ACGIH) has classified the chemical as a 'Confirmed animal carcinogen (A3)' with unknown relevance to humans (HSDB). The chemical is also on the ECHA 'Registry of submitted harmonised classification and labelling intentions' with category 1B for carcinogenicity (ECHA).

In a chronic study, Swiss mice (n = 50) were administered the chemical in drinking water at 100 mg/L. Lung adenomas developed in 12/50 females and 11/50 males. The chemical also reduced the survival rate of treated animals to 13/50 and 6/50 for females and males, respectively, compared with 96/110 and 67/110 for female and male untreated controls, respectively (DECOS, 2002).

Syrian golden hamsters (n = 50/sex) treated for life with the chemical in drinking water at 100 mg/L developed malignant histiocytomas (Kupffer cell sarcomas) in the liver (16/49 females, 27/50 males compared with none in control animals). Although the United States (US) Environment Protection Agency (EPA) concluded that 'no evidence of carcinogenicity was observed' in hamsters (DECOS, 2002), tumours in the caecum were observed in 9/49 and 7/50 treated females and males, respectively, compared with 1/99 and 1/97 in the control groups. The chemical also affected the survival rate (4/50 and 18/50 in females and males, respectively, aged 80 weeks compared with 31/100 and 42/100 in control females and males, respectively) (DECOS, 2002).

In a one-year inhalation study, rats, mice, hamsters and dogs were exposed to the chemical at 0, 0.38 and 3.8 mg/m^3 (0, 0.2 and 2.0 ppm), six hours a day for five days a week. Further exposure concentrations were used for a limited range of species: 0.04 mg/m^3 (0.02 ppm) for rats and mice only, and 9.6 mg/m^3 (5 ppm) for rats and hamsters only. Significant findings were reported for mice exposed to 3.8 mg/m^3, with higher incidences of lung tumours, nasal adenomas, nasal polyps, nasal osteomas, haemangiomas, and liver adenoma and carcinoma. In hamsters, the chemical induced nasal polyps and adrenal adenomas at 3.8 and 9.6 mg/m^3 and nasal cavity adenomas at 9.6 mg/m^3. Although no tumours were observed in rats and dogs, some toxic effects were reported (dose-related reductions in growth of rats and increased serum liver enzyme and methaemoglobin levels in dogs at the highest dose (DECOS, 2002).

Reproductive and Developmental Toxicity

Only limited data are available. The chemical has shown some reproductive or developmental toxicity effects in animal studies. However, most of these studies have not used a relevant route of exposure to humans or have limited information.

Daily oral doses of 20 mg/kg bw to female mice during gestation days (GD) 6–15 induced malformations in 36 % of the offspring. Similar effects (anencephaly, eye defects and complex facial bone deformities) were observed in rabbits following administration of a single oral dose of 200 mg/kg bw to pregnant rabbits on GD 14 (HSDB).

The chemical was reported to be cytotoxic to spermatogenic cells in B6C3F1 male mice, when administered at 3 mg/kg bw by i.p. injection (Wyrobek & London, 1973) (see **Genotoxicity**).

Two other studies in rats used i.p. or intravenous administration of the chemical. Pregnant Fischer 344 rats received the chemical (i.p. injection) at 2.5, 5, 10 or 20 mg/kg bw/day on GD 6–15 and showed a dose-dependent reduction in maternal body weight. There were some litter effects (e.g. ≥33 % resorptions) at 5 and 10 mg/kg bw/day, but these were considered non-significant by the study authors (Keller et al., 1984, cited in HSDB). The embryotoxic effects were observed only at maternally toxic doses (Keller, 1988).

Groups of pregnant SD rats that received continuous intravenous injections containing the chemical at 1.2–13.2 mg/kg bw/day on days six to 13 of pregnancy, or single intragastric bolus of the chemical at 1 or 5 mg/kg bw on day six of pregnancy showed a 'dose-dependent, statistically significant increase in the number of resorptions' along with a 'dramatic, dose-dependent decrease in the pregnancy rate as compared to controls'; no teratogenic effects were reported (Slanina et al., 1993, cited in HSDB).

Other Health Effects

Neurotoxicity

Some information was available on neurotoxic effects of the chemical. However, the available data were not conclusive.

In a behavioural study, Macaque monkeys (trained on a complex behavioural program) receiving the chemical exhibited emesis (vomiting) and impaired behaviour (HSDB).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic acute effects by oral, dermal and inhalation exposure; and
- systemic long-term effects (haemolytic anemia and carcinogenicity).

Only limited or no data are available on skin and eye irritation and skin sensitisation.

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 and inhalation exposure of workers to the chemical may occur, particularly where manual or open processes are used. These may include transfer and blending activities, quality control analysis, and cleaning and maintenance of equipment. Worker exposure to the chemical at lower concentrations may 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 may pose an unreasonable risk to workers unless adequate control measures to minimise dermal and inhalation exposure to the chemical 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 appropriate controls.

The data available support an amendment to the hazard classification in HSIS (refer to **Recommendation** section).

NICNAS Recommendation

Further risk management is required. Sufficient information is available to recommend that risks for workplace health and safety be managed through changes to classification and labelling.

Assessment of the chemical is considered to be sufficient provided that risk management recommendations are implemented and all 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 hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Toxic if swallowed (T; R25) Toxic in contact with skin (T; R24) Very toxic by inhalation (T+; R26)	Fatal if swallowed - Cat. 2 (H300) Fatal in contact with skin - Cat. 2 (H310) Fatal if inhaled - Cat. 1 (H330)
Repeat Dose Toxicity	Toxic: danger of serious damage to health by prolonged exposure through inhalation (T; R48/23)	Causes damage to organs through prolonged or repeated exposure through inhalation - Cat. 1 (H372)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)	May cause cancer - Cat. 1B (H350)

[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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