1H-Imidazole, 2-methyl-: Human health tier II assessment

21 April 2016

CAS Number: 693-98-1

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.

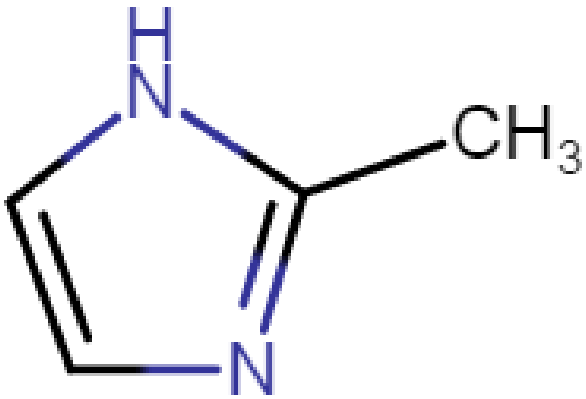
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	2-methylimidazole
Structural Formula	
Molecular Formula	C4H6N2
Molecular Weight (g/mol)	82.11
Appearance and Odour (where available)	White or colourless solid.
SMILES	C1(C)=NC=CN1

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 National Library of Medicine's Hazardous Substances Data Bank (HSDB); and international assessments (National Toxicology Program (NTP, 2004)).

The chemical has reported commercial use as a dyeing auxiliary for acrylic fibres and plastic foams.

The chemical has reported site-limited uses, including as:

- a component in polymers including epoxy resin pastes, acrylic rubber-fluororubber laminates, films, adhesives, textile finishes and epoxysilane coatings;
- an intermediate for manufacturing photographic and photothermographic chemicals, dyes, pigments, and rubber; and
- a polymerisation crosslinking accelerator and hardener for epoxy resin systems for semiconductor potting compounds and soldering masks.

The chemical has reported non-industrial uses for manufacturing pharmaceuticals; and agricultural products.

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

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

The chemical is readily absorbed orally and dermally. In Fischer 344 (F344) rats orally administered a single dose of the chemical at 25–100 mg/kg bw, the chemical was rapidly absorbed and widely distributed. Within 24 hours, 90 % of the chemical was excreted in the urine and 4 % in the faeces. The peak plasma concentrations were reached at 35–50 minutes. A gender difference in the urinary clearance rate was observed, with a faster clearance rate observed in males compared with females. Distribution to the tissues was proportional to dose and independent of the route of exposure (NTP, 2004b; IARC, 2012).

In rats, the chemical was not extensively metabolised. More than half of the parent compound was found unchanged in the urine and a mono-oxygenated urinary metabolite, 2-methylimidazolone was identified. Chemical modelling has shown that this metabolite can be formed via cytochrome P450 (CYP) dependent and independent pathways (NTP, 2004b; IARC, 2012).

The chemical is a toxic by-product in ammoniated hay for animals (see **Acute Toxicity - Oral**). It has been identified in the milk, plasma and urine of animals fed with ammoniated products (NTP, 2004b).

Acute Toxicity

Oral

The available information indicates that the chemical has moderate acute oral toxicity in animals, warranting hazard classification.

The oral median lethal dose (LD50) is 1400 mg/kg bw in mice and 1500 mg/kg bw in rats (HSDB; REACH).

The chemical is associated with acute convulsant activity. In mice, neurological effects including tremor, restlessness, sialorrhoea (excessive drooling), opisthotonus (spasm of the back muscles) and tonic extensor seizure at high doses, and balance disorders at lower doses were observed. A single oral convulsant dose (CD50) of 1300 mg/kg bw was determined for mice (NTP, 2004b).

Acute neurological effects were also reported in animals fed with commercially ammoniated food, for which the chemical is a toxic by-product. Facial twitching, body tremors followed by opisthotonus and convulsions were observed in ewes fed with ammoniated hay. Calves that were nursed by cows fed ammoniated hay showed signs of excitement to noise and touch, and would run in circles. Restlessness, bellowing, mouth-frothing and paralysis were observed in cattle (NTP, 2004b).

Dermal

Based on the available information, the chemical is considered to have low acute dermal toxicity.

The dermal LD50 is >2000 mg/kg bw in rats. The animals were reported to start displaying irregular breathing during exposure. A slight development of necrosis was observed at 24 hours following exposure, and progressed to a leathery necrosis within 14 days. No adverse effects were reported in rabbits (REACH).

Inhalation

No data are available.

Corrosion / Irritation

Corrosivity

Based on the available data, the chemical is considered corrosive to the skin and eyes, warranting hazard classification.

Skin effects

In a skin irritation study conducted according to the OECD Test Guideline (TG) 404, the chemical (0.5 g, 80 % concentration) was applied (occlusively) on the skin of Vienna white rabbits (n = 6) for four hours, and observed for 24 and 48 hours, and up to eight days. The mean erythema and oedema scores were 2.2 and 1.2, respectively. Necrosis was observed in three out of six animals after eight days, with an erythema score of 4 in two of these animals, and desquamation and spotted necrotic tissue in the other animal. The effects were fully reversible in the remaining three animals within the eight-day observation period. The chemical was interpreted as corrosive in this study (REACH).

In another skin irritation study (OECD TG 404), the chemical was applied occlusively on two Vienna white rabbits at a concentration of 80 % for 1, 5 and 15 minutes, and 20 hours. The animals were observed for up to eight days. No effects were reported between one to 15 minutes of exposure. At 20 hours of exposure, the mean erythema and oedema scores were observed to be 3, in both animals. Slight necrosis with marked erythema and oedema, which progressed to strong necrosis with marked desquamation eight days after treatment were observed. The chemical was interpreted as corrosive in this study (REACH).

Eye effects

In an eye irritation study (OECD TG 405), 50 µL of the chemical was administered into the conjunctival sac of two Vienna white rabbits. The eyes were not rinsed and observations were made at 10 minutes, one and three hours, and eight days following administration. Mean scores for the ocular lesions at one and 24 hours were 3 for corneal opacity, 3 for conjunctivae redness, and 4 for chemosis. At the one-hour time point, cauterisation of the mucous membrane was observed and was not reversible within the observation period. The chemical was determined as corrosive to the eye (REACH).

Sensitisation

Skin Sensitisation

The chemical is not a skin sensitiser.

A local lymph node assay (LLNA) (OECD TG 429) was conducted using the chemical at concentrations of 10, 25 and 50 % in ethanol/sterile water. The chemical was applied on the dorsal surface of the ears of female CBA mice (n = 5/sex/dose) on three consecutive days. No deaths or clinical signs of toxicity were observed during the study period. There were no prominent changes in lymph node weight or cell counts. A statistically significant increase in ear weights was observed but this was within the historical control range. The stimulation indices (SI) were reported to be 1.02, 1.07 and 1.14 for 10, 25 and 50 % concentrations, respectively, and no EC3 value could be determined (REACH).

Repeated Dose Toxicity

Oral

Based on the available data, the chemical is considered to cause severe effects in the thyroid at oral doses above 80 mg/kg bw/day. However hazard classification was not recommended as the thyroid effects are not considered relevant to humans.

The main adverse effects observed in rats and mice were in the thyroid, liver and spleen at doses ≥1250 ppm (80 mg/kg bw/day). A mode of action has yet to be established, but is potentially due to a neuroendocrine effect of the chemical on the thyroid, or an indirect effect on hepatic uridine diphosphate glucuronosyltransferase (UDPGT) enzyme levels (NTP, 2004a). Elevated UDPGT activity which eventually leads to thyroid hyperplasia and neoplasms in rodents is a mechanistic pathway extensively studied for microsomal enzyme inducers (e.g. phenobarbital), and is not relevant to humans (Capen, 1997). Thyroid lesions were less severe in mice compared with rats.

In a repeated dose toxicity study (OECD TG 408), F344 rats (n = 10/sex/dose) were administered the chemical in the diet at 0, 625, 1250, 2500, 5000 or 10000 ppm (equivalent to 0, 40, 80 160, 300 or 560 mg/kg bw/day), daily for 14 weeks. At doses ≥1250 ppm, thyroid hormone effects (decreased serum T3 and T4 concentrations, increased serum thyroid stimulating hormone (TSH) concentrations), and mild anaemia were observed. Significantly increased incidences of diffuse thyroid follicular cell hyperplasia were observed in males at ≥1250 ppm, and in females at ≥2500 ppm. Increased relative kidney and testicular weights were observed in males at ≥2500 ppm. At the highest dose (10000 ppm), significantly reduced body weight gains were observed in both sexes. Male rats displayed testicular degeneration and significantly decreased spermatid heads per testis and mean spermatid count, and female rats had a much elongated oestrous cycle at the highest dose. As no significant reproductive toxicity was observed (see **Reproductive & Developmental Toxicity**), effects on reproductive organs were postulated to be related to the reduced body weights. The no observed adverse effect level (NOAEL) was determined as 625 ppm (40 mg/kg bw/day) based on thyroid hyperplasia at the higher dose (NTP, 2004a; REACH).

In another 14-week repeated dose toxicity study (OECD TG 408), B6C3F1 mice (n = 10/sex/dose) were administered the chemical in the diet at the same concentrations as in the rat studies, equivalent to 100, 165, 360, 780, or 1740 mg/kg bw/day in males and 90, 190, 400, 800 or 1860 mg/kg bw/day in females. At the highest dose (10000 ppm), thyroid hormone effects (decreased concentrations of serum T4 (females only) and increased T3) were observed. TSH levels were unaffected. Mean body weight gains were significantly reduced in males at ≥2500 ppm and in females at ≥5000 ppm. Significantly increased absolute and relative liver and heart weights were observed in all treated males, and in females at ≥2500 ppm absolute and relative spleen weights were increased. Mild to moderate, macrocytic, hyperchromic and responsive anaemia (decreased red blood cell, haematocrit and haemoglobin numbers, and increased reticulocyte counts) was observed in all treated male groups, and in females at ≥1250 ppm. Other effects reported were significantly increased incidences of: diffuse thyroid follicular cell hyperplasia in both sexes (≥2500 ppm); haematopoietic cell proliferation in the spleen (≥1250 ppm); and renal tubule pigmentation (≥1250 ppm). The NOAEL was established as 625 ppm (90–100 mg/kg bw/day) based on splenic and renal lesions (NTP, 2004a; REACH).

In two short-term (15 day) dietary studies, rats and mice (n = 5/sex) were administered the chemical at concentrations ranging from 1200–10000 ppm for 15 days (115–770 mg/kg bw/day in rats, 220–2400 mg/kg bw/day in mice), and showed thyroid effects similar to those reported in 14-week studies from 3300 ppm in rats, and at all doses in mice (NTP, 2004a).

In two-year chronic oral studies (OECD TG 453) (see **Carcinogenicity**), F344 rats (n = 60/sex) were exposed to the chemical at 0, 300, 1000 or 3000 ppm (males) and 0, 1000, 2500 or 5000 ppm (females), and B6C3F1 mice at 0, 625, 1250, or 2500 ppm. Interim evaluations were conducted at six months for some animals. The effects observed were similar to the 90-day studies, with anaemia, reduced mean body weights for (mice at ≥1250 ppm and rats at ≥2500 ppm), and reduced feed consumption (high-dose female rats). At six months' exposure, effects observed for both species were primarily in the thyroid, liver and spleen. Thyroid effects included follicular cell hyperplasia (in most animals), increased thyroid weights (mice at ≥1250 ppm and rats at ≥2500 ppm), and follicular mineralisation of the thyroid (all rats). Liver effects observed primarily in mice included increased liver weights (high-dose female mice), hepatocellular karyomegaly, cytoplasmic alteration and Kupffer cell pigmentation (male mice ≥1250 ppm). Spleen effects included significantly increased incidences of haematopoietic cell proliferation (all treated male mice and in females at 2500 ppm), and granulomatous inflammation (high-dose female rats). Mice also showed bone marrow hyperplasia (≥1250 ppm) and renal proximal tubule pigmentation (2500 ppm), which were consistent with treatment-related anaemia. Hepatic UDPGT activity was significantly increased in all treated rats at six months, up to two-fold or more at the highest dose. Increases were less significant in mice compared with rats. Hepatic CYP450 levels decreased later in the study for rats, and after eight days in female mice (NTP, 2004b; IARC, 2012; REACH).

Dermal

No data are available.

Inhalation

No data are available.

Genotoxicity

The available in vitro and in vivo genotoxicity results are negative, except for one in vivo mouse study. In this mouse study, a dose-dependent increase in chromosomal damage was observed with repeated oral exposure to the chemical in the diet for 14 weeks.

The chemical gave negative results in bacterial reverse mutation assays with several strains of *Salmonella typhimurium* at concentrations up to 10000 µg/plate, with or without metabolic activation (NTP, 2004b).

The chemical gave negative results in two in vivo assays for induction of chromosomal damage (NTP, 2004b; REACH):

- did not induce micronucleated polychromatic erythrocytes (PCEs) in the bone marrow of rats or mice, administered the chemical intraperitoneally (i.p.) up to 400 mg/kg bw (rats) or 500 mg/kg bw (mice) (OECD TG 474); and
- negative results in a 14-day dominant lethal assay in mice administered (i.p.) the chemical at 90 and 180 mg/kg bw.

One other in vivo assay reported significantly increased frequency of micronucleated normochromatic erythrocytes (NCEs) in the peripheral blood of mice administered the chemical at doses up to 10000 ppm in the diet for 14 weeks. Results of this assay indicated increasing chromosomal damage with the duration of exposure, which is proposed as a ‘consequence of metabolic pathway overloading’ (NTP, 2004a). It is also postulated that elevated micronucleus frequencies can be attributed to the increased rate of haematopoiesis observed in mice in the 14-week study (see **Repeat dose Toxicity – Oral**). However, a comparison study with individual exposure groups have shown that elevated frequencies were not accompanied with changes in the percentage of PCEs and, thus, probably not solely a result of increased cell proliferation (NTP, 2004b).

Carcinogenicity

Based on the available data, the chemical is considered to have possible carcinogenic activity in animals, warranting hazard classification.

Studies in rats and mice have shown the presence of thyroid follicular cell neoplasms and liver neoplasms, at increasing incidences from six months until the end of exposure.

As tumours were observed in both the thyroid and liver, induction of hepatic microsomal enzymes and consequent thyroid hormone metabolism is suggested as a mechanistic link for the pathogenesis of thyroid follicular neoplasms and hepatocellular neoplasms in rodent carcinogenicity studies (NTP, 2004b). However, increased UDPGT activity observed in rats and mice was not accompanied with changes in total hepatic CYPs in mice (see **Repeat Dose Toxicity - Oral**) and, therefore, the microsomal enzyme mode of action is 'insufficient to account for the thyroid and liver neoplasms in these studies'. The possibility of an alternative (mutational) mechanism for the chemical may be involved in carcinogenesis (IARC, 2012). Available evidence from these studies is not sufficient to assign to the "phenobarbital" mode of action; however, liver tumours are still considered potentially relevant for humans.

In a two-year carcinogenicity study (OECD TG 453), F344 rats (n = 60/sex) were administered the chemical in the diet at 0, 300, 1000 or 3000 ppm (equivalent to 0, 13, 40, or 130 mg/kg bw/day) for males, and 0, 1000, 2500 or 5000 ppm (equivalent to 0, 50, 120 or 230 mg/kg bw/day) for females. After six months, increased incidences of combined thyroid gland follicular cell adenoma or carcinoma were observed and were particularly significant in high dose females. Increased incidences of combined hepatocellular adenoma or carcinoma were observed in mid and high dose animals. All of these incidences exceeded the historical control ranges. The authors reported that persistent TSH stimulation caused the thyroid gland cells to first become hyperplastic (as reported in the 14-week studies; see **Repeat dose Toxicity – Oral**), and eventually progress into tumours (NTP, 2004b; IARC, 2012), a mode of action not relevant to humans.

In a carcinogenicity study similar to the rat study as above, B6C3F1 mice were administered the chemical in the diet at 0, 625, 1250, or 2500 ppm (equivalent to 0, 75, 150 or 315 and 0, 80, 150 or 325 mg/kg bw/day for males and females, respectively). The observed effects were similar to those in rats, with increased incidences of thyroid neoplasms (adenoma or carcinoma) at the highest dose, and hepatocellular neoplasms that exceeded historical control ranges at ≥1250 ppm. These effects were observed at six months and significantly increased at two years (NTP, 2004b; IARC, 2012).

The International Agency for Research on Cancer (IARC) has classified the chemical as '*possibly carcinogenic to humans*' (Group 2B), based on sufficient evidence in experimental animals (IARC, 2012).

Reproductive and Developmental Toxicity

Based on the available data, the chemical does not show specific reproductive toxicity. However, developmental toxicity was observed in rats at ≥10 mg/kg bw/day, warranting hazard classification.

In a reproductive and developmental toxicity study (OECD TG 421), groups of Wistar rats (n = 10/sex/dose) were administered the chemical via oral gavage doses of 0, 50, 150 or 500 mg/kg bw/day during the premating period (two weeks), mating (maximum of two weeks), for a total of 28 days for males, and also during gestation and lactation (up to day 4) for females. For the parental animals (F0 generation), mortality was observed in two high-dose females during the lactation period. Some effects observed at mid to high doses, including transient salivation and discolouration of urine and faeces were determined to be due to irritation or metabolism, and considered non-adverse. Reproductive performance (fertility, gestation index, and implantation) was not affected. For the offspring, decreased numbers of liveborn pups and increased numbers of stillborn pups (statistically significant) were observed at 500 mg/kg bw/day. Histopathology revealed dissecting aneurysms of the aorta at all doses with increasing severity, and aorta and heart dilation at 500 mg/kg bw/day. Based on this study, a parental NOAEL of 150 mg/kg bw/day was determined based on mortality at the high dose. No NOAEL could be determined for developmental effects (REACH).

Subsequently, a developmental toxicity study (OECD TG 414) was conducted at lower doses. Groups of pregnant Wistar rats (n = 25/sex/dose) were administered (gavage) the chemical at 0, 2, 10 or 50 mg/kg bw/day, from gestation day (GD) 6 to postnatal day (PND) 3. Maternal toxicity (clinical signs, food consumption and reproductive and delivery parameters) was not observed up to the highest dose. In the offspring, dissecting aneurysms (focal bulging of arterial wall) and haemorrhages (characterised by focal accumulation of erythrocytes) were observed at ≥10 mg/kg bw/day. Based on these observations, the NOAEL values for maternal and developmental toxicity were determined to be 50 mg/kg bw/day and 2 mg/kg bw/day, respectively (REACH).

Other Health Effects

Endocrine Disruption

The chemical is listed on the US EPA's Universe of Chemicals list for potential endocrine disruptor screening and testing (US EPA, 2012). The data reported above indicate that the chemical has effects on the thyroid hormone system.

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic long-term effects (carcinogenicity and developmental toxicity);
- systemic acute effects (acute toxicity from oral exposure); and
- local effects (corrosivity).

The chemical may also cause harmful effects following repeated oral exposure.

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Although the public could come into contact with articles/coated surfaces containing the chemical, it is expected that the chemical will be bound within the article/coated surface and hence will not be bioavailable. Therefore, the chemical is not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

Given the critical systemic long-term, systemic acute and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise oral, dermal, ocular and inhalation 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 hazard classification of the chemical (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]
Acute Toxicity	Harmful if swallowed (Xn; R22)	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Causes burns (C; R34)	Causes severe skin burns and eye damage - Cat. 1B (H314)
Carcinogenicity	Carc. Cat 3 - Limited evidence of a carcinogenic effect (Xn; R40)	Suspected of causing cancer - Cat. 2 (H351)
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 industry

Control measures

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

- using closed systems or isolating operations;
- 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;
- 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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