Nitric acid: Human health tier II assessment

01 September 2015

CAS Number: 7697-37-2

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.

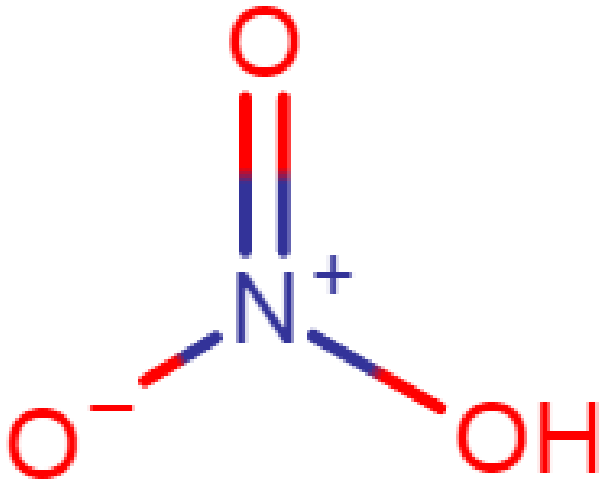
For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	hydrogen nitrate nitryl hydroxide aqua fortis azotic acid
Structural Formula	

	
Molecular Formula	HNO3
Molecular Weight (g/mol)	63.013
Appearance and Odour (where available)	Colourless crystals, or liquid with fumes in moist air above the melting point of around -41.6 deg C
SMILES	N(=O)(=O)O

Import, Manufacture and Use

Australian

The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 100000–999999 tonnes.

The chemical has reported commercial uses, including:

- in manufacturing electronic and optical products (synthetic fibres, electrical circuit boards, and semiconductors)
- as an oxidising agent;
- as a pH-regulating agent;
- for metal cleaning and etching;
- in the printing industry for photo-engraving;
- for manufacturing jewellery; and
- as an electroplating agent.

The chemical has reported site-limited uses, including:

- for manufacturing other chemicals (fertilisers and explosives); and
- for wet chemical etching.

The chemical has reported non-industrial uses for manufacturing pharmaceuticals

The National Pollutant Inventory (NPI) holds data for all sources or release of the chemical in Australia.

International

The following international uses have been identified through: the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossiers; the Organisation for Economic Co-operation and Development (OECD) Screening information data set International Assessment Report (SIAR); Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the US Household Products Database; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported domestic uses (SPIN):

- in adhesives and binding agents;
- in cleaning/washing agents (e.g. dishwashing detergent); and
- in paints, lacquers and varnishes.

It should be noted that SPIN does not distinguish between direct use of the chemical, or use of the materials that are produced from chemical reactions involving the chemical.

The United Kingdom Health Protection Agency (UK HPA) states that 'Nitric acid has a range of widespread uses in industry, but is not commonly found in household products.' (UK HPA, 2007)

The chemical has reported commercial uses, including:

- for metal cleaning and etching;
- in anti-freeze agents;
- in flux agents for casting or joining materials;
- as an oxidising agent;
- as a photochemical (photo-engraving);
- as a pH−regulation agent;
- as a corrosion inhibitor;
- for surface treatment (woodworking to artificially age pine and maple);
- as an electroplating agent;
- in process regulators; and
- in welding and soldering agents.

The chemical has reported site-limited uses, including:

- in ore flotation;
- for manufacturing organic and inorganic nitrates and nitro compounds for fertilisers, dye intermediates and explosives;
- for synthesising other chemicals (urethanes and rubber chemicals); and
- for reprocessing spent nuclear fuel.

The chemical has reported non-industrial uses, including in:

- non-agricultural pesticides and preservatives; and
- pharmaceuticals.

Restrictions

Australian

This chemical is listed in Schedules 5 and 6 of the *Poisons Standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP, 2015).

Schedule 6:

'NITRIC ACID (excluding its salts and derivatives) **except**:

(a) when included in Schedule 5; or

(b) in preparations containing 0.5 per cent or less of nitric acid (HNO_3).'

Schedule 5:

'NITRIC ACID (excluding its salts and derivatives) in preparations containing 10 per cent or less of nitric acid (HNO_3) **except** in preparations containing 0.5 per cent or less of nitric acid.'

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2015).

Schedule 5 chemicals are described as 'Substances with a low potential for causing harm, the extent of which can be reduced through the use of appropriate packaging with simple warnings and safety directions on the label.' Schedule 5 chemicals are labelled with 'Caution' (SUSMP, 2015).

International

No known restrictions have been identified.

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):

- C; R35 (corrosive)

Exposure Standards

Australian

The chemical has an exposure standard of 5.2 mg/m^3 (2 ppm) time weighted average (TWA) and 10 mg/m^3 (4 ppm) short-term exposure limit (STEL).

International

The following exposure standards are identified (Galleria Chemica).

TWA:

- 2–5.2 mg/m^3 (0.78–2 ppm) in Canada, Denmark, Germany, Japan, Latvia, Norway, Singapore, Switzerland, Taiwan, and the United States of America (USA); and
- 1.4 mg/m^3 in Poland.

STEL:

- 10–13 mg/m^3 (4–5 ppm) in Canada, Egypt, Greece, India, Mexico, South Africa, Sweden, the USA;
- 5 mg/m^3 (2 ppm) in Switzerland;
- 2.6 mg/m^3 (1 ppm) in Estonia, France, Hungary, Iceland, Japan, Malta, Poland, Spain and the United Kingdom; and
- 1.3 mg/m^3 in the Netherlands.

Health Hazard Information

Nitric acid is a highly corrosive, strongly oxidising aqueous solution which forms white fumes in moist air. Commercial nitric acid solutions are generally available in concentrations of 56–70 % (NAC/AEGL, 2008). Fuming nitric acid is a solution containing more than 86 % nitric acid and is characterised depending on the amount of dissolved nitrogen oxides:

- white fuming nitric acid (WFNA) is nearly pure anhydrous nitric acid (~97.5 %) containing 0.1–0.4 % nitrogen dioxide or dinitrogen tetroxide; and
- red fuming nitric acid (RFNA) is 82–85 % nitric acid containing 8–17 % nitrogen dioxide or dinitrogen tetroxide.

Nitric acid readily decomposes when heated or in the presence of light to form suffocating poisonous nitrogen oxide fumes and RFNA. The appearance of RFNA is as a yellow to brownish-red liquid (due to nitrogen dioxide formation as all others are colourless). Nitric acid also reacts violently with reducing agents (most metals and organic material) to release nitrogen oxide fumes (see **Acute Toxicity–Observations in humans**). Inhalation exposure to high concentrations of fumes from nitric acid or those produced during its use may cause toxic effects including respiratory irritation and pulmonary oedema (NAC/AEGL, 2008; OECD, 2008).

Due to the corrosive nature of the chemical, data are not available on specific toxicity endpoints such as skin sensitisation, repeat dose oral and dermal toxicity, genotoxicity, carcinogenicity and reproductive and developmental toxicity. Both of the constituent ions of nitric acid are naturally part of human physiological processes and are considered to have low systemic toxicity (NICNAS, 2014).

Toxicokinetics

Fumes from the chemical react immediately with the respiratory mucous membranes following inhalation exposure. As the chemical has a low molecular weight and is very water soluble, complete absorption (100 %) of the nitrate ion is expected following oral, inhalation and dermal exposure (HSDB; REACH).

The chemical dissociates into the nitrate anion and hydrogen ion in water. Distribution throughout the body is expected and the ions will diffuse through aqueous channels and pores. The nitrate anion enters the electrolyte pool in the body and, as shown in repeat dose animal studies, is not expected to play a significant toxicological role. Nitrate can also be reduced to nitrite by the bacteria of the intestines and by mammalian nitrate reductase activity. The hydrogen ion is largely responsible for irritation effects causing severe pH change locally (OECD, 2008; REACH), although, for concentrated nitric acid, the oxidising properties involving both pH and the nitrate ion lead to the severely corrosive properties.

Nitrate ion is not considered to be bioaccumulative and is rapidly excreted via urine (REACH).

Acute Toxicity

Oral

No data are available as the chemical is a corrosive compound.

Dermal

No data are available as the chemical is a corrosive compound.

Inhalation

Nitric acid atmospheres produced for inhalation exposure studies (animals and humans) result in a variety of physical states (gas, fume or vapour) (NAC/AEGL, 2008). The LC50s were obtained from exposure to fumes from the chemical (RFNA and WFNA) which consist of a mixture of nitrogen oxides, mainly nitrogen dioxide (CAS No. 10102-44-0; classified for acute inhalation toxicity (T+; R26) in the HSIS). The available data indicate high toxicity of the fumes, consistent with this classification.

In rats, the median lethal vapour concentrations (LC50) for red fuming nitric acid were reported to be $\leq$ 8 ppm (~21 mg/m^3) (four hour exposure, measured as NO_2) and 310 ppm (~799 mg/m^3) (30 minute exposure). The LC50 for white fuming nitric acid was 334 ppm (~861 mg/m^3) (30 minute exposure) (OECD, 2008; REACH).

Respiratory inflammation was observed particulary in the upper respiratory tract (rhinitis, tracheitis and pneumonitis) but subsided several weeks following cessation of exposure. However, lungs examined had 'localized areas of emphysema' (OECD, 2008). Other signs of toxicity included ocular or oral discharges, and burns on the skin. Neurological signs (lethargy, hunched posture), hair loss and partially closed eyes were observed at higher concentrations. Observed mortalities were due to pulmonary oedema (OECD, 2008; REACH).

The ECHA's Risk Assessment Committee (RAC) recommended classifying nitric acid based on the LC50 value for RFNA in rats and supporting evidence in humans (ECHA, 2013). The potential health effects of the nitric acid fumes are expected to be covered by the hazard classification (T+; R26) and exposure standard (TWA = 5.6 mg/m^3 (3 ppm)) in the HSIS for nitrogen dioxide. Therefore nitric acid is not recommended for classification. However, the classification for nitrogen dioxide should be referred to the users of nitric acid for protection from poisonous fumes.

Observation in humans

In several reported case studies, inhalation exposure to the fumes from the chemical or the decomposition product (nitrogen dioxide) caused immediate irritation of the respiratory tract and delayed acute respiratory distress syndrome. Some exposure situations involved contact or reaction with metals (e.g. copper), resulting in the release of toxic nitrogen oxides.

A man cleaning a copper chandelier with nitric acid at 60 % concentration (by placing the chandelier and nitric acid in a bowl) developed acute dyspnoea, including stages of extensive bronchiolitis and alveolitis obliterans. In another case report, a 66 year old man developed delayed-onset pulmonary oedema and fatal circulatory collapse 53 hours after occupational exposure to the fumes from the chemical (NAC/AEGL, 2008; HSDB). The exposure levels were not reported but were indicated to be at high concentrations.

In another case of delayed lung oedema, a bottle of nitric acid stored in the bathroom broke, and its contents came into contact with a metal shelf. The patient was exposed to nitrogen oxide fumes while wiping up the liquid. She was reported to suffer from severe dyspnoea. The concentration of nitric acid was not reported (ECHA, 2013).

In an accidental nitric acid explosion (a tank containing 1736 L of 68 % nitric acid), three men died of rapidly progressive pulmonary oedema with delayed onset following inhalation exposure. The men were in an area with the heaviest concentration of fumes and dust following the explosion, and escape took around 10–15 minutes. Respiratory difficulties occurred four to six hours later and deaths were within 21 hours. The concentrations of nitric acid aerosols or nitrogen oxides at the area of exposure were not determined (ECHA, 2013).

In Germany, between 1999 and 2010, a total of 134 cases of serious health damage with regard to handling specific nitric acid-containing (20-30 %) cleaning products at home were reported (in particular by using two limestone and rust removers), some with incorrect labelling. Among the cases, almost one quarter were caused by inhalation of fumes. The symptoms reported included prolonged cough, dyspnoea, respiratory tract obstruction, lung congestion, spasticity, and recurrent vomiting (ECHA, 2013).

Inhalation exposure to the chemical (stated as vapour) at much lower concentrations (0.08 ppm (~0.21 mg/m^3) for two hours, and 0.20 ppm (~0.52 mg/m^3) for four hours) did not cause adverse respiratory effects in healthy humans (Becker, 1996; HSDB).

In one experiment, two researchers exposed themselves to nitric acid fumes at 11.6–12.4 ppm (30–32 mg/m^3) for one hour. Acute toxicity symptoms included irritation of the nasal mucosa, stabbing pains in the trachea and larynx, marked secretion from the nose and salivary glands, burning and itching of the facial skin and eyes. Mild frontal headache developed after 20 minutes and most symptoms persisted one hour post-exposure. Another experiment was conducted at a higher concentration (85 ppm, 219 mg/m^3) where the researchers could only tolerate exposure to the chemical for two to three minutes as symptoms were more severe (NAC/AEGL, 2008).

Oral exposure to the chemical was reported to cause severe abdominal pain, burns to the skin or mouth, fever, a rapid drop in blood pressure, throat swelling and pain, and bloody vomiting (HSDB).

Corrosion / Irritation

Corrosivity

The chemical is classified as hazardous with the risk phrase 'Causes severe burns' (C; R35) in the HSIS (Safe Work Australia). The available data support this classification.

When rats were exposed to the fumes of the chemical, burns on the skin, ocular discharges and partially closed eyes were observed (see **Acute Toxicity: Inhalation**).

Following inhalation exposure to the fumes from red fuming nitric acid, widespread inflammation in the upper respiratory tract (rhinitis, tracheitis and pneumonitis), and localised areas of emphysema in the lungs were observed in rats (see **Acute Toxicity: Inhalation**). Delayed respiratory effects, including pulmonary oedema, occurred several hours after acute exposure, and were postulated to be related to inflammation caused by cellular necrosis in the lung tissues (HSDB).

In an acute exposure study, rats were exposed to white nitric acid fumes (97.5 %), nitrogen dioxide, or nitrogen dioxide (8–17 %) dissolved in RFNA. Nitrogen dioxide was reported to be the primary toxic constituent of RFNA, and its toxicity was potentiated by nitric acid following inhalation. Exposure to WFNA at an equivalent level to nitrogen dioxide and RFNA was reported to be less toxic (HSDB).

Observation in humans

In humans, inhalation from the fumes of the chemical at high concentrations caused immediate respiratory irritation (bronchoconstriction), followed by delayed acute respiratory distress syndrome and delayed-onset pulmonary oedema.

Nine adolescent asthmatics were exposed for 40 minutes to fumes from the chemical (a gas generated by passing clean air over a nitric acid solution) at 0.13 mg/m^3 (0.05 ppm) or 0.25 mg/m^3; or a combination of the gas at 0.13 mg/m^3 with 0.068 mg/m^3 sulfuric acid (aerosol) during exercise, and the respiratory function was measured at the end of exposure. Decreased forced expiratory volume (FEV) and increased respiratory resistance were observed under both treatment conditions (OECD, 2008; HSDB).

In several other respiratory studies, healthy human subjects exposed to fumes from the chemical were examined for biochemical and morphological changes, including bronchoalveolar lavage and pulmonary function. No adverse effects were observed when exposed to 0.08 ppm (0.20 mg/m^3) for two hours, or 0.20 ppm (0.50 mg/m^3) for four hours, during exercise (HSDB).

Humans exposed to concentrated nitric acid were reported to suffer from burns on the skin and severe eye irritation (opacification of the cornea and conjunctival epithelia) (OECD, 2008). Depending on the concentration and duration of exposure, effects on the eye including eyeball shrinkage, symblepharon (partial or complete adhesion of the eyelid to the eyeball), and visual impairment leading to blindness may occur (HSDB).

A 42 year old worker who was accidentally sprayed in the face with concentrated nitric acid suffered burns on the face and right cornea, despite bathing his face with water immediately after the accident. The patient was subjected to hydrotherapy for 24 hours and the injuries healed, except for some skin loss on the left auricle due to the gauze slipping off before completion of treatment. The remaining ulcers epithelialized within 10 days after injury (HSDB).

Repeated Dose Toxicity

Inhalation

Limited data are available. Based on the available data, the chemical is not considered to cause severe effects following repeated inhalation exposure at concentrations up to 0.45 mg/m^3. Erosion of the dental enamel has been reported in humans with long-term exposure to the chemical (aerosols and mist) (see **Observation in Humans**). However, the available data are insufficient to classify the chemical for repeated dose inhalation toxicity.

In an inhalation toxicity study (non-guideline), pulmonary effects of male rabbits (n = 6/dose) were examined when exposed (nose only) to the vapour from the chemical at concentrations of 0.05, 0.15 or 0.45 mg/m^3, four hours/day, three days/week for four weeks. A significant reduction of superoxide levels at all exposure concentrations and a reduction in bronchial response to pharmacologic agents (acetylcholine and histamine) indicated effects on both the conducting and respiratory airways. These effects were observed at all exposure concentrations (REACH). No systemic effects related to exposure were reported as only the pulmonary effect parameters were examined.

Observation in humans

In an occupational survey among workers exposed to mixed mineral acid aerosols and mist (32 exposed to the chemical alone), three out of 32 workers showed dental erosion. The duration of exposure and concentration of the chemical were not reported (OECD, 2008).

'Chronic inhalation exposure to nitric acid can cause respiratory irritation, leading to bronchitis and airways hyperreactivity and erosion of dental enamel' (UK HPA, 2007). The chemical was reported to be less potent in eroding teeth compared with sulphuric or hydrochloric acid (HSDB).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- local effects (corrosivity); and
- acute toxicity from inhalation of fumes from concentrated nitric acid (during storage or those produced in use).

When nitric acid is diluted (<10 %), the production of fumes such as nitrogen dioxide, is not likely.

Public Risk Characterisation

The chemical is on Schedules 5 and 6 of the SUSMP (2015). At concentrations greater than 0.5 %, a number of warning statements, first aid instructions and safety directions relating to products apply.

The current controls are considered adequate to minimise the risk to public health posed by any domestic use of the chemical. Therefore, the chemical is not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

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 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.

Also, the hazard classification in the HSIS should cross-reference the classifications and exposure standards for nitrogen oxides for users to be aware of the dangers of the fumes that may be produced during certain use scenarios such as working with metals (cleaning and etching).

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, 2015).

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.

Users of the chemical should be aware of the HSIS classification and exposure standard for nitrogen dioxide and its related hazard properties.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Causes severe burns (C; R35)*	Causes severe skin burns and eye damage - Cat. 1 (H314)

[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 dermal, ocular and inhalation exposure to the chemical or fumes produced during the use 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;
- 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 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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