Acetic acid: Human health tier II assessment

12 September 2013

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Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

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

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

Disclaimer

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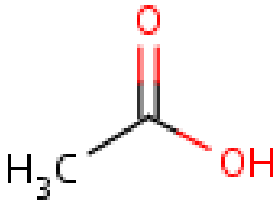
Chemical Identity

Synonyms	Ethanoic acid Acetic acid, glacial Vinegar acid Ethylic acid Methanecarboxylic acid
Structural Formula	

	
Molecular Formula	C2H4O2
Molecular Weight (g/mol)	60.05
Appearance and Odour (where available)	Clear colourless liquid with a pungent sour, vinegar-like odour.
SMILES	C(C)(=O)O

Import, Manufacture and Use

Australian

The following Australian industrial uses were reported under previous mandatory and/or voluntary calls for information.

The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 1000–9999 tonnes.

The National Pollutant Inventory (NPI) holds data for all sources of the chemical in Australia. The following use information was listed on NPI:

The chemical has reported cosmetic use (no details provided).

The chemical has reported commercial use including:

- in manufacturing of other chemicals;
- in research;
- in photographic chemicals;
- in latex coagulant;
- as an oil-well acidifier;
- in textile printing;
- as a solvent for gums, resins and volatile oils;
- in dyes; and
- as an antimicrobial agent.

The chemical has reported non-industrial uses including:

- in insecticides;
- in pharmaceuticals; and
- as a preservative in foods.

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; Galleria Chemical; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; and eChemPortal: OECD

High Production Volume chemical program (OECD HPV), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic use as a:

- buffering and masking agent; and
- fragrance compound.

The chemical has reported domestic use including as a:

- component of cleaning, washing and bleaching agents, paint, lacquers, varnishes, adhesive (binder), corrosion inhibitors, fillers, surface-active agents; and
- surface treatment.

The chemical has reported commercial use including:

- as a component of adhesives (binders), absorbents, adsorbents, fuels, paints, lacquers and varnishes, hydraulic fluids and additives, tanning dyes, colouring and bleaching agents, construction materials, corrosion inhibitors, anti-freeze agents, photochemicals, lubricants, additives, as a solvent for gums, resins, volatile oils and many other substances, in impregnation materials, fixing agents, stabilisers, and surface-active agents;
- as a laboratory reagent in chemical and biochemical analysis;
- in textile and dye industries as a dye catalyst, textile finishing, dye after-treatment, and in the production of nylon and acrylic fibres;
- as a flux agent for casting or joining materials;
- as an anti-static agent and viscosity adjustor;
- as a process regulator and softener;
- in printing calico and dyeing silk; and
- as reprographic, flotation and foaming agents.

The chemical has reported site-limited use including:

- as an intermediate in the manufacture of vinyl acetate monomer (VAM) (main use), acetate compounds, acetate rayon, plastics, rubber;
- as a constituent of photographic fixing baths;
- as an acidifying and neutralising agents in the chemical industry, as a stabiliser, as a complexing and flocculating agent, as a deliming agent during leather tanning; and
- as a laboratory reagent in chemical and biochemical analysis, in field testing of lead fumes, vinyl chloride determination, uric acid in urine, aniline vapours, and separation of gases.

The chemical has reported non-industrial use including:

- in medical preparations including lotions, ointments, and mouthwashes etc;
- as a non-agricultural pesticide and preservative;
- as an agricultural pesticide; and
- in food/feedstuff flavourings and nutrients, particularly as a componentt of vinegar.

Restrictions

Australian

The chemical is listed in the Poisons Standard (Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP, 2012)) in Schedule 2, Schedule 5, and Schedule 6.

The Schedule 2 entry states as follows:

ACETIC ACID (excluding its salts and derivatives) and preparations containing more than 80 per cent of acetic acid for therapeutic use.

Schedule 2 chemicals are labelled 'Pharmacy medicine' and may require advice from a pharmacist and which should be available from a pharmacy or, where a pharmacy service is not available, from a licensed person.

The Schedule 5 entry states as follows:

ACETIC ACID (excluding its salts and derivatives) in preparations containing more than 30 per cent of acetic acid (CH3COOH), **except**:

(a) when included in Schedule 2 or 6; or

(b) for therapeutic use.

Schedule 5 chemicals are labelled with 'Caution'. These are 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.

The Schedule 6 entry states as follows:

ACETIC ACID (excluding its salts and derivatives) and preparations containing more than 80 per cent of acetic acid (CH3COOH) **except** when included in Schedule 2.

Schedule 6 chemicals are labelled with 'Poison'. These are 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.

International

No known restrictions have been identified for cosmetic use in CosIng.

The US cosmetic ingredient review (CIR) concluded that a cosmetic ingredient can contain up to 0.3 % for safe use.

Existing Work Health and Safety Controls

Hazard Classification

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

R35 (corrosive)

Exposure Standards

Australian

The chemical has an exposure standard of 25 mg/m³ (10 ppm) time weighted average (TWA) and 37 mg/m³ (15 ppm) short-term exposure limit (STEL).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit (TWA) of 10–25 mg/m³ in countries such as China, Canada, Denmark, Germany, Ireland, South Africa, Spain, Sweden, Switzerland, and the USA.

An exposure limit (STEL) of 15–50 mg/m³ in countries such as China, Canada, France, Ireland, Singapore, South Africa, Spain, Sweden, Switzerland, and the USA.

Health Hazard Information

The acetate ion was considered to be of low systemic toxicity (NICNAS, 2012).

Toxicokinetics

Acetates are normal components in humans and animal diets. They are produced in small (molar) quantities daily in the gastrointestinal tract, where they are rapidly and completely metabolised (EFSA, 2012). Acetate is produced as a major intermediate in normal metabolic processes. Various isotope experiments have shown that the different carbon atoms of the chemical are used in glycogen formation, as intermediates of carbohydrates and fatty acid synthesis, as well as in cholesterol synthesis. In addition, the chemical also participates in the acetylation of amines and formation of proteins of plasma, the liver, kidney, gut mucosa, muscle and brain (IPCS, 1966).

The chemical is absorbed from the gastrointestinal tract and through the lungs. Following absorption, the chemical is almost completely metabolised by most tissues and may give rise to the production of ketone bodies as intermediates. An increased association with protein fractions of plasma in most major tissues was also noted. As only small amounts of sodium acetate were measured in the urine of dogs following large doses (1–2 g/kg) of sodium acetate administered intraperitoneally (i.p.), rapid utilisation of the acetate ion was indicated. In rats given radiolabelled acetate in the diet, 50 % of the radiolabel was excreted as carbon dioxide (EC, 2012; HSDB). The potential dermal absorption from a 100 mg/mL aqueous solution of acetic acid in humans was stated as 43 % (REACH). Following a single injection of acetic acid into the pylorus ligated stomach of rats, decreased absorption of the chemical was observed with increasing doses. In this case 100, 80, 75, and 30 % absorption of the chemical occurred at doses of 20, 50, 80, and 420 mg/kg bw, respectively (REACH).

The level of the acetate ion in humans has been estimated at about 50–60 µmol/L (3.0–3.6 mg/L) in plasma and 116 µmol/L (7 mg/L) in cerebrospinal fluid. Daily turnover of the acetate ion in humans is estimated at about 7.5 µmol/kg/min representing about 45 g/day (EC, 2012).

Acute Toxicity

Oral

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

The median lethal dose (LD50) observed in two rat studies is greater than 2000 mg/kg bw. Details regarding the concentration of the administered test substance were not provided (EC, 2012; HSBD; REACH).

Dermal

The chemical was of moderate acute toxicity in animal tests following dermal exposure. The median lethal dose (LD50) in rabbits was 1060 mg/kg bw. Details regarding the concentration of the administered test substance were not provided. Even though the chemical caused moderate acute dermal toxicity in this case, this is more likely due to shock from its corrosive effects rather than any systemic toxicity (see **Corrosivity**) (HSDB).

Inhalation

The chemical was of low acute toxicity in animal tests following inhalation exposure. The median lethal concentration (LC50) for a four-hour exposure in rats was 11.4 mg/L (HSDB).

Observation in humans

Severe health effects have been reported in humans following a single accidental exposure by different routes, mainly due to the local corrosive effects of the chemical leading to systemic effects (HSDB) (see **Corrosivity**). Dietary ingestion of vinegar, where the chemical is present at non-corrosive concentrations, is not associated with harm.

Perforation of the oesophagus has been reported following accidental ingestion of as little as 1 ml of the chemical at high concentrations. In other cases, ingestion has resulted in severe corrosion of the mouth, perforation of the oesophagus, severe corrosion of the gastrointestinal tract, bloody vomiting, diarrhoea, shock, haemolysis, haemoglobinuria and death.

Haemolysis, slight intravascular coagulation, and oliguric kidney insufficiency have been reported in only one patient (out of two) following ingestion of an 80 % solution of the chemical. Both patients also showed similar patterns of tubular proteinuria. The observations in the second patient suggested a direct toxic effect of the chemical on the proximal tubule of the kidney. Bronchopneumonia and pulmonary oedema have also been reported following acute overexposure.

Accidental rectal administration of 50 mL of 9 % chemical to a 5-year-old boy resulted in necrosis of the colon, acute renal failure, acute liver dysfunction, disseminated intravascular coagulopathy (DIC) and sepsis. The enhanced toxicity of the chemical, without the benefit of dilution and neutralisation in the upper intestine, is evident in this case (HSDB).

Corrosion / Irritation

Corrosivity

The chemical is classified as hazardous with the risk phrase 'Causes severe burns' (R35) in HSIS (Safe Work Australia). Human experience (see **Observation in humans**) and animal studies support this classification (IPCS, 2010; EC, 2012; HDSB; REACH).

In animal studies, severe skin burns were reported in guinea pigs at 80 % solution of the chemical, moderate to severe burns at 50–80 % solution, mild injury at 50 % solution, and no effect at 10 % solution to intact or abraded skin patches. In a study with rabbits, the chemical was considered to be slightly irritating at concentrations of 3.3 % and 10 %. In another study with rabbits, a concentration of 2.5 % of the chemical was not irritating while concentrations of 10–25 % caused moderate to severe erythema, slight to severe oedema, skin lesions over the application site and eschar formation. A 10 % solution was therefore considered a skin irritant. In an eye irritation study in rabbits, a 3 % solution of the chemical produced moderate irritation and a 10 % solution was classified as severely irritating or corrosive.

Observation in humans

Extreme eye and nasal irritation has been experienced by unaccustomed humans at vapour concentrations in excess of 25 ppm, and 50 ppm was stated to be 'unendurable'. Conjunctivitis from concentrations below 10 ppm has also been reported. It has also been reported that exposure to the chemical at 10 ppm is relatively non-irritating. A splash of vinegar (4 to 10 % solution) in the human eye caused immediate pain and conjunctival hyperaemia, and in some cases injury of the corneal epithelium. Contact with the concentrated form of the chemical can lead to severe skin and eye damage and vapours of the chemical can also damage nose, throat and lungs.

Although a case of chemical burns (necrosis, ulceration) has been reported in humans following treatment under occlusion with gauze consisting of a 50:50 mixture of flour and rice vinegar, containing 4.5 % of the chemical, a 10 % solution of the chemical in patch tests with humans over a period of 48 hours caused slight skin irritation (EC, 2012; HDSB; REACH).

Sensitisation

Respiratory Sensitisation

No data are available.

Skin Sensitisation

No data are available.

Observation in humans

Although limited information is available, repeated or prolonged contact of the chemical with the skin may cause dermatitis. Following on from the report of type-1 hypersensitivity-like reactions to a number of acid-based food items in a 68-year-old female, the authors concluded that the chemical was the likely causative agent for these reactions. This conclusion was based on the patient's history as well as the results of various allergy tests (HSDB).

Repeated Dose Toxicity

Oral

Based on the treatment-related effects reported in limited repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure. The effects observed in some cases could have been only due to the corrosive activity of the chemical.

In a repeated dose oral toxicity study in pigs for approximately six months, animals were initially fed the chemical at 155 mg/kg bw/day and the dose level was raised every 10–30 days to 380–450 mg/kg bw/day after 60 days. There was no mortality and no effects on body weight or acid-base balance noted in this study (REACH). In another similar study, pigs were fed daily diets containing the chemical at 0, 240, 720, 960 and 1200 mg/kg bw/day for successive 30-day periods to a total of 150 days. There were no significant differences in growth rate, weight gain, early morning urinary ammonia and terminal blood pH between controls and test groups (HSDB).

Repeated administration (intragastrically) of the chemical at a 3 % concentration in animals (unspecified) for six months resulted in chronic inflammation of the oesophageal mucosa. Similarly, intragastric administration to rats of 3 ml of a 10 % solution for 90 days produced a drop in haemoglobin concentration and erythrocyte count (HSDB).

Dermal

Although no data are available, repeated or prolonged exposure to chemical may cause skin darkening and may also cause dermatitis.

Inhalation

No data are available.

Observation in humans

Results from repeated oral, inhalation, and dermal exposure of humans to the chemical has been reported with effects on the gastrointestinal tract, digestive disorders including hearburn and constipation, chronic inflammation of the respiratory tract, pharyngitis, catarrhal bronchitis, darkening of skin, skin dermatitis, and erosion of the exposed front teeth enamel. In addition, skin on the palms of hands can become dry, cracked and hyperkeratotic. These observed effects were not associated with any systemic findings, suggesting the effects observed could be due to its corrosive activity (EC, 2012; HSDB).

Acetic acid present in vinegar and other items of food and drink has been consumed at about 1g/day by man for centuries, apparently without causing any adverse effects (IPCS, 1966). Furthermore, estimations of the daily intake of acetic acid has also been reported to vary from about 1 gram to 2.1 g/day for subjects older than two years. No adverse health effects at these intakes of the chemical have been reported (EC, 2012). This is likely to be due to the well-known function of the chemical in a number of normal cellular metabolic processes (IPCS, 1966). However, continued ingestion of large doses of the chemical has been regarded as a contributory factor in the development of the Laennec type of liver cirrhosis (IPCS, 1966).

In a study of five workers from a cellulose acetate chemical plant, reported effects included blackening and hyperkeratosis of the skin of the hands, conjunctivitis, pharyngitis, bronchitis, and blackening and erosion of the teeth. Specific details about exposure duration and the concentration of the chemical were not available (EC, 2012).

Specific studies have reported that workers exposed to concentrations of 60 ppm during their working hours, plus 1 hour daily at 100–200 ppm, for 7–12 years developed conjunctivitis, bronchitis, pharyngitis, and erosion of exposed teeth. In addition, workers exposed for a number of years to concentrations of up to 200 ppm have been found to suffer from palpebral oedema with hypertrophy of the lymph nodes, conjunctival hyperaemia, chronic pharyngitis, chronic catarrhal bronchitis, in some cases asthmatic bronchitis, and traces of erosion on the vestibular surface of teeth (incisors and canines) (HSDB). A further study of 12 workers exposed long term to the chemical (at least two years) with an average vapour exposure of 0.125 mg/L (including peaks of 0.44 mg/L) reported skin irritation (hyperkeratotic dermatitis with cracked and irritated lesions of the palmar skin). Respiratory and eye irritation was reported for eight of the workers. Five workers also had some erosion on their incisors (REACH).

Genotoxicity

As the chemical only produced mutations at low pH levels, where the effects are likely to be due to the acidic nature of the chemical rather than any underlying genotoxicity, the chemical is not considered to be genotoxic (EC, 2012; HSDB; REACH).

The chemical has been reported to be not mutagenic in bacterial reverse mutation assays using *Salmonella typhimurium* strains with and without metabolic activation.

The chemical also showed no mutagenic potential in *Saccharomyces cerevisiae*, with and without metabolic activation. The chemical, at concentrations close to those showing cytotoxicity (up to 16 mM), was also concluded not to be clastogenic when tested in cultured Chinese hamster K1 cells. Although chromosomal aberrations could be induced at these high concentrations, they were shown to be artifacts due to acidification of the culture medium and could be eliminated by neutralising the medium or enhancing its buffering ability. The chemical, in concentrations of 250–1500 µg/ml (LC50 1000 µg/ml), did not initiate transformation in C3H/10T1/2 cells. The chemical did not induce mutations or chromosomal recombination in *Drosophila melanogaster*.

Carcinogenicity

Based on the limited data available, the chemical is not likely to be a carcinogen.

Applying the chemical to the skin of mice was reported to stimulate epidermal hyperplasia, suggesting that it was a very weak tumour promotor for known carcinogens. In this case, the chemical was applied dermally 1–3 times per week (at doses of 1–40 mg/animal) for 32 weeks. Mice that were only initiated with 7,12-dimethylbenz[a]anthracene or β-propiolactone did not develop any tumours. Also, mice treated (promoted) once a week for 32 weeks with acetic acid developed no tumours. However, more than one weekly application of 10–40 mg of the chemical caused increased mortality. When 10 mg of the chemical was applied dermally three times a week, 33 % of mice died and when 20 mg was applied twice a week, approximately 50 % of mice died. Details regarding the concentration of the administered test substance were not provided (REACH).

In another study, oral administration of the chemical as a 3 % solution in rats (three times/week) for eight months did not induce tumours in the oesophagus and forestomach, although epithelial hyperplasia was observed. However, when dosed in combination with the known carcinogen N-nitrososarcosine ethyl ester to assess whether the chemical acts as an enhancer of the initiation and/or promotion of carcinogenesis, there was an increase in oesophageal/stomach tumour formation, presumably as a consequence of local irritation (REACH).

Reproductive and Developmental Toxicity

Based on the available data, the chemical does not show specific reproductive or developmental toxicity.

In developmental toxicity studies (EU Method B.31), the chemical was administered by gavage to pregnant female Wistar rats and CD-1 mice at 16, 74.3, 345, and 1600 mg/kg bw/day during gestation (days 6–15) with 10 consecutive doses. In a similar study, the chemical was administered (by gavage) to female Dutch rabbits at 16, 74.3, 345, and 1600 mg/kg bw/day during gestation (days 6–18) with 13 consecutive doses. The highest dose (1600 mg/kg bw/day) had no clearly discernible effect on nidation (implantation) or on maternal survival, or on foetal survival in any species. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the controls. The derived maternal as well as developmental NOAEL for each species was >1600 mg/kg bw/day, based on no adverse effects observed at the highest dose (REACH).

Risk Characterisation

Critical Health Effects

The critical health effect for risk characterisation is its local effect (corrosivity).

Public Risk Characterisation

The chemical is used only as a buffering and masking agent in cosmetics and therefore public exposure to high concentrations of the chemical is not expected through cosmetic uses. If the concentrations in cosmetics are low, corrosive effects are not expected and, therefore, the risk to public health is not considered to be unreasonable. Further risk management is not considered necessary for public safety.

Although the concentration of the chemical in domestic products is not known, the general public may be exposed to the chemical through dermal and/or inhalation routes when using domestic products containing the chemical. Acetic acid is listed in Schedule 2, Schedule 5, and Schedule 6 of the SUSMP for public use. A number of warning statements, safety directions and first aid instructions apply to any domestic products containing this chemical. The current controls are considered adequate to minimise the risk to public health posed by any domestic use of this chemical. Therefore, the risk to public health is not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, dermal, ocular 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 local health effect, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise dermal, ocular 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.

Based on the available data, the hazard classification in HSIS is considered appropriate.

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 hazards and environmental hazards.

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 instruction on the label.

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 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;
- 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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