# Benzonitrile: Human health tier II assessment

08 March 2019

# CAS Number: 100-47-0

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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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#### Acronyms & Abbreviations

# **Chemical Identity**

| Synonyms                               | benzene, cyano-<br>phenylcyanide<br>benzoic acid nitrile |  |
|--|--|--|
| Structural Formula                     |  |  |
| Molecular Formula                      | C7H5N  |  |
| Molecular Weight (g/mol)               | 103.12   |  |
| Appearance and Odour (where available) | Colourless oil with an almond odour.                     |  |
| SMILES                                 | C(#N)c1ccccc1  |  |

# 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) dossier; Galleria Chemica; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR); the US National Library of Medicine's Hazardous Substances Data Bank (HSDB), and the International Fragrance Association (IFRA) Transparency List.

The chemical has reported cosmetic use as a fragrance ingredient, with a reported maximum concentration of 0.2 % in perfumes (HSDB).

The chemical has reported site-limited uses, including:

- as a solvent and intermediate for the manufacture of rubber chemicals, specialty lacquers, resins, polymers, anhydrous metallic salts and dyes;
- for the manufacture of benzoguanamine; and
- as an additive in jet-fuels, cotton bleaching baths, nickel-plating baths, and acrylic fibres.

The chemical has reported non-industrial uses, including as an intermediate in pesticides and pharmaceuticals.

# Restrictions

#### Australian

No known restrictions have been identified.

### International

# **Existing Work Health and Safety Controls**

### **Hazard Classification**

The chemical is classified as hazardous, with the following hazard categories and hazard statements for human health in the Hazardous Chemical Information System (HCIS) (Safe Work Australia):

- Acute toxicity Category 4; H302 (Harmful if swallowed)
- Acute toxicity Category 4; H312 (Harmful in contact with skin)

### **Exposure Standards**

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

# **Health Hazard Information**

Although synonyms of the chemical contain the word 'cyanide', toxicokinetic data from animal studies show that the chemical does not form cyanides in vitro or in vivo (see **Toxicokinetics** section).

### **Toxicokinetics**

Animal studies indicate that the chemical can be absorbed via all routes of exposure: oral, dermal and inhalation. The major metabolic pathway for the chemical is aromatic hydroxylation to cyanophenols, and a small proportion can be hydrolysed to benzoic acid.

Following oral administration of 150 mg/kg bw of the chemical in rabbits, 50 % of the administered dose was excreted within 48 hours. The metabolites identified in the urine were cyanophenols (m-, and p-hydroxybenzonitrile), sulfate conjugates (23–27 %) and glucuronic acid conjugates (28–38 %) of hydroxylated compounds, mercapturic acid (~5 %) and benzoic acid (10 %) (HSDB; REACH).

Further studies conducted in rats and in rabbit liver microsomes confirmed biotransformation of the chemical to hydroxybenzonitrile (or cyanophenols). In rats, deuterated benzonitrile was converted via microsomal hydroxylation to 4-hydroxybenzonitrile with smaller amounts of 3-, and 2-hydroxybenzonitrile, and a deuterium retention rate of 41 %. In the in vitro study, trace amounts of 4- and 2-hydroxybenzonitrile were detected when rabbit liver microsomes were incubated with deuterated benzonitrile (HSDB; REACH).

Intraperitoneal administration of the chemical in rats resulted in significant decreases of hepatic glutathione levels at 3 and 8 hours after exposure, which is associated with the formation of mercapturic acid. However, this effect was reversed after 24 hours. Glutathione levels and cytochrome oxidase activity in the brain were not affected, indicating that the chemical does not pass the blood-brain barrier (REACH).

Hydrogen cyanide (HCN) was not identified as a metabolite of the chemical, and the cyanide ion was not found to be formed in vivo or in vitro. Therefore, there is no risk of HCN intoxication (HSDB; REACH). The chemical structure suggests the formation of an epoxide as an intermediate (NRC, 2014)

### **Acute Toxicity**

### Oral

The chemical is classified as hazardous with hazard category 'Acute Toxicity Category 4' and hazard statement 'Harmful if swallowed' (H302) in the HCIS (Safe Work Australia). The available data support this classification.

The median lethal dose (LD50) is 690–1500 mg/kg bw in rats, 971–1400 mg/kg bw in mice, and 800 mg/kg bw in rabbits and cats (BG Chemie; HSDB)

Sublethal signs of toxicity in rats, mice and rabbits included hyperactivity, muscular weakness, ruffled fur, prostration, dyspnea, tremor, and convulsions. Histopathological observations included lung congestion with oedema and intra-alveolar haemorrhages (BG Chemie; REACH).

### Dermal

The chemical is classified as hazardous with the hazard category 'Acute Toxicity Category 4' and hazard statement 'Harmful in contact with skin' (H312) in the HCIS (Safe Work Australia). The available data support this classification.

The dermal LD50 is 1200–2000 mg/kg bw in rats, and 1250–1400 mg/kg bw in rabbits.

In rabbits, sublethal signs of toxicity included salivation, muscular weakness, prostration, ataxia, tremors, loss of righting reflex and nasal discharge. Local skin irritation (pale red erythema and slight oedema) was observed at the end of the 24-hour exposure period, but subsided within 7 days after exposure (BG Chemie; REACH).

#### Inhalation

The median lethal concentration (LC50) is >8 mg/L in rats, and <2.95 mg/L in mice. Studies showed that mice are more sensitive to the chemical than rats following acute inhalation exposure. The available information indicates that the chemical is of moderate acute toxicity in mice and; therefore, hazard classification is warranted (see **Recommendation** section).

In a 4-hour acute inhalation toxicity study, all mice died (10/10) when exposed to a nominal concentration of 700 ppm (2.95 mg/L) for 4 hours. However, no mortality occurred in rats (0/6) when exposed to the same concentration. Therefore, a LC50 could not be determined (REACH).

In several acute inhalation toxicity studies in rats, there was no mortality within the tested concentration range for 4-hour exposures: 0.8–4 mg/L. In 1 study, 3 out of 10 animals died when exposed to 8 mg/L of the chemical vapour. Therefore, the LC50 was determined as >8 mg/L in this study (NRC, 2014; BG Chemie; REACH).

In mice, a LC50 of 6 mg/L was reported in 1 study; however, the duration of exposure was not stated. In another study, 1 out of 7 animals died when exposed to 890 ppm (3.8 mg/L) for 2 hours (NRC, 2014; BG Chemie; REACH).

Sublethal effects observed in both rats and mice included respiratory irritation, labored breathing, poor coordination, sedation, ataxia and prostration. The surviving rats showed subnormal weight gain. There were also changes in blood cholinesterase activity. No abnormal lesions were observed at autopsy, but microscopy examination revealed multifocal areas of lymphoid hyperplasia with foamy macrophage accumulations in the lungs of rats. In mice, congestion and oedema in the lungs, hepatic congestion and sinuisoidal dilation were observed (NRC, 2014; BG Chemie; HSDB).

#### Observation in humans

In an occupational case study, a male worker's head and clothing were reported to be accidentally drenched with the chemical (exposure concentration not reported). He was subsequently doused with water to prevent further dermal exposure, but his clothes were not removed. The worker then experienced periods of unconciousness with tonic contractions in his arm and face muscles, and severe respiratory distress for approximately 75 minutes. He was discharged without apparent symptoms the next day. Several years after this accident, he still experienced episodes of unconciousness (HSDB). However, differential diagnosis revealed the possibility of Pickwickian syndrome (obesity hypoventilation syndrome) and it was not possible to establish a causal relationship with exposure to the chemical (BG Chemie).

### **Corrosion / Irritation**

#### Skin Irritation

The available information indicates that the chemical is a slight skin irritant. However, the chemical is reported in case studies to be irritating to human skin, warranting classification (see **Observation in humans** section).

In a skin irritation study, undiluted benzonitrile (0.5 mL) was applied (occlusively) on intact and abraded skin of New Zealand White rabbits (n=6) for 24 hours, with observation up to 2 days. Effects were scored using the Draize system. For intact skin, very slight erythema was observed in 2 out of 6 rabbits. For abraded skin, very slight to well-defined erythema was observed in 5 out of 6 rabbits, and very slight oedema in 1 out of 6 rabbits. The chemical was considered to be a slight skin irritant with occlusive 24 hour exposure (REACH).

In a skin irritation study (according to a method adapted from the Draize procedure), undiluted benzonitrile (0.5 mL) was applied (occlusively) on intact and abraded skin of New Zealand albino rabbits (n=4) for 24 hours, with observation up to 72 hours. No evidence of irritation was observed at the 24- and 72-hour timepoints. The chemical was considered to be a non-irritant (REACH).

#### Eye Irritation

The available information indicates that the chemical is at most a slight eye irritant. The effects are not sufficient to warrant hazard classification.

In an eye irritation study (FDA guideline), undiluted benzonitrile (0.1 mL) was instilled into one eye of each New Zealand White rabbit (n=6). Observations were made at 24, 48 and 72 hours, and after 7 days. Slight lesions (conjunctival redness and chemosis) were observed in all animals upon application, but were reversible within 7 days. The chemical was not considered to be an eye irritant (REACH).

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In an eye irritation study (equivalent to OECD Test Guideline (TG) 405), undiluted benzonitrile (0.1 mg/L) was instilled into the right eye of New Zealand albino rabbits (n=5), with observation up to 7 days. Transient iridal and conjunctival irritation were observed upon exposure, but were reversible within 72 hours. The chemical was considered to be slightly irritating to the eye in rabbits (REACH).

### Observation in humans

The chemical as a liquid is reported to be irritating to the skin and eyes, and; as a vapour, is irritating to the eyes, nose and throat (HSDB).

Case studies with limited documentation have been reported. The chemical caused skin irritation (reddening and subsequent blistering) in humans following skin and mucous membrane contact after varying latency periods. The average healing time for skin lesions was 10 to 14 days. A worker was reported to develop erythema with scaly superficial detachment of the skin on both lower arms after the chemical flowed over his protective gloves. No further details are available (BG Chemie).

In an occupational study, employees working in benzonitrile production were observed over 13 years and 8 months. No skin irritation or damage, altered blood counts, allergies or other effects associated with handling of the chemical were observed. The chemical's unpleasant odour was 'tolerated' after a short period, and no irritancy of the nasal mucosa was observed (BG Chemie).

In a 48-hour patch test, no skin irritation was observed following exposure (occlusive) to the chemical in yellow vaseline at a concentration of 2 % (BG Chemie).

### Sensitisation

#### Skin Sensitisation

No data are available.

#### Observation in humans

A human maximisation test was conducted in 35 healthy subjects using the chemical in yellow vaseline at a concentration of 2 %. No sensitisation reactions were observed. The same results were obtained when the substance was retested in 27 subjects (BG Chemie).

In a case report, 6 patients were treated for allergy to the rubber components of their underwear that had been bleached with sodium hypochlorite. The hypochlorite had reacted with several components contained in the elastic, and benzonitrile was identified as one of the components. Volunteers (n=25) were then tested for sensitisation with the reaction mixture (0.1 mL), for which 14 showed a positive response. No allergic reactions were observed when 7 subjects were tested with benzonitrile (0.5 % of the mixture) (BG Chemie).

### **Repeated Dose Toxicity**

Oral

Based on the available data, the chemical is not considered to cause serious damage to health from repeated oral exposure.

The main adverse effects observed in rats and mice were in liver and kidney. Male rats were more sensitive to the chemical with kidney effects observed at  $\geq$ 75 mg/kg bw/day, compared with female rats and mice at  $\geq$ 150 mg/kg bw/day. These effects were attributed to 'hydrocarbon nephropathy', which is unique to the male rat.

In a 13-week repeat dose oral toxicity study (OECD TG 408), Fischer 344 rats (n=10/sex/dose) were administered the chemical in corn oil at doses of 0, 19, 37.5, 75, 150 or 300 mg/kg bw/day by gavage, once a day. There was no mortality. At the highest dose, decreased body weight gain (both sexes), reduced strength in the hind legs and delayed reaction to thermic stimuli (females only) were observed. At  $\geq$ 150 mg/kg bw/day, neurotoxicity symptoms (hyperactivity and aggressive behaviour) were observed in both sexes, and kidney effects (vacuolar degeneration of the cortex tubuli) in females only. At  $\geq$ 75 mg/kg bw/day, males showed significantly increased kidney weights (relative and absolute), and histopathology showed dose-dependent hyaline droplet degeneration and dilated renal tubules. The authors attributed these effects to 'hydrocarbon-nephropathy'. The no observed adverse effect level (NOAEL) was determined as 37.5 and 75 mg/kg bw/day in male and female rats, respectively (BG Chemie; REACH).

In a 13-week repeat dose oral toxicity study, B6C3F1 mice (n=10/sex/dose) were administered the chemical daily at 0, 37.5, 75, 150, 300 or 600 mg/kg bw/day. No mortality occurred. At the highest dose, decreased body weight gain (both sexes), and delayed startle responses to acoustic signals (females only) were observed. All animals showed hyperactivity and aggressive behaviour at  $\geq$ 300 mg/kg bw/day. Observed liver effects included centrilobular hypertrophy, increased Kupffer cells, mineralisation and cell necrosis (males:  $\geq$ 300 mg/kg bw/day, females: 600 mg/kg bw/day), increased liver weights (relative and absolute) at  $\geq$ 75 mg/kg bw/day (both sexes), and increased absolute liver weight at 37.5 mg/kg bw/day (females only). Kidney effects (dose-related dilations of the tubuli of the inner cortex) were observed in males at =150 mg/kg bw/day and in females at  $\geq$ 300 mg/kg bw/day. The NOAEL was reported to be 37.5 mg/kg bw/day (BG Chemie; REACH).

In two short term repeat dose oral toxicity studies (14 days) with limited documentation, neurotoxicity symptoms (ataxia, loss of motor function, hypoactivity) were observed in rats at 1000 mg/kg bw/day, and in mice at 600 mg/kg bw/day. Renal tubule degeneration in rats at 1000 mg/kg bw/day, and hepatotoxicity

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(karyomegaly and necrosis in hepatocytes) in mice at 600 mg/kg bw/day were reported. The NOAELs for these studies were determined to be 300 and 200 mg/kg bw/day, for rats and mice, respectively. In another 28-day study, there were no adverse effects in rats at 40 mg/kg bw/day (REACH)

Dermal

No data are available.

#### Inhalation

Limited data are available. Mortalities seen in mice and cats are likely to be manifestations of acute toxicity, severe repeat dose effects were not reported. Therefore, hazard classification is not warranted.

In a repeat dose inhalation toxicity study, rats (n=20) and rabbits (n=20) were exposed to the chemical vapour at 0.01 mg/L or 0.07 mg/L, 4 hours/day, 5 days/week for 4.5 months. Effects observed included changes in blood chemistry (decreased red blood cell counts and haemoglobin, increased leucocyte counts), and increased cytochrome oxidase activities. At 0.01 mg/L, the observed effects were considered transient, and there were no morphological changes in visceral organs. Based on these results, a maximum allowable concentration of the chemical in the air of work areas of 0.001 mg/L was assigned (BG Chemie; REACH). No further details are available.

Several effects were observed to be age-dependent in rats. Following exposure to 0.07 mg/L of the chemical vapour for 4.5 months, significant histopathological changes were observed in the inner organs of adult and aged rats, but not in juvenile rats. Specifically, changes were observed in the lungs, liver (necrosis, pericholangitis with granular epithelial dystrophy, accumulation of erythrocytes in the bile duct), adrenal gland (inflammation of the cortex), and spleen (hyperplasia of follicles). Blood chemistry (erythrocyte and leukocyte counts and haemoglobin, shift of the albumin-globulin ratio) was observed to change earlier in adult rats than in juvenile or aged rats, and was also 'compensated' more rapidly (HSDB; REACH).

Species-specific effects were observed in another short-term study, where mice appeared to be particularly sensitive to effects following repeated inhalation exposure. Cats (n=2), rabbits (n=2), guinea pigs (n=2), rats (n=4) and mice (n=10) were exposed to the chemical vapour at 1.9 mg/L, 6 hours/day for 3 days. Mortalities occurred in 1 cat after 7 days, 1 rat after 3 days and 8 mice within the exposure period. The cats displayed imbalance, jumpiness, mydriasis (dilation of the pupil), and decreased body weight. However, these effects were reversible in the surviving cat after 5 days. The mice displayed apathy, imbalance, lateral position and narcosis. Autopsy revealed atelectasis of the lungs, and liver effects such as a marked lobular pattern and increased fat deposits. No effects were observed in rabbits, guinea pigs and the surviving rats. When tested at a lower concentration (0.41 mg/L) up to 2 weeks, no mortalities or gross abnormalities were observed in cats, rabbits, guinea pigs and rats (REACH).

### Genotoxicity

Based on the available data, the chemical is not considered to be genotoxic; and therefore, hazard classification is not warranted.

The following results were reported in in vitro assays (REACH):

- negative in several bacterial reverse mutation assays (OECD TG 471) with several strains of Salmonella typhimurium (TA98, TA100, TA1535, TA1537 and TA1538) at concentrations up to 5000 µg, with or without metabolic activation;
- did not induce chromosomal losses in a mitotic recombination assay (OECD TG 481) with Saccharomyces cerevisiae under standard conditions (16-hour incubation, 30 °C) up to 2190 µg/mL. However, it was positive under modified experimental conditions (16-hour cold treatment at 0 °C);
- negative in a mammalian cell gene mutation assay (OECD TG 476) in mouse lymphoma L5178Y cells up to cytotoxic concentrations, with or without metabolic activation;
- negative in a sister chromatid exchange (SCE) assay (OECD TG 479) in Chinese hamster ovary (CHO) cells up to cytotoxic concentrations, with or without metabolic activation;
- induced chromosomal aberrations in Chinese hamster ovary (CHO) cells at 1495 μg/mL with metabolic activation. However, this effect could not be verified in a second experiment. A concentration of 1993 μg/mL was found to be cytotoxic.

The chemical was not considered to be genotoxic in an in vivo mammalian erythrocyte micronucleus test (OECD TG 474) in B6C3F1 mice. This test was conducted following a 13-week repeat dose toxicity study (see **Repeated dose toxicity: Oral** section), where the animals were orally administered the chemical at 0, 37.5, 75, 150, 300 or 600 mg/kg bw/day. There was no increase in the incidence of micronuclei in polychromatic erythrocytes up to the maximum tested dose. Systemic toxicity effects were reported at 37.5 mg/kg bw/day (REACH).

### Carcinogenicity

No data are available.

### **Reproductive and Developmental Toxicity**

The chemical was reported to have no adverse effect on sperm morphology and vaginal cytology. No further details are available and there is no information about the study conducted (REACH).

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# Other Health Effects

#### Neurotoxicity

In a neurotoxicity study, male Swiss mice (n=10/dose) were administered (gavage) the chemical in olive oil at doses of 240, 480 or 720 mg/kg bw/day. Observed effects at all doses included low levels of motor and exploratory activity, dose-dependently impaired neuromotor co-ordination (rotating rod test) and catalepsy. At the highest dose, the animals exhibited significantly reduced body temperature and analgesia. The low and mid-dose group showed hyperaesthesia at 30 and 60 minutes. Based on these effects, it was concluded that the chemical could affect catecholaminergic neurons (BG Chemie; REACH).

The chemical was tested for anticonvulsive action in mice treated with pentetrazol (a drug used in convulsive therapy), and suppressed convulsions in 7 out of 10 mice at a dose of 300 mg/kg bw (route of exposure not stated) (BG Chemie; HSDB).

Acute and repeated dose toxicity studies in animals have reported neurological symptoms including hyperactivity, tremor, loss of motor function, and ataxia (see **Acute** and **Repeated dose toxicity** sections).

# **Risk Characterisation**

### **Critical Health Effects**

The critical health effects for risk characterisation include systemic acute effects (acute toxicity from oral, dermal and inhalation exposure).

Undiluted benzonitrile may also cause irritation to the skin upon dermal exposure.

### **Public Risk Characterisation**

Although use in cosmetic products in Australia is not known, the chemical is reported to be used overseas in perfumery at concentrations up to 0.2 %. The general public could be exposed through the skin or inhalation when using cosmetic products containing the chemical. However, based on the maximum reported concentration used in perfumery, the concentration is not considered to be sufficiently high to cause acute or local effects. Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

#### **Occupational Risk Characterisation**

During product formulation, dermal 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 systemic acute health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, and inhalation exposure are implemented. Good hygiene practices to minimise oral exposure are expected to be in place. 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 an amendment to the hazard classification in the HCIS (Safe Work Australia) (see Recommendation section).

## **NICNAS Recommendation**

Assessment of the chemical is considered to be sufficient, provided that the recommended amendment to the 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 aligned with the Globally Harmonized System of Classification and Labelling of Chemicals (GHS) as below. This does not consider classification of physical hazards and environmental hazards.

From 1 January 2017, under the model Work Health and Safety Regulations, chemicals are no longer to be classified under the Approved Criteria for Classifying Hazardous Substances system.

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|  | Hazard                   | Approved Criteria (HSIS) <sup>a</sup> | GHS Classification (HCIS) <sup>b</sup>   |
|--|--------------------------|---------------------------------------|--|
|  | Acute Toxicity           | Not Applicable* *                     | Harmful if swallowed - Cat. 4 (H302)*<br>Harmful in contact with skin - Cat. 4<br>(H312)* Toxic if inhaled - Cat. 3 (H331) |
|  | Irritation / Corrosivity | Not Applicable                        | Causes skin irritation - Cat. 2 (H315)   |

<sup>a</sup> Approved Criteria for Classifying Hazardous Substances [NOHSC:1008(2004)].

<sup>b</sup> 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 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;
- 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[s] 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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Last update 08 March 2019

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