2-Propanol: Human health tier II assessment

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CAS Number: 67-63-0

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.

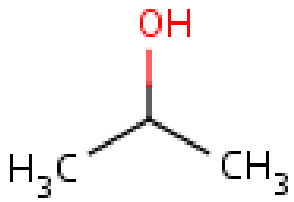
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	Isopropanol Isopropyl alcohol Propan-2-ol Dimethyl carbinol
Structural Formula	
Molecular Formula	C3H8O
Molecular Weight (g/mol)	60.09
Appearance and Odour (where available)	Colourless liquid with a pleasant odour.
SMILES	C(C)(C)O

Import, Manufacture and Use

Australian

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

The chemical has reported cosmetic use including in:

- cosmetics (details not specified), hair sprays and colours.

The chemical has reported domestic use including in:

- printing inks and surface coatings (major use);
- adhesives (binding agents);
- cleaning/washing agents, including in domestic detergents; and
- colouring agents.

The chemical has reported commercial use including as:

- a solvent (major use);
- an industrial detergent;
- a dry cleaning agent;
- fuel and lubricant additives; and
- welding and soldering agents.

The chemical has reported site-limited use including:

- as a chemical intermediate in manufacturing polyacrylic acid and other chemicals; and
- in analytical laboratory work.

The following non-industrial uses have been identified in Australia including:

- as a solvent in pharmaceuticals products.

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

International

The following international uses have been identified through European Union Registration, Evaluation and Authorisation of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; US Household Products Database; and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic use as a:

- solvent for cosmetics with a concentration of up to 10 % (liquid) (e.g. lotions, perfumes, shampoos, skin cleansers, nail polishes, makeup removers, deodorants, body oils, shampoos, hair dye rinses preparations and permanent wave lotions,

and skin lotions); and

- antifoaming agent, fragrance ingredient, and viscosity decreasing agent.

The chemical has reported domestic use including in:

- adhesives and binding agents;
- anti-condensation agents;
- aerosol propellants;
- colouring agents;
- cleaning/bleaching/washing agents;
- flame retardants and extinguishing agents;
- fillers;
- insulating materials and corrosion inhibitors;
- paints, lacquers and varnishes;
- odour agents; and
- surface-active agents.

The chemical has reported domestic use in the SPIN database. However, it should be noted that SPIN does not distinguish between direct use of the chemical or using the materials that are produced from chemical reactions involving the chemical. The US Household Products Database states a concentration of up to 25 % (liquid) for use in arts and crafts, a concentration of up to 60 % for home maintenance use, and a concentration of up to 95 % for use as auto products and inside the home, including aerosolised and liquid cleaning products.

The chemical has reported commercial use including:

- as a process solvent; coating and dye solvent; cleaning and drying agent; and an aerosol solvent;
- in absorbents and adsorbents;
- in anti-freezing and anti-adhesive agents;
- in conductive agents;
- in construction materials and in cutting agents;
- in dust binding agents;
- in electromechanical components;
- in fuels & fuel additives;
- in lubricants and additives;
- in impregnation materials;
- as a flux agent for casting or joining materials;
- in process regulators, softeners and fixing agents;
- in reprographic, flotation and foaming agents;

- in photo chemicals and pH-regulation agents;
- as a viscosity adjustor and anti-static agent;
- in tanning agents; and
- in welding and soldering agents.

The chemical has reported site-limited use including:

- as a chemical intermediate to produce acetone, methyl isobutyl ketone, methyl isobutyl carbinol, isopropylamine, and isopropyl acetate;
- in laboratory chemicals;
- in heat transferring agents;
- as electroplating agents;
- as a complexing and flocculating agent;
- as a vulcanising agent; and
- in stabilisers.

The chemical has reported non-industrial use including:

- in pesticides;
- in food/feedstuff flavourings and nutrients;
- as a preservative for pathological specimens, for dehydrating tissues, and in icepacks; and
- as a solvent in pharmaceuticals products (medical and veterinary) such as rubbing alcohols, antiseptic and local anaesthetic (e.g. tincture of iodine, bathing solutions for surgical sutures and dressing), skin soothers, veterinary pink eye, wound and dehorning sprays, house and garden type insecticides.

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

Xi; R36 (Irritation)

R67 (Vapours may cause drowsiness and dizziness)

Exposure Standards

Australian

The chemical has an exposure standard of 983 mg/m³ (400 ppm) time weighted average (TWA) and 1230 mg/m³ (500 ppm) short-term exposure limit (STEL).

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit (TWA) of 245–999 mg/m³ (100–400 ppm) in countries such as Canada, Denmark, Iceland, Germany, Norway, Sweden, Spain, Switzerland, UK, and USA.

An exposure limit (STEL) of 600–1250 mg/m³ (250–500 ppm) in countries such as Canada, France, Spain, Sweden, Switzerland, UK, and USA.

Health Hazard Information

Toxicokinetics

The chemical is readily absorbed and distributed throughout the body in animals and humans following ingestion, inhalation, and dermal application. The chemical was recovered from the tissues of dogs half an hour after gastrointestinal tract injection, and in rats three hours after a single oral administration.

The chemical is metabolised to acetone predominantly by the enzyme alcohol dehydrogenase in both animals and humans. Dose-related increases in blood levels of the chemical and acetone were reported following oral and inhalation exposure in rats. The data available indicate saturation of the oxidative metabolic pathway at levels above approximately 4000 ppm (9840 mg/m³). A minor metabolic pathway is the conjugation of the chemical by glucuronic acid and the conjugate has been detected in the urine in animals and humans.

The majority of the absorbed chemical is exhaled as acetone, carbon dioxide and unmetabolised chemical, with smaller amounts excreted in the urine and less again in the faeces. There were no main differences in the rates of excretion between sexes or administration routes. Although limited details were available, the chemical has been reported to be excreted in the gastric juice and saliva in the dog and through breast milk in the rat. Although details were not provided, elimination half-lives of 2.5–3 hours and 6.4 hours in blood of humans have been reported in two studies following ingestion of the chemical (WHO, 1990a; WHO, 1990b; REACH).

Acute Toxicity

Oral

The chemical was of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) in rats is greater than 2000 mg/kg bw. Observed effects included irritation and respiratory arrest while under narcosis (OECD, 2002; WHO, 1990a; HSDB).

Dermal

The chemical was of low acute toxicity in an animal test following dermal exposure. The median lethal dose (LD50) in rats is greater than 2000/kg mg/kg bw. Observed effects were not reported (OECD, 2002; WHO, 1990a; HSDB).

Inhalation

The chemical was of low acute toxicity in animal tests following inhalation exposure with reported median lethal concentrations (LC50) >20 mg/L in rats (OECD, 2002; HSDB). Observed effects included severe irritation of the mucous membranes and central nervous system depression as indicated by ataxia, prostration and narcosis.

The chemical is currently classified with the risk phrase 'Vapours may cause drowsiness and dizziness (R67)' in Australia (Safe Work Australia—HSIS). The available data in animals and humans (see **Acute toxicity: Observation in humans**) support this classification (OECD, 2002; WHO, 1990a; WHO, 1990b; REACH).

In an acute inhalation toxicity study (OECD TG 403), Fischer 344 (F344) rats were exposed (whole-body exposure) to the chemical at 500, 1500, 5000, and 10000 ppm for six hours (instead of the standard four hours). Transient concentration-related narcosis and/or central nervous system sedation was noted in the study and the motor activity was decreased at 1500 ppm (males only), 5000 ppm (both sexes). Severe central nervous system depression was seen in the 10000 ppm group. After one and six hours exposure at 10000 ppm, prostration, severe ataxia, decreased arousal, slowed or laboured respiration, decreased neuromuscular tone, hypothermia, and loss of reflex function was observed (OECD, 2002; REACH).

Observation in humans

Acute intoxication incidents in humans with the chemical have been reported (WHO, 1990b; OCED, 2002; HSDB).

Ingestion and inhalation are the common routes of poisoning in humans. Acute intoxication of the chemical has a rapid onset (30–60 minutes) following ingestion, and reported symptoms included drowsiness, poor coordination, abdominal pain, cramps, nausea, vomiting and diarrhoea, with unconsciousness and death following massive exposure. Inhaling high concentrations of the chemical can cause nausea, headache, light headedness, drowsiness, ataxia and deep narcosis (WHO, 1990b; OECD, 2002; HSDB).

The odour threshold for the chemical has been reported to be quite low at 22 ppm (OECD, 2002). Significant absorption of the chemical has been reported from sponging children with the chemical for fever control. The chemical has also been reported to be twice as toxic as ethanol, with respect to its acute potency to depress central nervous system (HSDB). Specific cases of poisoning include: a two year old boy who ingested a small amount of the chemical at 70 % concentration (rubbing alcohol); an 18 months old child who was wrapped in towels soaked with the chemical at 70 % concentration (rubbing alcohol) for four hours to control a fever due to otitis media (middle ear infection); a 55 year old woman with massive skin lesions who was bathed with approximately one litre of the chemical at 70 % concentration, possibly absorbed through the skin and by inhalation (WHO, 1990b).

Corrosion / Irritation

Skin Irritation

The chemical was reported not to be a skin irritant.

In skin irritation studies, irritation was not observed following patch application (occlusive) of undiluted chemical for four hours to intact and abraded skin of rabbits and guinea pigs (OECD, 2002; WHO, 1990a; REACH).

Eye Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia). The available data support this classification (OECD, 2002; WHO, 1990a; REACH).

In an eye irritation study (OECD TG 405), the undiluted chemical was applied to the conjunctival sac of three male and three female New Zealand White rabbits. While conjunctival responses included redness, chemosis (oedema of the conjunctiva), and clear/white discharge, corneal opacity, stippling and corneal ulceration were also noted. Reported average scores were: corneal opacity (1.89), iris lesion (0.78), redness of the conjunctivae (2.95), and chemosis (2.0). While the incidence of redness was noted in all six animals at the day 10 following application, redness was still present in three animals at the day 14 observation. The incidence of chemosis was noted in only one animal at the day 10 observation and was not present in any animal at the day 14 observation. Given the observation period did not extend to 21 days, it is difficult to conclude on the reversibility of the conjunctival redness. All corneal and iris responses were cleared by the day seven and 10 observations, respectively.

Other reported eye irritation studies also concluded that the chemical is irritating to rabbits' eyes (OECD, 2002; WHO, 1990a; REACH).

Observation in humans

Mild irritation of the eyes, nose and throat of humans has been reported following exposure to the chemical vapours (400 ppm) for 3–5 minutes (WHO, 1990b; OECD, 2002; HSDB). Even though the chemical has not been reported to be a skin irritant in humans, a prolonged (four-hour) contact with the skin could lead to a significant absorption resulting in central nervous system effects. Although the chemical has been reported to be mildly irritating to the eyes at 400 ppm concentration, severe irritation and even corneal abrasion has resulted following direct contact with the liquid chemical (WHO, 1990b; HSDB; REACH).

Sensitisation

Skin Sensitisation

No human or animal data are available. The chemical does not contain a structural alert for skin sensitisation (OECD Toolbox).

Repeated Dose Toxicity

Oral

Considering the lowest observed adverse effect levels (LOAELs) available from a 12-week rat study (1390 mg/kg bw/day), and based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure.

Male Wistar rats were administered the chemical at concentrations of 0, 1, 2, 3, or 5 % (0, 870, 1390, 1700, or 2500 mg/kg bw/day) in drinking water for 12 weeks. The top dose was reduced to 4 % due to unpalatability after two weeks. Significantly decreased bodyweights were seen at the two highest doses and dose-related increases in relative liver and kidney weights were also significant at 1390 mg/kg bw/day and above. Relative adrenal weights were also significantly increased at the two highest doses; increased testis weight was noted only at the top dose. A dose-dependent increase of hyaline casts and hyaline droplet formation in the proximal tubules of the kidneys was also noted. The no observed adverse effect level (NOAEL) was determined to be 870 mg/kg bw/day, based on liver and kidney effects observed at the LOAEL of 1390 mg/kg bw/day (OECD, 2002; EFSA, 2005).

In another repeated dose study, rats (strain not specified) were administered the chemical in drinking water at doses of 600 or 2300 mg/kg bw/day for males and 1000 or 3900 mg/kg bw/day for females for 27 weeks. Male rats showed decreased bodyweight gain during the first 13 weeks and increased bodyweight gain for the remainder of the treatment. Female rats showed decreased bodyweight gain throughout the dosing period. No other effects were reported. The NOAELs were 2300 and 1000 mg/kg bw/day for males and females, respectively. The LOAEL in females was 3900 mg/kg bw/day but could not be established in males (OECD, 2002).

Dermal

No data are available.

Inhalation

Several repeated dose inhalation studies were available in rats and mice. Considering the no observed adverse effect concentrations (NOAECs) available from these studies (500 ppm), and based on the treatment-related effects reported, the chemical is not considered to cause serious damage to health from repeated inhalation exposure.

The kidney appears to be the target organ with kidney lesions and changes in urine chemistry indicative of impaired kidney function observed at doses ≥2500 ppm in animals exposed to the chemical for ≥78 weeks (effects not observed in 13-week studies). Transient signs of narcosis were observed for both mice and rats at doses ≥1500 ppm (OECD, 2002; REACH; US EPA, 1986).

Observation in humans

Although limited information is available, it has been reported that oral intake of low doses of the chemical (2.6 or 6.4 mg/kg bw/day) by groups of eight men for six weeks had no effect on their blood cells, serum or urine and also produced no clinical symptoms (HSDB).

Genotoxicity

Based on the available information, the chemical is not considered to have mutagenic or genotoxic potential as reported below. All reported in vitro and in vivo genotoxicity assays for the chemical have been negative (OECD, 2002; WHO,1990a; EFSA, 2005; REACH).

The chemical did not induce gene mutations in vitro (with or without metabolic activation) in Ames assays, sister chromatid exchange assay using cultured Chinese hamster V79 fibroblasts, and hypoxanthine-guanine phosphoribosyltransferase (HGPRT) assay using Chinese hamster ovary cells. The chemical was also negative in vitro for aneuploidy in *Neurospora crassa* and did not induce cell transformation in Syrian hamster embryos infected with Simian SA7 virus. The chemical did not increase micronuclei in an in vivo micronuclei assay in mice at levels up to 2500 mg/kg.

Carcinogenicity

Based on available data, the chemical is not considered to be carcinogenic (OECD, 2002; WHO, 1990a; EFSA, 2005; REACH).

The International Agency for Research on Cancer (IARC) has concluded that there is inadequate evidence for the carcinogenicity of isopropanol in laboratory animals and humans, placing the chemical in Group 3 (Not classifiable as to its carcinogenicity to humans) (IARC, 1999). Although there are no carcinogenicity studies available for the chemical by oral exposure, studies are available for inhalation exposure in rats and mice.

In a carcinogenicity study (OECD TG 451), F344 rats were exposed (whole-body) through inhalation to vapours of the chemical at concentrations of 0, 500, 2500, and 5000 ppm for six hours a day, five days a week for two years. The only neoplastic lesion found was stated to be increased frequency of interstitial (Leydig) cell adenoma of the testis (77.3, 86.7 and 94.7 % at low, mid and top dose groups, respectively). The authors did not consider the tumours to be treatment related as testicular adenomas are a common finding in aged male rats and that incidence of this spontaneous tumour reported for the control group (64.9 %) of this study was lower than the historical incidence (88 %) of control F344 rats of numerous two-year National Toxicology Program (NTP) carcinogenicity studies. In a similar carcinogenicity study, CD-1 mice were also exposed (whole-body) through inhaltion to vapours of the chemical at concentrations of 0, 500, 2500, and 5000 ppm for six hours a day, five days a week for 18 months. No increased frequency of neoplastic changes was reported in any of the treated groups (OECD, 2002; EFSA, 2005; REACH).

Reproductive and Developmental Toxicity

The chemical does not show specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity.

Several one or two-generation reproductive toxicity studies (rats) and developmental studies (rats and rabbits) were available. Other than a statistically significant reduction in the male mating index observed in a recent two generation study (high dose, 1000 mg//kg bw/day second generation males), there were no other effects on reproductive indices, including fertility and gestational indices and histopathology of the reproductive organs. The NOAELs for reproductive toxicity were reported as ≥500 mg/kg bw/day (OECD, 2002; EFSA, 2005; REACH). Developmental effects, including a reduction in postnatal survival and decreased foetal bodyweights, occurred only at maternally toxic doses. No accompanying malformations were observed.

In a developmental toxicity study (US EPA TSCA Guidelines), pregnant Sprague Dawley (SD) rats were administered the chemical by gavage at 0, 400, 800 or 1200 mg /kg bw/day on gestational days 6–15. In the same study, pregnant New Zealand white rabbits were dosed orally with the chemical at 0, 120, 240 or 480 mg/kg bw/day during gestational days 6–18. There was no evidence of developmental toxicity in rats and rabbits at any tested dose. There was mortality of two dams (8%) at 1200 mg/kg and one dam (4%) at 800 mg/kg. Reduced maternal gestational weight gain associated with significantly reduced gravid uterine weights was noted in the higher dose group. The NOAEL for maternal toxicity in rats was reported to be 400 mg/kg bw/day. The NOAEL for developmental toxicity in rats was established as 400 mg /kg bw/day, based on significantly reduced foetal litter body weights at the 800 and 1200 mg/kg dose levels. The NOAEL for maternal toxicity in rabbits was determined to be 240 mg/kg bw/day, based on decreased maternal bodyweight and profound clinical signs (peripheral vasodilatation, cyanosis, lethargy, laboured respiration) of toxicity seen at the top dose. There was no evidence of any developmental toxicity and the NOAEL for developmental toxicity was established as the highest dose: 480 mg/kg bw/day. There was no evidence of any teratogenicity in either studies in rats and rabbits (US EPA, 1995; OECD, 2002; EFSA, 2005; HSDB; REACH).

Other Health Effects

Neurotoxicity

Based on the available neurotoxicity studies on adult rats, inhaling vapours of the chemical produces reversible central nervous system depression. Increased motor activity was seen in adult rats exposed to high doses of the chemical.

In a neurotoxicity study, the chemical was administered to F344 rats via inhalation at doses of 0, 500, 1500, 5000, and 10000 ppm for six hours. The exposure of rats to the chemical resulted in transient, concentration-related narcosis and/or CNS sedation. A concentration-dependent decrease in motor activity was observed for males exposed to >1500 ppm and for females exposed to >5000 ppm. While a NOEL of 1500 ppm was established for clinical signs of intoxication (narcosis and neurobehavioural function) at 5000 ppm, a NOEL for neurobehavioural effects was determined to be 500 ppm as motor activity decreased at 1500 ppm in males (OECD, 2002; REACH).

In a study to examine the effect of the chemical on developmental neurotoxicity (US EPA TSCA Guidelines), SD rats were administered the chemical at doses of 0, 200, 700 or 1200 mg/kg bw/day from gestational day six through to postnatal day 21. The treatment did not have any effect on motor activity, weights of the four regions of the brain, developmental landmarks, or morphological changes to the tissues of the central nervous tissue. The NOAEL for developmental neurotoxicity was established as 1200 mg/kg bw/day (OECD, 2002; EFSA, 2005; REACH).

In another neurotoxicity study, F344 rats were administered the chemical via inhalation at doses of 0, 100, 500, 1500, and 5000 ppm, six hours a day, five days a week, for 13 weeks. Treatment-related effects on functional observation battery or neuropathologic lesions in the central or peripheral nervous systems were not observed. The narcotic effects of the chemical were noted only during exposures at the top two doses and were typically absent following exposures. Swollen periocular tissue, perinasal encrustation, and ataxia were observed following exposure for rats of the 5000 ppm group. Increased motor activity was noticed in females of the top dose group at 9 and 13 weeks. A NOAEC of 1500 ppm was established in this study for neurotoxicity (OECD, 2002; US EPA, 1995). In another study, female rats exposed to the chemical at 5000 ppm for 13 weeks also showed increased motor activity (US EPA, 1995).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include the potential for eye irritation and intoxication symptoms following inhalation of high vapour concentrations.

Public Risk Characterisation

Considering the range of domestic and cosmetic products that may contain this chemical, the main route of public exposure is expected to be through the skin and eyes, and inhalation from products applied as cosmetics and from using domestic products.

The use of the chemical in cosmetics is stated to be up to a concentration of 10 % (see **Import, manufacture and use**). Eye irritation and inhalation toxicity is not expected from exposure to these concentrations of the chemical. Even though a much higher concentration of the chemical has been stated to be used for domestic uses (up to 95 %), provided that normal precautions are taken to avoid eye contact and inhaling chemical vapours, the risk from the use of domestic products is not considered to be unreasonable. Spray application of products containing the chemical may result in reversible eye irritation, although the likelihood is low. The effects are likely to be slight and reversible.

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, skin, eye, 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 equipment cleaning and maintenance. Worker exposure to the chemical at lower concentrations may also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical health effects, the chemical may pose an unreasonable risk to workers unless adequate control measures to minimise skin, eye, 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.

Work Health and Safety

The chemical is recommended for classification and labelling under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical hazards and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Vapours may cause drowsiness and dizziness (R67)*	May cause drowsiness or dizziness - Specific target organ tox, single exp Cat. 3 (H336)
Irritation / Corrosivity	Irritating to eyes (Xi; R36)*	Causes serious eye irritation - Cat. 2A (H319)

[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 eye 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 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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