Benzenemethanol: Human health tier II assessment

01 July 2016

CAS Number: 100-51-6

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.

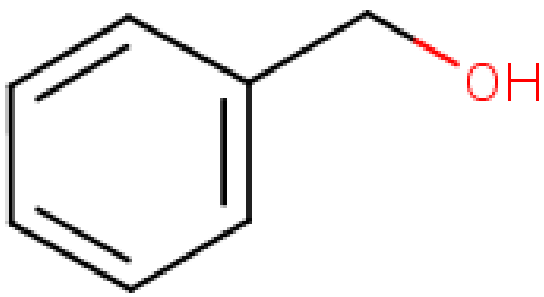
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	benzyl alcohol phenylmethanol phenylcarbinol benzenecarbinol hydroxytoluene
Structural Formula	
Molecular Formula	C7H8O
Molecular Weight (g/mol)	108.14
Appearance and Odour (where available)	Colourless liquid with a faint aromatic odour.
SMILES	c1(CO)ccccc1

Import, Manufacture and Use

Australian

No specific Australian use, import, or manufacturing information has been identified.

The chemical is listed on the 2006 High Volume Industrial Chemicals List (HVICL) with a total reported volume of 10000–99999 tonnes.

International

The following international uses have been identified through:

- the European Union (EU) Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) dossier;
- the Organisation for Economic Co-operation and Development Screening information data set International Assessment Report (OECD SIAR);
- Galleria Chemica;
- the Substances and Preparations in Nordic countries (SPIN) database;
- the European Commission Cosmetic Ingredients and Substances (CosIng) database;
- the United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary; and
- the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic uses including as:

- a fragrance ingredient;
- a solvent and preservative;
- a viscosity decreasing agent; and
- an oral health care agent.

The chemical has reported cosmetic uses in the United States of America (USA), with identified use in 2042 products (Personal Care Products Council, 2011).

The chemical has reported domestic uses including in:

- adhesives, binding agents;
- bleaching agents;
- cleaning/washing agents;
- colouring agents;
- corrosion inhibitors;
- fillers;
- odour agents;

- surface treatments;
- surface-active agents; and
- paints, lacquers and varnishes.

The US Household Products Database states a concentration of up to: 30 % (paste) and 20 % (fluid) for home maintenance use; 10 % (paste) for inside the home use; and 1.5 % (cream, liquid and wipes) for personal care uses. One personal care product (Aveeno Moisturizing Shower and Bath Oil, liquid form) contains the chemical up to 5 % and another personal care product (Old Spice cologne, liquid form) contains the chemical up to 30 % (US Household Products Database).

The chemical has reported commercial uses including:

- in absorbents and adsorbents;
- in anti-freezing and anti-static agents;
- in construction and impregnation materials;
- as a fixing agent;
- in lubricants and additives;
- as a solvent and viscosity adjustor;
- in welding and soldering agents;
- in reprographic agents; and
- in photo chemicals and process regulators.

The chemical has reported site-limited uses including as intermediates.

The following non-industrial uses have been identified internationally:

- in food/feedstuff flavourings and nutrients;
- in non-agricultural pesticides and preservatives; and
- in pharmaceuticals.

Restrictions

Australian

No known restrictions have been identified.

International

The chemical is listed on the following (Galleria Chemica):

- EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products—Annex III—List of Substances which cosmetic products must not contain except subject to the restrictions laid down;
- New Zealand Cosmetic Products Group Standard—Schedule 5—Table 1: Components cosmetic products must not contain except subject to the restrictions and conditions laid down;

- The Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex III—Part 1 List of substances which cosmetic products must not contain except subject to restrictions and conditions laid down;
- EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products—Annex V List of preservatives allowed in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 7: Preservatives cosmetic products may contain with restrictions—Table 1: List of preservatives allowed; and
- ASEAN Cosmetic Directive Annex VI—Part 1—List of preservatives allowed for use in cosmetic products.

The US Cosmetic Ingredient Review (CIR) Expert Panel concluded that the chemical is safe for use in cosmetic formulations at concentrations up to 5 %. The chemical is also safe for use in hair dyes at concentrations up to 10 %. The available data are sufficient to support the safety of these ingredients in cosmetic products in which a primary route of exposure is inhalation (CIR, 2001).

Restrictions for the chemical for certain types of cosmetic products, according to Annex III of the REACH Regulations (List of restricted substances), are that they are not to be used. The presence of the substance must be indicated in the list of ingredients referred to in Article 19(1)(g) when its concentration exceeds:

- 0.001 % in leave-on products; and
- 0.01 % in rinse-off products.

Restriction for the chemical for certain types of cosmetic products, according to Annex V of the REACH Regulations (List of preservatives allowed in cosmetic products) is that a maximum concentration in ready-for-use preparations is 1 %.

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):

- Xn; R20/22 (acute toxicity)

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available

Health Hazard Information

Toxicokinetics

The chemical is readily and totally absorbed from the alimentary tract in humans following an oral application of 1.5 g; the chemical is metabolised within six hours and about 75–85 % is excreted in the urine. The absorbed chemical is readily oxidized to benzaldehyde as an intermediate, and then to benzoic acid, which is conjugated with glycine to hippuric acid and excreted via the kidneys. Higher doses of the chemical result in excretion as a glucuronide conjugate (NTP, 1989; SCF, 2002; REACH).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase 'Harmful if swallowed' (Xn; R22) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). The available data (median lethal dose (LD50) is 1620 mg/kg bw) support this classification.

Reported signs of toxicity were noted at all doses and included sedation, side and prone-position, bloody eyes and a reduced general condition. The noted mortality during the study was 1/10, 2/10, 6/10, 8/10 and 10/10 animals at 1254, 1463, 1467, 2090 and 2195 mg/kg bw, respectively (REACH). Similar conclusions have also been reported in other studies (NTP, 1989; WHO, 2001; SCF, 2002).

Dermal

Although no data are available for the chemical, chemicals in this category demonstrated low acute toxicity based on results from animal tests following dermal exposure. The reported LD50 in rats is >2000 mg/kg bw (WHO, 2001).

Inhalation

The chemical is classified as hazardous with the risk phrase 'Harmful by inhalation' (Xn; R20) in the HSIS (Safe Work Australia). While the available data do not support the classification, in the absence of more comprehensive information, amendment of the current HSIS classification is not warranted.

In an acute inhalation toxicity study, Wistar rats (five/dose/sex) were subjected to a single four-hour head/nose-only exposure to aerosol concentrations of the chemical at 3297 and 4178 mg/m^3 and observed for 14 days post exposure. The concentration of 4178 mg/m^3 was proven to be the maximum technically achievable concentration.

At 4178 mg/m^3, transient clinical signs (piloerection, slight bradypnoea; recovery within one day) were seen as causally related to the slight irritant effect of the chemical to the upper respiratory tract. There was no indication of any lasting respiratory damage in this study. No mortality was noted in the study. The median lethal concentration (LC50) over four hours was concluded to be >4178 mg/m^3 for male and female rats (REACH).

Observation in humans

There have been reports of premature infants, who had received benzyl alcohol in medications administered intravenously, suffering serious effects including central nervous system dysfunction, coma and death. Death occurred between 6–46 days in infants who had received 99–234 mg/kg bw of benzyl alcohol, while a matched control group of infants did not develop the syndrome, had received doses of 27–99 mg/kg bw of benzyl alcohol (SCF, 2002).

Corrosion / Irritation

Skin Irritation

The chemical is reported to slightly irritate the skin in animal studies. The effects were not sufficient to warrant hazard classification.

In a skin irritation study, the chemical (0.5 mL) was applied to the dorsal area of the trunk of three New Zealand White rabbits for four hours under observation for seven days. Slight irritation, which fully resolved within 72 hours, was seen in one animal (WHO, 2001; REACH).

Eye Irritation

Although the chemical produced only slight eye irritation in one animal study where the eye was washed out after application, the eye irritation effects reported in a second eye irritation study without washing after application support the chemical being classified (refer to **Recommendation** section).

In an eye irritation study, the chemical (0.1 mL) was applied on the conjunctival sac of one eye of each of three New Zealand White rabbits, with observation at 24, 48 and 72 hours after the administration and then daily until the ocular reactions were reversed. The average scores following grading at 24, 48 and 72 hours for three rabbits were 2 for corneal opacity, 1 for iritis, 2.67 for conjunctivitis, and 2.23 for chemosis. The average scores for all the end points are sufficient to support classification (REACH).

In an eye irritation study, the chemical (0.1 mL) was applied on the conjunctival sac of one eye of each of three New Zealand White rabbits. The treated eyes were washed 24 hours after instillation of the chemical. Irritation responses were observed at 24, 48 and 72 hours and at 14 and 21 days following application. The average scores following grading at 24, 48 and 72 hours for three rabbits were 1 for corneal opacity, 0.3 for iritis, 2 for conjunctivitis, and 0.7 for chemosis (REACH).

Sensitisation

Skin Sensitisation

While the chemical gave both positive and negative responses in animal studies, the maximisation test conducted, which was similar to the current test guidelines, was negative. The information presented here in humans also indicated that the chemical is unlikely to be a skin sensitiser (see **Sensitisation: observation in humans**). No sensitisation has been reported with the chemical among workers who used it over several decades. Therefore, the weight of evidence approach would indicate that the chemical is not a skin sensitiser in humans (WHO, 2001; REACH).

In a guinea pig skin sensitisation test, Himalayan guinea pigs (10/dose) were induced (intradermally) with doses of 0.05 mL of the undiluted chemical mixed with the same volume of Freund's complete adjuvant (FCA) on days 0, 2, 4, 7 and 9. The control animals were similarly treated with 0.05 mL of FCA alone. All the animals were challenged epicutaneously on days 21 and 35 in a patch test with a non-irritant concentration of the chemical. The chemical yielded a positive result for skin sensitisation.

In another guinea pig skin sensitisation test, Himalayan guinea pigs (4–6/group) were induced (epicutaneously) with 0.1 mL of undiluted chemical up to several diluted concentrations (up to 3 %) daily for 21 days. Animals were challenged epicutaneously on days 21 and 35, with the lowest irritant and a non-irritant concentration. Evaluations were performed at 42, 48, and 72 hours after the last challenged dose. The chemical tested positive for skin sensitisation in this test.

In an old skin sensitisation test (Draize test), Himalayan guinea pigs were induced (intradermally) with a dose of 0.05 mL of a 0.1 % solution of the chemical in isotonic saline. Further doses of 0.1 mL each were injected on alternate days for the following nine days. The treated animals and the untreated controls were challenged intradermally with 0.05 mL of a 0.1 % solution on days 35 and 49. The evaluation criterion was the mean diameter of the papular reactions. The guinea pigs showed no reaction in this test for skin sensitisation.

In a guinea pig maximisation test, Himalayan guinea pigs (10/dose) were induced (intradermally) with: 0.1 mL of a 5 % solution of the chemical, also with 0.1 mL of a 5 % emulsion of the chemical in FCA; each injection being given twice. In addition, 250 mg of the compound dissolved in petrolatum at a concentration of 25 %, which caused mild to moderate skin irritation under occlusion, was applied on day eight to the clipped skin area of the neck and was kept under occlusive bandage for two days

(total dose 20 mg intradermally plus 250 mg epicutaneously). On day 21, the chemical at a subirritant concentration of 25 % in petrolatum was applied to the flank epicutaneously for 24 hours. The reactions were read at 24 and 48 hours after removing the patch. The chemical tested negative for skin sensitisation.

Observation in humans

A repeat-insult patch test (HRIPT) was reported to be negative in 110 patients. In this study, patients were tested with two formulations containing 0.65 % of the chemical. A negative reaction was also reported for a 10 % concentration of the chemical in petrolatum using 25 male volunteers in a human maximisation study.

Three cutaneous reactions to 5 % of the chemical in petrolatum were noted among 713 cosmetic dermatitis patients over a period of six years from results compiled by 12 dermatologists. In another study, four positive patch tests to a 6.5 % concentration of the chemical were reported among 242 patients with histories of contact allergy of varying origin (CIR, 2001; REACH).

Repeated Dose Toxicity

Oral

The chemical is not considered to cause serious damage to health from repeated oral exposure. A no observed adverse effect level (NOAEL) of ≥200 mg/kg bw/day for mice and ≥400 mg/kg bw/day for rats can be established for the chemical from long-term studies.

In a repeated dose toxicity study, the chemical was administered (gavage) in corn oil to groups of B6C3F1 mice (10/sex/dose) and Fischer 344/N (F344/N) rats (10/sex/dose) at doses of 0, 50, 100, 200, 400, or 800 mg/kg bw/day, five days/week for 13 weeks. Eight out of ten male rats dosed with 800 mg/kg bw/day died during weeks seven and eight; four of these mortalities were related to gavage. While mortalities were scattered among all dose levels in mice, all mortalities (four males and six females), apart from one, were related to administration of the chemical through gavage. Clinical signs of neurotoxicity, including staggering, respiratory difficulty, and lethargy, were noted in rats rats dosed with 800 mg/kg bw/day. Haemorrhages around the mouth and nose were noted and there were pathological lesions in the brain, thymus, skeletal muscle, and kidney. Mice dosed with 800 mg/kg bw/day also showed staggering after dosing during the first two weeks of the study.

In the same study, reduced relative weight gain was noted in male rats dosed with 800 mg/kg bw/day; female rats dosed with 200 mg/kg bw/day or more; male mice dosed with 400 or 800 mg/kg bw/day; and female mice dosed with 200 mg/kg bw/day or more. Notable changes in body weight gain or chemically-related histopathologic lesions were not observed in rats or mice from the lower dose groups. Some of the deaths in the rats and mice could have been caused by a combination of the gavage procedure and chemical toxicity, since there was evidence that benzyl alcohol induced neurotoxic effects. An NOAEL of 200 mg/kg bw/day was established for mice, based on clinical signs and reduced body weight development in males and females. An NOAEL of 400 mg/kg bw/day was established for rats, based on clinical signs and reduced body weight development in males and females and histopathological changes in the brain at 800 mg/kg bw (NTP, 1989; REACH).

In a chronic toxicity/carcinogenicity study, the chemical was administered to groups of B6C3F1 mice (50/sex/dose) and F344/N rats (50/sex/dose) at a dose of 0, 100, or 200 mg/kg bw/day, and 0, 200, or 400 mg/kg bw/day, respectively, in corn oil on five days a week for 103 weeks (see **Carcinogenicity**). There were no effects on body weight gain and no chemically-related clinical signs were observed throughout the study in either mice or rats. Survival in both dose groups of female rats was reduced to 50 % compared with vehicle controls, primarily due to an increased number of gavage-related deaths. Gross necropsy and histopathology revealed no apparent chemically-related non-neoplastic responses in mice and rats. Therefore, an NOAEL of 200 and 400 mg/kg bw/day, respectively, in mice and rats was established from this study (NTP, 1989).

Dermal

No data are available.

Inhalation

Limited information indicates that the chemical is not likely to cause serious damage to health from repeated inhalation exposure.

In a repeated dose inhalation study, the aerosolised chemical was administered via the nose for six hours/day on five days/week for four weeks (a minimum of 20 exposures/animal) to Sprague Dawley (SD) rats (10/sex/dose) at target exposure concentrations (mean analytical concentrations) of 30 (41), 100 (102), 300 (290), and 1000 (1072) mg/m^3. A concurrent control group was exposed to filtered air on a comparable regimen. All animals were euthanised on the day following the last exposure. There were no clinical test-substance related effects on mortality, body weight or body gain, food consumption, ophthalmoscopic examination, haematology, clinical chemistry, organ weight, gross pathology, or histopathology. As an adverse effect was not noted at any concentration, no observed adverse effect concentration (NOAEC) was considered to be 1072 mg/m^3 (REACH).

Genotoxicity

The weight of the evidence of the in vitro and in vivo genotoxicity data indicates that the chemical does not have mutagenic or clastogenic potential (NTP, 1989; SCF, 2002; REACH).

The chemical was reported to be negative in several Ames tests, with and without metabolic activation, and also negative in an in vitro mammalian chromosome aberration test using Chinese hamster lung (CHL) cells without metabolic activation.

The chemical produced a significant increase in chromosomal aberrations with Chinese hamster ovary (CHO) cells in the presence, but not the absence, of S9. The chemical also produced an increase in sister chromatid exchanges (SCEs) in CHO cells in a sister chromatid exchange assay in mammalian cells, which was judged to be equivocal both with and without metabolic activation. While the chemical produced a positive response in mammalian cells in the mouse lymphoma thymidine kinase forward mutation assay without metabolic activation, negative results were reported for tests with metabolic activation.

The chemical was found to be negative in the in vivo micronucleus assay on mouse bone marrow cells, in an assay on replicative DNA synthesis in rats, and in the sex-linked recessive lethal assay in *Drosophila melanogaster*.

Carcinogenicity

The available information indicates that the chemical is not likely to have carcinogenic potential.

In a chronic toxicity/carcinogenicity study, the chemical was administered to groups of B6C3F1 mice (50/sex/dose) and F344/N rats (50/sex/dose) at a dose of 0, 100, or 200 mg/kg bw/day, and 0, 200, or 400 mg/kg bw/day, respectively, in corn oil for five days a week for 103 weeks (see **Repeat dose toxicity: oral**). Dose-related negative trends were noted in the incidences of anterior pituitary gland neoplasms in female rats (control 29/50; low dose 17/47; high dose 9/49) and of Harderian gland adenomas in male mice (8/50; 3/50; 2/50). Increased incidence of hyperplasia of the forestomach epithelium was seen (not statistically significant) in male rats (0/48; 0/19, 4/50). The increased incidence of adenomas of the adrenal cortex noted in the high-dose male mice (0/48; 0/44; 3/48) was within the historical range and not considered compound-related. The study concluded that the chemical showed no evidence of carcinogenic activity in male or female F344/N rats dosed with 200 or 400 mg/kg, or male or female B6C3F1 mice dosed with 100 or 200 mg/kg for two years (NTP, 1989).

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.

In a developmental toxicity study, 50 CD-1 mice were administered (gavage) the chemical at 550 mg/kg bw/day on gestation days (GD) 6–15. A further 50 mice received the corn oil vehicle only. No clinical signs were reported in the treatment group and maternal body weight and body-weight gain during treatment and up to day three days post-partum were not affected. Mortality was not significantly increased in treated animals compared with the control group. All other parameters examined, including the

gestation index, average number of live pups per litter, and postnatal survival and pup body weight on days zero and three post-partum were not significantly different from the control values. As no significant effects were noted in the study, the NOAEL was determined to be <550 mg/kg bw/day (CIR, 2001: OECD, 2001; REACH).

In another developmental toxicity study, 50 CD-1 mice were administered (gavage) the chemical at 750 mg/kg bw/day on GD 7–14. A control group of 50 animals received distilled water only. Clinical signs of toxicity were reported in up to 20 mice, and included hunched posture, tremors, inactivity, prostration, hypothermia, ataxia, dyspnoea, swollen or cyanotic abdomen, and piloerection. There were 19 deaths attributed to the treatment during the study. Maternal body weights were statistically significant as they reduced on day 18 of gestation and on day three post-partum. Significant reductions in pup body weight were reported on days one and three post-partum, including a lower mean pup weight per litter, mean litter weight change between day one and day three post-partum, and mean pup weight change between days one and three post-partum. No differences in pup survival were observed by day three post-partum. As effects were seen on dams and foetuses at the only dose used, the lowest observed adverse effect level (LOAEL) was 750 mg/kg bw/day for both maternal and foetal effects (CIR, 2001: WHO, 2001; REACH).

Risk Characterisation

Critical Health Effects

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

The chemical is also irritating to the eyes.

Public Risk Characterisation

Although the use in cosmetic and domestic products in Australia is not known, the chemical has reported cosmetic and domestic uses overseas (see **Import, manufacture and use**). Considering the range of domestic and cosmetic products that could contain this chemical, the main route of public exposure is expected to be through the skin and eyes, inhalation from products applied as cosmetics, and from using domestic products.

While the chemical has various uses (as a fragrance ingredient, solvent, preservative, viscosity decreasing agent and oral health care drug), the chemical is not expected to be present in sufficiently high concentrations in these products to lead to toxic effects. The use of the chemical in personal care products is stated to be up to a concentration of 5 % as cream, liquid and wipes. A single personal care product reportedly has a much higher concentration of the chemical (up to 30 %) (see **Import, manufacture and use**), but this product is not expected to be used at high volumes on large skin areas.

It has been concluded that the chemical is safe for use in cosmetic formulations at concentrations up to 5 %, and in hair dyes at concentrations up to 10 % (CIR, 2001). Although a much higher concentration of chemical has been reported for a single personal care product (up to 30 %) and for home maintenance use (up to 30 %) (see **Import, manufacture and use)**, 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.

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, ocular and inhalation exposure might occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

Given the critical systemic acute local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise ocular and inhalation exposure are implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU) at a workplace (such as an employer) has adequate information to determine the appropriate controls.

The data available support an amendment to the hazard classification in the HSIS (Safe Work Australia) (refer to **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 under the current approved criteria and adopted GHS as below. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful by inhalation (Xn; R20)*	Harmful if swallowed - Cat. 4 (H302) Harmful if inhaled - Cat. 4 (H332)
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 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 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;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing risks of hazardous chemicals in the workplace—Code of practice* available on the Safe Work Australia website.

Personal protective equipment should not solely be relied upon to control risk and should only be used when all other reasonably practicable control measures do not eliminate or sufficiently minimise risk. Guidance in selecting personal protective equipment can be obtained from Australian, Australian/New Zealand or other approved standards.

Obligations under workplace health and safety legislation

Information in this report should be taken into account to help meet obligations under workplace health and safety legislation as adopted by the relevant state or territory. This includes, but is not limited to:

- ensuring that hazardous chemicals are correctly classified and labelled;
- ensuring that (material) safety data sheets ((M)SDS) containing accurate information about the hazards (relating to both health hazards and physicochemical (physical) hazards) of the chemical are prepared; and
- managing risks arising from storing, handling and using a hazardous chemical.

Your work health and safety regulator should be contacted for information on the work health and safety laws in your jurisdiction.

Information on how to prepare an (M)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of safety data sheets for hazardous chemicals—Code of practice* and *Labelling of workplace hazardous chemicals—Code of practice*, respectively. These codes of practice are available from the Safe Work Australia website.

A review of the physical hazards of the chemical has not been undertaken as part of this assessment.

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