1,4-Butanediol: Human health tier II assessment

11 April 2014

CAS Number: 110-63-4

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	Tetramethylene glycol 1,4-butylene glycol 1,4-dihydroxybutane 1,4-tetramethylene glycol Butane-1,4-diol
Structural Formula	
Molecular Formula	C4H10O2
Molecular Weight (g/mol)	90.12
Appearance and Odour (where available)	Colourless, waxy solid to viscous liquid depending on temperature.
SMILES	C(O)CCCO

Import, Manufacture and Use

Australian

The total volume of the chemical introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was between 1000 and 9999 tonnes.

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

The chemical has reported site-limited use including:

- as an intermediate and chain extender in the production of urethane prepolymers and polyether diols; and
- as a plasticiser (e.g. polyesters and cellulosics).

The chemical was found in toy beads (‘Bindeez') marketed in Australia (Gunja et al., 2008).

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; and eChemPortal: OECD High Production Volume chemical program (OECD HPV), the US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic use in:

- eye shadow (as a solvent);
- sunscreens (US Household Products Database (HHPD); and
- deodorants.

The chemical has reported commercial use including in:

- paints, lacquers and varnishes; and
- adhesives and binders.

The chemical has reported domestic use including in:

- cleaning and washing products.

The chemical has reported site-limited use including:

- as a reprographic agent;
- as a process regulator;
- in manufacturing tetrohydrofuran, gamma-butyrolactone and derivatives;
- as a co-monomer in classical diol-condensation reactions to produce polyurethane elastomers and polybutylene terephthalate; and
- as a laboratory analytical reagent.

Additionally, the use of the chemical in toy beads in North America was reported by a study from the University of California (Irvine) (Suchard et al., 2009).

Restrictions

Australian

The chemical is listed in the Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) in Appendix C.

Appendix C:

1,4-butanediol (excluding its derivatives) in non-polymerised form in preparations for domestic use.

Appendix C chemicals are substances of such danger to health as to warrant prohibition of sale, supply and use.

The main metabolite of this chemical, 4-hydroxybutanoic acid or gamma-hydroxybutyrate (GHB) and its salts, are listed in Schedule 9 of the SUSMP. Schedule 9 chemicals are prohibited substances, which may be abused or misused. The use of Schedule 9 chemicals is only allowed for specific purposes, such as for medical or scientific research, with approval from Commonwealth and/or State or Territory Health Authorities.

International

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

- Canada Controlled Drugs and Substance Act Schedule VI Part 1 – Class A Precursors, including synthetic and natural forms;
- European Union (EU) Restrictions on the Marketing and Use of Certain Dangerous Substances and Preparations; and,
- Japan Chemical Substances Control Law – Type II Monitoring Chemical Substances.

Existing Work Health and Safety Controls

Hazard Classification

The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica):

- An occupational exposure limit (OEL) of 100 mg/m^3 in Italy and Portugal; and 200 mg/m^3 in Austria and Germany; and

- A short term exposure value (STV) of 800 mg/m^3 in Austria.

Health Hazard Information

The chemical is a substance of abuse which acts primarily as a transient central nervous system (CNS) depressant. It is readily metabolised in the body to GHB which is a controlled substance due to its sedative and anaesthetic effects (Gunja et al., 2008).

Toxicokinetics

The toxicokinetics of the chemical are similar in man and in experimental animals. The chemical is rapidly and efficiently absorbed and is metabolised to gamma-hydroxybutyraldehyde by aldehyde dehydrogenase and then to GHB by alcohol dehydrogenases (Taberner et al., 1972; Fung et al., 2008). After oral or intravenous administration, it is converted to GHB in the liver; and then it is eliminated through the tricarboxylic acid cycle as carbon dioxide.

In an oral metabolism and disposition study, male Fischer 344/N (F344/N) rats were administered with the radiolabelled chemical [^{14}C 1,4-BD] by gavage at single doses of 4, 40, 120, or 400 mg/kg bw (NTP, 1996). Changes in urine, faeces, breath and tissues in rats were evaluated between two to 72 hours after administration. Approximately 50 % of the radiolabelled chemical was eliminated within the first two hours after administration as exhaled $^{14}CO_2$ and 85 % eliminated within 72 hours. The gastrointestinal tract and kidneys were minor routes of excretion with approximately 4 % and 0.6 % of the administered radioactivity (94 % as $^{14}CO_2$) excreted over 72 h in the urine and the faeces, respectively. There was no evidence of bioaccumulation in any tissue. Seventy-two hours after administration, a total of 2.3 % of the dose remained in the carcass, with majority in the liver and skin.

In animals and humans, the major biotransformation pathway is oxidation to GHB in the brain, liver, heart and kidneys. Following intravenous administration to humans, the conversion of the chemical occurs rapidly with the plasma concentration-time profile of GHB as a metabolite very similar to that obtained after intravenous injection of GHB itself. However, an equivalent oral study has not been conducted (OECD, 2000).

In a further study in humans, Thai et al. (2007) demonstrated extensive conversion of the chemical to GHB after oral administration, indicating that ingestion of the chemical is essentially equivalent to GHB intake. The average maximum plasma concentration of GHB following administration of the chemical to eight subjects at 25 mg/kg bw was found to be 45.6 $\pm$ 19.7 mg/L. This was comparable to the maximum plasma concentration of GHB (39.4 $\pm$ 25.2 mg/L) following oral administration of GHB at 25 mg/kg bw in eight volunteers under similar conditions (Thai et al., 2007; OECD, 2000).

Several studies describe the interaction of the chemical with ethanol. Simultaneous ingestion of the chemical and ethanol increased mortality and both renal and hepatic damage in rats. The action of alcohol dehydrogenase (ADH) from rat liver or brain in the conversion of the chemical to GHB was competitively inhibited by ethanol. In rodents, pyrazole (an inhibitor of ADH) blocked the reaction catalysed by the liver enzyme and antagonised the pharmacological response (induction of sleep or sedation) to the chemical (OECD, 2000).

Acute Toxicity

Oral

The chemical has moderate acute oral toxicity in rats and guinea pigs and warrants a hazard classification.

The median lethal dose (LD50) was reported to be 1525-1830 mg/kg bw in rats, 2060 mg/kg bw in mice, 1200 mg/kg bw in guinea pigs and 2531 mg/kg bw in rabbits (OECD, 2000).

Dermal

The chemical has low acute toxicity following dermal exposure.

The dermal LD50 in rats (under occlusive conditions) has been reported as >5000 mg/kg bw (OECD, 2000).

Inhalation

The chemical has low acute toxicity following inhalation.

In an inhalation study conducted according to the OECD Test Guideline (TG) 403, Wistar rats were exposed to 5.1 mg/L of a liquid aerosol of the chemical for four hours. No deaths were noted in the animals during the course of the study. Slight respiratory distress, manifested as accelerated and shallow respiration, was observed in animals during and immediately after exposure but was resolved by day one. On gross pathological examination, no abnormalities could be detected (OECD, 2000).

Male Crl:CD rats were exposed (nose only) to the chemical aerosol at 4.6, 9.4 or 15.0 mg/L for four hours. All rats survived at 4.6 or 9.4 mg/L up to 14 days after exposure, but 1/10 treated with 15 mg/L of the chemical died one day after exposure. Rats from 4.6 and 9.4 mg/L dose groups were lethargic and showed laboured breathing (REACH).

Observation in humans

Eight human volunteers (five males and three females) who received a single oral dose of the chemical at 25 mg/kg bw reported feeling less awake and less alert and more light-headed within 90 minutes of ingestion. Mild respiratory depression and transient increases in blood pressure were also observed (Thai et al*.,* 2007).

Numerous case reports are available describing the neurological consequences (including agitation, combativeness, respiratory depression, a labile level of consciousness, vomiting, seizures and death) in patients known to have ingested illicit products containing the chemical at unkown concentrations (Dyer et al., 1997, Zvosec et al., 2001; Theron et al., 2003; OECD, 2000).

In early November 2007, two children (a two-year-old boy and a 10-year-old girl) were admitted to an intensive care unit at the Children's Hospital at Westmead, Australia, with a decreased level of consciousness. The girl also had persistent vomiting and a four-minute generalised seizure. Upon toxicological screening, ?-hydroxybutyrate (GHB) was identified in the urine of both children. The source of the GHB was subsequently identified as its metabolic precursor, 1,4-BD, which was present in 'Bindeez' brand toy beads that had been ingested by both children (Gunja et al., 2008). Quantification of the amount of 1,4-butanediol ingested was not reported. During the same period, media sources in Australia reported that an 18-month-old New South Wales (NSW) boy and a 19-month-old Queensland boy had both become ill after allegedly swallowing the same beads. The NSW Division of Analytical Laboratories tested six sets of 'Bindeez' beads provided by the Office of Fair Trading (purchased at retailers) and confirmed the presence of 1,4-butanediol in all samples.

Following media reports from Australia and the United States on children becoming ill upon ingestion of the toy beads, the US Consumer Product Safety Commission issued a voluntary recall of some toy beads (Aquadots) in November 2007 (Suchard et al., 2009).

Corrosion / Irritation

Skin Irritation

The chemical is not reported to be a skin irritant in animal studies.

White Vienna rabbits were topically administered the chemical (in gauze patches under occlusive conditions to intact or abraded skin) for 24 hours. No skin reactions were seen at one, 24, 48 and 72 hours after patch removal (REACH).

In another experiment, the internal areas of the right ears of rabbits were painted with either 100 % or 50 % of the chemical in water for 10 consecutive days. The left ear of each rabbit painted with water served as a control. After 10 days of exposure, a minimal reddening was observed in the group exposed to 100 % concentration (OECD, 2000).

Eye Irritation

The chemical is reported to be a slight eye irritant in animal studies.

Slight reddening of the conjunctivae and small amounts of discharge were observed in four rabbits, one hour after ocular application of a single dose of the chemical (0.1 mL). The effects diminished after 24 and 48 hours and no abnormalities were observed at 72 hours (REACH).

Observation in humans

No skin irritation was seen in 200 human volunteers following treatment with the chemical (details not available) (OECD, 2000).

Sensitisation

Skin Sensitisation

The chemical is not reported to be a skin sensitiser in animal studies.

In a maximisation test, the chemical was applied to Hartley guinea pigs at a concentration of 10 % (intradermal injections) and 30 % (topical application) at the induction phase. The challenge procedure was conducted with 10 % and 30 % of the chemical. No allergic contact dermatitis was reported (REACH; OECD, 2000).

Repeated Dose Toxicity

Oral

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

In a 28-day study, groups of eight male and female Wistar rats were administered the chemical at oral gavage doses of 0, 5, 50 or 500 mg/kg bw/day. During necropsies, slight alterations to haematology profiles were observed, but not considered to be treatment related. Only 5/8 animals in each group were histopathologically examined. The results indicated histopathological changes in the bile ducts and periportal infiltrations with fibroblasts and mononuclear cells in the liver at all doses. These changes were suggestive of mild to moderate inflammation of the liver. However, the incidence of proliferation of the bile ducts was statistically significant only at the top dose (500 mg/kg bw/day) when both sexes were pooled. Therefore, the no observed adverse effect level (NOAEL) was determined to be 50 mg/kg bw/day in both sexes based on effects on the liver (OECD, 2000; REACH).

In a combined repeat dose and reproductive toxicity study (OECD TG 422), male SD rats were administered the chemical by gavage at 0, 200, 400 or 800 mg/kg bw/day for 45 days before mating. The female SD rats were given the same doses from 14 days before mating to day three of lactation. The results indicated that all treated groups showed transient dose dependent behavioural signs such as hyperactivity (see **Neurotoxicity**). All treated animals showed full recovery within five hours after each exposure. Body weight gains were reduced at 400 and 800 mg/kg bw/day with an associated decrease in food consumption. Reproductive outcomes are discussed under '**Reproductive & developmental toxicity'** section. The transient neurotoxic effects were observed at the lowest dose tested and a LOAEL of 200 mg/kg bw/day for both sexes was established based on the dose-related trend in central nervous system effects (OECD, 2000).

Dermal

No data are available.

Inhalation

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

Groups of male rats (strain not specified) were exposed to the chemical (aerosol) at doses between 1.5 and 2.0 mg/L for two hours per day (calculated dose: 85–110 mg/kg bw/day) for four months. Clinical signs of toxicity, such as inactivity and sleepiness, were observed after the first three or four weeks of the study. However, these signs were reversible within 10 to 20 minutes after exposure. Histopathological examination revealed extensive pulmonary emphysema, mild lung oedema and, in a few animals, inflammatory changes of single alveolar cells and slight hyperplasia of alveolar septum with proliferation of lymphocytes and histiocytes were observed. These changes were considered to be local irritant effects and were not accompanied by treatment related pathological changes in other organs. The lowest observed adverse effect concentration (LOAEC) of 1.5 mg/L (85 mg/kg bw/day) was determined in male rats based on signs of clinical toxicity immediately following exposure observed at all doses (OECD, 2000).

In another four-month study, male rats (strain not specified) were exposed to the chemical as an aerosol at doses between 0.3–0.5 mg/L for two hours a day, six days a week (calculated dose: 15–24 mg/kg bw/day). No clinical signs of toxicity were observed after the exposure, and body weight, nervous system function (neuromuscular response), haemogenesis, liver function and kidney function were not changed. The no observed adverse effect concentration (NOAEC) was determined to be 0.5 mg/L (equivalent to 24 mg/kg bw/day) (OECD, 2000).

In a two-week study, groups of 10 male Crl:CD rats were exposed (nose only) to an aerosol of the chemical at 0, 0.2, 1.1 or 5.2 mg/L for six hours a day, five days a week (REACH; OECD, 2000). Compared to controls, lower (7 % to 9 %) mean body weights, increased erythrocyte counts and haematocrit were seen in rats at the highest dose. Slight atrophy of the lymphoid cells of the thymus was seen in three out of five rats after 10 exposures. Body weights and thymic changes returned to normal during the recovery period. As no adverse effects were observed in rats exposed to 0.2 or 1.1 mg/L, the NOAEC was determined to be 1.1 mg/L (calculated to be 134 mg/kg bw/day) based on changes in haematology parameters at the highest dose (OECD, 2000).

Genotoxicity

Based on the negative results indicated in the genotoxicity studies, the chemical is not considered to be genotoxic.

The following in vitro studies with the chemical produced negative results (OECD, 2000; REACH):

- Ames assay (OECD TG 471 and 472) with four strains of *Salmonella typhimurium* (TA100, TA1535, TA98, TA1537) at doses 313, 625, 1250, 2500 or 5000 µg/plate, with or without metabolic activation;
- hypoxanthine-guanine phosphoribosyl transferase (HPRT) forward mutation test (OECD TG 476) in Chinese hamster ovary (CHO) cells, with or without metabolic activation; and
- chromosomal aberration (CA) test (OECD TG 473) in V79 Chinese hamster lung cells exposed to 26 and 41 hours, with or without metabolic activation.

Negative results were observed in the in vivo *Drosophila melanogaster* sex-linked recessive lethal assay. However, this result was considered inconclusive due to the limited sample size used in this study (Lee et al., 1983; OECD, 2000).

Carcinogenicity

No data are available for the chemical. However, based on the information available for gamma-butyrolactone (which also metabolises to GHB), and in the absence of genotoxicity, this chemical is not considered to be carcinogenic.

The chemical gamma-butyrolactone, which also rapidly converts to GHB, has shown no carcinogenic response in rats and mice (OECD, 2000). Except for behavioural changes, no study has identified organ-specific toxicity or other significant pathological

effects. Based on the absence of evidence for genotoxicity and the negative result of the carcinogenicity bioassay for gamma-butyrolactone, the chemical 1,4-BD is not considered to be carcinogenic in animals (OECD, 2000; REACH).

Reproductive and Developmental Toxicity

Based on the data available, the chemical is not considered to have reproductive and developmental toxicity.

In a combined repeated dose and reproductive toxicity study (OECD TG 422), male SD rats were administered the chemical at 0, 200, 400 or 800 mg/kg bw/day for 45 days before mating. The female SD rats were given the same doses from 14 days before mating to day three of lactation. The chemical did not induce changes in any of the reproductive parameters in mating pairs. However, there was a slight, but significant reduction in pup body weight at 800 mg/kg bw/day, which was considered to be attributable to maternal toxicity (reduced food consumption and body weight gain) (OECD, 2000).

In a developmental toxicity study, pregnant Swiss (CD-1) mice were administered the chemical by gavage doses of 0, 100, 300 or 600 mg/kg bw/day on gestation days six to 15. No maternal mortality was observed during the study; however, signs of CNS depression (including hypoactivity, immobility, and loss of righting reflex) occurred at doses 300 and 600 mg/kg bw/day, but usually resolved within four hours after dosing. In the same groups, other indications of maternal toxicity included lower body and liver weights and food consumption compared with the controls. Reduced kidney weights were seen in the top dose group only. In groups dosed with 300 and 600 mg/kg bw/day, significant reductions in live foetal body weights (8 % and 17 %, respectively) were observed. An increasing trend (not statistically significant) towards skeletal malformations (missing or branched ribs and fused thoracic vertebrae) was also observed; this was considered as secondary to maternal toxicity. Furthermore, the exposure to the chemical did not increase incidence of resorptions. The percentage of litters with one or more late deaths was lower in the 300 and 600 mg/kg bw/day groups compared with the control and 100 mg/kg bw/day groups. The NOAEL for maternal toxicity is 100 mg/kg bw/day based on CNS effects at higher doses. The NOAEL for developmental toxicity was established as 600 mg/kg bw/day. The chemical is not determined to show specific developmental toxicity (OECD, 2000; REACH; NTP, 1996).

Other Health Effects

Neurotoxicity

Transient effects on the central nervous system of rats have been observed soon after oral exposure to the chemical. These effects are attributable to its conversion to GHB. A hazard classification is warranted under the adopted GHS.

The neurotoxic effects are similar to the effects seen following alcohol exposure (REACH; OECD, 2000). Administration of the chemical at 496 mg/kg bw/day to male SD or Holtzman rats caused CNS depression and induced a state resembling sleep or anaesthesia. This is characterised by loss of righting reflex, struggle response, and voluntary motor activity, but maintained the ability to respond to pain and tactile stimuli (OECD, 2000).

Daily treatment of Wistar rats with the chemical for 28 days resulted in dose-related acute toxicity in the CNS (see **Repeat dose toxicity**). Immediately after administration, the rats in the 200 mg/kg bw/day group showed transient hyperactivity. The severity of the toxic effects were more prominent in animals dosed with 800 mg/kg bw/day. These effects include loss of consciousness after showing hypoactivity and recumbency; however, these effects were reversible after each dose within five hours (REACH).

No clinical or pathological changes were observed in the central and peripheral nervous systems of SD rats receiving the chemical at 0.5 % in drinking water (approx. 508 mg/kg bw/day) daily for 10 days (OECD, 2000).

In a six-month oral study investigating the influence of the chemical on conditioned reflexes and biochemical profile, male rats (species not specified) were administered the chemical at 0, 0.25, 3 or 30 mg/kg bw/day. The animals at 30 mg/kg bw/day lagged with respect to the appearance and fixation of the reflex and had a longer latent period before responding to the stimulus; there were also changes to clinical chemistry parameters and histopathological changes in the brain and liver. Only limited information was provided (OECD, 2000).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include:

- systemic acute toxicity following oral exposure; and
- transient neurotoxic effects soon after oral exposure.

The chemical may cause transient central nervous system effects following ingestion due to its rapid conversion to GHB (main metabolite), a controlled substance with sedative and anaesthetic properties.

Public Risk Characterisation

The low concentrations of the chemical found in toy beads have caused adverse effects in young children following oral exposure (Gunja et al., 2008). Following identification of the chemical in toy beads in Australia and considering its adverse effects on young children, the use of this chemical in non-polymerised form in preparations for domestic use was prohibited by adding it to Appendix C of the SUSMP to prohibit such uses. Therefore, it is unlikely that the public will be exposed to the chemical or its main metabolite through domestic products/uses and the public risk is not considered to be unreasonable.

Occupational Risk Characterisation

Workers may be exposed to the chemical through inhalation of vapours and through eye and skin contact. The level and route of exposure will vary depending on the work practices employed.

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.

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

Public Health

The Appendix C entry in the SUSMP to prohibit the use of this chemical in non-polymerised form in preparations for domestic use is appropriate to mitigate the risk of unintended ingestion of the chemical from domestic articles (such as toys).

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]

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)	Harmful if swallowed - Cat. 4 (H302)
Other Health Effects		May cause drowsiness or dizziness - Specific target organ tox, single exp Cat. 3 (H336)

[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 label instructions.

Advice for industry

Control measures

Control measures to minimise the risk from 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:

- 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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