1,3-Propanediol, 2-bromo-2-nitro-: Human health tier II assessment

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CAS Number: 52-51-7

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.

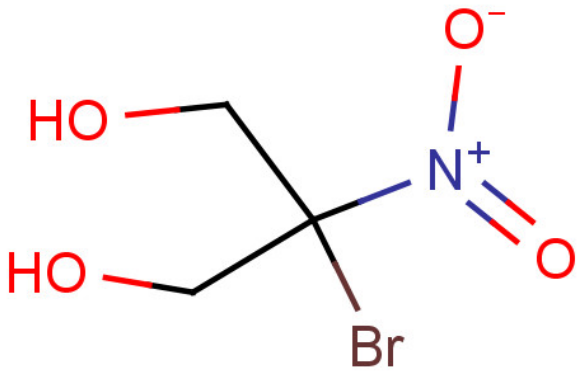
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	2-bromo-2-nitro-1,3-propanediol bronopol 2-bromo-2-nitropropane-1,3-diol
Structural Formula	
Molecular Formula	C3H6BrNO4
Molecular Weight (g/mol)	200
Appearance and Odour (where available)	White crystalline powder with faint odour.
SMILES	C(Br)(CO)(CO)N(=O)=O

Import, Manufacture and Use

Australian

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

The chemical has a reported cosmetic use as a preservative.

Bronopol has a reported site-limited use in hydraulic fracturing.

International

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

The chemical has a reported cosmetic use as a preservative.

The chemical has reported domestic uses, including in:

- adhesives;
- bleaching agents;
- cleaning and washing agents;
- colouring agents;
- corrosion inhibitors;
- fillers;
- flame retardants and extinguishing agents;
- insulating materials;
- odour agents;
- paints, lacquers and varnishes;
- surface treatment agents; and
- surface-active agents.

The chemical has reported commercial uses, including in:

- anti-set-off and anti-adhesive agents;
- anti-static agents
- conducting agents;
- construction materials;
- dust binding agents;
- hydraulic fluids and additives;
- impregnation materials;
- lubricants and additives;

- photochemicals;
- pH regulating agents;
- process regulators;
- reprographic agents;
- softeners;
- solvents; and
- viscosity adjustors.

The chemical has reported site-limited uses, including in:

- complexing and flocculating agent in;
- stabilisers; and
- hydraulic fracturing.

The chemical has reported non-industrial uses, including in:

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

Restrictions

Australian

No known restrictions have been identified.

International

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

- EU Cosmetic Directive 76/768/EEC Annex VI Part 1, List of preservatives allowed (at up to 0.1%);
- New Zealand Cosmetic Products Group Standard—Schedule 7: Preservatives cosmetic products may contain with restrictions;
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient 'Hotlist')—Permitted at concentrations equal to or less than 0.1%. Not permitted in formulations that contain amines or amides; and
- US Cosmetic Ingredient Review (CIR) Cosmetic ingredients found safe, with qualifications—should not be used in cosmetic products in which N-nitroso compounds can be formed.

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; R21/R22 (acute toxicity); and
- Xi; R37/38; R41 (irritation).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

No data are available for oral, dermal or inhalational absorption of bronopol.

In metabolism studies in rats, rapid absorption of gavage-administered bronopol from the gastro-intestinal tract was reported (US EPA, 1995). The International Chemical Safety Card (ICSC) for bronopol indicated that the substance can be absorbed into the body by inhaling it as an aerosol, through the skin and by ingestion (IPCS, 2012). Acute inhalation toxicity studies in rats demonstrated that bronopol is absorbed by the inhalational route (Collins, 1986, Binns et al., 1971).

Information on the distribution of bronopol in the body is not available. Oral repeated dose toxicity studies in rats have reported treatment-related effects in numerous organs, suggesting wide distribution of the chemical within the body.

In three metabolism studies analysed by the United States Environmental Protection agency (US EPA) (details of the studies not provided), Sprague Dawley (SD) rats were treated by gavage with ^{14}C-bronopol. In the first study, animals received a single dose of 10 mg/kg bw. The second study employed a higher dose of 50 mg/kg bw; the dose in the third study was 10 mg/kg bw (14 daily doses of non-radioactive bronopol, followed by one dose of ^{14}C-bronopol). Urine, faeces and exhaled CO_2 were collected for seven days after dosing, at which time the rats were euthanised and the tissues examined for radioactivity. Irrespective of the dose, most of the administered ^{14}C was excreted in urine (64–78% in 24 hours and 68–83% in seven days). Faeces, exhaled CO_2 and tissues represented minor routes of excretion of ^{14}C. Very little ^{14}C was detected in the blood (US EPA, 1995).

Based on the results of the study, the US EPA concluded that orally administered bronopol was rapidly absorbed and excreted by rats of both sexes, with urine being the major route of excretion. The only metabolite identified in the urine was 2-nitropropane-1,3-diol (desbromo-bronopol), accounting for 45–50% of the radioactivity. Unchanged bronopol was not detected.

Formaldehyde has been identified as a degradant of bronopol under aqueous alkaline conditions. However, exposure to formaldehyde is not likely to present a risk because of the slow decomposition rate of bronopol.

Bronopol does not hydrolyse under normal conditions. However, at higher temperatures (30, 40, 50, and 60 °C), and/or higher pH (as encountered in some industrial applications or under atypical environmental conditions), rapid hydrolysis can occur. At high temperatures and pH, hydrolysis products include formaldehyde and lesser amounts of other degradants.

At ambient laboratory temperatures and at concentrations of 2000 ppm or higher, the half-life of bronopol was extrapolated to be about 18 years at pH 4, about 1.5 years at pH 6, and approximately two months at pH 8. At 60 °C, half-lives range roughly from four days at pH 4 to only three hours at pH 8.

Other degradants produced under these circumstances are 2-hydroxymethyl-2-nitropropane-1,3-diol (tris); 2-bromo-2-nitroethanol; unidentified products, which were possibly polymeric; bromide; nitrite (but not nitrate); and other trace products such as aliphatic nitro compounds and lightweight gases, but not carbon dioxide (US EPA, 1995).

Acute Toxicity

Oral

Bronopol has moderate acute toxicity by the oral route. Rats administered bronopol orally exhibited clinical signs of sedation, nasal exudate, gasping, wheezing, cyanosis, and convulsions. The study reported acute oral median lethal doses (LD50) of 307 and 342 mg/kg bw for males and females, respectively (Inolex Chemical Company, 1976).

Dermal

Bronopol has moderate to high acute toxicity from dermal exposure. When applied to rat skin at doses of 0, 64, 160, 400 or 1000 mg/kg bw, it produced oedema, haemorrhage, laboured breathing, prostration, and lung congestion. Acute dermal LD50 values of 64–160 mg/kg bw were derived from this study. No details of the study were provided (Smithson, 1984).

Inhalation

Bronopol has low acute toxicity from inhalational exposure. In an acute inhalation study (details not provided), rats were exposed to various aerosolised concentrations of bronopol. No mortalities occurred. At a low concentration of 0.089 mg/L bronopol (particle size 1.3–6.7 µm), piloerection, hunched posture and hydronephrosis were observed. At higher concentration (0.588 mg/L), diffused red lungs, sore eyelids and severe dermatitis and ulceration on the head were reported (Collins, 1986).

In another study, clinical signs observed in rats exposed to 0, 0.05, 0.5 or 5 mg/L bronopol included eye irritation, dyspnoea, profuse mucus production and lethargy. Chronic pneumonitis was also observed after the test duration. There were no mortalities even at the highest exposure dose. The acute inhalation median lethal concentration (LC50) for this study was identified by the author as >5 mg/L (Binns et al., 1971).

Observation in humans

No data are available.

Corrosion / Irritation

Respiratory Irritation

No data are available. Acute inhalation toxicity studies indicated some respiratory irritation signs following bronopol exposure.

Skin Irritation

Bronopol is considered to be a severe skin irritant. In a primary dermal study evaluated by the US EPA (1995), 0.5, 2, or 5 % bronopol solution in aqueous methylcellulose was applied to the skin of rats. Slight to moderate erythema and slight to severe oedema were noted on the intact skin of females 24 hours after the six-hour exposure was terminated. The effects observed at the low concentrations of bronopol in this study demonstrated the strong skin irritation potential of bronopol (Inolex Chemical Company, 1976).

Eye Irritation

Bronopol is a severe eye irritant. Instillation of a 5 % bronopol solution in polyethylene glycol in rabbit eyes produced redness and swelling of the conjunctivae with moderate discharge one hour after dosing. The effects subsided in most of the animals after seven days (Liggett & Parcell, 1984). No details of the study were provided. The effects observed in this test at low bronopol concentrations demonstrate the severe irritation potential of the chemical to eyes.

Observation in humans

No data are available.

Sensitisation

Skin Sensitisation

Bronopol is not a skin sensitiser in guinea pigs. In a study to determine the dermal sensitisation potential of bronopol (>98.8 %), guinea pigs received dermal applications of 1 % bronopol in acetone. The three induction treatments on the outer surface of each ear and, one week later, one challenge treatment on the back and flank, did not produce any sensitisation effects (Maibach, 1977; Smithson, 1984).

Observation in humans

In a large clinical study, patch testing of 8149 individuals with bronopol was conducted in seven European contact clinics. The results reported low response rates with only 0.47 % of the cases showing allergic reactions. The study concluded that the sensitisation rate to bronopol is quite low (Frosch et al., 1990).

Repeated Dose Toxicity

Oral

Male and female SPF rats were given bronopol by gavage for 13 weeks at doses of 0, 20, 80, or 160 mg/kg bw/day. In the 20 mg/kg bw/day group, one female died in week 10 and dilated tubules in the kidneys were observed in two males. In the 80 mg/kg bw/day group, mortality was 35 % in males and 45 % in females. The effects observed at this dose were respiratory distress, such as gasping and wheezing, decreased bodyweight gain (7–20 % in males and 12–16 % in females), and dilated tubules in the kidney of one male. In the 160 mg/kg bw/day group, the study reported high mortality. The effects observed at the highest dose included decreased bodyweight gain (29 % and 13 % less compared with controls for males and females, respectively), severe respiratory distress, and gaseous or fluid distension of the gastrointestinal (GI) tract in most rats. GI lesions such as superficial ulceration with underlying inflammation, epithelial hyperplasia and hyperkeratosis were also observed in the animals. The effects on the kidneys (dilated tubules) observed at the lowest and mid-dose groups were not considered dose-related, since these effects were not reported at the highest dose tested. The no observed adverse effect level (NOAEL) was 20

mg/kg bw/day based on mortalities at the lowest observed adverse effect level (LOAEL) of 80 mg/kg bw/day (Hunter et al., 1973a).

Beagle dogs were administered bronopol by gavage for 13 weeks at doses of 0, 4, 8, or 20 mg/kg bw/day. The NOAEL identified was 8 mg/kg bw/day based on increased liver (15 %) and spleen (39 %) weights at the LOAEL of 20 mg/kg bw/day (Rivett et al., 1973).

In a chronic feeding/carcinogenicity study, SD rats were administered 0, 10, 40, or 160 mg/kg bw/day bronopol in drinking water for 104 weeks. In the 40 mg/kg bw/day group, treatment-related effects included reduced bodyweight gain (20–52 %), squamous metaplasia and atrophic acini in the salivary glands (48 % in males and 13 % in females). In the 160 mg/kg bw/day group, treatment-related effects included: reduced grooming activity; high mortality (80 % in males and 67 % in females); decreased bodyweight gain (13–48 % in males and 11–53 % in females); increased relative organ weights (adrenals, brain, kidneys, liver, and lungs—proportions were not reported in the study); decreased absolute organ weights (29 % for heart, 35 % for liver, 12 % for lungs, 47 % for seminal vesicles, 20 % for testes, and 26 % for thyroid); stomach lesions (37 % for males and 29 % for females); squamous metaplasia; dilatation of the ducts; acinar atrophy and/or inflammation of the salivary glands in 92 % of the males and 55 % of the females. The NOAEL was 10 mg/kg bw/day for both sexes based on the body weight at the LOAEL of 40 mg/kg bw/day (Hunter et al., 1973b; Hunter et al., 1976).

Dermal

Bronopol suspended in aqueous methyl cellulose was applied to New Zealand White rabbit skin at doses of 0, 0.2, or 0.5 % (w/v) for three weeks. Effects observed at the highest dose included moderate erythema and oedema; and thickened, hardened, and sloughed skin at the application site. No systemic effects were observed. The NOAEL established in this study for local effects was 0.2 % (w/v) bronopol (equivalent to 2 mg/kg bw/day), based on dermal irritation at the highest dose (Davies et al., 1973).

In another study, bronopol was applied on the skin of CFLP mice three days a week for 80 weeks at doses of 0, 20, or 50 mg/kg bw/day. At the highest dose, the only treatment-related effects observed were hair loss at the edge of the shaved area in some mice (numbers not reported), and reduced bodyweight gain (percentages not reported) (Hunter et al., 1975).

Inhalation

No data are available.

Observation in humans

No data are available.

Genotoxicity

Bronopol is not considered to be genotoxic based on the available data. Bronopol was negative for mutagenicity in an Ames test in *Salmonella typhimurium* (up to 125 µg/plate) (Everest & Williams, 1986a) and in a cell mutation assay in Chinese hamster lung fibroblast V79 cells at up to 8 µg/mL concentration (Everest & O'Donovan, 1986).

In a human lymphocyte culture cytogenetic assay, bronopol was not clastogenic in the presence of the metabolic activation system (S-9 microsomal fraction) and was clastogenic in the absence of S-9, but only at 30 µg/mL, the highest concentration allowed by cytotoxicity. The observed clastogenicity (significant increases in the percentage of cells with aberrations, relative to the negative control values) was attributed by the testing facility to formaldehyde, one of the degradation products of bronopol and a known clastogen (**Toxicokinetics**) (Everest & Williams, 1986b).

In an in vivo micronucleus assay in mice, the chemical was negative at up to the maximum tolerated dose of 160 mg/kg bw/day (Everest & Williams, 1986c).

Carcinogenicity

Bronopol is not considered to be carcinogenic. In the previously described chronic feeding/carcinogenicity study (**Repeat dose oral toxicity**) by Hunter et al., 1976, the tumours most frequently observed in the SD rats were pituitary adenoma in males (23, 32, 17, and 5 %) and females (45, 47, 55, and 37 %) at the 0, 10, 40, and 160 mg/kg bw/day group, respectively), and fibroadenoma in the females (79, 80, 71, and 50 % at the 0, 10, 40, and 160 mg/kg bw/day group, respectively). No dose responses were seen.

In the previously described dermal study (**Repeat dose dermal toxicity**) by Hunter et al. (1975) on CFLP mice, the tumour incidences were 48, 42, and 46 % (in males) and 49, 36, and 45 % (in females) at the 0, 20, and 50 mg/kg bw/day dose groups, respectively. The tumours most frequently observed were lymphoma and lung tumours. The authors indicated that the tumour incidences observed in mice of both sexes were not statistically significant compared with controls.

The US EPA (1995) indicated that bronopol was classified as a Group E carcinogen (evidence of non-carcinogenicity in humans) based on a lack of carcinogenicity evidence from acceptable studies in rats and mice. The dose levels used in the tests were considered satisfactory for carcinogenicity evaluation. The conclusion was based on increased mortality, stomach lesions and reduced bodyweight gain in rats, and no statistically significant increases in tumour incidences in the treated animals.

Reproductive and Developmental Toxicity

Reproductive toxicity

In a two-generation reproductive study (authors and year of the study not available), bronopol was administered to Charles River COBS CD rats in drinking water during pre-mating, mating, gestation and lactation periods. The doses tested were 0, 25, 70, and 200 mg/kg bw/day (US EPA, 1995).

No treatment-related effects were observed in the 25 mg/kg bw/day group. Systemic toxicity was observed in the mid-dose (70 mg/kg/day) and high-dose (200 mg/kg/day) groups, in both F0 and F1 generation rats.

In the mid-dose group, toxic signs observed included increased kidney weights in F0 females, decreased liver weight in F1 males and females, and an increased incidence of nephropathy (kidney tubule degeration) in F0 males and females.

Toxic signs noted in the high-dose group were: (a) decreased body weights of the F0 and F1 females during the premating, gestation and lactation periods; (b) decreased body weights of the F1 males; (c) increases in adrenal (F0 females), kidney (F0 females and F1 males) and thyroid/parathyroid (F1 males); (d) decreases in liver weight (F1 males); and (e) an increased incidence of nephropathy in the F0 males and females. Reproductive toxicity, a slight decrease in pup weight and in the female fertility index in the F1 generation (75 % vs 87.5 % in the controls), were observed only in the high-dose group. The NOAELs for systemic and fertility effects were 25 and 70 mg/kg bw/day, respectively (as cited in US EPA, 1995).

Developmental toxicity

In a developmental toxicity study, bronopol was administered to SD rats by gavage in doses of 0, 10, 28, or 80 mg/kg bw/day from gestation days 6 to 15. There were no dose-related clinical signs observed in the animals and no developmental effects observed in the pups. No maternal toxicity was noted apart from a 1 % decrease in bodyweight gain. The NOAEL for both maternal and developmental toxicity was >80 mg/kg bw/day (Palmer, 1995; Steele, 1994).

In another developmental toxicity study, New Zealand White rabbits were administered the chemical by gavage at doses of 5, 20, 40, or 80 mg/kg bw/day. Maternal effects observed were a 13 % decrease in bodyweight gain and 38 % decrease in food consumption at the highest dose. Developmental effects, observed only at the highest dose, were 10 % decrease in foetal bodyweight in both sexes, 6.9 % increase in foetuses with major external/visceral and skeletal abnormalities, 19.3 % increase in foetuses with minor skeletal abnormalities, and increased incidence of foetuses with skeletal variants (8 % unossified forelimb and 16 % hind limb epiphyses). There were no developmental effects observed in the absence of maternal toxicity. The NOAEL for both maternal and developmental toxicity was 40 mg/kg bw/day based on the effects noted above (Irvine, 1992a; Irvine, 1992b).

Risk Characterisation

Critical Health Effects

Bronopol has moderate acute dermal and oral toxicity, is a skin and respiratory system irritant, and severely damaging to the eyes. The chemical is not a skin sensitiser in animals and humans.

Public Risk Characterisation

The general public could be exposed through the skin or by inhalation when using cosmetic or domestic products containing the chemical. However, based on limited USA information derived from the National Library of Medicine (NLM) Household Products Database, the concentration in these products is not considered to be sufficiently high to cause corrosive effects. Therefore, the risk to public health is not considered to be unreasonable and further risk management is not considered necessary for public safety.

Occupational Risk Characterisation

During product formulation, oral, dermal, 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 and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal, 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

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 and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Toxic in contact with skin (T; R24)	Harmful if swallowed - Cat. 4 (H302) Fatal in contact with skin - Cat. 2 (H310)
Irritation / Corrosivity	Risk of serious eye damage (Xi; R41)* Irritating to skin (Xi; R38)* Irritating to respiratory system (Xi; R37)*	Causes serious eye damage - Cat. 1 (H318) Causes skin irritation - Cat. 2 (H315) May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335)

[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 oral, dermal, ocular and inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate, or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures which 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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