Ethane, 1,2-dibromo-: Human health tier II assessment

17 May 2013

CAS Number: 106-93-4

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	Ethylene dibromide 1,2-dibromoethane
Structural Formula	
Molecular Formula	C2H4Br2
Molecular Weight (g/mol)	187.86
Appearance and Odour (where available)	Liquid
SMILES	C(Br)CBr

Import, Manufacture and Use

Australian

The following potential Australian uses were reported (National Pollutant Inventory (NPI)):

The chemical has reported site-limited use including:

- used as a preparation for dyes and waxes;
- used in the production of some plastics and latex; and
- used in the manufacture in leaded petrol.

The chemical has reported non-industrial use including:

- in the treatment of logs for pests; and
- fumigant in soil, grains, fruits, and vegetables.

International

The international uses have been identified through the 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 Substances and Ingredients (CosIng) database, United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) directory and other data sources via eChemPortal including the US Environmental Protection Agency's (EPA) Aggregated Computer Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported commercial use including:

- used as a lead scavenger in tetraalkyllead petrol and in anti-knock preparations; and
- solvent for resins and gum waxes.

The chemical has reported site-limited use including:

- used as a chemical intermediate in dye synthesis.

The chemical has reported non-industrial use including:

- used as a soil and grain fumigant.

Restrictions

Australian

This chemical is listed in the Poisons Standard (Standard for the Uniform Scheduling of Medicines and Posions (SUSMP)) in Schedule 7.

Schedule 7 chemicals are labelled as a "Dangerous Poison". These are substances with a high potential for causing harm at low exposure and which require special precautions during manufacture, handling or use. These poisons should be available only to specialised or authorised users who have the skills necessary to handle them safely. Special regulations restricting their availability, possession, storage or use may apply.

The chemical is listed in the Model Work Health and Safety (WHS) Regulations; Schedule 10 Restricted carcinogens – when used as a fumigant.

Australia Customs (Prohibited Exports) Regulations 1958 - Schedule 2: Goods, being certain chemicals, the exportation of which is prohibited unless permission is granted under regulation 4A.

International

International restrictions include (Galleria Chemica):

Association of Southeast Asian Nations (ASEAN) Cosmetic Directive Annex II Part 1: List of substances which must not form part of the composition of cosmetic products;

European Union (EU) Cosmetic Directive 76/768/EEC Annex II: List of Substances which must not form part of the Composition of Cosmetic Products;

European Union (EU) List of Chemicals Subject to the PIC (Prior Informed Consent) Procedure under the Rotterdam Convention;

European Union (EU) Restrictions on the Marketing and Use of Certain Dangerous Substances and Preparations;

New Zealand Cosmetic Products Group Standard—Schedule 4: Components Cosmetic Products Must Not Contain – Table 1;

United Nations List of Prior Informed Consent Chemicals under the Rotterdam Convention;

US Environment Protection Agency (US EPA) Banned or Severely Restricted Pesticides.

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):

T; R45 (Carcinogenicity Cat. 2)

T; R23/24/25 (Acute toxicity)

Xi; R36/37/38 (Irritation)

Exposure Standards

Australian

The chemical has no exposure standard. However, there is a skin notation and footnote "d".

Skin notation – Absorption through the skin may be a significant source of exposure.

Footnote "d" – For a few substances, usually the more potent probable and established human carcinogens, it is not currently possible to assign an appropriate exposure standard. For these substances, exposure should be controlled to the lowest practicable level.

International

The following esposure standards are identified (Galleria Chemica):

Canada (Bristish Columbia) – time weighted average (TWA) 0.5 ppm, skin notation carcinogen 2A;

United Kingdom Workplace Exposure Limit – long term exposure limit 0.5 ppm (3.9 mg/m³), skin and carcinogen;

US National Institute of Occupational Safety and Health (NIOSH) Recommended Exposure Limit – 0.045 ppm TWA, 0.13 ppm Ceiling; and

New Zealand Workplace Standards – TWA 0.5 ppm or 3.9 mg/m³, skin notation; confirmed carcinogen.

Health Hazard Information

Toxicokinetics

Absorption

Absorption of the chemical through the gastrointestinal tract (GIT) is assumed to be from the intestines to the liver. Absorption was reported to be fast and within 30 minutes (US EPA, 2004).

Following dermal exposure to 1 mL of 1,2 dibromoethane in guinea pigs, blood levels increased rapidly to 2.1 ug/mL after 1 hour and decreased slightly to 1.8 ug/mL after 6 hours (US EPA, 2004).

Inhalation studies in rats showed that 1,2 dibromoethane is also absorbed by inhalation and distributed systemically with 50 % absorbed in the upper or lower respiratory tract, at a flow rate equivalent to 53 mL/min (US EPA, 2004).

Metabolism

The chemical is metabolised by two major pathways, cytochrome P450 monooxygenase and glutathione (GSH) conjugation via glutathione-S-transferase (GST). Oxidative metabolism by cytochrome-P450 leads to the formation of the reactive metabolite 2-bromoacetaldehyde. This pathway accounts for 80 % of the metabolism of the chemical in rats.

The chemical can also conjugate directly with glutathione to form S-(2-bromoethyl)glutathione that can spontaneously rearrange to an episulfonium ion. It is further hydrolysed to S-(β-hydroxyethyl)GSH or it can bind to DNA to generate miscoding adducts. The episulfonium ion is believed to be responsible for the genotoxicity of 1,2 dibromoethane (US EPA, 2004).

Distribution

In rats, 3 % of the administered radiolabel was retained in the kidney, liver and spleen 24 hours after oral administration. After 48 hours, 1.1 % of the administered label was found in the liver. Urinary elimination was decreased significantly with increased tissue binding.

In mice, irreversibly bound metabolites of the chemical were reported to be highest in the nasal mucosa, followed by the liver, lung and kidney, 3 hours after intraperitoneal (ip) injection. Similar observations were reported for rats. In the cynomolgous monkey, the liver and kidney had the highest amount of radioactivity and very little binding was observed in the lungs and the respiratory tract after ip injection (US EPA, 2004).

Excretion

The chemical is eliminated mainly in the urine. Following oral administration in rats, 72 % of the chemical was excreted in the urine and 1.65 % in the faeces. After 48 hours, 73 % of the dose was found in urine and 3 % in the faeces. Similar results were reported following intravenous (iv) administration, except for a much higher percentage of the dose being eliminated in the expired air.

Following oral administration, a number of urinary metabolites have been reported from in vivo metabolic studies in rats including S-(hydroxyethyl)mercapturic acid and S-(β-hydroxyethyl)cysteine, thiodiacetic acid and thiodiacetic acid sulfoxide. (US EPA, 2004).

Mode of action

Microsomal cytochrome P-450 dependent oxidative metabolism of 1,2-dibromoethane produces the metabolite 2-bromoacetaldehyde.The cytotoxic mechanisms for the chemical may be attributed to lipid peroxidation and/or protein binding induced by the metabolite 2-bromoacetaldehyde. In addition, the conjugation of the chemical with glutathione may also contribute to cytotoxicity. Studies have shown that 1,2-dibromomethane-induced depletion of hepatic mitochondrial glutathione, correlated with liver toxicity and perturbations in mitochondrial Ca^{2+} homeostasis (US EPA, 2004).

Repeated oral administration of the chemical in rats has been shown to increase the content of glutathione in the liver and kidney. Lipid peroxidation may have a role in the liver cell necrosis seen in animals treated with the chemical (US EPA, 2004).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in HSIS (Safe Work Australia). The data available (median lethal dose LD50) is 117 – 146 mg/kg bw) support this classification (US EPA, 2004). Reported signs of toxicity include centrilobular necrosis and sinusoidal dilations in the liver.

Dermal

The chemical is classified as hazardous with the risk phrase ‘Toxic in contact with skin’ (T; R24) in HSIS (Safe Work Australia). The toxicokinetic data confirming that the chemical is absorbed through the skin together with its high systemic toxicity supports this classification.

Inhalation

The chemical is classified as hazardous with the risk phrase ‘Toxic by inhalation’ (T; R23) in HSIS (Safe Work Australia). The available data support this classification. The rat LC50 was 200 ppm (approximately 1.54 mg/m^3) over 4 hours (USEPA-IRIS; REACH, 2013).

Corrosion / Irritation

Corrosivity

Results of the in vitro EPISKIN model test indicate that the chemical is not corrosive to the skin (US EPA, 2004 and REACH, 2013).

Respiratory Irritation

The chemical is classified as hazardous with the risk phrase ‘Irritating to respiratory system’ (Xn; R37) in HSIS (Safe Work Australia). The available data support this classification.

In a 13 week repeat dose inhalation study (OECD TG 413 or similar), rats were exposed to 3, 10 and 40 ppm of the chemical for 6 hours/day, 5 days/week. At 40 ppm, male rats showed eye and nasal irritation during the first exposure period. At 10 ppm and above, scattered to diffuse hyperplasia of the respiratory epithelium of the turbinate was reported but appeared to be reversible. The no observed adverse effect concentration (NOAEC) from this study is 3 ppm (23 mg/m^3) (US EPA, 2004 and REACH, 2013).

Skin Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to skin' (Xi; R38) in HSIS (Safe Work Australia). The available data support this classification. The chemical on unabraded rabbit skin produced erythema and oedema progressing to necrosis and sloughing of the superficial layers of skin. Irritation effects without scarring were reversible within 7 days (US EPA, 2004 and REACH, 2013).

Eye Irritation

The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia). The available data support this classification. In an eye irritation study in rabbits, the undiluted chemical caused slight superficial necrosis of the cornea, corneal irritation and conjunctival irritation. Effects were reversible within 48 hours. At 10 % concentration in propylene glycol, the chemical caused moderate conjunctival irritation and moderate to severe corneal irritation. Irritation effects persisted for 48 hours and were fully reversible after 12 days. The study showed that the undiluted chemical is not likely to cause permanent eye injury. However, strong solutions (10 %) of the chemical in propylene glycol may cause more serious eye injury than the undiluted chemical (US EPA, 2004 and REACH, 2013).

Sensitisation

Skin Sensitisation

No animal or human data are available. The chemical was predicted not to be a skin or respiratory sensitiser using the OECD QSAR Toolbox (QSAR Toolbox).

Repeated Dose Toxicity

Oral

In rats and mice, long term exposure to the chemical by oral route (13-weeks) was associated with excessive mortality, decreased body weight gain, testicular atrophy, forestomach lesions (hyperkeratosis and acanthosis) and adrenocortical degeneration. Peliosis hepatis (mottled-blue liver) and inflammation of the liver were also oberved in rats. These effects occurred at doses that also produced tumours

(US EPA, 2004 and REACH, 2013).

Dermal

No data are available.

Inhalation

In a 13-week repeat dose inhalation study (OECD TG 413), rats were exposed to 3, 10 and 40 ppm of the chemical for 6 hours/day, 5 days/week. At 40 ppm, decreases in body weight and body weight gain were observed in male rats throughout the study. The relative liver and kidney weights were increased. Scattered to diffuse hyperplasia of the respiratory epithelium of the turbinate was also reported. Some males of this group showed very slight focal individual epithelial cell necrosis of the respiratory epithelium. At 10 ppm, male rats showed isolated to scattered hyperplasia of the respiratory epithelium. In females, relative liver weights were increased at 10 and 40 ppm. The no observed adverse effect concentration (NOAEC) from this study is 3 ppm (23 mg/m^3) (US EPA, 2004 and REACH, 2013).

The toxicity of the chemical was examined in rats, rabbits, guinea pigs and monkeys following inhalation exposure to 25, 50 or 100 ppm (7 hours/day, 5 days/week). The number of exposures depend on the species of the experimental animals used.

At 100 ppm (0.77 mg/L), high mortality was observed in rats and rabbits. In rats, lung, liver and kidney weights were significantly increased. Histopathological examination showed thickening of the alveolar walls with leucocytic infiltration of the lungs, cloudy swelling of the liver, and slight congestion and haemosiderosis of the spleen. In rabbits, the liver tissues showed widespread central fatty degeneration with necrosis and slight cloudy swelling (US EPA, 2004; REACH, 2013).

At 50 ppm (0.38 mg/L), high mortality was observed in rats due to pneumonia and infections of the upper respiratory tract. Liver and kidney weights were significantly increased. Histological changes included patches of pneumonic consolidation in the lungs. In guinea pigs, decreased body weight gain, fatty degeneration of the liver, and slight interstitial congestion and oedema of the tubular epithelium of the kidney were reported. Monkeys showed central fatty degeneration of the liver and increased relative kidney weights. They appeared to be unwell and unkempt throughout the study. No toxicological effects were reported for rabbits (US EPA, 2004; REACH, 2013).

There was no evidence of adverse effects in all animals following exposure to 25 ppm (0.19 mg/L), except for high mortality in rats due to pneumonia and infections of the upper respiratory tract.

In chronic inhalation studies (US EPA, 2004), rats and mice were exposed to 0, 10 or 40 ppm (0, 77 or 307 mg/m^3) of 1,2-dibromoethane for 88 weeks, 6 hours/day, 5 days/week. In both species, high mortality and reduced weight gain were reported at the highest dose. In rats, dose-dependent hepatic necrosis, toxic nephropathy, testicular degeneration and atrophy, spermatic granulomas and degeneration of the adrenal cortex were reported. In female mice, dose-dependent epithelial hyperplasia of the lung alveoli, bronchioles, and bronchi were observed. Adenomatous hyperplasia of the lung and hyperplasia of the nasal cavity were reported in high dose females. Suppurative inflammation of the nasal cavities and hepatic necrosis were also reported from the lowest dose (10 ppm).

Proliferative lesions of the nasal epithelium in mice exposed to 10 or 40 ppm were also reported in another study. The target tissue following inhalation exposure in both species appears to be the nasal epithelium (US EPA, 2004).

Genotoxicity

This chemical is not classified for mutagenicity in the HSIS. Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies, the chemical is considered genotoxic. The chemical warrants classification as hazardous, Mutagen Category 3, with the risk phrase "Possible risk of irreversible effects (R68)" in the HSIS (Safe Work Australia).

A sufficient genotoxicity database is available for the chemical. Overall, in vitro bacterial mutation, mammalian choromosome aberration, mammalian gene mutation, sister chromatid exchange and mammalian micronucleus assays showed positive results. However, equivocal results were observed in germ cell in vivo assays (positive results for sex linked recessive lethal mutations in spermatozoa of *Drosophila melanogaster* but negative results when tested for dominant lethal and electrophoretically detectable specific locus mutations in germ cells of mice) (US EPA, 2004; REACH, 2012).

Cytogenetic examination (sister chromatic exchanges or chromosomal aberrations) of the peripheral lymphocytes of workers exposed to 1,2-dibromoethane gave equivocal results (US EPA, 2004).

Carcinogenicity

The chemical is classified as hazardous, Category 2 Carcinogen, with the risk phrase "May cause cancer (R45)" in the HSIS (Safe Work Australia). The data available support this classification.

Animal Studies

The carcinogenicity potential of the chemical was determined on the basis of long term oral and inhalation studies in rats and mice.

Oral

Male rats administered with 40 and 80 mg/kg of the chemical, 5 days/week for 2 weeks showed tumours in the forestomach and haemangiosarcomas. The lowest observed adverse effect level (LOAEL) was determined to be 40 mg/kg in this study (REACH, 2013).

In a combined rat and mice study, rats were administered up to 80 mg/kg bw while mice were administered up to 120 mg/kg bw of the chemical by gastric intubation, 5 days/week for 13 weeks. Rats showed increased incidence of squamous cell carcinomas of the forestomach in both sexes, hepatocellular carcinomas in females and haemangiosarcomas in males. Mice showed squamous cell carcinomas of the forestomach and alveolar/bronchiolar adenomas in both sexes. A no observed adverse effect level (NOAEL) could not be established in this study (REACH, 2013).

Dermal

Female mice were exposed dermally to 25 and 50 mg/kg of the chemical, 3 times a week for 440 to 594 days. There was a significant increase in the incidence of benign lung papillomas at doses ≥ 25 mg/kg, and a significant increase in the incidence of stomach tumours (papillomas and squamous cell carcinomas of the forestomach) above 25 mg/kg. The LOAEL in this study was determined to be 25 mg/kg (US EPA, 2004 and REACH, 2013).

Inhalation

In chronic inhalation studies, rats and mice were exposed to 10 and 40 ppm of the chemical 6 hours/day, 5 days per week for up to 103 weeks. Rats showed increased incidence of carcinoma, adenocarcinoma, adenomas of the nasal cavity and haemangiosarcoma of the circulatory system in both sexes. Increased mesotheliomas of the tunica vaginalis and increased in adenomatous polyps of the nasal cavity were observed in male rats only, while increased fibroadenomas of the mammary glands and alveolar/brionchiolar adenomas and carcinomas (combined) were seen in female rats.

In mice, alveolar/bronchiolar carcinomas and adenomas were seen in both sexes. In females, haemagiosarcomas of the circulatory system, fibrosarcomas in the subcutaneous tissue, carcinomas of the nasal cavity and adenocarcinomas of the mammary gland were observed (US EPA, 2004 and REACH, 2013).

Human studies

Several studies have been conducted to investigate the carcinogenic effects of the chemical in occupationally exposed workers. In two production facilities, two deaths (compared with 3.6 expected) from malignant neoplasms occurred in the first facility while five deaths (compared with 2.2 expected) were reported for the same effect in the second facility. Neoplasms in the lung, stomach and pancreas, and prostate reticulum-cell sarcoma were reported. The first facility did not manufacture other organic bromides and the exposure was primarily to the chemical. However, the second facility manufactured other organic bromide chemicals to which workers were also exposed (US EPA, 2004; IARC, 1999).

The results of other mortality studies of workers exposed to the chemical were inconclusive regarding its potential carcinogenicity in humans (US EPA, 2004; IARC, 1999).

The International Agency for Research on Cancer (IARC) has classified the chemical as ‘Probably carcinogenic to humans’ (Group 2A), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity based on animal studies.

Reproductive and Developmental Toxicity

This chemical is not classified for fertility and developmental effects in the HSIS. Based on the weight of evidence from the available reproductive and developmental studies, the chemical warrants classification as hazardous, Reproductive toxin Category 3, with the risk phrase "Possible risk of impaired fertility (R62)" in the HSIS (Safe Work Australia).

Oral

Bulls exposed to 10 doses of 4 mg/kg 1,2-dibromoethane on alternate days showed altered sperm morphology, decreased sperm motility, depleted number of sperm from seminiferous tubules and testicular abnormalities (US EPA, 2004).

Inhalation

The reproductive effects of the chemical in rats and mice have been examined in several studies. At 89 ppm (684 mg/m^3), reproductive effects included decrease testicular weight and serum testosterone levels; atrophy of the testis, epididymis, prostate and seminal vesicle; and impairment of reproductive performance. No treatment reproductive effects were seen at 19 or 30 ppm (146 or 300 mg/m^3). A NOAEC of 39 ppm was established in a reproductive study in rats and mice following inhalation exposure of 5 days /week for 10 weeks and for 7 days/week for 3 weeks, respectively (US EPA, 2004 and REACH, 2013).

Reported developmental effects in rats and mice consisted of decreased foetal body weight, increased resorptions, decreased foetal survival, and/or skeletal abnormalities at all doses (20, 38 and 80 ppm). High mortality was also reported for both species at 38 ppm and above. The lowest observed adverse effect concentration (LOAEC) in this study was established at 20 ppm (US EPA, 2004 and REACH, 2013).

Human studies

In humans, inhalation exposures to the chemical in various workplaces have been investigated. Inhalation exposure was investigated in:

- agricultural workers (unknown exposure level);
- forestry workers (short term exposure of up to 2165 ppb; 16.6 mg/m^3);
- workers employed in a chemical plant (up to 5 ppm; up to 38 mg/m^3) or production facility (unknown exposure level); and
- workers exposed to the chemicals when used as a fumigant (geometric mean of 88 ppb; 0.68 mg/m^3).

Overall, there were decreases in average sperm count, percentage of viable sperm and percentage of motile sperm following long term exposure. There were also increases in certain types of sperm morphological abnormalities such as tapered heads and abnormal tails. Short term exposure caused decreases in semen volume.

Decrease in reproductive performance, assessed by the number of live births in exposed workers' wives following long term exposure was reported. However, there were inadequate worker exposure data in this study, and potential exposure to other reproductive toxins and other confounding factors, such as dermal exposure cannot be excluded (US EPA, 2004).

Other Health Effects

Neurotoxicity

There were two animal studies reported examining developmental neurotoxicity. The effects observed, including cliff avoidance, circular swimming direction, slower ability to raise head and repressed open-filled ambulation, did not show a dose-response. No conclusive results were drawn from these studies (US EPA, 2004).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity, mutagenicity and possible effects on fertility) and systemic acute effects (acute toxicity by the oral, dermal and inhalation routes of exposure). The chemical can also cause skin, eye and respiratory irritation.

Public Risk Characterisation

The chemical is currently listed on Schedule 7 of the SUSMP. The current controls are considered adequate to minimise the risk to public health posed by domestic products containing the chemical. Therefore, the risk to public health is not considered to be unreasonable.

Occupational Risk Characterisation

Given the critical (systemic long term, systemic acute and local) health effects of the chemical, the chemical may pose an unreasonable risk to workers if adequate control measures to minimise dermal, ocular and inhalation exposures are not implemented. The chemical should be appropriately classified and labelled to ensure that a person conducting a business or undertaking (PCBU), e.g. employer, at a workplace has adequate information to determine appropriate controls. The data available support the need to amend the hazard classification in HSIS (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 and used in accordance with state and territory legislation (SUSMP).

Work Health and Safety

Products containing the chemical should be used according to label instructions.

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

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Toxic if swallowed (T; R25)* Toxic in contact with skin (T; R24)* Toxic by inhalation (T; R23)*	Toxic if swallowed - Cat. 3 (H301) Toxic in contact with skin - Cat. 3 (H311) Fatal if inhaled - Cat. 1 (H330)
Irritation / Corrosivity	Irritating to skin (Xi; R38)* Irritating to eyes (Xi; R36)* Irritating to respiratory system (Xi; R37)*	Causes skin irritation - Cat. 2 (H315) Causes serious eye irritation - Cat. 2A (H319) May cause respiratory irritation - Specific target organ tox, single exp Cat. 3 (H335)
Genotoxicity	Muta. Cat 3 - Possible risk of irreversible effects (Xn; R68)	Suspected of causing genetic defects - Cat. 2 (H341)
Carcinogenicity	Carc. Cat 2 - May cause cancer (T; R45)*	May cause cancer - Cat. 1B (H350)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)	Suspected of damaging fertility - Cat. 2 (H361f)

[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 industry

Control measures

Control measures to minimise the risk from dermal/ocular/inhalation exposure to the chemical should be implemented in accordance with the hierarchy of controls. Approaches to minimise risk include substitution, isolation and engineering controls. Measures required to eliminate or minimise risk arising from storing, handling and using a hazardous chemical depend on the physical form and the manner in which the chemical is used. Examples of control measures which may minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- using local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- air monitoring to ensure control measures in place are working effectively and continue to do so;
- minimising manual processes and work tasks through automating processes;
- work procedures that minimise splashes and spills;
- regularly cleaning equipment and work areas; and
- using protective equipment that is designed, constructed, and operated to ensure that the worker does not come into contact with the chemical.

Guidance on managing risks from hazardous chemicals are provided in the *Managing Risks of Hazardous Chemicals in the Workplace—Code of Practice* available on the Safe Work Australia website.

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

Obligations under workplace health and safety legislation

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

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

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

Information on how to prepare an (m)SDS and how to label containers of hazardous chemicals are provided in relevant codes of practice such as the *Preparation of Safety Data Sheets for Hazardous Chemicals— Code of Practice* and *Labelling of Workplace Hazardous Chemicals—Code of Practice*, respectively. These codes of practice are available from the Safe Work Australia website.

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

References

Environmental Health Criteria (EHC, 177) for dibromoethane (1996). Accessed October 2012 at http://www.inchem.org/documents/ehc/ehc/ehc177.htm#SubSectionNumber:3.2.2.

Galleria Chemica. Accessed April 2013 at http://jr.chemwatch.net/galleria/.

IARC (1999). Volume 71- Re-evaluation of some organic chemicals, hydrazine, and hydrogen peroxide. Accessed October 2012 at http://monographs.iarc.fr/ENG/Monographs/vol71/mono71-27.pdf.

National Pollutant Inventory (NPI) 1,2-dibromoethane. Accessed October 2012 at http://www.npi.gov.au/substances/dibromoethane/index.html.

OECD QSAR Toolbox version 3.1. http://www.oecd.org/chemicalsafety/assessmentofchemicals/theoecdqsartoolbox.htm

REACH Dossiers. Dibromoethane (106-93-4). Accessed april 2013 at http://echa.europa.eu/web/guest/information-on-chemicals/registered-substances.

Safe Work Australia (SWA). Guidance on the Classification of Hazardous Chemicals Under the WHS Regulations: Implementation of the Globally Harmonised System of Classification and Labelling of Chemicals (GHS). Accessed January 2013 at http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents.

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed September 2012 at http://hsis.safeworkaustralia.gov.au/HazardousSubstance.

U.S Environmental Protection Agency (EPA) (2004). Toxicological Review of 1,2-Dibromoethane (CAS No. 106-93-4) In Support of Summary Information on the Integrated Risk Information System (IRIS). Accessed November 2012 at http://www.epa.gov/iris/toxreviews/0361tr.pdf.

Work Health and Safety (WHS) Regulations 2011. Chapter 7 - Using, handling and storing prohibited carcinogens. Accessed October 2012 at http://www.comlaw.gov.au/Details/F2011L02664.

Work Health and Safety (WHS) Regulations 2011. Schedule 10 - Prohibited carcinogens, restricted carcinogens and restricted hazardous chemicals. Accessed October 2012 at http://www.comlaw.gov.au/Details/F2011L02664.

Last update 17 May 2013