Acetic acid, trichloro-: Human health tier II assessment

03 July 2015

CAS Number: 76-03-9

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.

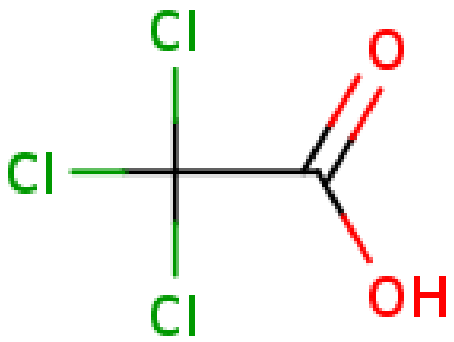
For more detail on this program please visit:www.nicnas.gov.au

Disclaimer

NICNAS has made every effort to assure the quality of information available in this report. However, before relying on it for a specific purpose, users should obtain advice relevant to their particular circumstances. This report has been prepared by NICNAS using a range of sources, including information from databases maintained by third parties, which include data supplied by industry. NICNAS has not verified and cannot guarantee the correctness of all information obtained from those databases. Reproduction or further distribution of this information may be subject to copyright protection. Use of this information without obtaining the permission from the owner(s) of the respective information might violate the rights of the owner. NICNAS does not take any responsibility whatsoever for any copyright or other infringements that may be caused by using this information.

Acronyms & Abbreviations

Chemical Identity

Synonyms	trichloroacetic acid aceto-caustin Tecane
Structural Formula	
Molecular Formula	C2HCl3O2
Molecular Weight (g/mol)	163.386
Appearance and Odour (where available)	colourless to white, crystalline solid with a sharp, pungent odour
SMILES	C(Cl)(Cl)(Cl)C(=O)O

Import, Manufacture and Use

Australian

The chemical is used as an etching agent in the glass industry.

It is not manufactured in Australia. The total volume introduced into Australia, reported under previous mandatory and/or voluntary calls for information, was 0.4 tonnes.

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorization and Restriction of Chemicals (REACH) dossiers; the Organisation for Economic Co-operation and Development Screening information data set International Assessment Report (OECD SIAR); Galleria Chemica; the Substances and Preparations in Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; the OECD High Production Volume chemical program (OECD HPV) and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB)

The chemical has reported cosmetic uses a:

- a fragrance agent; and
- a skin peel agent.
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The chemical has reported commercial use as a solvent.

The chemical has reported non-industrial use as a human and veterinary pharmaceutical.

Restrictions

Australian

This chemical is listed in the *Poisons standard—the Standard for the Uniform Scheduling of Medicines and Poisons* (SUSMP) in Schedules 4 and 6 (SUSMP, 2015).

Schedule 4:

TRICHLOROACETIC ACID for human dermal use **except** when in preparations containing 12.5 per cent or less of trichloroacetic acid for the treatment of warts other than anogenital warts.

Schedule 6:

'TRICHLOROACETIC ACID except:

(a) when included in Schedule 4 or 5; or

(b) in human dermal preparations containing 12.5 per cent or less of trichloroacetic acid for the treatment of warts other than anogenital warts.

Schedule 6 chemicals are described as 'Substances with a moderate potential for causing harm, the extent of which can be reduced through the use of distinctive packaging with strong warnings and safety directions on the label'. Schedule 6 chemicals are labelled with 'Poison' (SUSMP, 2015)'.

Schedule 4. Substances, the use or supply of which should be by or on the order of persons permitted by State or Territory legislation to prescribe and should be available from a pharmacist on prescription. Schedule 4 chemicals are labelled with 'Prescription Only Medicine, or Prescription Animal Remedy' (SUSMP, 2015).

International

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

- Association of South East Asian (ASEAN) Cosmetic Directive Annex II Part 1—List of substances which must not form part of the composition of cosmetic products
- EU Cosmetics Regulation 1223/2009 Annex II—List of substances prohibited in cosmetic products;
- New Zealand Cosmetic Products Group Standard—Schedule 4: Components cosmetic products must not contain; and
- Health Canada List of prohibited and restricted cosmetic ingredients (The Cosmetic Ingredient 'Hotlist').

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):

- C; R35 (corrosive)

Exposure Standards

Australian

The chemical has an exposure standard of 6.7 mg/m^3 (1 ppm) time weighted average (TWA) and 5 mg/m^3 (1 ppm) maximum workplace concentration (MAK).

International

The following exposure standards are identified (Galleria Chemica).

An exposure limit of 1-2 mg/m^3 TWA in different countries such as Denmark, Iceland and Portugal.

An exposure limit of 5 mg/m^3 TWA in Egypt, France, Ireland, Latvia, Norway, Russia, South Africa and the USA.

An exposure limit of 6.7-7.0 mg/m^3 (1 ppm) TWA in Abu Dhabi, Belgium, Bulgaria, Canada, Columbia, Croatia, Indonesia, Italy, Korea, Malaysia, New Zealand, Nicaragua, Peru, Singapore, Spain, Switzerland, Taiwan, the UAE, Uruguay and Venezuela.

Health Hazard Information

Trichloroacetic acid (TCA) is a relatively stable organic compound which dissociates into the trichloroacetate ion (Cl_3COO^-) and hydrogen ion (H^+) in aqueous solution.

Health hazard information for sodium trichloroacetate (CAS No. 650-51-1) has been included in this report, when data for the chemical being assessed are not available,. Similar to the chemical being assessed, sodium trichloroacetate is expected to exist almost entirely as the trichloroacetate ion in biological solutions, and therefore is considered to be a suitable analogue for the chemical for systemic effects.

Toxicokinetics

The chemical is rapidly absorbed orally and dermally, in humans and animals. The chemical is metabolised minimally and accumulates to a steady-state after successive exposures. Elimination from internal compartments is fast from plasma, red blood

cells, muscle and fat, moderate from the kidneys and skin and slow from the liver and small and large intestines. The majority of elimination is via the urine (up to 85 %) as the parent compound, with minor elimination in faeces (2-8 %) and metabolised to carbon dioxide in expired air (4-12 %) (IARC, 2014; IRIS; OECD).

Acute Toxicity

Oral

The chemical has low acute toxicity based on results from animal tests following oral exposure. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Observed sub-lethal effects included narcosis, lethargy, increasing weakness, changes in motor activity, salivation, collapse and coma (REACHa).

Dermal

No data are available for the chemical. However, sodium trichloroacetate has low acute toxicity based on results from animal tests following dermal exposure. No mortalities were observed and the LD50 in rats is >2000 mg/kg bw (REACHb).

Inhalation

No data are available for the chemical. However, sodium trichloroacetate has low acute toxicity in animal tests following inhalation exposure. No mortalities were observed (median lethal concentration (LC50) >20 mg/L) (REACHb).

Corrosion / Irritation

Corrosivity

The chemical is classified as hazardous with the risk phrase 'Causes severe burns' (C; R35) in the HSIS. No animal data are available to evaluate this classification. Humans exposed to the chemical (35 %) have reported cases of severe eye irritation, which was fully reversible within 3 days. Scarring has resulted from use of the chemical in dermal peels (35-50 %) (REACHa).

While the available data do not support this classification, in the absence of more comprehensive information, particularly at higher concentrations, there is insufficient evidence to support a recommendation to amend this classification.

Sensitisation

Skin Sensitisation

Of the five skin sensitisation studies reported in REACH dossier (REACHa) only one study gave positive results with TCA. All other sensitisation studies were negative. The chemical is not considered to be a skin sensitiser.

Repeated Dose Toxicity

Oral

No serious adverse effects were reported in repeated dose toxicity studies with TCA. Studies in rats and mice indicate that TCA primarily affects the liver, although effects on the lungs and kidneys have also been noted in rats. Observed hepatic effects in rodents include increased size and weight, collagen deposition, indications of altered lipid and carbohydrate metabolism, histopathological changes, peroxisome proliferation, and evidence of lipid peroxidation (USEPA, 2011).

In a 90-day oral repeat dose toxicity study (OECD TG 408), TCA (0, 50, 500 and 5000 ppm; corresponding to 0, 4.1, 35.5 and 355 mg/kg bw/d) was administered in the drinking water to male Sprague Dawley (SD) rats. A no observed adverse effect level (NOAEL) of 35.5 mg/kg bw/day was established, based on increased liver and kidney weight relative to body weight and histopathological changes to the liver and kidneys observed at higher concentrations (355 mg/kg bw/day) (REACHa).

In a 90-day oral repeat dose toxicity study (TG 408) sodium trichloroacetate (0, 250, 630, 1600, 4000 and 10000 ppm) was administered in feed to SPF-Wistar rats (n=10/sex/group), a NOAEL of 4000 ppm (equivalent to 365 mg/kg bw/day) was reported. Effects observed at higher concentrations (10000 ppm) were limited to decreased body weight (REACHa).

In a 90-day oral repeat dose toxicity study (TG 409) sodium trichloroacetate (0, 500, 2000, 4000 and 8000 ppm) was administered in feed to Beagle dogs (n=3/sex/group), a NOAEL of 500 ppm (equivalent to 30 mg/kg bw/day) was reported. Effects observed at higher concentrations (2000 ppm) included: anaemia and changes to white blood cell count, liver and heart tissue and spermatogenesis. Muscle atrophy and the presence of protein and bilirubin were observed in the two highest dose groups (REACHb).

In a two-year oral chronic toxicity study (TG 452) sodium trichloroacetate (0, 250, 630, 1600 and 10000 ppm) was administered in feed to Wistar rats (n=25/sex/group). A NOAEL of 1600 ppm (equivalent to 80 mg/kg bw/day) was reported. Effects observed at higher concentrations (10000 ppm) were limited to decreased body weight. No neoplasms, histopathological or morphological changes were observed (REACHb).

Dermal

No data are available for long term repeated dose effects.

In a 5-day non-guideline dermal exposure study, sodium trichloroacetate (100, 300 and 900 mg/kg bw/day) was applied to the skin of New Zealand White rabbits (n=3/sex/dose) under semiocclusive conditions for 8 hours. No effect on behaviour, food consumption or body weights were observed (REACHb).

Inhalation

No data are available.

Genotoxicity

Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies the chemical gave equivocal results for genotoxicity.

The chemical gave mixed results in an in vitro mammalian chromosome aberration assay (OECD TG 473) in human lymphocytes with and without metabolic activation. The chemical gave positive results when administered unbuffered, but when neutralised to pH 7, it gave negative results up to cytotoxic concentrations. The authors concluded that the chemical has no intrinsic potential to induce cytogenic damage (REACHa).

The chemical showed positive results in the following in vitro genotoxicity tests (REACHa):

- bacterial reverse mutation assay (Ames test) with *Salmonella typhimurium* (TA100), with or without metabolic activation;
- mammalian cell gene mutation assay (OECD TG 476) with mouse lymphoma L5178Y cells, with metabolic activation.

The chemical showed negative results in the following in vitro genotoxicity tests (REACH):

- bacterial reverse mutation assay (Ames test, OECD TG 471) with *Salmonella typhimurium* (TA 98, 100, 1535 and RSJ100) and *Escherichia coli* (PQ37 and WP2S), with or without metabolic activation;
- DNA damage by single strand breaks with F344 rat hepatocytes, without metabolic activation; and
- mammalian cell gene mutation assay (OECD TG 476) with mouse lymphoma L5178Y cells, without metabolic activation.

Positive results were reported for TCA in the following in vivo assays (IARC, 2014; REACHa) :

- chromosome aberration assays in mammalian bone marrow cells (OECD TG 475) in mice, chickens and newt larvae exposed to the chemical by intraperitoneal (i.p.) injection or oral administration;
- DNA damage by single strand breaks in SD rats and B6C3F1 mice treated by oral gavage at doses of 98 and 0.98 mg/kg bw, respectively; and
- a sperm morphology test in male Swiss mice treated by i.p. injection at doses up to 500 mg/kg;

Negative results were reported for trichloroacetic acid in the following in vivo assays (IARC, 2014; REACHa):

- a mammalian erythrocyte micronucleus assay (OECD TG 474) in mice at i.p. doses up to 1300 mg/kg bw; and
- a genome mutation assay (non-guideline) in male SD rats at oral gavage doses up to 1500 mg/kg bw.

OECD concluded that, on balance, the available evidence does not suggest that TCA is a mutagen (OECD).

Carcinogenicity

No human data are available for carcinogenicity of TCA. In several mouse chronic studies, reviewed by IARC, substantial increases in the incidences of hepatocellular adenomas and hepatocellular carcinomas were observed when TCA was administered in drinking water (IARC, 2014).

Four drinking-water studies in male mice and two studies in female mice showed increased incidences of hepatocellular adenoma and/or hepatocellular carcinoma. Two initiation–promotion studies in mice showed that TCA is an efficient promoter of hepatocellular tumours initiated by *N*-ethyl-*N* nitrosourea and *N*-methyl-*N*-nitrosourea (IARC, 2014; REACHa).

In rats the incidence of liver tumours was not greatly increased. In a 104-week rat study, male Fischer 344 (F344/N) rats were given drinking-water containing TCA at a concentration of 0, 0.05, 0.5,or 5 g/L (DeAngelo *et al.*, 1997). The data were reported as the percentage of rats examined with hepatocellular adenoma (4.4 %, 4.2 %, 15 %, and 4.6 % for 0, 0.05, 0.5 and 5 g/L group, respectively) and hepatocellular carcinoma (i.e. 0 %, 0 %, 0 %, and 4.6 % for each dose group, respectively). Although there were increases in the incidence of adenoma in the group receiving the intermediate dose, and in the incidence of carcinoma in the group receiving the highest dose, these increases were not statistically significant.

Based on the evidence for carcinogenicity in mice studies, the International Agency for Research on Cancer (IARC) has classified TCA as ‘Possibly carcinogenic to humans’ (Group 2B).

There is evidence suggesting that TCA may act through multiple nongenotoxic mechanisms, leading to liver carcinogenesis. The IARC has suggested, (a) epigenetic effects (global DNA hypomethylation), (b) oxidative stress (oxidative DNA damage and lipid peroxidation, activation of phagocytic cells that may lead to generation of oxidants), (c) increase in cell proliferation (an effect not observed in PPARa-null mice in a 7-day study), (d) induction of the peroxisome proliferation response (strong direct and indirect evidence for activation of PPARa in rodents, limited evidence for TCA as a ligand of human PPARa), and (e) disruption of gap-junctional intercellular communications (limited evidence from several studies in rat cells in vitro*)* as the probable mechanisms of carcinogenicity in rodents (IARC, 2014).

In a 14-week dermal study, male SKH/HR1 mice (n=5/group) were treated with the chemical (35 %) in a 15 x 15 mm treatment area for 20 minutes, 3 days per week. The mice were exposed to UVB radiation every 4 weeks for 18 weeks. Increased susceptibility to tumours was reported in the treatment area compared to controls while a paradoxical decrease in tumours was reported in the non-treatment area of treated mice (REACHa).

The mouse, and in particular the B6C3F1 mouse, is relatively susceptible to liver tumours, and the background incidence of this tumour is generally high. For these reasons, use of mouse liver tumour data in risk assessment has been a subject of controversy (King-Herbert and Thayer, 2006). Thus, although the consistent positive evidence in B6C3F1 mice raises a concern for carcinogenic effects in humans, this assessment attaches greater weight to the lack of evidence in other strains or species than to the replication of positive results in this one strain. Accordingly, this assessment concludes that there is insufficient evidence to recommend classification of TCA for carcinogenicity.

Reproductive and Developmental Toxicity

TCA did not show any effects on the reproductive system in experimental animals. Developmental effects, especially soft tissue malformation, were only noted at doses that also resulted in maternal toxicity.

In a sperm head morphology study in male B6C3F1 mice (n=24/group), sodium trichloroacetate (0, 625, 1250 and 2500 mg/kg bw/day) was administered by oral gavage for five days. No morphological changes related to reproductive toxicity were observed in animals sacrificed at 21 and 35 days (REACHa).

In a prenatal developmental toxicity study (OECD TG 414), female SD rats (n=18/dose) were administered the chemical by oral gavage at concentrations of 0, 300 mg/kg bw/day during gestation days (GD) 6–15 and foetuses assessed for ocular malformation. No gross malformation was observed. Non-statistically significant decreases in lens and globe areas were reported with statistically significant decreases in foetal mean body weight compared to controls (REACHa).

Pregnant Long-Evans rats were dosed with 0, 330, 800, 1200 or 1800 mg/kg bw/day TCA (adjusted to pH 7 by NaOH) by gavage on GDs 6–15. Clinical signs of toxicity and body weight gain were monitored throughout the exposure period. The dams were sacrificed on GD 20. Evidence of maternal toxicity was observed in all TCA treatment groups as indicated by a significant increase in spleen (up to 74 % increase) and kidney (up to 24 % increase) weights when compared with the control group. Dams exposed to 800, 1200, or 1800 mg/kg bw/day had significantly decreased body weight gains. The number of litters totally resorbed was significantly increased (5/21 and 12/20, respectively), and the number of viable litters (14/21 and 8/20, respectively) was significantly decreased at 1200 and 1800 mg/kg bw/day. Developmental effects were observed at all doses and included significant decreases in mean foetal weight; significant decreases in foetal crown-rump length; and increased percentages of foetuses affected per litter with total soft-tissue malformations. The maternal and developmental LOAELs in this study are 330 mg/kg bw/day. Maternal and developmental NOAEL values for TCA could not be determined because adverse effects were observed at all tested doses (Smith et al., 1989).

In a battery of studies in rats, the effect of TCA on the developing testes (Singh, 2005a), developing ovaries (Singh, 2005b), and developing brains were studied (Singh, 2006). Charles Foster rats were treated with 0, 1000, 1200, 1400, 1600, or 1800 mg/kg bw/day TCA by gavage on GDs 6–15.The pregnant rats were sacrificed on GD 19, and the foetuses and placenta were collected for examination. Maternal weight gains were statistically significantly decreased at TCA doses of ≥1200 mg/kg bw/day (38–46 %).

The percentage of post implantation loss in rats was significantly increased in a dose-related manner (22 % at 1000 mg/kg bw/day versus 3 % for control group); however, no external abnormalities were observed. The average weights of the foetal testes were significantly reduced when compared to the control at ≥1200 mg/kg bw/day. Histological examination of foetal rat testes of the 1200 mg/kg bw/day dose group showed a reduction in the diameter of the seminiferous tubules. At the higher doses, reduction in length of the seminiferous tubules was also reported. Examination of the testes at higher magnification revealed increased apoptosis of the gonocytes as well as the Sertoli cells within the seminiferous tubules in comparison to the controls at ≥1200 mg/kg bw/day.

The average weights of the foetal ovaries in rats were significantly reduced for the dose groups ≥1400 mg/kg bw/day (Singh, 2005b). Histological examination of the foetal ovaries showed small size cells with less prominent nuclei at the coelomic epithelium with ≥1,400 mg/kg bw/day TCA. The cortical cords proliferating from the coelomic epithelium traversing the gonads were either shortened or lacking. Oocytes in the ovarian stroma showed shrinkage in size with distorted cell membrane and indistinct nucleus, suggestive of cell apoptosis. The number of oocytes and the size of ovary were reduced. Singh (2005b) suggested that the gonadal changes were due to anoxia and oxidative stress resulting from TCA exposure.

Foetal brains of rats in the different dose groups from the above study were evaluated. Mean foetal weight and foetal brain weight decreased significantly at TCA doses ≥1000 mg/kg bw/day; while the length of the foetal brain increased significantly at 1,000 and 1200 mg/kg bw/day (about 10 % at 1000 mg/kg bw/day) but decreased significantly (8–16 %) at TCA doses ≥1400 mg/kg bw/day when compared with controls (Singh, 2006). At doses ≥1000 mg/kg bw/day, the foetal brains showed hydrocephalus with breach of the ependymal lining, altered choroid plexus architecture, and increased apoptosis. Vacuolation of the neutrophils was a prominent feature with TCA exposure, with an incidence of 26 % at 1000 mg/kg bw/day (0 % in controls) and reached 100 % in the 1600 and 1800 mg/kg bw/day dose groups. The incidence of brain haemorrhages increased to 30 % at TCA doses ≥1200 mg/kg bw/day (0 % in controls) and reached 100 % at 1800 mg/kg bw/day. The rat foetal brain was concluded to be susceptible to the toxic effects of TCA. However, maternal toxicity was also noted at these doses, which are higher than the dose range for which developmental toxicity classification is normally applied.

Risk Characterisation

Critical Health Effects

The critical health effect for risk characterisation is corrosivity. There is limited evidence of carcinogenic potential for TCA based on significantly increased incidences of liver tumours in B6C3F1 mice exposed via drinking water for 52–104 weeks, and lack of treatment-related tumours in a study of male F344/N rats following lifetime exposure in drinking water.

Public Risk Characterisation

Although use in cosmetic and domestic products in Australia is not known, the chemical is reported to be used in cosmetic and domestic products overseas.

The chemical is currently listed on Schedules 4 and 6 of the SUSMP. A number of warning statements, first aid instructions and safety directions relating to TCA apply. The current controls are considered adequate to minimise the risk to public health posed by domestic and cosmetic products containing the chemical; therefore, the chemical is not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

Given the critical systemic long-term and local health effects, the chemical could pose an unreasonable risk to workers unless adequate control measures to minimise dermal and ocular 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

Current risk management measures are considered adequate to protect public and workers' health and safety, provided that all requirements are met under workplace health and safety, and poisons legislation as adopted by the relevant state or territory. No further assessment is required.

Regulatory Control

Public Health

Products containing the chemical should be labelled in accordance with state and territory legislation (SUSMP, 2014).

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]
Irritation / Corrosivity	Causes severe burns (C; R35)*	Causes severe skin burns and eye damage - Cat. 1A (H314)

[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 dermal and ocular 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 that 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;
- health monitoring for any worker who is at risk of exposure to the chemical[s], if valid techniques are available to monitor the effect on the worker's health;
- 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 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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