Ethanol, 2-[(2-aminoethyl)amino]-: Human health tier II assessment

21 April 2016

CAS Number: 111-41-1

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.

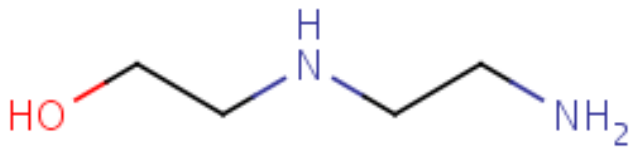
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	N-(aminoethyl)ethanolamine 2-(2-aminoethylamino)ethanol N-(2-hydroxyethyl)ethylenediamine AEEA
Structural Formula	
Molecular Formula	C4H12N2O
Molecular Weight (g/mol)	104.15
Appearance and Odour (where available)	colourless to yellowish liquid with an amine-like odour
SMILES	C(O)CNCCN

Import, Manufacture and Use

Australian

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

International

The following international uses have been identified through the European Union (EU) Registration, Evaluation, Authorisation 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 US Environmental Protection Agency's Aggregated Computer Toxicology Resource (ACToR) and; the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

The chemical has reported cosmetic use as an impurity in surfactants in shampoos and other cosmetic products.

The chemical has reported domestic use as an impurity in surfactants in dishwashing detergents and other cleaning products.

The chemical has reported commercial uses, including as:

- a surface smoothing agent for polystyrene and polyvinyl chloride resins;
- an additive in paints; and
- a textile finishing compound.

The chemical has reported site-limited use, including as an intermediate in the manufacture of corrosion inhibitors, chelating agents, detergents/emulsifiers, oils used in metal processing/manufacturing and amide wax.

The chemical has non-industrial uses in pharmaceuticals and pesticides.

Restrictions

Australian

No known restrictions have been identified.

International

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

- US Food and Drug Administration (FDA) Indirect food additives: Adhesives and components of coatings - Substances for use only as components of adhesives; and
- European Union (EU) Annex I to Directive 67/548/EEC on Classification and Labelling of Dangerous Substances - Reprotoxic Substances.

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; R34 (corrosion);
- Xi; R43 (sensitisation);
- T; R61 Repr. Cat. 2 (developmental toxicity); and
- T; R62 Repr. Cat. 3 (reproductive toxicity).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica).

An exposure limit of 3 mg/m^3 time weighted average (TWA) in various countries such as Latvia.

Health Hazard Information

The chemical is commonly known as aminoethylethanolamine (AEEA) and will be referred to as AEEA in the report.

Toxicokinetics

In a study in accordance with OECD Test Guideline (TG) 417, female Wistar rats were administered ^{14}C-radiolabelled AEEA by oral gavage at doses of 0.5 or 50 mg/kg bw in non-pregnant rats or 50 mg/kg bw in pregnant rats (three animals/dose). The chemical was well absorbed by oral administration at > 85 % within 48 hours in all dose groups. Maximum plasma levels were achieved at 30 minutes and one hour after dosing in non-pregnant and pregnant rats, respectively. The elimination half-life was biphasic with values of between 1.6 — 1.8 hours and 16.7 — 17.3 hours, respectively, with no significant differences in non-pregnant and pregnant rats. The chemical was well eliminated, with remaining radioactivity in tissues between 2.3 — 3.0 % of the administered dose. The main route of excretion was via the urine. Between 85 — 98 % was excreted in the urine within 48 hours, 5.2 — 11 % recovered in faeces and 0.02 — 0.03 % as expired CO_2. The majority of the chemical was excreted as the unchanged parent chemical (55 — 65 %), followed by the N-acetylated metabolite (5 — 11 %). The pregnancy status of the animals did not cause significant differences in absorption, elimination, excretion, or the metabolic profile following oral administration of the chemical (OECD, 2009; REACH).

In the same study, the dermal toxicokinetics were also assessed in female Wistar rats (eight animals/dose), using a 25 % solution containing ^{14}C-radiolabelled AEEA at 480 mg/kg bw. The chemical was applied under semi-occlusive conditions for 8 hours before washing. The chemical was absorbed to a moderate extent through the skin with a reported absorption of 7.73 % of the total applied dose. Total recovery of the absorbed dose was 90.97 %, and the main route of elimination was through the urine at 3.04 % of the administered dose (OECD, 2009; REACH).

In a study in pregnant Wistar rats (three animals/dose), ^{14}C-radiolabelled AEEA was orally administered at 300 mg/kg bw daily during gestation day (GD) 17 through 19 to two groups, with observation periods of six and 48 hours, respectively, after the final dose. The chemical was able to cross the placenta such that the blood level in the foetus was 80 % of the level measured in the dam after 6 hours. Levels were reduced after 48 hours. The chemical was reported to be distributed evenly in the foetus after 6 hours between the aortic arch, descending aorta, and the remainder of the tissues. After 48 hours the concentration was greater in the aortic vessels compared to the carcass (REACH).

The ability of the chemical to be transferred by lactation was assessed in the same study. Female Wistar rats (three animals/dose) were administered ^{14}C-radiolabelled AEEA by gavage at 250 mg/kg bw/day on lactation day (LD) one to five or 300 mg/kg bw/day on LD six to 12. Pup exposures were calculated to be 9.6, 8.8 and 4.5 % of the maternal daily administered dose on LD 4, 8 and 12, respectively (REACH).

Acute Toxicity

Oral

The chemical has low acute toxicity based on results from animal studies following oral exposure. The reported effects are consistent with the corrosivity of the chemical and hazard classification for acute toxicity is not considered warranted.

The acute toxicity of the chemical was assessed in a study conducted in accordance with OECD TG 401 in Wistar rats (five animals/sex/dose). The chemical was administered by oral gavage at doses of 1470, 2150, 3160 or 5000 mg/kg bw. The median lethal dose (LD50) was reported at 2150 mg/kg bw. Observed sub-lethal effects included dyspnoea, apathy, staggering and poor general state. Congestive hyperaemia was reported. Additional effects attributed to the corrosive effects of the chemical were at the local site of action, including red-liquid contents and dilation on the stomach and haemorrhagic gastritis at the highest dose. Intestinal effects included red-liquid contents and red coloured mucosa (OECD, 2009; REACH).

Further studies have reported LD50 values in rats to be > 2000 mg/kg bw (OECD, 2009; HSDB), with similarly reported sub-lethal effects and local effects in the gastric and intestinal mucosa attributed to the corrosivity of the chemical (OECD, 2009).

Dermal

The chemical has low acute toxicity based on results from animal studies following dermal exposure. The reported effects are consistent with the corrosivity of the chemical and hazard classification for acute toxicity is not considered warranted.

The acute toxicity of the chemical was assessed in a study in accordance with OECD TG 402 in Sprague Dawley (SD) rats at 400 (five animals/sex/dose) or 2000 mg/kg bw (10 animals/sex/dose) for 24 hours under occlusive conditions. The LD50 value was determined to be > 2000 mg/kg bw. Local skin irritation effects were reported; however, no mortalities or systemic effects were observed (REACH). Other LD50 values were reported to be > 2000 mg/kg bw in rabbits (Vienna White and New Zealand White) and 1800 — 1854 mg/kg bw in guinea pigs (HSDB; OECD, 2009).

Inhalation

The chemical is not acutely toxic at its saturated vapour pressure based on results from animal tests following inhalation exposure. The median lethal concentration (LC50) was reported to be > 51.3 mg/m³ air for multiple studies. The reported effects are consistent with the corrosivity of the chemical. The data are insufficient to determine hazard classification for acute toxicity.

The acute toxicity of the chemical was assessed in a study conducted similarly to OECD TG 403. Rats (strain unspecified; six animals/sex/dose) were exposed (whole-body) to saturated vapours of the chemical for eight hours. Substance-related eye irritation was reported. No mortalities or other treatment-related findings were found upon necrospy (REACH).

In a study in female Wistar rats (six animals/dose), animals were exposed (whole-body) to near saturated vapours of the chemical for six to eight hours. No mortalities, signs of systemic toxicity or abnormalities were reported at necropsy (OECD, 2009; REACH).

Corrosion / Irritation

Corrosivity

The chemical is classified as hazardous with the risk phrase ‘Causes burns’ (C; R34) in the HSIS (Safe Work Australia). The available data support this classification.

Eye Corrosivity

The eye irritation potential of the chemical was assessed in a study in accordance with OECD TG 405 in one rabbit (strain unspecified). A dose of 0.05mL of the chemical was instilled into one eye. The mean scores for erythema/oedema/corneal (opacity) at both one and 24 hours were 2,3,2, respectively. These effects were not reversible within eight days. Bleeding and formation of pus and staphyloma occured after eight days (OECD, 2009; REACH).

Skin Corrosivity

The skin irritation potential of the chemical was assessed in accordance with OECD TG 404 in Vienna white rabbits (six animals/dose). A dose of 0.5 mL of the chemical was applied to the clipped intact dorsal skin area for one or four hours. The mean scores for erythema and oedema were 2.09/1.33 and 3/2, for the one-hour and four-hour exposure groups, respectively. Animals displayed necrosis at both doses and this was not reversible within eight days (OECD, 2009; REACH).

In a study in accordance with OECD TG 404, New Zealand White rabbits (six animals/dose) were administered 0.5 mL of the chemical to the shaved dorsal flank areas under semi-occlusive conditions for three minutes or four hours. The mean scores for one, 24, 48 and 72 hours after exposure for erythema and oedema were 1.33/0.67 and 2.57/2.80, after an exposure time of three minutes and four hours, respectively. The development of necrosis leading to scar formation after 14 days was observed in the four-hour exposure group (REACH).

Sensitisation

Respiratory Sensitisation

No animal data are available; however, evidence in humans suggest that the chemical may cause allergic respiratory responses (See **Observations in Humans**).

Skin Sensitisation

The chemical is classified as hazardous with the risk phrase ‘May cause sensitisation by skin contact’ (R43) in the HSIS (Safe Work Australia). Available observations in humans, and positive results reported in local lymph node assays (LLNA) and a guinea pig maximisation test (GPMT) support this classification.

The skin sensitisation potential was assessed in an LLNA study conducted similarly to OECD TG 429 in female Balb/c mice (four animals/dose). The chemical was topically administered on the dorsal surface of each ear lobe once daily, for three consecutive days at 0, 2.5, 5, 10 or 20 % in acetone/olive oil (4:1 v/v). The extent of ^{3}H-thymidine incorporation in the lymph nodes was used to calculate stimulation indices (SI). The SI for the dose groups were 2.2, 2.8, 6.3 and 14.7, respectively, and the calculated estimated concentration required to produce a 3-fold increase in lymphocyte proliferation (EC3) was 5.3 %. On the basis of these findings, AEEA is considered to be a skin sensitiser (OECD, 2009 ; REACH).

In another LLNA study conducted in accordance with OECD TG 429, female CBA mice (six animals/dose) were administered the chemical at 0, 3, 10 or 30 % in acetone. The SI for the dose groups were 2, 1.72 and 6.6, respectively, and the calculated EC3 value was 15.2 % (REACH).

The chemical was also reported to produce a positive result in a GPMT at 5 % in water for intradermal injection at 50 % at for topical induction, and challenge at 25 %. (OECD, 2009; TSCATS).

Observation in humans

The chemical has industrial uses as a component in soldering flux and as a surfactant in cosmetic products; thus, most human exposures are attributed to these exposure scenarios.

Skin Sensitisation

In a study of cable joiners with contact dermatitis, the chemical produced a positive response in all 23 patients during patch testing for both neat AEEA and in a common flux formulation. The severity of the sensitisation response was reported to be stronger after 96 hours than 48 hours' exposure (OECD, 2009).

In a study on 26 electronics industry workers with contact dermatitis, 22 cases were attributed to soldering flux containing AEEA at 5 %. Four patients tested positive for sensitisation to 5 % AEEA when tested dermally (OECD, 2009).

In a study of patients sensitised to cosmetics containing sodium lauroamphoacetate, of which AEEA is present as a manufacturing impurity, four suspected cases and 20 control patients were patch tested with sodium lauroamphoacetate (containing AEEA as an impurity) at 1 % in various vehicles, and AEEA in water at 0.5, 0.1, 0.05, 0.01, and 0.005 % concentration. The chemical did not induce a sensitisation response at any concentration in control patients; however, all sensitised patients responded positively to the two highest doses of AEEA. In addition, two patients responded positively to 0.05 and 0.01 % and one patient to the lowest dose (OECD, 2009).

Respiratory Sensitisation

Two patients, both cable joiners by occupation, were exposed to the fumes produced from AEEA or soldering flux containing AEEA, dropped onto a heated sheet of aluminium. Following inhalation of the chemical, delayed but severe bronchoconstriction lasted for several days in patients. This effect was also observed in the control patient, who was believed not to be previously sensitised to the chemical. Onset of symptoms occured approximately three hours after exposure (OECD, 2009).

Three patients, also cable joiners, displayed severe allergic asthma following inhalation of fumes from the flux or AEEA, with delayed onset of symptoms occuring between two - four hours in two patients, and after 14 hours in the third patient. The symptoms persisted for several days. The asthma was characterised by reduction of functional expiratory volume in one-second (FEV1) by 14 % to 58 % (OECD, 2009).

Repeated Dose Toxicity

Oral

Based on the data available, the chemical is not considered to cause serious damage to health through repeated exposure by the oral route. The toxic effects of the chemical are attributed to its corrosivity and are not considered relevant for classification for this endpoint.

In a sub-chronic study conducted in accordance with OECD TG 407, SD rats (six animals/dose) were administered the chemical by oral gavage at doses of 0, 60, 250 or 1000 mg/kg bw/day for 28 days. A recovery group (six animals) was sacrificed after an additional 14 days. Clinical chemistry findings included a significant increase in aspartate aminotransaminase (AST) in the top two dose groups, and decreases in chloride and cholesterol in males and females, respectively, in the highest dose group. Haemoglobin was reduced in both sexes at the highest dose. Urinalysis parameters were only different in females, with the urine specific gravity and protein increased at the two highest doses, and decreased in volume at the highest dose. Organ weight changes included an increase in the relative weight of kidneys and a decrease in the relative adrenal weight at the highest dose. Histopathological changes included deposition of amphophilic bodies and swelling of the renal proximal tubules in the cortico-medullary junction in the 250 mg/kg bw/day group in males and in both sexes at the highest dose. Thickening of the limiting ridge of the stomach at the top two doses was attributed to the corrosivity of the chemical. The kidney and stomach lesions were

still apparent in the 14 day recovery groups; however, other treatment-related changes were reversible within 14-days. The no-observed-adverse-effect-level (NOAEL) was 60 mg/kg bw/day in both sexes. (OECD, 2009; REACH).

Dermal

Based on the data available, the chemical is not considered to cause serious damage to health through repeated exposure by the dermal route. The toxic effects of the chemical are attributed to its corrosivity and are not considered relevant for classification of this endpoint.

The repeated-dose toxicity of the chemical was assessed in a 28-day dermal study in accordance with OECD TG 410 in Fischer 344 rats (five animals /sex/dose). The chemical was applied at 0, 100, 300 or 1000 mg/kg bw/day for six hours/day, five days/week under semi-occlusive conditions. The only treatment-related effects were localised skin effects at the site of application consistent with the corrosivity of the chemical, including scabs at the highest dose. Microscopic examination at the test site revealed ulcer formation and inflammation of the dermis and epidermis. The no-observed-effect-level (NOEL) was determined to be 1000 mg/kg bw/day (OECD, 2009; REACH).

Inhalation

No data are available.

Genotoxicity

Based on the weight of evidence from the available well-conducted in vitro and in vivo genotoxicity studies, the chemical is not considered to be genotoxic.

In vitro

The chemical tested negative in a bacterial reverse mutation assay in *Salmonella typhimurium* in strains TA98, TA1537, and TA100 with or without metabolic activation, and a weakly positive result was obtained in TA1535 with metabolic activation, at concentrations above 3333 µg/plate (OECD, 2009; REACH). In another reverse gene mutation assay, the chemical tested negative in strains TA 1535, TA 1537, TA 98 and TA 100, with or without metabolic activation at concentrations up to 8000 µg/plate (OECD, 2009).

The chemical tested negative in the following in vitro studies;

- Mammalian cell gene mutation test (OECD TG 476) in Chinese hamster ovary (CHO) cells with and without metabolic activation (OECD, 2009; REACH).
- Sister chromatid exchange (OECD TG 479) in CHO cells, with and without metabolic activation at concentrations up to 4.12 mg/mL (OECD, 2009; REACH)
- Mammalian chromosome aberration test (OECD TG 473) in Chinese hamster lung cells with and without metabolic activation at concentrations between 0.25 - 1 mg/ml. No increase in structural changes was reported and an increase in polyploidy was only significant at higher doses after 48 hours (OECD, 2009; REACH).
- Unscheduled DNA synthesis (UDS) (OECD TG 482) in rat hepatocytes at doses up to 1030 µg/ml (OECD, 2009; REACH).

In vivo

The genotoxicity potential of the chemical was assessed in a mammalian erythrocyte micronucleus test conducted according to OECD TG 474 in Crj:BDF1 mice (five animals/sex/dose). The chemical was administered as single dose by oral gavage at 0, 500, 1000 and 2000 mg/kg bw. No increases in frequency of micronucleated polychromatic erythrocytes were reported (OECD, 2009; REACH).

The chemical produced a negative result in male *Drosophila melanogaster* in a study in accordance with OECD TG 477 (sex-linked recessive lethal assay) in both feeding and injection tests, at concentrations of 52 000 and 3 400 ppm, respectively.

(OECD, 2009; REACH).

Carcinogenicity

No data are available.

Reproductive and Developmental Toxicity

The chemical is classified as hazardous—Category 2 substance toxic to reproduction—with the risk phrase 'May cause harm to the unborn child' (T; R61) and as hazardous—Category 3 substance toxic to reproduction—with the risk phrase 'Possible risk of impaired fertility' (Xn; R62) in the HSIS (Safe Work Australia). The available data support these classifications.

Reproductive toxicity

In a study in accordance with OECD TG 421, Wistar rats (10 animals/sex/dose) were administered the chemical by oral gavage at doses of 0, 50, 250 or 1000 mg/kg bw/day. Animals were dosed two weeks prior to mating with continued dosing of females until postnatal day (PD) four. No parental mortalities occurred. Observed sub-lethal signs including salivation and impairment of the regular care of the fur were seen at the highest dose only. Fertility and number of implantations per dam were reduced at the top dose and no live pups were born. Statistically significant reduced organ weights of the epididymides and ovaries were reported; however, no histopathological findings were reported in these organs. The parental NOAEL for reduced fertility was reported to be 250 mg/kg bw/day (OECD, 2009; REACH).

Developmental toxicity

The developmental toxicity of AEEA is well established in animal studies with no evidence of maternal or paternal toxicity. Studies in rats for maternal and paternal or maternal-only dosing of the chemical were available, with dosing occurring over various developmental periods including 2 weeks prior to mating, and continued until postnatal day (PD) 28. The doses range from 0.1 — 1000 mg/kg bw/day, with evidence of embryotoxicity from doses of 0.2 mg/kg bw/day and greater (OECD, 2009; REACH; TSCATS).

The target tissues for AEEA in the foetus were the aorta and pericardial blood vessels, with effects reported to show strong dose dependency at doses of 50 mg/kg bw/day and greater. The most commonly reported treatment-related effects were both dissecting aortic and/or pericardial aneurysm. Other reported aortic effects included dilation of the aorta, thickening or focal necrosis of the aortic wall and high aortic arch. Dissection or abnormal courses of the pulmonary and carotid artery were also reported. Haemorrhage of the mediastinal or pericardial tissues as well as irregular elastin fibres/scar tissue in the aorta, pulmonary or carotid arteries were also observed in some studies (OECD, 2009; REACH; TSCATS).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (reproductive toxicity, developmental toxicity), and local effects (corrosivity, skin sensitisation).

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. Considering the range of domestic and cosmetic products that may contain the chemical, the main route of public exposure is expected to be through the skin.

Amphoteric surfactants are generally present at concentrations of 10 — 50 % in detergents and shampoos; however, as AEEA is only present as an unreacted intermediate, the actual concentration present in these products is generally below 5 ppm

(OECD, 2009). Given the low concentration of the chemical in domestic and cosmetic products, 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.

Based on the available data, the hazard classification in the HSIS (Safe Work Australia) is considered appropriate.

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, 2016).

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 burns (C; R34)*	Causes severe skin burns and eye damage - Cat. 1 (H314)
Sensitisation	May cause sensitisation by skin contact (Xi; R43)*	May cause an allergic skin reaction - Cat. 1 (H317)
Reproductive and Developmental Toxicity	Repro. Cat 3 - Possible risk of impaired fertility (Xn; R62)* Repro. Cat 2 - May cause harm to the unborn child (T; R61)*	May damage the unborn child. Suspected of damaging fertility - Repr. 1B (H360Df)

[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 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, 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.

References

European Commission Cosmetic Ingredients and Substances (CosIng) Database. Accessed March 2016 at http://ec.europa.eu/consumers/cosmetics/cosing/

Galleria Chemica. Accessed March 2016. http://jr.chemwatch.net/galleria/

Organisation for Economic Cooperation and Development (OECD) 2002. SIAR on Ethanol, 2-((2-aminoethyl)amino)-—(CAS No. 111-41-1). Accessed March 2016 at http://webnet.oecd.org/Hpv/UI/SIDS_Details.aspx?id=E9491E63-A3AA-470D-9560-6D045BB6CF0F

Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) Dossier. 2-(2-aminoethylamino)ethanol (CAS No. 111-41-1). Accessed March 2016 at http://echa.europa.eu/information-on-chemicals/registered-substances

SPIN (Substances in Preparations in Nordic Countries) Database. Accessed March 2016. Available: http://195.215.202.233/DotNetNuke/default.aspx

The Poisons Standard (the Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)) 2016. Accessed March 2016 at https://www.comlaw.gov.au/Details/F2016L00036

Toxic Substance Control Act Test Submission (TSCATS). Low detail Report Aminoethylethanolamine (CAS No. 77-58-7) Accessed March 2016 at https://yosemite.epa.gov/oppts/epatscat8.nsf/ReportSearch?OpenAgent&CASNumber=111-41-1

US Environmental Protection Agency Aggregated Computational Toxicology Resource (ACToR). Accessed March 2016 at http://actor.epa.gov/actor/faces/ACToRHome.jsp

US National Library of Medicine's Hazardous Substances Data Bank (HSDB). Accessed March 2016 at http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

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