Acetic acid, methoxy-: Human health tier II assessment

01 September 2015

CAS Number: 625-45-6

Preface

This assessment was carried out by staff of the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) using the Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework.

The IMAP framework addresses the human health and environmental impacts of previously unassessed industrial chemicals listed on the Australian Inventory of Chemical Substances (the Inventory).

The framework was developed with significant input from stakeholders and provides a more rapid, flexible and transparent approach for the assessment of chemicals listed on the Inventory.

Stage One of the implementation of this framework, which lasted four years from 1 July 2012, examined 3000 chemicals meeting characteristics identified by stakeholders as needing priority assessment. This included chemicals for which NICNAS already held exposure information, chemicals identified as a concern or for which regulatory action had been taken overseas, and chemicals detected in international studies analysing chemicals present in babies' umbilical cord blood.

Stage Two of IMAP began in July 2016. We are continuing to assess chemicals on the Inventory, including chemicals identified as a concern for which action has been taken overseas and chemicals that can be rapidly identified and assessed by using Stage One information. We are also continuing to publish information for chemicals on the Inventory that pose a low risk to human health or the environment or both. This work provides efficiencies and enables us to identify higher risk chemicals requiring assessment.

The IMAP framework is a science and risk-based model designed to align the assessment effort with the human health and environmental impacts of chemicals. It has three tiers of assessment, with the assessment effort increasing with each tier. The Tier I assessment is a high throughput approach using tabulated electronic data. The Tier II assessment is an evaluation of risk on a substance-by-substance or chemical category-by-category basis. Tier III assessments are conducted to address specific concerns that could not be resolved during the Tier II assessment.

These assessments are carried out by staff employed by the Australian Government Department of Health and the Australian Government Department of the Environment and Energy. The human health and environment risk assessments are conducted and published separately, using information available at the time, and may be undertaken at different tiers.

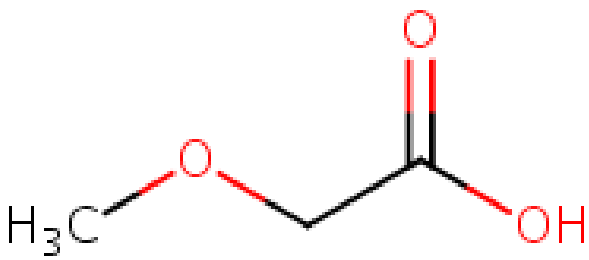
This chemical or group of chemicals are being assessed at Tier II because the Tier I assessment indicated that it needed further investigation.

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

Disclaimer

Acronyms & Abbreviations

Chemical Identity

Synonyms	2-methoxyacetic acid methoxyethanoic acid
Structural Formula	
Molecular Formula	C3H6O3
Molecular Weight (g/mol)	90.08
Appearance and Odour (where available)	Colourless liquid with a pungent odour
SMILES	C(=O)(O)COC

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, Authorization and Restriction of Chemicals (REACH) dossiers, Galleria Chemica, the Substances and Preparations in Nordic countries (SPIN) database, and the United States Environmental Protection Agency's (US EPA) Aggregated Computer Toxicology Resource (ACToR).

The chemical has reported commercial uses as industrial solvents, including in:

- cleaning and washing agents;
- paints, lacquers and varnishes; and
- surface treatment.

The chemical has reported site-limited use as an intermediate.

Restrictions

Australian

No known restrictions have been identified.

International

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

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

Existing Work Health and Safety Controls

Hazard Classification

The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

- Xn; R22 (acute toxicity);

- C; R34 (corrosivity); and
- Repr. Cat. 2; R60-61 (reproductive and developmental toxicity).

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica):

An exposure limit of 19 mg/m^3 (5 ppm) time weighted average (TWA) in Germany, and 3.7 mg/m^3 (1 ppm) TWA and 29.6 mg/m^3 (8 ppm) short-term exposure limit (STEL) in Switzerland.

Health Hazard Information

Toxicokinetics

The chemical, a short chain alkoxy acetic acid, is a major metabolite of ethylene glycol monomethyl ether (EGME; CAS No. 109-86-4). The chemical is rapidly converted from EGME by the action of alcohol dehydrogenase (ADH) and is considered to mediate the systemic toxicity of EGME and other glycol ethers, including monoglyme (CAS No. 110-71-4) and diglyme (CAS No. 111-96-6). The chemical can undergo activation to methoxyacetyl coenzyme A and enter the Krebs cycle or fatty acid biosynthesis. The chemical is hypothesised to cause cell disruption by interfering with essential metabolic pathways, leading to reproductive and developmental toxicity, including testicular lesions and malformations (WHO, 2002; ECETOC, 2005; NICNASa; NICNASb; NICNASc).

The chemical is excreted renally, partially in a conjugated form. The biological half-life of the chemical in rats is approximately 9–13 hours. Slow accumulation of the chemical was observed in non-human primates (*Macaca fascicularis*) and humans, with an average half-life of 77.1 hours in humans exposed to EGME by inhalation. The concentration of the chemical in mice embryos and in extra-embryonic fluid was 20 % higher than in maternal serum after a single gavage treatment of EGME at 250 mg/kg bw (WHO, 2002; ECETOC, 2005; REACH).

Acute Toxicity

Oral

The chemical is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in the HSIS (Safe Work Australia). The available data support this classification.

The chemical has moderate acute toxicity based on results from an animal test following oral exposure. The median lethal dose (LD50) in Sprague Dawley (SD) rats is between 1000–1500 mg/kg bw. Reported signs of toxicity included breathing difficulty, apathy, muscle weakness, spastic gait, tonic spasms, ruffled fur, cyanosis, dehydration, bulging eye, abducted extremities and voluntary movement weakness (ECETOC, 2005; REACH).

Dermal

No data are available for the chemical.

Inhalation

The chemical has low acute toxicity based on results from an animal test following inhalation exposure.

In an inhalation risk test, 12 rats (species unspecified) were exposed to the chemical as vapour for seven hours. The test atmosphere was generated by passing 200 L air/hour through a 5 cm deep layer of the chemical at 20 °C. The concentration of the chemical was 7.86 mg/L when converted to a four-hour inhalation test equivalent. Observed sub-lethal effects included closed eyes, snout-wiping and rhinitis. No mortalities were observed in the study (ECETOC, 2005; 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.

In a primary dermal irritation study, the chemical was applied occlusively to the shaved dorsal skin of six Vienna White rabbits for three minutes, one hour or four hours. There were four females in total for the three minutes and one hour exposures, while two males were exposed for four hours. The chemical was washed off after each exposure. In the three-minute exposure group, slight to intense reddened skin and oedema were observed 24 hours after application. The effects were fully reversed after 48 hours. The animals exposed to the chemical for three minutes were also exposed for one hour. Severe reddened skin, oedema and necrosis were observed 24 hours after the one-hour exposure. Three of the four animals were euthanised due to the corrosive effects observed. The effects were not reversed after 48 hours in the surviving animal. In the four-hour exposure group, oedema and necrosis were observed 24 hours after application, which developed into blistering necrosis 48 hours after application. Both animals were euthanised due to the corrosive effects observed. The chemical was concluded to be corrosive to the skin (ECETOC, 2005; REACH).

Sensitisation

Skin Sensitisation

No data are available.

Quantitative Structure Activity Relationship (QSAR) models can be used to predict the activities of data-poor chemicals, based on known relationships between chemical structures and biological activity that have previously been reported. The chemical has no structural alerts for binding to protein or skin sensitisation as profiled by the Organisation for Economic Co-operation and Development (OECD) QSAR Toolbox v3.2.

Repeated Dose Toxicity

Oral

The available data indicate that the testis is the main target organ for toxicity following repeated oral exposure.

Male Fischer 344 (F344) rats (five animals/group) were administered eight daily doses of the chemical at 0, 30, 100 or 300 mg/kg bw/day by gavage over a period of two weeks. Significant reductions in body weight, absolute and relative spleen and testicular weights, and leukocyte count were observed at 300 mg/kg bw/day. At 100 and 300 mg/kg bw/day, significant dose-dependent reductions in absolute and relative thymus weight, erythrocyte count, and haemoglobin and haematocrit levels were observed. Histopathological examinations showed degeneration of the testicular germinal epithelium at 100 and 300 mg/kg bw/day. At 300 mg/kg bw/day, testicular giant cells and reduced cell density in the bone marrow were also observed. A no observed adverse effect level (NOAEL) of 30 mg/kg bw/day was established (ECETOC, 2005; REACH).

In a published study on the effects of the metabolic precursor EGME, male SD rats were administered the chemical using an equimolar dose to EGME at 500 mg/kg bw/day (calculated as 592 mg/kg bw/day) by gavage for four consecutive days. Significant reductions in relative body, liver and testicular weights were observed in the treated animals. Histopathological examinations showed testicular damage in the treated animals, including degeneration and subsequent loss of spermatocytes (ECETOC, 2005; REACH).

Dermal

No data are available.

Inhalation

The available data suggest that the chemical has low repeated dose toxicity based on results from an animal test following inhalation exposure, except the testicular effects which are treated as effects on fertility.

In a repeated dose inhalation toxicity study conducted according to the OECD Test Guideline (TG) 412, Wistar rats (five animals/sex/group) were exposed by nose only to the chemical at concentrations of 0, 22.8, 58.8 or 156.9 mg/m^3 for six hours/day, five days/week for 28 days. No mortalities were observed in the study. At 156.9 mg/m^3, significant reductions in thymus weights were observed in the males and histopathological examinations showed substance-related changes in the testes. No other effects were reported following histopathological examinations. Investigation on the reproductive effects in male rats was inconclusive. A no observed adverse effect concentration (NOAEC) of 58.8 mg/m^3 was established in this study (REACH).

Genotoxicity

Based on the available data from in vitro studies with the chemical and genotoxicity studies using the metabolic precursor EGME, the chemical is not considered to be genotoxic.

A bacterial reverse mutation assay was conducted in four *Salmonella typhimurium* strains (TA97a, TA98, TA100 and TA102) up to a maximum concentration of 2 mg/plate of the chemical, in the absence or presence of a rat liver metabolic activation system. Negative findings were reported in this study (REACH).

A mammalian cell gene mutation assay was conducted in two Chinese hamster ovary (CHO) cell lines (AS52 and K1-BH4) using the chemical at concentrations of 5, 10, 50, 100, 150 or 200 mM (in the absence of a rat liver metabolic activation system) and 100 mM (in the presence of the metabolic activation system). The chemical was not mutagenic in the xanthine-guanine phosphoribosyl transferase (xgpt) gene on the autosome AS52 cell line and in the hypoxanthine-guanine phosphoribosyl transferase (hgprt) gene on the X chromosome of the K1-BH4 cell line (ECETOC, 2005; REACH).

A sister chromatid exchange (SCE) assay was conducted in human lymphocytes using the chemical. The chemical increased the frequencies of SCE at concentrations of 1 and 10 mmol/L. No further details were provided (ECETOC, 2005; REACH).

The chemical did not induce chromosomal aberrations in the Chinese hamster lung fibroblast (V79) cells and human lymphocytes. However, weakly positive results were observed in the V79 cells in a micronucleus assay and a test for the induction of aneugenic effects. No inhibition of metabolic co-operation between the V79 cells was observed (ECECTOC, 2005; REACH).

The NICNAS assessment on the metabolic precursor EGME indicated that EGME could have, at most, weak genotoxic potential (NICNASa).

Carcinogenicity

No data are available. The metabolic precursor EGME is not considered to be a human carcinogen (NICNASa). Experimental in vitro genotoxicity data (refer to **Genotoxicity** section) showed that the chemical is not considered to be genotoxic and there are no structural alerts from OECD QSAR Toolbox v3.2 for carcinogenicity. Therefore, the chemical is not considered to be carcinogenic.

Reproductive and Developmental Toxicity

The chemical is classified as hazardous—Category 2 substance toxic to reproduction—with the risk phrases ‘May impair fertility’ (T; R60) and ‘May cause harm to the unborn child’ (T; R61) in the HSIS (Safe Work Australia). The available data support this classification.

In a two-generation continuous breeding study, Swiss CD-1 mice (20 animals/sex/group) were administered the chemical at concentrations of 0, 0.1, 0.2 or 0.4 % (approximately 0, 140, 240 or 390 mg/kg bw/day) via drinking water for 98 days. During the continuous breeding phase, male body weights were reduced by 9 % (0.4 % group) while female body weights were reduced by 19 % (0.1 % group), 24 % (0.2 % group) and 32 % (0.4 % group). The reductions in body weights were related to dose-dependent reductions (19–44 %) in water consumption. In the 0.1 and 0.2 % groups, significant reductions in the number of litters per pair and live pups per litter, and live pup weights, were observed. In the 0.2 % group, the pups died by day four. No dams in the 0.4 % group delivered any live pups. In a subsequent cross-mating experiment, fertility was reduced by 92 % even though all animals showed equal evidence of mating. Significant reductions in the testes, epididymis and seminal vesicle weights were observed in the 0.4 % group. Sperm number and motility were significantly reduced and sperm abnormalities were significantly increased in this group. In the surviving first-filial (F1) offspring exposed to 0.1 % of the chemical in drinking water, the fertility index was zero (NTP, 1986; REACH).

Male rats (six animals/group; species not specified) were administered the chemical as single oral doses at 0, 118, 296 or 592 mg/kg bw by gavage. The animals were euthanised on days 1, 2, 4 and 14 post-treatment and the testes, epididymides, seminal vesicles, prostate and liver were histopathologically examined. Significant reductions in the relative testes weights were observed at 592 mg/kg bw. In all treated animals, dose-dependent damage specific to spermatocytes undergoing meiotic maturation and division was observed 24 hours after treatment. The number of immature and abnormal cells was increased in the epididymides of the treated animals. No effects were observed in the seminal vesicles, prostates or livers of the animals (REACH).

In a reproductive toxicity study, male SD rats (three or four animals/group) were treated with single intraperitoneal (i.p.) injections of the chemical at doses of 0, 60, 300, 600 or 900 mg/kg bw. Significant reductions in relative testes weights were observed at 900 mg/kg bw. At doses of 300 mg/kg bw and above, dose-dependent depletions of spermatocytes, mainly restricted to the early pachytene spermatocytes in stages I and II of the spermatogenic cycle, and to the late pachytene and dividing spermatocytes in stages XII-XIV, were observed. This effect was associated with significant increases in urinary creatine excretion (REACH).

Three male SD rats were treated with single i.p. injections of the chemical at a dose of 650 mg/kg bw. Reduced serum testosterone and increased follicle stimulating hormone (FSH) concentrations were observed in the treated animals, although the effects were not statistically significant. A near complete depletion of the spermatocytes at stages IX–II caused by an apoptotic mechanism was observed after treatment with the chemical (REACH).

Male Syrian golden hamsters (12 animals/group) were administered the chemical at doses of 0, 8, 32 or 64 mg/kg bw/day by gavage for five weeks. The animals were euthanised and spermatozoa were recovered from the epididymis for in vitro fertilisation analysis. A significant decline in sperm fertility was observed in all treated animals. The sperm fertilising ability was fully recovered five weeks after treatment at 8 mg/kg bw/day, whereas recovery was incomplete at 32 and 64 mg/kg bw/day. Persistent damage to the seminiferous tubules, including the loss of spermatocytes and other cell types, was observed at 64 mg/kg bw/day (ECETOC, 2005; REACH).

Mixed cultures of Sertoli and germ cells from the testes of four-week-old rats were treated with the chemical at concentrations of 0, 5 or 10 mM (duration unspecified). Dose-dependent increases in initial degeneration and subsequent loss from the culture of the spermatocyte populations were observed. At 5 mM, the effects were restricted to the pachytene spermatocytes, but earlier stages of the spermatocyte populations were also affected at 10 mM (REACH).

The chemical has also been shown to cause adverse developmental effects in several published studies. Effects included foetal mortality, digit malformation and skeletal anomalies. In one published study, Wistar rats (8–10 animals/group) were treated with i.p. injections of the chemicals at doses of 0 or 225 mg/kg bw on GDs 8, 10, 12 or 14. Foetal mortality was significantly increased after injection of the chemical on days 8 (93 %), 10 (61 %), 12 (16 %) and 14 (3 %) when compared with controls (0 %). Increased skeletal malformations and hydrocephalus (abnormal enlargement of the brain) were also observed in a dose-dependent manner in all treatment groups (ECETOC, 2005; REACH).

Pregnant CD-1 mice were administered single doses of the chemical at 3.4 or 4.6 mmol/kg bw (approximately 306 or 414 mg/kg bw, respectively) by oral gavage on GD 11. High incidences of digit malformations (52–100 %) were observed in the foetuses following chemical treatment (ECETOC, 2005; REACH).

In a separate study, the authors compared the developmental effects of the chemical in CD-1 mice when exposed by different routes. The animals were administered single doses of the chemical at 2.9 or 3.8 mmol/kg bw (approximately 260 or 340 mg/kg bw, respectively) either by oral gavage or intravenous (i.v.) administration. Digit malformations were increased in a dose-dependent manner for both treatments, with slightly higher incidences observed following gavage administration of the chemical (ECETOC, 2005; REACH).

In a developmental toxicity study, artificially inseminated female New Zealand White rabbits (20 animals/group) were administered the chemical at doses of 0, 2.5, 7.5 or 15 mg/kg bw/day by oral gavage on GDs 7–19. Reductions in faecal production, food consumption and body weights, and increased relative liver weights were observed in the treated animals at 15 mg/kg bw/day. Developmental effects including malformations of the limbs, digits, ribs and decreased foetal body weights were observed at 7.5 and 15 mg/kg bw/day. Increased resorptions and decreased litter size and gravid uterine weights were also observed at 15 mg/kg bw/day. A NOAEL of 2.5 mg/kg bw/day for both maternal and developmental toxicity was established in this study (TSCATS, 1996).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic long-term effects (reproductive and developmental toxicity), systemic acute effects (acute toxicity from oral exposure) and local effects (corrosivity).

Public Risk Characterisation

Given the uses identified for the chemical, it is unlikely that the public will be exposed. Hence, the public risk from this chemical is not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, oral, dermal and ocular exposure may occur, particularly where manual or open processes are used. These could include transfer and blending activities, quality control analysis, and cleaning and maintaining equipment. Worker exposure to the chemical at lower concentrations could also occur while using formulated products containing the chemical. The level and route of exposure will vary depending on the method of application and work practices employed.

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

Work Health and Safety

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

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)
Irritation / Corrosivity	Causes burns (C; R34)*	Causes severe skin burns and eye damage - Cat. 1B (H314)
Reproductive and Developmental Toxicity	Repro. Cat 2 - May impair fertility (T; R60)* Repro. Cat 2 - May cause harm to the unborn child (T; R61)*	May damage fertility. May damage the unborn child - Cat. 1B (H360FD)

[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 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;
- 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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Last update 01 September 2015

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