Oxalic acid: Human health tier II assessment

18 September 2014

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Ethanedioic acid	144-62-7
Ethanedioic acid, dihydrate	6153-56-6

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

Grouping Rationale

Oxalic acid does not exist in its anhydrous form in nature. It is generally available commercially as oxalic acid dihydrate (CAS No. 6153-56-6, molecular weight 126.06). On the AICS (Australian Inventory of Chemical Substances), hydrates are not required to be linked seperately, as they are regarded as a mixture of the anhydrous form and water; therefore, these chemicals should be considered equivalent.

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 European Union Registration, Evaluation, Authorisation and Restriction of Chemicals (EU REACH) dossiers; the Organisation for Economic Cooperation and Development Screening Information Dataset Initial Assessment Report (OECD SIAR); Galleria Chemica; Substances and Preparations in the Nordic countries (SPIN) database; the European Commission Cosmetic Ingredients and Substances (CosIng) database; United States (US) Personal Care Product Council International Nomenclature of Cosmetic Ingredients (INCI) dictionary; and eChemPortal: OECD High Production Volume chemical program (OECD HPV), the US Environmental Protection Agency's Aggregated Computational Toxicology Resource (ACToR), and the US National Library of Medicine's Hazardous Substances Data Bank (HSDB).

Oxalic acid (identified by the CAS No. 144-62-7) is included in the CosIng database and US Personal Care Products Council's INCI dictionary with the identified functions of chelating agent (CosIng) and pH adjuster (INCI). However, no documented use of the chemicals in cosmetics in the US was identified (Personal Care Products Council, 2011).

The chemicals have reported domestic use including in:

- anti−freezing agents;
- adhesives, binding agents;
- bleaching agents;
- cleaning/washing agents;
- colouring agents;
- corrosion inhibitors;
- paints, lacquers and varnishes; and
- surface treatments.

The chemicals are reported to be present in a range of domestic products (household and auto cleaning products, available as powder/liquid/pump spray formulations) up to a concentration of 20 % (Household Products database, US Department of Health and Human Services).

The chemicals have reported commercial use including in:

- anti−freezing agents;
- fixing agents;
- photo chemicals;
- reprographic agents;
- fabric printing and dyeing and bleaching; and
- tanning agents.

The chemicals have reported site-limited use including:

- in electroplating agents; and
- as chemical intermediates.

The following non-industrial uses have been identified internationally for the chemicals:

- as disinfectants;
- as veterinary medicines; and
- as ingredients in pesticide and agricultural products.

Restrictions

Australian

The chemicals are listed in the *Poisons Standard (Standard for the Uniform Scheduling of Medicines and Poisons 2013*— SUSMP) in Schedule 6. This entry excludes the derivatives and insoluble salts (SUSMP, 2013).

International

Oxalic acid (identified by the CAS No. 144-62-7) is listed on the following (Galleria Chemica):

- ASEAN Cosmetic Directive Annex III – Part 1 (List of substances which cosmetic products must not contain except subject to restrictions and conditions laid down);
- EU Regulation (EC) No 1223/2009 Annex III (List of substances which cosmetic products must not contain except subject to the restrictions laid down); and
- New Zealand Cosmetic Products Group Standard—Schedule 5—Table 1 (Components cosmetic products must not contain except subject to the restrictions and conditions laid down).

For the above, the chemicals are restricted to use in hair products, for professional use only, at a maximum concentration of 5 %.

Existing Worker Health and Safety Controls

Hazard Classification

Oxalic acid (identified by the CAS No. 144-62-7) is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia):

Xn; R21/22 (acute toxicity)

Oxalic acid dihydrate is not listed in the HSIS.

Exposure Standards

Australian

Oxalic acid (identified by the CAS No. 144-62-7) has an exposure standard of 1 mg/m^3 time weighted average (TWA) and 2 mg/m^3 short-term exposure limit (STEL).

International

The following exposure standards are identified (Galleria Chemica) for oxalic acid (identified by the CAS No. 144-62-7).

An exposure limit of 1 mg/m^3 (TWA) and 2 mg/m^3 (STEL) is recommended by the National Institute for Occupational Safety and Health (NIOSH) and the American Conference of Governmental Industrial Hygienists (ACGIH).

Health Hazard Information

Oxalic acid is generally available commercially in the dihydrate form. In a number of the studies it was not clear whether the anhydrous or dihydrate form had been tested. However, these chemicals should be considered equivalent. Data have been included for sodium oxalate and the oxalic acid esters, which are considered suitable analogues for systemic effects, where relevant.

Toxicokinetics

Oxalic acid is an organic acid that occurs naturally in food (e.g. spinach, rhubarb, coffee, chocolate, tea etc.). Dietary intake is reported as 5–500 mg daily, with intake sometimes exceeding 1 g/day (EMEA, 2003). Oxalic acid is also produced

endogenously in the normal human body as an end product of the metabolism of glycine, glycolate and ascorbic acid. Endogenous sources constitute 30–70 % (20–30 mg) of oxalic acid excreted daily (EMEA, 2003).

Oxalic acid is reported to be rapidly cleared from the plasma pool. Oxalate absorption in rats and humans is 2–30 % and mainly occurs in the small and upper intestine (Hanes et al., 1999; EMEA, 2003).

Oxalic acid is mainly excreted unchanged in the urine as the parent compound or as calcium oxalate. Degradation by intestinal bacteria to CO_2 can occur (EMEA, 2003).

Acute Toxicity

Oral

Oxalic acid (identified by the CAS No. 144-62-7) is classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in HSIS (Safe Work Australia). The available data for an oxalic acid solution (median lethal dose—LD50 of 425 mg/kg bw) support this classification for both chemicals in the group. Reported signs of toxicity include kidney damage, gastric haemorrhage, central nervous system depression, convulsions and coma (US EPA, 2005).

Dermal

Oxalic acid (CAS No. 144-62-7) is classified as hazardous with the risk phrase 'Harmful in contact with skin' (Xn:R21) in HSIS (Safe Work Australia). While the available animal data do not support this classification, in the absence of more comprehensive information, there is insufficient evidence to support a recommendation to amend this classification.

Oxalic acid (CAS No. not specified) had low toxicity in rabbits following dermal exposure. No mortalities occurred in three rabbits topically administered 20000 mg/kg bw of the chemical (EMEA, 2003).

Inhalation

No data are available.

Observation in humans

Oxalic acid can be fatal in humans, with fatal doses reported at 3–30 g/person (EMEA, 2003; ACGIH, 2011; HSDB). The probable oral lethal dose for humans has been estimated at 50–500 mg/kg (US EPA, 1992). Deaths have been reported to be rapid as a result of cardiac arrest (ACGIH, 2011).

Inadvertant intravenous doses of approximately 25 mg/kg bw/day have resulted in kidney failure, cardiac arrest and death in patients.

Other reported signs of toxicity in humans include nausea and vomiting, headaches, abdominal pain, diarrhoea, bloody stool, numbness and tingling of fingers and toes, muscular irritability, tetany, convulsions, shock, oliguria, anuria, haematuria, albuminuria, cardiac irregularities and circulatory collapse (HSBD).

Corrosion / Irritation

Corrosivity

Skin

A diverse range of information sources is available relating to skin irritation including reported human data (see **Observation in humans**), in vivo animal test data, in vitro assay data and pH information. Whilst consistent evidence of the severity of skin irritant responses is not available, on the weight of evidence, the chemicals are considered to be corrosive to the skin.

The chemicals are strong acids with a pH <2 in saturated solutions (IUCLID, 2000); therefore, the chemicals could produce significant corrosive effects on the skin. The chemicals are predicted to be corrosive when the acid reserve is taken into account (Willems et al., 2011).

In an irritation test, rabbit skin was exposed to two concentrations of oxalic acid: 10 % (100 mg/mL) and 3.3 % (33 mg/mL), once daily for five consecutive days. Redness and bloodshot skin were observed in all the animals exposed to the 10 % solution and in 2/5 animals exposed to the 3.3 % solution. Furthermore, the skin was hardened after treatment with the 10 % solution. Thus, the chemical was highly irritating to the skin under the tested conditions (REACH).

Using an alternative method for assessing skin irritancy, albino guinea pigs (Hartley strain) were exposed to 1%, 5% or 10% oxalic acid (0.1 mL test solution was applied and repeated once daily for 10 days) and skin-fold thickness was used to measure oedema. Significant increase in skin-fold thickness was measured from day three for the 5 % and 10 % solution of oxalic acid in water. Erythema was also observed from day three for these concentrations. Erythema was also observed on day nine for the 1% solution (REACH).

In an in vivo study conducted according to OECD Test Guideline (TG) 404, oxalic acid dihydrate (CAS No. 6153-56-6) was applied to rabbit skin after humidification with saline (500 mg) for four hours, after which the exceeding substance was cleansed off. No erythema or oedema was observed at 1, 24, 48 and 72 hours after the application (REACH).

The chemical oxalic acid dihydrate (CAS No. 6153-56-6) was found to be corrosive in the in vitro EpiDerm™ dermal corrosivity assay (OECD TG 431). Relative viabilities of 83.6 % after three minutes and 8 % after 60 minutes were observed. The chemical was predicted to be corrosive based on the 60-minute observation (Willems et al., 2011).

Eye

Based on the available data, the chemicals are considered to cause severe eye damage. This classification is implicit within the proposed corrosivity classification.

Corrosive effects to the eye have been reported in humans (see **Observation in humans)**.

In an eye irritation study (OECD TG 405), oxalic acid caused irreversible effects in the rabbit eye. Maximum scores were reported for corneal opacity, iris lesions, redness of the conjunctivae, and chemosis, 24 and 48 hours after application, and the effects persisted for the duration of the observation (up to 72 hours) (REACH).

The ocular irritancy of the chemical was tested in the rabbit eye, with and without rinsing, using three different protocols (OECD TG 405, Association Francaise de Normalisation (AFNOR), and a guideline by the French authorities for cosmetics and toiletries). The authors concluded that substances with an acidic pH (pH <2) are extremely irritating to the eye (REACH).

Respiratory

Based on reported effects in humans following inhalation exposure (see **Observation in humans)**, the chemical is considered to be corrosive to the respiratory tract. This classification is implicit within the proposed corrosivity classification, although, the Globally Harmonized System of Classification (GHS) classification 'Corrosive to the respiratory tract' (AUH071) should also apply (refer to **Recommendation** section).

Observation in humans

Corrosion and irritant effects of the mouth and digestive tract, skin, eyes and respiratory tract have been reported following exposure to either the solid or concentrated solutions of oxalic acid (US EPA, 2005; ACGIH, 2011; NIOSH).

With the skin, limited information regarding corrosive effects is available; however, gangrenous ulcerations of skin have been reported. In the eye, oxalic acid was reported to cause burns when a solution accidentally came in contact with an eye. Conjunctivitis and corneal damage has also been reported following exposure to oxalic acid (HSBD). After inhalation exposure, irritation of the respiratory tract and ulceration of the mucous membrane has been reported (HSBD). Burning and corrosion of the mouth, oesophagus and stomach have been reported following oral exposure (HSDB).

Sensitisation

Skin Sensitisation

The available data suggests that the chemicals might be, at most, weak sensitisers. Classification is not considered warranted.

Oxalic acid was not a sensitiser in a guinea pig maximisation test (ICCVAM, 2011). Two murine local lymph node assay (LLNA) studies reported EC3 values at 6.3 and 2.4 (Montelius et al., 1994). The positive results seen with the chemical were concluded to be due to its irritant properties. The level of false positives associated with LLNA has also been reported elsewhere as one of the limitations of the assay with non-sensitising irritants (Anderson et al., 2011).

In another LLNA conducted according to OECD TG 429, none of the three concentrations tested (up to 50 %) reached the stimulation index (SI) of three (REACH).

Furthermore, a more extensive LLNA dataset has been made available on 211 chemicals to evaluate skin sensitisers using alternative methods. The study predicted oxalic acid to be a weak sensitiser. However, the SI values obtained in the LLNA for oxalic acid were most often low and close to the threshold level of three (Gerberick et al., 2005).

No human sensitisation studies have been reported.

Repeated Dose Toxicity

Oral

The toxicity of oxalic acid is believed to be due to its ability to chelate free calcium ions, upsetting the calcium-potassium ratio in tissues and, ultimately, causing precipitation as calcium oxalate crystals. Accumulation of the chemicals (as crystals) in the renal tubules causes nephrotoxicity, and accumulation in the testes could be linked to impaired sperm quality (refer **Reproductive and developmental toxicity**). Based on these effects, the chemicals are recommended for classification (refer to the **Recommendation** section).

Rats (Long-Evans) fed diets supplemented with 2.5 or 5 % oxalic acid (1250 and 2500 mg/kg bw/day) for 70 days had decreased water intake, decreased body weights, restricted growth and disrupted oestrous cycles. At the highest dose, the animals also had reduced thyroid weights and changes in iodine and hormone levels. This was accompanied by renal toxicity (increased kidney weight, abnormal gross appearance of the kidneys at necropsy and stone formation), and reduced thyroid function. The lowest observed adverse effect level (LOAEL) was 1250 mg/kg bw/day based on renal toxicity (US EPA, 1992; US EPA, 2005).

The chemical ethanedioic acid diethyl ester (CAS No. 95-92-1), which hydrolyses to oxalic acid, produced oxalate nephrolithiasis in a 28-day repeated dose oral gavage toxicity study. The severity of effects increased with exposure with slight to mild oxalate nephrolithiasis observed in animals exposed to 60 mg/kg bw/day and severe nephrolithiasis observed in animals exposed to 180 mg/kg bw/day. The no observed adverse effect level (NOAEL) was established as 20 mg/kg bw/day (NICNASa).

Dermal

No repeated dose dermal toxicity data are available.

The analogue chemical, sodium oxalate was administrated subcutaneously to rats at 25, 50 and 75 mg/kg bw/day (five days/week) for two, three and one week respectively. Effects included mainly haematuria, with increased white blood cells, epithelial cells and casts excreted into the urine. Histopathological examination of the kidneys indicated a small number of oxalate deposits. The effects were indicative of a mild nephrotoxicity due to tubular obstruction following the administration of the chemical at >25 mg/kg bw/day (EMEA, 2003).

Inhalation

No data are available.

Observation in humans

Oxalic acid is a natural component of food and is also produced in the body endogenously (see **Toxicokinetics**). Prolonged exposure to oxalic acid may lead to urinary stones as crystals of calcium oxalate are a major constituent of kidney stones (HSBD).

Weight of evidence from animal studies and human incidents indicates that significant toxic effects in the kidneys, similar to the effects seen following acute exposures can be produced by cumulative dosing. The chemicals are likely to cause serious damage to health from cumulative exposure and is recommended for classification with the risk phrase R33 (Danger to cumulative effects) in HSIS.

Genotoxicity

Based on the results from the available in vitro genotoxicity studies, the chemicals are not considered to be genotoxic.

For oxalic acid (CAS No. 144-62-7), reverse mutation assays using *Salmonella typhimurium* were negative with and without metabolic activation (US EPA, 2005). The chemical was also reported as negative in a chromosome aberration test in Chinese hamster lung fibroblasts (EMEA, 2003).

No in vivo test reports are available.

Carcinogenicity

A limited two-year feeding study in rats gave no evidence of carcinogenicity.

In a carcinogenicity study, rats (Osborne-Mendel) were exposed to oxalic acid in the diet for two years (50–600 mg/kg bw/day). The treatment produced no effects on body weight, body weight gain and food consumption during the first 52 weeks. There were no significant differences in the mortality rate at any dosage level in the treated groups and in the controls. Pathological examination indicated a slight periportal hypertrophy of the hepatic cells. No difference in tumour incidence was observed. Within the limitations of the study, which did not meet the criteria of current guidelines, there was no evidence of carcinogenicity in rats at doses of approximately 50–600 mg/kg bw/day (EMEA, 2003).

Reproductive and Developmental Toxicity

The chemicals do not show specific reproductive or developmental toxicity. Any reproductive and developmental effects are considered secondary to parental toxicity.

In a multigenerational reproductive toxicity study, mice (CD-1) were exposed to oxalic acid (89, 162 or 275 mg/kg bw/day) in drinking water. In the F0 mice, reduced water consumption was seen in the middle and high dose groups (by 25 %). Decreased prostate weight (19 %) was observed in high dose males (organ weights in lower dose animals were not investigated). A reduction in the number of pups/litters (5 %) and live pup weight (4 %) were observed for the high dose animals. No apparent reproductive effects were observed at the lower doses.

The last litter from the high dose and control groups (F1 mice) was tested in a one-week mating trail and then euthenised and necropsied. An increase in kidney weight (females) and increased incidence of abnormal sperm (males) were observed in the treated F1 mice. Similar to the F0 mice, a reduction in the number of live pups per litter was observed (NTP, 1985; EMEA, 2003; US EPA, 2005; HSDB). Due to the design of the study, it was not possible to establish an NOAEL for the effects in the F1 mice.

Similar effects were observed in a reproduction/developmental toxicity study with ethanedioic acid diethyl ester (CAS No. 95-92-1), which hydrolyses to oxalic acid (NICNASa). Decreased prostate weight and impaired sperm quality, together with oxalate crystals in testes, were observed in the males. In the females, a decrease in the mating index and occurrence of pre-implantation and post implantation loss were reported. A reduction in the number of live pups per litter was observed. The NOAEL was established as 30 mg/kg bw/day (NICNASa).

Pregnant rats exposed to the oxalic acid by gavage developed renal oxalosis and gastritis, as well as increased mortality. There was also a decrease in mean litter size, but no teratogenic effects in pups (US EPA, 2005).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include local effects (corrosion, eye damage) and systemic acute effects (acute toxicity from oral exposure). The chemicals can also cause harmful cumulative effects following repeated exposure.

Public Risk Characterisation

Public exposure to high concentrations of the chemicals is not expected through cosmetic use as the chemicals are used only as a chelating (CosIng) and buffering (INCI) agents. It is predicted that cosmetic formulations will be buffered to achieve a neutral pH. Therefore, if the concentrations in cosmetics are low and the formulations are neutralised, corrosive effects are not expected with their use.

Although particular domestic uses of the chemicals in Australia were not identified, the chemicals are reported to be used in domestic products (available as liquid/powder/pump spray formulations) overseas at concentrations up to 20 % (refer to the **Use** section). Therefore, dermal, ocular and inhalation exposure to concentrations that could cause corrosive/irritant effects is possible through using domestic products.

However, the chemicals are currently listed in Schedule 6 of the SUSMP. A number of warning statements, first aid instructions and safety directions relating to corrosivity, contact with skin and breathing dusts and mists apply.

The current controls are considered adequate to minimise the risk to public health posed by domestic and cosmetic products containing the chemicals. Therefore, no further risk management is necessary for public safety.

Occupational Risk Characterisation

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

Given the critical systemic acute, cumulative, and local health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise oral, dermal, and ocular exposure to the chemical are implemented. The chemicals 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 appropriate controls. The data available support an amendment to the hazard classification in HSIS (refer to **Recommendation** section).

NICNAS Recommendation

The assessment of the chemicals 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 chemicals should be labelled in accordance with state and territory legislation (SUSMP).

Work Health and Safety

The chemicals are 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.

Note: Acute toxicity (oral) is the existing classification for oxalic acid.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)* Harmful in contact with skin (Xn; R21)*	Harmful if swallowed - Cat. 4 (H302) Harmful in contact with skin - Cat. 4 (H312)
Irritation / Corrosivity	Causes burns (C; R34)	Causes severe skin burns and eye damage - Cat. 1B (H314) Corrosive to the respiratory tract (AUH071)
Repeat Dose Toxicity	Danger of cumulative effects (R33)	May cause damage to organs (kidney) through prolonged or repeated exposure - Cat. 2 (H373)

[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 chemicals should be used according to the instructions on the label.

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal, ocular and inhalation exposure to the chemicals 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 chemicals has not been undertaken as part of this assessment.

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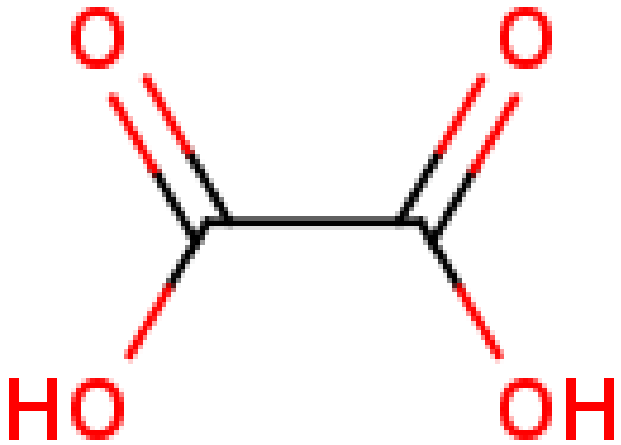
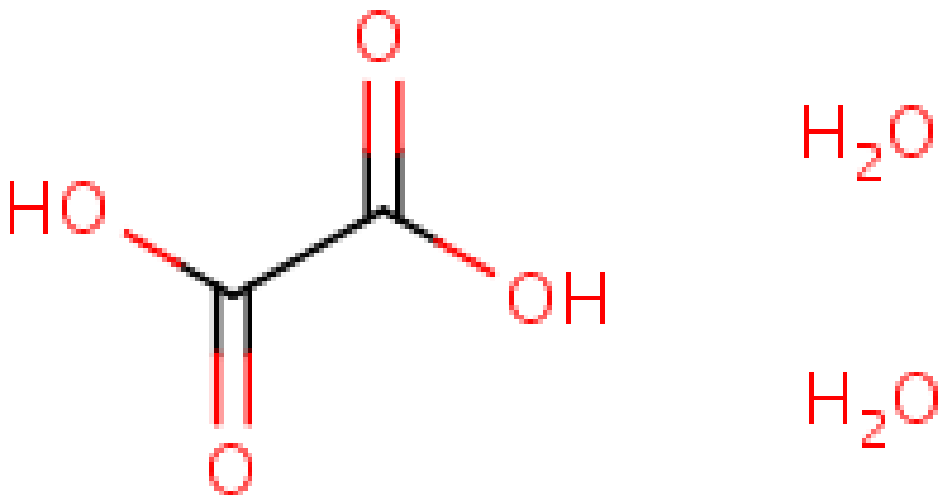
Last Update 18 September 2014

Chemical Identities

Chemical Name in the Inventory and Synonyms	**Ethanedioic acid** Oxalic acid Ethane-1,2-dioic acid Oxiric acid Ethanedionic acid
CAS Number	144-62-7
Structural Formula	

	
Molecular Formula	C2H2O4
Molecular Weight	90.03

Chemical Name in the Inventory and Synonyms	**Ethanedioic acid, dihydrate** Oxalic acid, dihydrate
CAS Number	6153-56-6
Structural Formula	

	
Molecular Formula	C2H2O4.2H2O
Molecular Weight	126.06

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