Propanoates: Human health tier II assessment

03 July 2015

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
Propanoic acid, sodium salt	137-40-6
Propanoic acid, calcium salt	4075-81-4
Propanoic acid, ammonium salt	17496-08-1

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

Grouping Rationale

The chemicals in this group are salts of propanoic acid (CAS No. 79-09-4), a simple carboxylic acid. Propanoic acid occurs naturally as a by-product of normal intermediary metabolism in animals and humans and is generated during the oxidation of odd-number carbon fatty acids and the catabolism of some amino acids. The resulting propionyl-coenzyme A is further metabolised via methylmalonyl-coenzyme A to succinyl-coenzyme A, an intermediate of the tricarboxylic acid cycle. As a result, propanoic acid and its salts are efficiently metabolised in different organisms.

Sodium propanoate (CAS No. 137-40-6) and calcium propanoate (CAS No. 4075-81-4) are freely soluble in water and ethanol. The pH values for 10 % aqueous solutions of sodium and calcium propanoates are between 7.5 and 10.5. Sodium propanoate is produced from the reaction of propanoic acid and an aqueous solution of sodium hydroxide, followed by filtration and drying of the product. Calcium propanoate is produced from the reaction of propanoic acid and calcium oxide in water in the presence of a flocculating agent. The product is then filtered and dried. Ammonium propanoate (CAS No. 17496-08-1) is produced from partial neutralisation of propanoic acid with ammonia or an aqueous solution of ammonium hydroxide.

An IMAP Tier II assessment report has been published on propanoic acid (NICNASa). When data on propanoate salts are limited, read across data from propanoic acid are applied to the assessment of the chemicals in this group as the toxicokinetics and toxicity of the salts are similar on a propanoic acid-equivalent basis (WHO, 1974; US EPA, 2007; EFSA, 2011; EFSA, 2014).

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, the European Commission Cosmetic Ingredients and Substances (CosIng) database, the United States (US) Personal Care Products Council International Nomenclature of Cosmetic Ingredients (INCI) Dictionary, the Organisation for Economic Co-operation and Development High Production Volume chemical program (OECD HPV), the United States Environmental Protection Agency's (US EPA) Aggregated Computer Toxicology Resource (ACToR), the US National Library of Medicine's Hazardous Substances Data Bank (HSDB), the International Programme on Chemical Safety (IPCS), and the Canada Natural Health Products Ingredients Database (NHPID).

The chemicals have reported cosmetic use as preservatives.

The chemicals have reported domestic uses, including in:

- fillers;
- paints, lacquers and varnishes; and
- surface treatments.

The chemicals have reported commercial use in photo chemicals.

The following non-industrial uses have been identified internationally for chemicals in this group:

- in pharmaceuticals (antifungal);
- as a mould inhibitor in foods and tobacco;
- as food/feedstuff flavourings and nutrients; and
- as non-agricultural pesticides and preservatives.

Restrictions

Australian

Propanoic acid, sodium salt (CAS No 137-40-6) is listed in the *Poisons standard*—the *Standard for the uniform scheduling of medicines and poisons* (SUSMP, 2015) Appendix B; a; 1.3. The chemical is considered not to require control by scheduling due to low toxicity when used as a fungicide.

International

The chemicals in this group are listed on the EU Regulation (EC) No 1223/2009 of the European Parliament and of the Council of 30 November 2009 on cosmetic products Annex V—List of preservatives allowed in cosmetic products with maximum concentration in ready for use preparation of 2 % (as propanoic acid) (Galleria Chemica).

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).

Exposure Standards

Australian

No specific exposure standards are available.

International

No specific exposure standards are available.

Health Hazard Information

Toxicokinetics

Limited toxicokinetic data on the chemicals in this group suggest that there is significant oral absorption and the chemicals are rapidly metabolised.

In a rat study, a single oral dose of sodium propanoate was administered to the animals and within three days, 77 % of the chemical was eliminated by exhalation. Therefore, at least 77 % of the chemical was absorbed and systemically available. Faecal and urinary excretion accounted for approximately 7 % of the total elimination during the three days. There were no marked differences in the levels detected in the different organs investigated (i.e. liver, spleen, gastrointestinal (GI) tract, kidney and brain) (EFSA, 2014).

In another rat study, sodium propanoate was fed to animals that were fasted for 24 hours. The total excretion as carbon dioxide after two hours was 24 % of the amount fed, indicating that the chemical was rapidly metabolised (REACH).

Propanoic acid metabolises by interacting with co-enzyme A and is carboxylated to form methylmalonyl-coenzyme A. It is then trans-carboxylated to succinyl-CoA and enters the Krebs cycle to be metabolised to carbon dioxide and water. Approximately 80 % of the propanoate is oxidised to carbon dioxide after extensive metabolism and is excreted by exhalation (NICNASa).

Acute Toxicity

Oral

Limited data are available for the chemicals in this group. The available data suggest that the chemicals have low acute toxicity based on results from animal tests following oral exposure.

The median lethal dose (LD50) for sodium propanoate is >5100 mg/kg bw in both rats and mice. The LD50 values for calcium propanoate are >3920 and >2350 mg/kg bw in rats and mice, respectively. Observed sub-lethal effects included dyspnoea, apathy and piloerection (IUCLID, 2000; US EPA, 2007; HSDB).

Dermal

Limited data are available for the chemicals in this group. The available data suggest that the chemicals have moderate acute toxicity based on results from an animal test following dermal exposure. However, limitations in the report do not allow hazard classification to be determined.

The dermal LD50 for calcium propanoate was reported as 500 mg/kg bw in rabbits. No further details were provided (IUCLID, 2000; US EPA, 2007).

Inhalation

Limited data are available for the chemicals in this group. The available data suggest that the chemicals have low acute toxicity based on results from animal tests following inhalation exposure.

The median lethal concentration (LC50) for both sodium and calcium propanoate is >5.4 mg/L in rats following exposure to the chemicals as dust aerosols for four hours. No further details were provided (IUCLID, 2000; US EPA, 2007).

Corrosion / Irritation

Skin Irritation

The available data indicate that the chemicals in this group are not likely to be skin irritants.

In a skin irritation study conducted according to OECD Test Guideline (TG) 404, calcium propanoate did not cause skin irritation when applied to the skin of Himalayan rabbits at a concentration of 500 mg for four hours (REACH).

Sodium and calcium propanoate were found to be non-irritating in the Draize skin irritation tests with rabbits. No further details were provided (IUCLID, 2000; US EPA, 2007).

Eye Irritation

The chemicals in this group are considered to be irritating to the eyes, warranting hazard classification. The results for calcium propanoate showed irreversible damage.

In an eye irritation study conducted according to OECD TG 405, 0.1 mL of calcium propanoate was instilled into the conjunctival sac of one eye each in two male New Zealand White rabbits. An initial slight pain was observed but no corneal effects were noted during the study. Iridial inflammation (grade one) was observed in both animals one hour after treatment and the effect was fully reversed within 48 hours. Moderate chemosis (scores of 1.33/4 in both animals) was observed one hour after treatment and the effect was fully reversed within 14 days. Moderate conjunctival irritation (scores of 1.33/3 and 1.67/3) was observed in both animals one and 24 hours after treatment. Petechial haemorrhage on the nictitating membrane (third eyelid) was observed in both animals on days 7, 14 and 21 after treatment and this ocular damage was not fully reversed within 21 days. The chemical was concluded to cause serious eye damage in rabbits (REACH).

In an eye irritation study conducted according to OECD TG 437, bovine eyes were isolated from >9-month-old donor cattle and applied with 0.75 mL of calcium propanoate in 0.9 % (w/v) sodium chloride in deionised water for four hours. The chemical caused a slight increase in corneal opacity but permeability effects were not observed. The mean in vitro eye irritation score was 13.01 (threshold for corrosivity/severe irritancy is ≥55.1). Based on this study, the chemical is not severely irritating to the eye (REACH).

Sodium and calcium propanoate were found to be non-irritating in the Draize eye irritation tests in rabbits. No further details were provided (IUCLID, 2000; US EPA, 2007).

Sensitisation

Skin Sensitisation

The available data suggest that the chemicals in this group are not skin sensitisers.

In a guinea pig maximisation test conducted according to OECD TG 406 using sodium propanoate, sensitisation was induced by intradermal injections (two 0.1 mL of Freund's complete adjuvant, two of 0.1 mL of the chemical at 2 % concentration in distilled water, and two of a 1:1 mixture of the chemical and adjuvant) on the shoulder of albino specific pathogen-free guinea pigs. One week after the injection, 20 % of the chemical in vaseline was applied occlusively for 48 hours to the same area. Two weeks later the animals were challenged by an occlusive dermal application of 20 % of the chemical for 24 hours. The chemical did not cause positive responses at challenge (REACH).

Two similar guinea pig maximisation tests were conducted with calcium propanoate; the chemical was also concluded to be non-sensitising in these tests (IUCLID, 2000; REACH).

Repeated Dose Toxicity

Oral

The available data suggest that the chemicals in this group have low repeated dose toxicity based on results from animal tests following oral exposure. The main effects seen in repeated dose toxicity studies were slight reductions in growth rates and forestomach lesions. It is noted that humans lack a forestomach and there is no correlation between the forestomach in rats and oesophageal lesions in humans. Therefore, the forestomach lesions are not considered relevant to human health.

In a repeated dose toxicity study, rats were fed 0, 830 or 2490 mg/kg bw/day of sodium or calcium propanoate in their diet for 3–4 weeks. The only endpoint measured in this study was growth and no effect was observed (IUCLID, 2000).

Groups of 40 female Wistar rats were fed 0 or 1320 mg/kg bw/day of sodium propanoate in their diet for a year. No changes were observed in the blood, clinical chemistry or urine tests. No changes in organ weights were observed in the study. The average body weight at the end of the study was 290 g in treated animals compared with 299 g in control animals, but the slight reduction in growth rate was not considered to be significant (IUCLID, 2000; US EPA, 2007).

Wistar rats were maintained on a normal diet, or diet consisting of 75 % bread that was baked with the addition of a 50-fold amount of four bread additives and bleached flour for a year. Sodium propanoate at a concentration of 5 % was one of the additives. Although it would not be possible to determine the attribution of effects given the complex mixture of substances in this study, no clinical or pathological effects were observed. Therefore, the chemical was concluded to not cause toxic effects (IUCLID, 2000).

In a repeated dose toxicity study, male Wistar rats were fed 0, 20000 or 40000 ppm of calcium propanoate in their diet for eight weeks. The diets were also supplemented with vitamin B12. The treated animals showed a reduction in body weight compared with control animals. No other toxicological endpoints were measured (IUCLID, 2000).

Male Wistar rats were fed 0 or 40000 ppm (approximately 3320 mg/kg bw/day) of sodium propanoate in their diets daily for four weeks. In the forestomach of the treated animals, a slightly thickened limiting ridge was observed in 3/5 animals. Hyperkeratosis of mucosa and hyperplasia of basal cells at the limiting ridge were also observed in 1/5 animals (IUCLID, 2000).

In a separate study, Wistar rats (five animals/sex/group) were fed 0 or 40000 ppm (approximately 3320 mg/kg bw/day) of calcium propanoate in their diet for four (females) and eight (males) weeks. Following four weeks of oral exposure to the chemical, the treated animals showed slightly thickened limiting ridge in the forestomach and more pronounced hyperkeratosis and hyperplasia of mucosa. More pronounced lesions were observed in the forestomach after eight weeks of exposure. The effects were reversed following an eight-week treatment-free period (IUCLID, 2000).

In a repeated dose toxicity study in beagle dogs, calcium propanoate at concentrations of 0, 14500 or 43500 ppm in the diet was fed to the animals for 90 days. Diarrhoea and vomiting were observed in all animals at the highest concentration and one animal in the 14500 ppm group. Spontaneous epithelial hyperplasia of the oesophageal mucosa was observed in all groups and was concluded to be not related to treatment. No further details were provided (IUCLID, 2000).

In a repeated dose toxicity study in 12 monkeys, sodium propanoate was fed at a concentration of 2 % (equivalent to 420 mg/kg bw/day) in the diet for nine weeks. Haematological and liver effects were studied and no toxic effects were observed (IUCLID, 2000).

Dermal

No data are available for the chemicals in this group.

Inhalation

No data are available for the chemicals in this group.

Genotoxicity

The negative results from the available in vitro and in vivo genotoxicity studies indicate that the chemicals in this group are not likely to be genotoxic.

In vitro studies

In a bacterial point mutation assay, sodium and calcium propanoate were assayed for gene mutation with six *Salmonella typhimurium* strains (TA92, TA94, TA98, TA100, TA1535 and TA1537) in the absence or presence of a rat liver metabolic activation system. The maximum concentrations of sodium and calcium propanoate studied were 5 and 10 mg/mL, respectively. Both chemicals tested negative in the assay (IUCLID, 2000; REACH).

In a separate bacterial point mutation assay, calcium propanoate was assayed for gene mutation with *S. typhimurium* strains (TA1535, TA1537 and TA1538) and for gene conversion with *Saccharomyces cerevisiae* strain D4 in the absence or presence of exogenous mouse, rat and monkey liver metabolic activation systems. Negative results were obtained, but it is noted that the study had poorly reported concentration levels and experimental designs (EFSA, 2014).

In a separate study, calcium propanoate was tested for mutagenicity in *S. typhimurium* strains G-46 and TA1530 and for recombinogenic properties in *S. cerevisiae* strain D3. Negative findings were reported, but it is noted that the study had poorly reported concentration levels and experimental designs (EFSA, 2014).

In a mammalian chromosomal aberration study conducted according to OECD TG 473, sodium and calcium propanoate were tested for their clastogenic potential in Chinese hamster lung fibroblasts at concentrations of 0, 500, 1000, 2000 µg/plate for 24 or 48 hours. Results were negative for sodium propanoate, but an equivocal clastogenic response was reported for calcium propanoate at the highest dose. However, the results were not considered to be biologically relevant as the findings were only observed at 48 hours, which was excessively long for the study (EFSA, 2014; REACH).

In a DNA repair test, the DNA-modifying effects of calcium propanoate at a concentration of 10 mg/mL were studied in the *Bacillus subtilis* mutant strain M45 (rec^-). The wild type strain H17 (rec^+) was used as a control. Negative findings were reported in the study (IUCLID, 2000; US EPA, 2007; EFSA, 2014).

In a separate DNA repair test, the DNA-modifying effects of calcium propanoate were studied using the *B. subtilis* mutant strain M45 (rec^-). The wild type strain H17 (rec^+) was used as a control. Concurrently, the mutagenicity of the chemical was studied in a reverse mutation assay with *Escherichia coli* WP2 hcr trp strain and *S. typhimurium* strains (TA98, TA100, TA1535, TA1537 and TA1538). Both studies were conducted in the absence or presence of a rat liver metabolic activation system. Negative results were reported in the studies (IUCLID, 2000; US EPA, 2007; EFSA, 2014).

In vivo studies

In a bone marrow chromosomal aberration assay, rats were administered 0, 50, 500 or 5000 mg/kg bw of calcium propanoate by oral gavage as a single dose (59 animals) or daily for five consecutive days (18 animals). The animals were euthanised six, 24 or 48 hours after dosing in the single treatment groups and six hours after the last dose in the repeated treatment groups. Chromosomal aberration and mitotic index were determined in this study and the chemical did not induce statistically significant increases at any treatment (IUCLID, 2000; US EPA, 2007; EFSA, 2014).

In a rat bone marrow cytogenetic assay, sodium propanoate was tested for genotoxicity effects and negative findings were reported. No further details were provided for the study (IUCLID, 2000).

In a host-mediated assay, male mice were administered 50, 500 or 5000 mg/kg bw of calcium propanoate by oral gavage as a single dose or daily for five consecutive days. Positive and negative control groups were included in both single and repeated treatments. The *S. typhimurium* strains (G-46 and TA1530) and *S. cerevisiae* strain D3 were used as indicator organisms for inducing reverse mutation and mitotic recombination, respectively. In the single dose treatment, all animals received 2 mL of the indicator organisms (3×10^8 cells for *S. typhimurium* and 5×10^8 cells for *S. cerevisiae*) immediately after treatment by intraperitoneal (i.p.) injection. The animals were euthanised three hours later with the indicator organisms removed from the peritoneal cavity and plated for colony scoring. Results indicated a non-dose-related increase in the reversion frequency of *S. typhimurium* strain G-46. Therefore, the results were considered not biologically relevant. Negative findings were reported for strains TA1530 and D3 (IUCLID, 2000; EFSA, 2014).

In a dominant lethal assay, male rats were administered 50, 500 or 5000 mg/kg bw of calcium propanoate by oral gavage as a single dose, or daily for five consecutive days. Positive and negative control groups were included in both single and repeated treatments. Following treatments, the animals were sequentially mated with two untreated virgin females, five days/week for eight weeks. At the end of the treatment every week, the females were replaced with two untreated virgin females. The treated females were removed from the males, housed separately and euthanised 14 days after separation. The uteri were analysed for early and late foetal deaths and total implantations. Negative findings were reported in the study (IUCLID, 2000; US EPA, 2007; EFSA, 2014).

Carcinogenicity

No data are available for the chemicals in this group. However, based on data from the analogue chemical, propanoic acid, the chemicals in this group are not expected to be carcinogenic.

In a lifetime study, male Wistar rats (30 animals/group) were administered propanoic acid in their diet at doses of 0, 264 or 2640 mg/kg bw/day for 20 weeks or for their lifetime. Ten animals from each group were euthanised at week 20 and the remaining animals were fed with their respective diets until death. No treatment-related effects were observed in the animals treated at 264 mg/kg bw/day. In the animals treated at 2640 mg/kg bw/day, forestomach epithelial changes such as hyperplasia and hyperkeratosis were observed at week 20. The other permanent effects included hyperplasia with ulceration, dyskeratosis and papillomatous elevations. One animal euthanised two years after treatment had hyperplasia with ulceration and unspecified ‘carcinomatous changes’ along with erosive changes in the glandular region of the stomach (NICNASa). It was concluded that the effects observed were due to chronic irritation and inflammation and the associated hyperplastic proliferative repair response (IUCLID, 2000; US EPA, 2007; EFSA, 2014; NICNASa; REACH).

In the above study, only forestomach lesions related to chronic irritation were reported. Humans lack this organ and there is no correlation between the forestomach in rats and oesophageal lesions in humans. Considering that the chemicals in this group are less likely to be irritating compared with propanoic acid, it is unlikely that the chemicals in this group are carcinogenic.

Reproductive and Developmental Toxicity

No data are available on the reproductive toxicity of the chemicals in this group. The available data on developmental toxicity indicate that the chemicals in this group are not developmental toxins.

In a developmental toxicity study conducted according to OECD TG 414, calcium propanoate was fed to pregnant Wistar rats (24 animals/group) and CD-1 mice (25–30 animals/group) during gestational days (GDs) 6–15 at concentrations of 0, 3, 14, 65 or 300 mg/kg bw/day, and to pregnant hamsters (22 animals/group) during GDs 6–10 and pregnant rabbits (22 animals/group) during GDs 6–18 at concentrations of 0, 4, 19, 86 or 400 mg/kg bw/day. In all species, no effects on maternal or foetal survival, or litter size were reported. There were no increases in foetal or skeletal abnormalities in the study. It was concluded that the chemical did not cause developmental toxicity and the no observed adverse effect levels (NOAELs) were 300 mg/kg bw/day in rats and mice and 400 mg/kg bw/day in hamsters and rabbits (IUCLID, 2000; US EPA, 2007; EFSA, 2014; REACH).

In a separate developmental toxicity study, calcium propanoate was injected into the air cell or yolk sac of chicken eggs at concentrations of 0, 5, 10 or 100 mg/kg. The exposure period was during pre-incubation or 96 hours post-incubation of the chicken embryos. High mortality rates were observed at 5 mg/kg (administered pre-incubation via the yolk) and 10 mg/kg. The chemical was concluded to be non-teratogenic to chicken embryo development at concentrations up to 100 mg/kg (US EPA, 2007).

Risk Characterisation

Critical Health Effects

The critical health effect for risk characterisation is eye irritation.

Public Risk Characterisation

Although use in cosmetic products in Australia is not known, the chemicals are reported to be used in cosmetic products overseas, but only as a preservative (refer to **Import, manufacture and use** section). In such cosmetic formulations, the chemicals are expected to be used at low concentrations that will not cause any significant human health concerns.

The chemicals in this group also have reported domestic uses overseas as fillers, paints, and surface treatments. Exposure could occur from products containing the chemicals in this group. The main route of exposure is via skin contact. Provided that normal precautions are taken to avoid prolonged skin contact, the risk to the public posed by cosmetic/domestic products containing the chemicals are not considered to be unreasonable.

Occupational Risk Characterisation

During product formulation, ocular exposure might 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 chemicals at lower concentrations could 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 health effects, the chemicals could pose an unreasonable risk to workers unless adequate control measures to minimise ocular exposure 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 the appropriate controls.

NICNAS Recommendation

Assessment of these 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

Work Health and Safety

The chemicals are recommended for classification and labelling under the current approved criteria and adopted GHS as below. The classification for calcium propanoate should be ‘Risk of serious eye damage’ (Xi; R41)/'Causes serious eye damage - Cat. 1 (H318)'. This assessment does not consider classification of physical and environmental hazards.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Irritation / Corrosivity	Irritating to eyes (Xi; R36)	Causes serious eye irritation - Cat. 2A (H319)

[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 ocular 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 chemicals are used. Examples of control measures which could minimise the risk include, but are not limited to:

- using closed systems or isolating operations;
- 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 chemicals.

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 chemicals 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 these chemicals has not been undertaken as part of this assessment.

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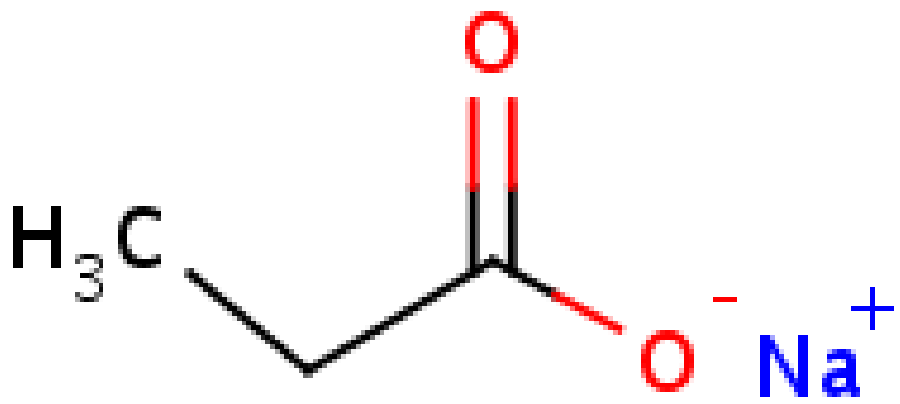
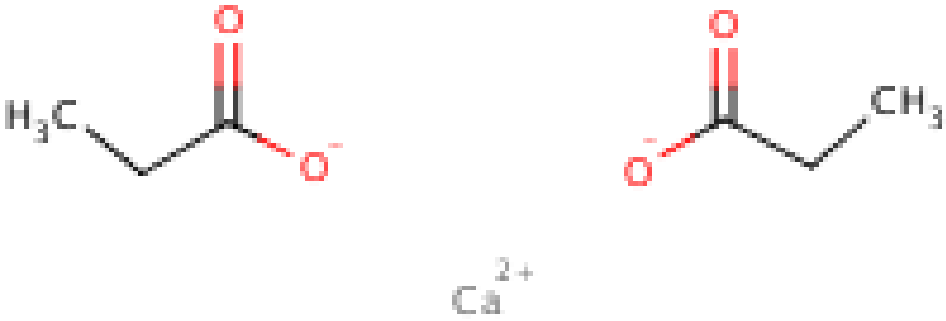
Chemical Identities

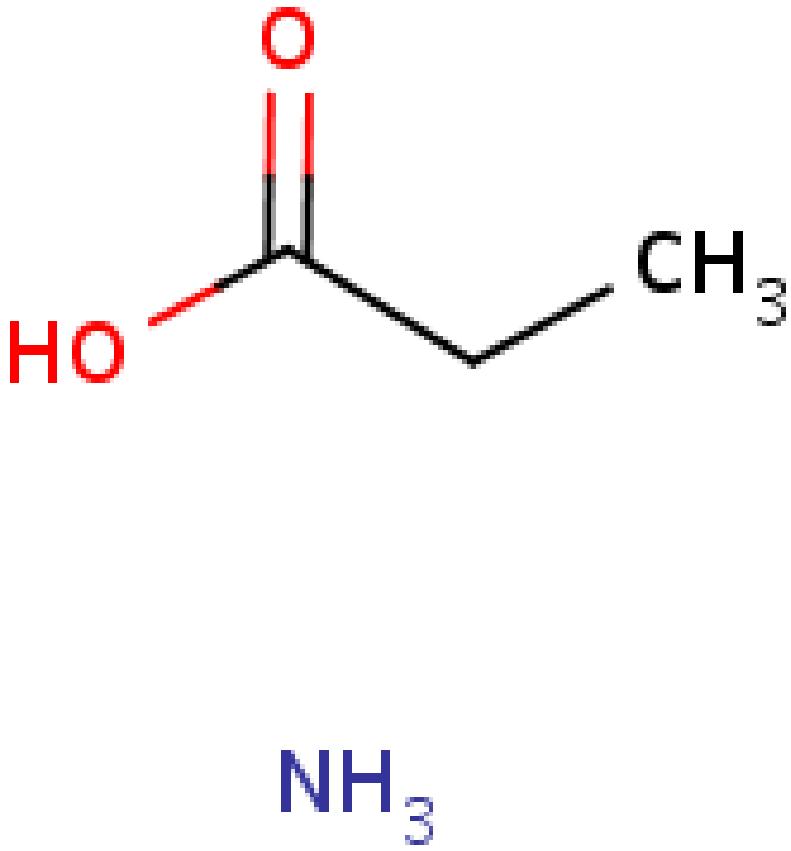
Chemical Name in the Inventory and Synonyms	**Propanoic acid, sodium salt** sodium propionate sodium propionate hydrate sodium propanoate
CAS Number	137-40-6

Structural Formula	
Molecular Formula	C3H6O2.Na
Molecular Weight	96.06

Chemical Name in the Inventory and Synonyms	**Propanoic acid, calcium salt** calcium propionate calcium dipropionate calcium propanoate Bioban-C
CAS Number	4075-81-4
Structural Formula	

	
Molecular Formula	C3H6O2.1/2Ca
Molecular Weight	186.22

Chemical Name in the Inventory and Synonyms	**Propanoic acid, ammonium salt** ammonium propanoate
CAS Number	17496-08-1
Structural Formula	

	
Molecular Formula	C3H6O2.H3N
Molecular Weight	91.11

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