Dehydroacetic acid and its sodium salt: Human health tier II assessment

27 November 2014

Chemicals in this assessment

Chemical Name in the Inventory	CAS Number
2H-Pyran-2,4(3H)-dione, 3-acetyl-6-methyl-	520-45-6
2H-Pyran-2,4(3H)-dione, 3-acetyl-6-methyl-, ion(1-), sodium	4418-26-2
4H-Pyran-4-one, 3-acetyl-2-hydroxy-6-methyl-	16807-48-0

Preface

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

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

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

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

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

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

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

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

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

Disclaimer

NICNAS has made every effort to assure the quality of information available in this report. However, before relying on it for a specific purpose, users should obtain advice relevant to their particular circumstances. This report has been prepared by NICNAS using a range of sources, including information from databases

ACRONYMS & ABBREVIATIONS

Grouping Rationale

The chemical, sodium dehydroacetate (Na-DHA) (CAS No. 4418-26-2) is a salt resulting from dehydroactic acid (DHA) (CAS No. 520-45-6) reacting with one molecule of sodium hydroxide. The speciation of these two chemicals in biological fluids will be dependent on pH, but independent of the original form. There is also a keto-enol tautomeric form of dehydroacetic acid (CAS No. 16807-48-0). Keto- and enol forms of chemicals do not have separate existence and, therefore, this should be considered the same chemical as CAS No. 520-45-6. The three chemicals are considered together in this assessment report.

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 Galleria Chemica; 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).

The chemicals have reported cosmetic use:

- as preservatives;
- as antimicrobial agents; and
- in antienzyme toothpastes.

These three chemicals are listed in the EU Cosmetics Regulation 1223/2009 Annex V—List of Preservatives Allowed in Cosmetic Products. They are listed as safe to use in cosmetics up to a maximum concentration of 0.6 % as dehydroacetic acid, but prohibited from use in aerosol dispensers (sprays).

The chemicals have reported domestic/commercial use including:

- in adhesives; and
- as wood preservatives.

The chemicals have reported site-limited use including as plasticisers in synthetic resins.

The following non-industrial uses have been identified internationally:

- as preservatives in food; and
- as agrichemicals, fungicides and bactericides.

Restrictions

Australian

No known restrictions have been identified.

International

The chemicals are listed on the following (Galleria Chemica):

- Philippines Restricted Ingredients For Use in Cosmetics—List of preservatives which cosmetic products may contain subject to restrictions and conditions laid down. Lists a maximum allowable concentration for DHA and its salts of 0.6% (as DHA) and prohibited use in aerosol dispensers.

Existing Worker Health and Safety Controls

Hazard Classification

The chemicals are classified as hazardous, with the following risk phrase for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia), although the alternate CAS number for the keto-enol tautomeric form (CAS No. 16807-48-0) has not been included:

Xn; R22 (acute toxicity)

Exposure Standards

Australian

No specific exposure standards are available.

International

The following exposure standards are identified (Galleria Chemica).

A time weighted average (TWA) for dehydroacetic acid of:

- 2 mg/m³ in Hungary
- 30 mg/m³ in Finland

Health Hazard Information

Toxicokinetics

The chemicals are rapidly absorbed following oral administration in humans, monkeys, rats and dogs. In a study involving three men, oral doses of 500 mg/day DHA for 115–153 days gave a steady state plasma concentration of 10–15 mg/100 mL, after 15–20 days. In humans and rats, following absorption, approximately 90–98 % of DHA was found to be bound to plasma protein (6 mg/100 mL) with the majority associated with serum albumin, and serum globulin to a lesser extent. In a dog study, DHA administered at 80 mg/kg bw/day to a pregnant animal was shown to cross the placenta and was also found in the milk (CIR, 1985).

DHA and Na-DHA were readily absorbed dermally in rabbits. Dermal absorption was high (50 % more) from a washable base medium (composition not stated) compared with from an aqueous solution (CIR, 1985).

Metabolism studies in rats and rabbits with ^{14}C-labelled DHA identified three main metabolites: triacetic acid lactone, hydroxy-dehydroacetic acid and 'metabolite X' (presumed to be the salt of triacetic acid lactone 3-carboxylic acid). Rabbits excreted 70–80 % of the ^{14}C label in urine, 7–10 % as exhaled carbon dioxide and 2–3 % in faeces (after 3–7 days) leaving 8–11 % in tissues. Excretion in rats was 20–40 % in urine, 10–25 % by respiration, 10–20 % in faeces (after 4–5 days) and 5–26 % in tissues. The percentage of DHA and hydroxy-DHA excreted was found to increase with an increasing dose, while metabolite X levels diminished as the dose increased (CIR, 1985).

Urinary excretion of DHA was found to be 22.2 % in humans that received the chemical at 6.1–12.5 mg/kg bw/d and a maximum of 20 % in monkeys and dogs. Faecal elimination in dogs varied with the route of administration, with excretion of 5 and 1.3 % of the dose administered orally and intravenously, respectively (CIR, 1985).

Acute Toxicity

Oral

The chemicals (DHA and Na-DHA) are classified as hazardous with the risk phrase ‘Harmful if swallowed’ (Xn; R22) in HSIS (Safe Work Australia). The available data support this classification.

Several median lethal dose (LD50) values are reported for rats and mice. For DHA, the LD50 in rats was 500 mg/kg bw (RTECS). Reported signs of toxicity include salivation, vomiting, changes in motor activity, weakness, convulsions and respiratory failure.

Other LD50 values available are:

- 1500 and 1450 mg/kg bw in male and female Wistar rats, respectively for DHA (Uchida O et al., 1985);
- 1000 mg/kg bw for DHA and 570 mg/kg bw for Na-DHA, in rats (CIR, 1985);
- 1330 mg/kg bw in mice for DHA (RTECS); and
- 1050 mg/kg bw in mice for Na-DHA (CIR, 1985).

Dermal

Based on the limited data available, the chemicals are considered to have low acute dermal toxicity.

The lowest published dermal lethal dose (LDLo) was 5000 mg/kg bw in rabbits for DHA (RTECS).

Inhalation

No data are available.

Corrosion / Irritation

Skin Irritation

The chemicals are not considered to cause skin irritation.

The chemicals produced minimal skin irritation in studies which were performed in accordance with OECD Test Guideline (TG) 404. An average irritation index of 0.25/4 was reported for Na-DHA (CIR,1985).

Eye Irritation

The chemicals are considered to cause minimal eye irritation.

No data are available for DHA.

The chemical, Na-DHA, is reported to be 'minimally irritating' to eyes in animal studies. Average irritation scores of 5, 1, 2 and 0 (out of 110) were reported on days 1, 2, 3 and 4, respectively (CIR, 1985). The irritation scores do not warrant a hazard classification for the chemical.

Observation in humans

The chemicals were non-irritating when applied as a paste (Na-DHA 60 % in water and DHA 65 % in sesame oil) under occlusive patch conditions to the intact skin of male and female volunteers (n = 100/sex) for a period of five days (CIR, 1985).

Sensitisation

Skin Sensitisation

No animal data are available.

Observation in humans

The chemicals may have some potential to cause skin sensitisation in humans when absorbed through the broken skin. However, the data available are not sufficient to warrant a hazard classification.

The chemicals, DHA and Na-DHA were applied as a paste (Na-DHA 60 % in water and DHA 65 % in sesame oil) under occlusive patch conditions to the intact skin of male and female volunteers (n=100/sex) for a period of five days. The challenge application was conducted, three weeks after the induction. No skin reactions were observed (CIR, 1985).

A study of patients receiving a hyaluronic acid cream containing Na-DHA to treat leg ulcers showed some evidence of contact eczema. During patch testing, four out of seven patients treated with Na-DHA (3 %) showed positive skin sensitisation reactions. The study authors stated that the application of the cream on broken skin, and the potentiating role of propylene glycol also found in the cream, could have facilitated its absorption, contributing to the skin sensitising potential (Milpied et al., 2011).

Allergic contact reactions have been reported in humans exposed to these chemicals, but were considered rare (Timmer, 2000; HSBD).

Repeated Dose Toxicity

Oral

The studies available are of limited value due to short treatment periods and the number of animals tested. The short-term studies in rats indicate anticoagulant activity and haemorrhage from doses around 100 mg/kg bw/d.

In a 34-day oral gavage study, groups of male rats (n=5) were administered DHA at concentrations of 0, 10, 30, 100 or 300 mg/kg bw/d in olive oil, for a total of 24 doses. A no observed adverse effect level (NOAEL) of 100 mg/kg bw/d was determined based on the effects observed at 300 mg/kg bw/d (20–30 % reduced body weight compared with controls, increased mortality after 11 days, emaciation, contracted stomach containing some blood, congested mucosa, slight haemorrhaging and slight liver swelling) (CIR, 1985).

In a 14-day oral gavage study, groups of male Sprague Dawley (SD) rats (n=5) were administered Na-DHA at concentrations of 0, 50, 100, 200 or 400 mg/kg bw/d. Increased mortality (5/5 at 400 mg/kg bw/d and 2/5 at 200 mg/kg bw/d), haemorrhage, decreased body weight and food consumption were observed in animals of the two highest dose groups. Severe haemorrhaging was observed in various organs, including the stomach, intestines, testes, epididymides, subcutaneous tissue and subdural area in the animals that died in the study. The study authors attributed this to vitamin K deficiency. Blood coagulation, measured as prolonged prothrombin time (PT), was prolonged in surviving rats of the 100 and 200 mg/kg bw/d dose groups (19 and 25 seconds, respectively, compared with 15 seconds for the control group). The chemical, Na-DHA, was shown to inhibit the enzyme vitamin K epoxide reductase (VKOR), with an inhibitory concentration (IC50) of 3.15 mM, compared with warfarin, which has an IC50 of 2.15 µM for VKOR (Sakaguchi et al., 2008).

In an oral gavage study, dogs (n=9) were administered Na-DHA at 80 mg/kg bw/d in water. Toxic effects included anorexia, salivation, weight loss (13–33 % reduction compared with controls), convulsions and death (after 10–23 days). Two dogs were force-fed daily to counteract the anorexia observed in the treatment group, and these two animals survived for 73 days and were reported to be 'in excellent health' prior to sacrifice (CIR, 1985; Seevers MH, et al., 1950). In another study, dogs fed 50 mg/kg bw/d of DHA for six days a week tolerated the chemical for 200 days with no ill effect (CIR, 1985).

In a one-year oral gavage study, monkeys (*Macaca mulatta*) were administered DHA (5 % in olive oil) or Na-DHA (10 % (as acid) in water) at concentrations of 0, 50, 100, 200 or 300 mg/kg bw/d (n=1/dose). A NOAEL of 100 mg/kg bw/d was established based on lack of appetite and weight loss at 200 mg/kg bw/d. Monkeys that received the chemicals at 200 mg/kg bw/d were able to recover from the treatment-related effects when dosing was stopped for three days; the dosing regime of 3–4 doses/week was tolerated for one year. At 300 mg/kg bw/d, reduction in appetite and weight loss (23 % reduction with DHA and 13 % reduction with Na-DHA), along with changes in motor activity, vomiting and convulsions occurred with monkeys either dying or euthanised on day 18 (after 11 doses) or 26 (after 20 doses) during treatment with DHA and Na-DHA, respectively. Moderate degeneration of the renal tubular epithelium and inflammation of the small intestine were observed at 300 mg/kg bw/d DHA (CIR, 1985).

Dermal

No data are available.

Inhalation

No data are available.

Observation in humans

Three men ingested daily doses of DHA at 500 mg for 115–118 days and then 750 mg for 35–38 days, and showed no adverse effects (CIR, 1985).

Genotoxicity

No data are available for DHA and only limited data are available for Na-DHA. The studies on Na-DHA indicated mixed results and, therefore, no conclusion can be made on the potential genotoxicity of these chemicals.

Na-DHA gave mostly negative results in several in vitro tests for gene mutation and clastogenicity (CIR, 1985):

- bacterial reverse mutation assay using *Salmonella typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 and *Escherichia coli* WP2 hcr, with or without metabolic activation up to 5 mg/plate;
- chromosome aberration test in Chinese hamster cells (unspecified) at 0.5 mg/mL;
- sister chromatid exchange assay in Chinese hamster cells (unspecified) at up to 1 mM; and
- a DNA repair test (rec assay) in *Bacillus subtilis* H17 and M45 with or without metabolic activation up to 5 mg/plate.

Na-DHA at 3.0 mg/mL gave positive results in one in vitro chromosome aberration assay in Chinese hamster fibroblast cells (Hayashi et al., 1988; HSDB).

Na-DHA gave negative results (dose not specified) in an in vivo chromosome aberration test in rats (CIR, 1985).

Equivocal results are reported in an in vivo micronucleus assay in male ddY mice that received intraperitoneal (i.p.) injections of Na-DHA. Induction of micronucleated polychromatic erythrocytes was observed at 125–1000 mg/kg bw, but the statistical significance of this increased induction was reported to be borderline (Hayashi et al., 1988).

Carcinogenicity

The two available carcinogenicity studies on DHA are of limited value due to an irrelevant route of exposure or due to the low number of animals tested. However, these two studies indicate no carcinogenic potential for DHA. No data are available on Na-DHA.

In an 85-week carcinogenicity study, male rats (n = 6) were administered DHA (2 mg in 0.5 mL arachis oil, equivalent to 592 mg/kg bw/d), two days a week by subcutaneous injection. Local tumours at the injection site were reported in 5/6 treated rats, while no other tumours were observed. The study authors stated that

localised sarcomas observed are 'notoriously unreliable as an indicator of carcinogenicity' (CIR, 1985; RTECS).

In a 64-week study, male rats (n = 6) were administered DHA in drinking water at a concentration of 10 mg/100 mL (equivalent to 6.6 mg daily). No tumours were reported (CIR, 1985).

In a co-administration study in rats, DHA was shown to have an inhibitory effect on the induction of hepatomas by the known carcinogen 4-(dimethylamino)azobenzene (DAB). Groups of rats were administered (in the diet) DAB alone (0.06 %), DAB (0.06 %) followed by DHA (0.25 %), DAB (0.06 %) with DHA (0.25 %) or DHA alone (0.25 %), over a one-year period. Hepatomas and cholangiocarcinomas were observed in the DAB alone and DAB followed by DHA groups (27.3 and 42.9 %, respectively). No tumours were observed in either the DHA alone group or the DAB and DHA co-administered group (CIR, 1985).

Reproductive and Developmental Toxicity

No reproductive toxicity studies are available. Based on the information available the chemicals are not expected to have developmental toxicity.

In a developmental toxicity study (guideline not stated), Wistar rats were administered Na-DHA orally at concentrations of 0, 25, 50 or 100 mg/kg bw/d during gestation days (GD) 6–17. Reduced maternal body weight gain and food consumption were observed at 50 and 100 mg/kg bw/d. The foetuses from these groups had reduced body weight, high incidence of skeletal variations and retarded ossification. Increased foetal mortalities were observed in the 100 mg/kg bw/d dose group, which was considered to be due to retardation of intrauterine development (Tanaka et al., 1988). The developmental effects are considered to be secondary to maternal effects (i.e. reduced food consumption) at 50 and 100 mg/kg bw/d.

In a developmental toxicity study, mice were administered Na-DHA orally at concentrations of 0, 50, 100 or 200 mg/kg bw/d during GD 6–15. High mortality and decreased foetal body weight were observed in the 200 mg/kg bw/d group. Skeletal abnormalities were observed, including a supernumerary rib (in all treated groups), sternabrae deformities and rib malformation, but the study authors stated that these effects were not significant compared with controls (CIR, 1985).

In a repeated dose oral toxicity study, a dog that received DHA at 80 mg/kg bw/d orally and intravenously for 49 days gave birth to a litter of puppies. No developmental abnormalities were reported (CIR, 1985).

Risk Characterisation

Critical Health Effects

The critical health effects for risk characterisation include systemic acute effects by oral exposure.

The chemicals are powders when manufactured, but data are lacking for acute or repeated dose inhalation toxicity. The information available indicates some potential for skin sensitisation in humans when used on damaged skin.

Public Risk Characterisation

The main uses of the chemicals in cosmetic/domestic products are as preservatives or antimicrobial agents. Therefore, high concentrations of these chemicals are not expected to be present in consumer products.

Internationally, the chemicals are listed as safe for use in cosmetics up to a maximum concentration of 0.6 % (as dehydroacetic acid) (CosIng). They are also prohibited from use in aerosol dispensers (sprays), probably due to a lack of data on inhalation toxicity. There are no restrictions in Australia on using these chemicals in cosmetics or domestic products.

Although the public may be exposed to the chemicals through cosmetic/domestic uses, given the low hazards identified for the chemicals and low concentrations expected in consumer products, the chemicals are not considered to pose an unreasonable risk to public health.

Occupational Risk Characterisation

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.

Based on the available data, the hazard classification in HSIS is considered appropriate. The keto-enol tautomeric form of dehydroacetic acid (CAS No. 16807-48-0) should also included in the HSIS entry for DHA.

NICNAS Recommendation

Assessment of these chemicals is considered to be sufficient, provided that the keto-enol tautomeric form of dehydroacetic acid (CAS No. 16807-48-0) is included in the HSIS entry for DHA, 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.

Hazard	Approved Criteria (HSIS)[a]	GHS Classification (HCIS)[b]
Acute Toxicity	Harmful if swallowed (Xn; R22)*	Harmful if swallowed - Cat. 4 (H302)

[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 label instructions.

Advice for industry

Control measures

Control measures to minimise the risk from oral, dermal or 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 local exhaust ventilation to prevent the chemical from entering the breathing zone of any worker;
- 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.

References

Approved Criteria for Classifying Hazardous Substances [NOHSC: 1008(2004)] Third edition. Accessed at **http://www.safeworkaustralia.gov.au/sites/SWA/about/Publications/Documents/258/ApprovedCriteria_Classifying_Hazardous_Substances_NOHSC1008-2004_PDF.pdf**

Cosmetic Ingredient Review (CIR) 1985. Final report on the safety assessment of Sodium Dehydroacetate and Dehydroacetic Acid. International Journal of Toxicology 4(3) pp. 123-159.

eChemPortal. Accessed May 2014 at **http://www.echemportal.org/echemportal/substancesearch/substancesearchlink.action**.

Galleria Chemica. Accessed May 2014 at **http://jr.chemwatch.net/galleria/**

Hayashi M, Kishi M, Sofuni T, Ishidate M Jr. 1988. Micronucleus tests in mice on 39 food additives and eight miscellaneous chemicals. Food and Chemical Toxicology 26(6) pp. 487–500.

Hazardous Substances Data Bank (HSDB). National Library of Medicine. Accessed on May 2014 at **http://toxnet.nlm.nih.gov**.

Milpied B, Collet E, Genillier3 N& Vigan M 2011. Allergic contact dermatitis caused by sodium dehydroacetate, not hyaluronic acid, in Ialuset® cream. Contact Dermatitis, 65, pp. 359–368.

Registry of Toxic Effects of Chemical Substances (RTECS). Accessed at **http://www.cdc.gov/niosh/rtecs/**

Safe Work Australia (SWA). Hazardous Substances Information System (HSIS). Accessed May 2014 at **http://hsis.safeworkaustralia.gov.au/HazardousSubstance**

Sakaguchi Y, Suga S, Oshida K, Miyamoto-Kuramitsu K, Ueda K, Miyamoto Y 2008. Anticoagulant effect of sodium dehydroacetate (DHA-S) in rats. Journal of Applied Toxicology 28 pp. 524-529.

Seevers MH, Shideman FE, Woods A, Weeks JR and Kruse WT 1950. Dehydroacetic acid (DHA) II. General pharmacology and mechanism of action. Journal of Pharmacology and Experimental Therapeutics 99(1) pp. 69–83.

Tanaka S, Kawashima K, Nakaura S, Djajalaksana S, Takanaka A 1988. Studies on the teratogenic potential of sodium dehydroacetate in rats. Eisei Shikenjo Hokoku 106 pp. 54-61.

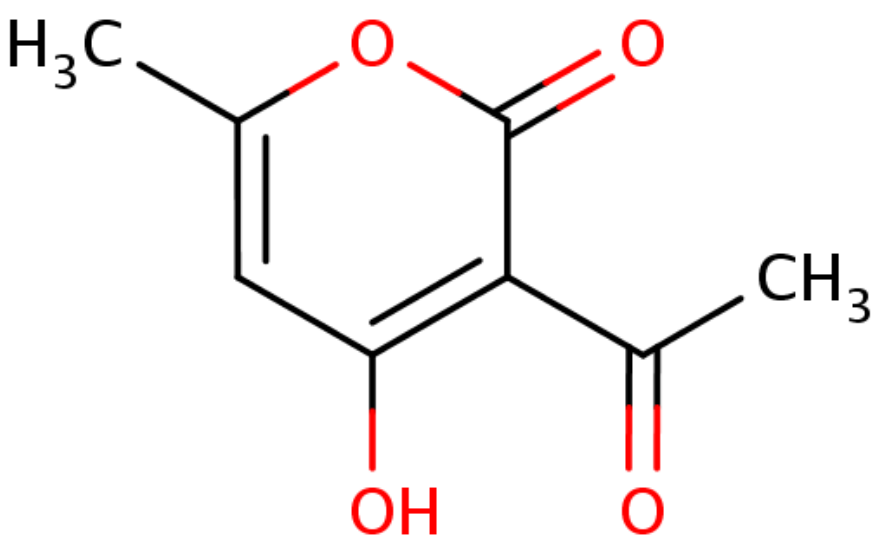
Timmer C 2000. 'Antimicrobials and disinfectants' in L Kanerva Handbook of Occupational Dermatology, pp. 462-473. Springer. Accessed June 2014 at **http://books.google.com.au/books?id=ZrclHh9Ep7AC&pg=PA462&source=gbs_toc_r&cad=3#v=onepage&q&f=false**

Uchida O, Naito K, Yasuhara K, Ohba S, Sato C, Shimonuri S, Kobayashi K, Tobe M. 1985 Studies on the acute oral toxicity of dehydroacetic acid, sorbic acid and their combination compound in rats. Eisei Shikenjo Hokoku. 103 pp. 166-71.

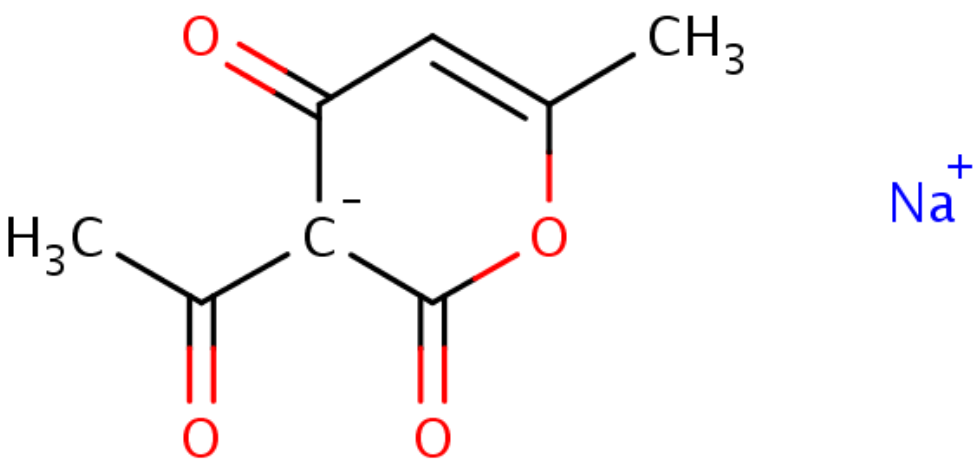
Last Update 27 November 2014

Chemical Identities

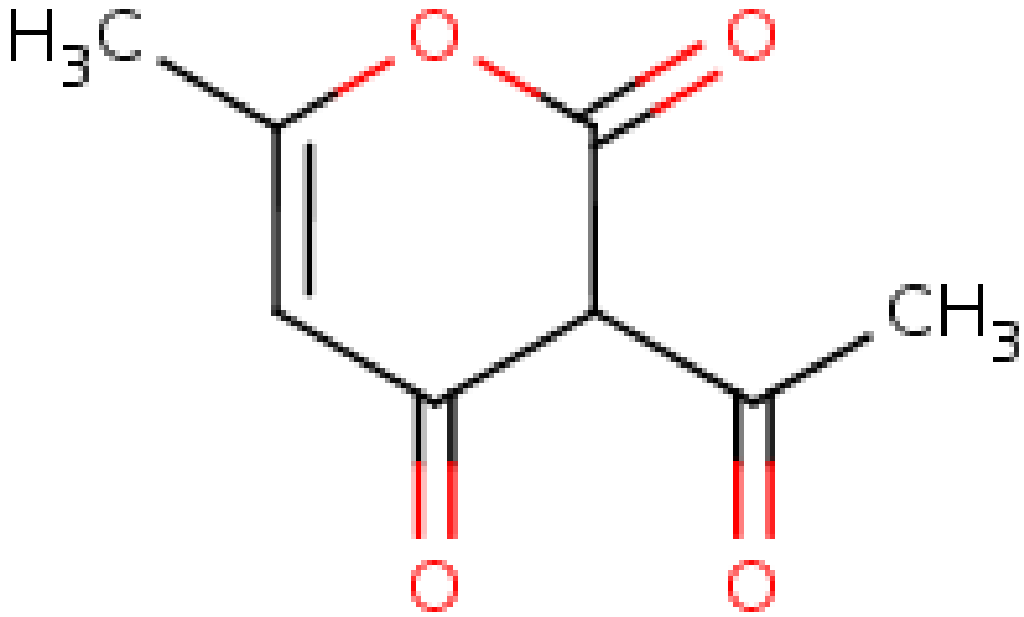
Chemical Name in the Inventory and Synonyms	**2H-Pyran-2,4(3H)-dione, 3-acetyl-6-methyl-** 3-acetyl-6-methyl-2H-pyran-2,4-(3H)-dione dehydroacetic acid (DHA)
CAS Number	520-45-6
Structural Formula	

	
Molecular Formula	C8H8O4
Molecular Weight	168.15

Chemical Name in the Inventory and Synonyms	**2H-Pyran-2,4(3H)-dione, 3-acetyl-6-methyl-, ion(1-), sodium** sodium dehydroacetate (Na-DHA)
CAS Number	4418-26-2
Structural Formula	

	
Molecular Formula	C8H7O4.Na
Molecular Weight	190.13

Chemical Name in the Inventory and Synonyms	**4H-Pyran-4-one, 3-acetyl-2-hydroxy-6-methyl-** Dehydroacetic acid
CAS Number	16807-48-0
Structural Formula	

	
Molecular Formula	C8H8O4
Molecular Weight	168.15

Share this page