1 Identification

GHS Product Identifier

CITRIC ACID

Other means of identification

CAS:	77-92-9 5949-29-1
EC:	201-069-1
RTECS:	GE7350000
ICSC:	0855
FEMA:	2306
NSC:	759606 626579 30279
Chemical Family:	Organic Acid
Synonyms:	Anhydrous Citric Acid Citrate Citric Acid Citric Acid Monohydrate Citric Acid, Anhydrous
Proper Shipping Name:	Not regulated for transport
Chemical Formula:	$C_6H_8O_7$

Recommended use of the chemical and restriction on use

CITRIC ACID is used in Antimicrobial Actives, Chelating Agents, Processing Aids and Additives. Its used as a flavouring Agent, food additive, antioxidant synergist and sequestrant.

Supplier's details

AQUATRADE WATER TREATMENT CHEMICALS (PTY) LTD

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2 Hazard(s) identification

Classification of the substance or mixture

Classification according to Regulation (EC) No 1272/2008
Serious Eye Damage/Eye Irritation (Category 2), H319

For the full text of the H-Statements mentioned in this Section, see Section 16.

GHS label elements

Warning

Causes serious eye irritation

Wash thoroughly after handling.

Wear protective gloves/protective clothing/eye protection/face protection.

IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.

IF eye irritation persists: Get medical advice/attention.

Other hazards which do not result in classification

The substance is not PBT / vPvB.

3 Composition/information on ingredients

Description	CAS Number	EINECS Number	%	Note
2-Hydroxypropane-1,2,3-tricarboxylic acid	77-92-9	201-069-1	0 - 99	

4 First-aid measures

Description of necessary first-aid measures

Eyes

First check the victim for contact lenses and remove if present. Flush victim's eyes with water or normal saline solution for 20 to 30 minutes while simultaneously calling a hospital or poison control center. **DO NOT** put any ointments, oils, or medication in the victim's eyes without specific instructions from a physician. If symptoms (such as redness or irritation) develop, immediately transport the victim to a hospital.

Skin

IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Gently wash all affected skin areas thoroughly with soap and water. If symptoms such as redness or irritation develop, IMMEDIATELY call a physician and be prepared to transport the victim to a hospital for treatment.

Inhalation

IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. If symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop, call a physician and be prepared to transport the victim to a hospital. Provide proper respiratory protection to rescuers entering an unknown atmosphere. Whenever possible, Self-Contained Breathing Apparatus (SCBA) should be used; if not available, use a level of protection greater than or equal to that advised under Protective Clothing.

Ingestion

DO NOT INDUCE VOMITING. If the victim is conscious and not convulsing, give 1 or 2 glasses of water to dilute the chemical and IMMEDIATELY call a hospital or poison control center. Be prepared to transport the victim to a hospital if advised by a physician. If the victim is convulsing or unconscious, **DO NOT** give anything by mouth, ensure that the victim's airway is open and lay the victim on his/her side with the head lower than the body. **DO NOT** INDUCE VOMITING. IMMEDIATELY transport the victim to a hospital.

Most important symptoms/effects, acute and delayed

Inhalation

Inhalation of dust irritates nose and throat. Cough. Shortness of breath. Sore throat. Lungs, thorax, or respiration: other changes; musculoskeletal: other changes.

Eye contact

Contact with eyes causes irritation. Redness.

Skin contact

A severe eye and moderate skin irritant. Redness. Pain.

Ingestion

Abdominal pain. Sore throat. Behavioral: tremor; behavioral: convulsions or effect on seizure threshold; behavioral: muscle contraction or spasticity).

Indication of immediate medical attention and special treatment needed, if necessary

Immediate first aid
Ensure that adequate decontamination has been carried out. If patient is not breathing, start artificial respiration, preferably with a demand-valve resuscitator, bag-valve-mask device, or pocket mask, as trained. Perform CPR as necessary. Immediately flush contaminated eyes with gently flowing water. Do not induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain an open airway and prevent aspiration. Keep patient quiet and maintain normal body temperature. Obtain medical attention.

Basic treatment
Establish a patent airway (oropharyngeal or nasopharyngeal airway, if needed). Suction if necessary. Watch for signs of respiratory insufficiency and assist respirations if necessary. Administer oxygen by nonrebreather mask at 10 to 15 L/min. Monitor for pulmonary edema and treat if necessary. Monitor for shock and treat if necessary. For eye contamination, flush eyes immediately with water. Irrigate each eye continuously with 0.9% saline (NS) during transport. Do not use emetics. For ingestion, rinse mouth and administer 5 mL/kg up to 200 mL of water for dilution if the patient can swallow, has a strong gag reflex, and does not drool. Activated charcoal is not effective. Do not attempt to neutralize because of exothermic reaction. Cover skin burns with dry, sterile dressings after decontamination.

Advanced treatment
Consider orotracheal or nasotracheal intubation for airway control in the patient who is unconscious, has severe pulmonary edema, or is in severe respiratory distress. Early intubation, at the first sign of upper airway obstruction, may be necessary. Positive-pressure ventilation techniques with a bag valve mask device may be beneficial. Consider drug therapy for pulmonary edema. Consider administering a beta agonist such as albuterol for severe bronchospasm. Monitor cardiac rhythm and treat arrhythmias as necessary. Start IV administration of D5W "To keep open", minimal flow rate. Use 0.9% saline (NS) or lactated Ringer's (LR) if signs of hypovolemia are present. For hypotension with signs of hypovolemia, administer fluid cautiously. Consider vasopressors if patient is hypotensive with a normal fluid volume. Watch for signs of fluid overload. Use proparacaine hydrochloride to assist eye irrigation.

5 Fire-fighting measures

Suitable extinguishing media

Suitable extinguishing media
Water, water spray, dry powder, foam, carbon dioxide (CO2).

Extinguishing media which must not be used
None.

Specific hazards arising from the chemical

Melts and decomposes. The reaction is not hazardous. Combustible. Finely dispersed particles form explosive mixtures in air.

Hazardous decomposition products
carbon oxides.

Special protective actions for fire-fighters

Protection of fire-fighter
Use personal protective equipment.

Specific methods
Standard procedure for chemical fires.

6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

Use personal protective equipment. Particulate filter respirator adapted to the airborne concentration of the substance. Wear eye protection . **Avoid** contact with skin and eyes.

Environmental precautions

Prevent further leakage or spillage if safe to do so. No special environmental precautions required.

Methods and materials for containment and cleaning up

Cleanup Methods
Sweep spilled substance into covered labelled containers. If appropriate, moisten first to prevent dusting. Wash away remainder with plenty of water.

Disposal Methods
Wastewater from contaminant suppression, cleaning of protective clothing/equipment, or contaminated sites should be contained and evaluated for subject chemical or decomposition product concentrations. Concentrations shall be lower than applicable environmental discharge or disposal criteria. Alternatively, pre-treatment and/or discharge to a permitted wastewater treatment facility is acceptable only after review by the governing authority and assurance that "pass through" violations will not occur. Due consideration shall be given to remediation worker exposure (inhalation, dermal and ingestion) as well as fate during treatment, transfer and disposal. If it is not practicable to manage the chemical in this fashion, it must be evaluated in accordance with EPA 40 CFR Part 261, specifically Subpart B, in order to determine the appropriate local, state and federal requirements for disposal.

Inject at base of incinerator equipped with afterburner. Flammable solvent may be added.

7 Handling and storage

Precautions for safe handling

Avoid dust formation. Take precautionary measures against static discharges. Use personal protective equipment.

Conditions for safe storage, including any incompatibilities

Technical measures/Storage conditions
Keep tightly closed in a dry and cool place.

Incompatible products
Strong oxidizing agents, strong bases.

Packaging material
Polyethylene coated paper bags, Polyvinyl or Polyethylene/propylene big bags.

SANS10236-0 : 2009 provisions
None

8 Exposure controls/personal protection

Control parameters

Threshold Limit Values (TLV)
(inhalable fraction): 2 mg/m^3

Peak limitation category: I(2)

Pregnancy risk group: C

Allowable Tolerances
Unless specifically excluded, residues resulting from the use of the following substance as either an inert or an active ingredient in a pesticide chemical formulation, including antimicrobial pesticide chemicals, is exempted from the requirement of a tolerance under FFDCA section 408, if such use is in accordance with good agricultural or manufacturing practices. Citric acid is included on this list.

Appropriate engineering controls

Fire Prevention
NO open flames. Closed system, dust explosion-proof electrical equipment and lighting. Prevent deposition of dust.

Exposure Prevention
PREVENT DISPERSION OF DUST!

Inhalation Prevention
Use ventilation (not if powder).

Ingestion Prevention
Do not eat, drink, or smoke during work.

Individual protection measures

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors. Recommendations below is advisory only and must be evaluated by an industrial hygienist and safety officer familiar with the specific situation of anticipated use by our customers. It should not be construed as offering an approval for any specific use scenario.

Eye/face protection

Safety glasses with side shields or safety goggles. Use equipment for eye protection tested and approved under appropriate government standards such as NIOSH (US) or EN 166 (EU). Contact lenses should not be worn as they may contribute to severe eye injury.

Hand protection

Handle with gloves. Gloves must be inspected prior to use. Use proper glove removal technique (without touching glove's outer surface) to avoid skin contact with this product. Dispose of contaminated gloves after use in accordance with applicable laws and good laboratory practices. Wash and dry hands. The selected protective gloves have to satisfy the specifications of EU Directive 89/686/EEC and the standard EN 374 derived from it.

If used in solution, or mixed with other substances, and under conditions which differ from EN 374, contact the supplier of the CE approved gloves.

Body Protection

Complete suit protecting against chemicals, The type of protective equipment must be selected according to the concentration and amount of the dangerous substance at the specific workplace.

Respiratory protection

Where risk assessment shows air-purifying respirators are appropriate use a moulded respirator type EN 149:2001 FFP1/FFP2 as a backup to engineering controls. Use respirators and components tested and approved under appropriate government standards such as NIOSH (US) or CEN (EU).

9 Physical and chemical properties

Physical and chemical properties

Appearance (physical state, colour etc) @ 20°C and 1013 hPa:	Solid White organic
Odour:	Odourless
Odour threshold:	No data
pH @ 5 w% at 25°C:	1.8
Melting/Freezing Point @ 101 325 Pa:	426 K / 153 °C
Initial boiling point and boiling range:	Decomposes before boiling
Flash point:	Not relevant

Evaporation rate:	Not relevant
Flammability (solid, gas):	Non flammable
Upper/lower flammability or explosive limits:	Not explosive
Vapour pressure @ 25 °C:	2.21E-6 Pa
Vapour density:	Not relevant
Relative density @ 20 °C:	1.665
Solubility(ies) @ 20 °C:	590 g/L
Partition coefficient: n-octanol/water:	Log Kow: -0.2 to -1.8
Auto-ignition temperature:	Not relevant
Decomposition temperature:	1 850 ° F
Viscosity @ 20 °C:	2.549 cP 30% aqueous solution
Oxidising properties:	Not oxidizing
Dissociation constant @ 25 °C:	pKa: 3.13, 4.76 and 6.4
Particle size distribution (Granulometry):	D50 of the fraction below 100 µm was at 31.99 µm

NOTE: The physical data presented above are typical values and should not be construed as a specification.

10 Stability and reactivity

Reactivity

Air and Water Reactions
The pure material is moisture sensitive (undergoes slow hydrolysis) Water soluble. queous form is corrosive to copper, zinc, aluminum and their alloys.

Reactive Group
Acids, Carboxylic, Alcohols and Polyols.

Chemical stability

Stable under normal operating, handling, storage and transport situations.

Possibility of hazardous reactions

CITRIC ACID reacts with oxidizing agents, bases, reducing agents and metal nitrates (NTP, 1992). Reactions with metal nitrates are potentially explosive.

Conditions to avoid

Direct sunlight. Extremely high or low temperatures. **Avoid** dust formation. Heat, flames and sparks.

Incompatible materials

Amines. Heavy metals. Strong oxidizing agents. Strong acids. Strong bases.

Hazardous decomposition products

Heating to the point of decomposition causes emission of acrid smoke and fumes. Carbon monoxide. Carbon dioxide.

11 Toxicological information

Toxicological (health) effects

Acute Toxicity
Musculoskeletal: other changes. Lungs, thorax, or respiration: cyanosis. Behavioral: convulsions or effect on seizure threshold.

Corrosion/Irritation
The key study reports citric acid to be not irritating as assessed by a 4 hour semiocclusive application to intact skin (Haarmaan & Reimer, 1990). This is further supported by three reliability 2 supporting studies which are all equivalent or similar to guideline and also concluded that citric acid is not irritating to skin (Miles 1979, Roche 1984).

The key study for eye irritation reports that a 30% aqueous solution of citric acid caused well-defined to moderate conjunctival irritation that had not fully resolved after 14 days. A 10% solution was associated with weak to moderate

conjunctival effects, resolved after 7 days (Roche 1984). The reported eye reaction data was re4 -evaluated with respect to the recovery period and whether there would have been complete recovery at the 30% concentration if a longer recovery period had been used (21 days rather than 14 days). It was found that the score level of 1 for conjunctival redness in 1/3 animals in day 14 would have completely resolved if there had been a further 7 day observation period. This opinion was based on the recovery profile in the 2 other rabbits at this concentration and the ongoing recovery profile for the rabbit in question (significant recovery had already occurred by day 14 with only a low grade of conjunctival rednedd still remaining).

Sensitisation
No published sensitisation studies were identified for citric acid in the literature search.

In accordance with REACH Annex XI Section 2, the sensitisation study (required in Section 8.3) does not need to be conducted because citric acid and its salts have been used for many years as a permitted additive for human food, medicines and cosmetics. During this time, there has been no documented evidence that citric acid could be a sensitiser. Therefore there is no scientific basis for recommending animal studies to investigate this endpoint.

There aren't any structural alerts that suggest citric acid is a sensitiser. Similarly, other acids, e.g. the phosphonates groups, are also not known to be sensitisers.

Repeated dose toxicity
In accordance with Annex 11, Section 1 of REACH, it is not necessary to conduct the study based on:

1. The available repeat dose toxicity studies. For example, a reliable five day study in rats gave NAOEL value of 4000 mg/kg (Bächtold, 1976a); a 10 day study in mice and rats gave NOAEL (mortality) values of 1000 mg/kg and 4000 mg/kg respectively (Bächtold, 1978a). Although the other numerous repeat dose studies that have been conducted do not meet current testing requirements, they still indicate a lack of any significant toxicological effects under the test conditions used;
2. The low acute toxicity (LD50 = 5400 mg/kg with mice Roche 1981);
3. A long history of human exposure. For example, Citric Acid has wide dispersive use, being naturally present in common fruit and vegetables. It is also added to processed food and beverages. (HERA 2005). In addition Citric Acid has well established and documented metabolic pathways in humans. (WHO Food Additives, Series 5, 1973);

No dermal or inhalation repeated dose animal studies related to citric acid were located.

This is probably because citric acid is commonly used as a food additive and therefore the oral route is the most significant (and therefore studied) route of exposure. Similarly the low vapour pressure of citric acid means that it is unlikely to be inhaled unless used in a spray, or powder form comprising respirable sized particles. Public and private usage of the liquid form indicates that exposure by the dermal route would be the most significant. Based on the chemical structure of citric acid (hydrophilic, ionisable), it is unlikely that citric acid would penetrate the stratum corneum of the skin. If it did, two sub-cutaneous toxicity studies (Yokotani 1971) indicate low toxicity (>2500 mg/kg bw).

Genetic Toxicity
Citric acid (CAS number 77 -92 -9) has been tested in a number of bacterial assays, all of which gave negative results. There is also information from a lower reliability study that citric acid does not cause chromosome aberrations in vitro: this result does not agree with a recently published study. Evidence for genetic toxicity has been described in a recent publication of results from an in vitro micronucleus study. An in vivo chromosome aberration study does not support the conclusion of the recently reported in vitro studies in mammalian cells, and an in vivo rodent dominant lethal assay also showed no evidence of chromosome damage.

Citric acid is negative in in vivo genotoxicity testing, although effects have been observed in some in vitro studies. Moreover, it has been used as a food additive over a long period. In addition, citrate plays a central role in cellular metabolism, so it is considered that classification for mutagenicity is not required.

Carcinogenicity
No data is available for the carcinogenicity of citric acid. However, further testing is not considered necessary because:

- The substance is not classified for mutagenicity; and
- There is no evidence from long term human exposure to citric acid that it is a carcinogen

Toxicity to reproduction

In accordance with Annex 11, Section 1 of REACH, the evidence based on:

1. The available developmental toxicity studies. A non standard repeat dose study by Wright, Hughes: Nutr. Rep. Int. 13: 563, (1976) where 5% citric acid was administered in the feed to rats and mice did not give rise to any reproductive effects. In addition a study by Bonting (1956) where 1.2% w/w citric acid in feed given daily to male and female rats over a period of 90 weeks did not give rise to any reproductive effects. Although these studies are not reliable, they help provide supporting evidence that citric acid is not expected to cause reproductive effects.
2. A long history of human exposure. For example, Citric Acid has wide dispersive use, being naturally present in common fruit and vegetables. It is also added to processed food and beverages. (HERA 2005). In addition Citric Acid has well established and documented metabolic pathways in humans. (WHO Food Additives, Series 5, 1973) is sufficient to fulfil the requirements for this endpoint.

Developmental toxicity/Teratogenicity
In accordance with Annex 11, Section 1 of REACH, the evidence based on:

1. The available developmental toxicity studies. A study by the Food & Drug Research Laboratories (1973) researched the teratogenic effects of citric acid in mice (NAOEL > 241 mg/kg/d), rats (NAOEL > 295 mg/kg/d), rabbits (NAOEL > 425 mg/kg/d), and hamsters (NAOEL > 272 mg/kg/d), There were no reported teratogenic effects in any of the species tested.
2. A long history of human exposure. For example, Citric Acid has wide dispersive use, being naturally present in common fruit and vegetables. It is also added to processed food and beverages. (HERA 2005). In addition Citric Acid has well established and documented metabolic pathways in humans. (WHO Food Additives, Series 5, 1973) is sufficient to fulfil the requirements for this endpoint.

Information on the likely routes of exposure

The substance can be absorbed into the body by inhalation and by ingestion.

Symptoms related to the physical, chemical and toxicological characteristics

Inhalation Symptoms
Cough. Shortness of breath. Sore throat.

Skin Symptoms
Redness.

Eye Symptoms
Redness. Pain.

Ingestion Symptoms
Abdominal pain. Sore throat.

Human exposure studies
While presumably aqueous solutions (2% in one case, not stated in the other) may produce pain or "sting", patch testing of 60 eczema patients with 2.5% citric acid in petrolatum did not produce any irritant or allergic reactions; thus, the reaction appears to reflect mainly the acid effect of the substance.

Repeated exposure of up to 15 g/d of potassium and sodium citrate as medications did not cause any reported marked side effects, but minor gastrointestinal disturbances (diarrhea, indigestion, nausea, "burning") were experienced by 22 out of 81 patients taking potassium citrate in water and 7 out of 75 taking solid potassium citrate (doses not stated in both groups) for the treatment of renal calculi.

Signs and symptoms
Injection of large volumes of citrated blood during transfusion may lead to hypocalcemia and changes in blood composition with concomitant nausea, muscle weakness, breathing difficulties and even cardiac arrest.

Within a concentration range of 0.625-320.0 mg/ml, inhaled citric acid caused cough in all subjects. Geometric mean (range) cough threshold was 13 (2.5-160) in normal subjects, 14 (5-40) in patients with mild, and 32 (20-40) mg/mL in patients with moderate to severe asthma, 40 (20-80) in current smokers, and 119 (80-160) in occasional smokers.

Citric acid is generally considered innocuous, although hypocalcemic effects were reported during transfusion of large

volumes of citrated blood. Frequent or excessive intake of citric acid may cause erosion of teeth and local irritation of mucous membranes. This effect also occurs with lemon juice, which contains about 7% citric acid and has a pH <3.

Case reports
In one patient a splash of large quantity of saturated solution of citric acid in eyes caused severe conjunctival reaction & ulceration of cornea, resulting in extensive adherent leukoma.

Two patients who suffered cardiac arrests after dialysis using hypertonic citrate are discussed. Both received anticoagulation as described in the literature, although the citrate infusion rate was lower than recommended. EKG obtained during the first such session showed no change in the Q-Tc interval with initiation of the infusion in either patient. Both were noted to have cardiac arrest within 5 minutes of discontinuation of dialysis, without warning symptoms, following the second and fifteenth treatments, respectively. The initial rhythm of ventricular fibrillation did not respond to standard advanced cardiac life support therapy, and the patients were not successfully resuscitated until they received intravenous calcium. It was postulated that the loss of positive calcium flux from the dialysate, in conjunction with circulating unmetabolized citrate, caused an electrolyte imbalance leading to the potentially fatal arrhythmia. Caution is recommended in using this method of regional anticoagulation.

After ingesting a single dose of 25 g citric acid (approx. 417 mg/kg) a young woman vomited and almost died.

Ingestion of a massive oral citric acid load included metabolic acidosis accompanied by an increase in the plasma anion gap that was not caused by L -lactic acidosis, hyperkalemia, and the abrupt onset of hypotension.

The authors report a case of severe citrate toxicity during volunteer donor apheresis platelet collection. The donor was a 40-year-old female, first-time apheresis platelet donor. Past medical history was remarkable for hypertension, hyperlipidemia, and depression. Reported medications included bumetanide, pravastatin, and paroxetine. Thirty minutes from the start of the procedure, the donor noted tingling around the mouth, hands, and feet. She then very rapidly developed acute onset of severe facial and extremity tetany. Empirical treatment with intravenous calcium gluconate was initiated, and muscle contractions slowly subsided over approximately 10 to 15 minutes. The events are consistent with a severe reaction to calcium chelation by sodium citrate anticoagulant resulting in symptomatic systemic hypocalcemia. Upon additional retrospective analysis, it was noted that bumetanide is a loop diuretic that may cause significant hypocalcemia. The authors conclude that careful screening for medications and underlying conditions predisposing to hypocalcemia is recommended to help prevent severe reactions due to citrate toxicity. Laboratory measurement of pre-procedure serum calcium levels in selected donors may identify cases requiring heightened vigilance. The case also illustrates the importance of maintaining preparedness for managing rare but serious reactions in volunteer apheresis blood donors.

Epidemiology studies
This study aims to investigate the possible effects of acute citrate administration on bone metabolism in healthy men. A placebo-controlled, crossover trial was conducted on 10 male volunteers. The volunteers received either a standardized infusion of citrate at 1.5 mg/kg body weight/min or the equal volume of placebo, separated by a washout period of 14 days. Serial blood and urine samples were collected and analysed for bone biochemical markers and electrolytes. Infusion of citrate resulted in increased serum levels of the bone formation marker osteocalcin (OC) and bone resorption marker C-telopeptide of type 1 collagen (CTX). Increases in CTX and OC were positively correlated to the surge in the serum concentration of intact parathyroid hormone (iPTH) but only OC showed correlation to changes in ionized calcium. Citrate infusion showed no effect on serum concentrations of bone alkaline phosphatase, osteoprotegerin, and bone tartrate-resistant acid phosphatase 5b, or the expression of receptor activator of nuclear factor kappa B ligand. Variations in OC and CTX were short-term as both bone markers gradually declined within 90 min following citrate exposure. Acute citrate load resulted in profound alterations of the bone markers OC and CTX. The short-term increase of CTX suggests a temporary shift to a higher bone turnover rate, although the clinical consequence of the observed changes in bone markers remains open.

Alternative and in vitro tests
Incubation of cultured human dental pulp cells in medium containing 0.5% (pH 4.74) or 1.0% (pH 3.42) of citric acid for 2 hr lead to 25% and 48% of cell death, respectively. Cytotoxicity of citric acid was associated with its acidity. Exposure of cells to pure 1% citric acid (pH 2.26) for 60 s lead to immediate cell death. Cytotoxicity was usually preceded by cell retraction, cell surface blebbing, and finally uptake of trypan blue. A medium containing 0.05% citric acid can retard the growth of pulp cells.

The authors investigated the effects of citric acid (CA) on cultured human osteoblastic (HOB) cells by evaluating cell

adhesion, proliferation, and cytotoxicity. (3)H-Thymidine-labeled HOB cells were incubated in culture medium supplemented or not with 4%, 6%, 8%, or 10% CA for 1 minute. After incubation, cell morphology was evaluated by Nomarski interferential light microscopy, cell proliferation was accessed by measurements of (3)H-thymidine associated to the cells, and cell lysis was monitored by measuring the amount of (3)H-thymidine released by cells. We observed that most of the CA-treated cells presented numerous atypical vacuoles, and such cells were also highly polymorphic, exhibiting round-shaped cells. Nonetheless, CA at all concentrations assayed did not yield cytotoxicity as measured by (3)H-containing DNA release, although significant decrease in cell proliferation was observed (P >.05). Furthermore, cells which were treated with CA at the lowest concentration assayed (4%) restored normal proliferation rates 3 days after treatment.

Other toxicity information
The erosive action of buffered and unbuffered aspirin (acetylsalicylic acid) on dental enamel was compared in vitro with that of citric acid and a cola soft drink. The degree of erosion depended on the duration of exposure and the concn of the acidic agent used. The unbuffered acetylsalicylic acid caused a slightly lower degree of erosive changes of enamel surfaces than citric acid. Calcium carbonate as buffer completely prevented erosion of the dental enamel induced by acetylsalicylic acid.

Non-Human Toxicity Excerpts
Laboratory animals: acute exposure
Coughing is reported for guinea pigs exposed for 30 minutes to atmospheric citric acid concentrations of 81 mg/cu m (aerosolised 6% solution). Coughing was also produced in guinea pigs exposed to 75 mg citric acid/mL as an aerosol for 3 minutes. Coughing was also caused by instillation of 1 mL of an approx. 5.2% solution to the lower trachea in lambs, but not by instillation to the mid-trachea or laryngeal area.

The application of a 50% citric acid solution to the tongue of dogs for 5 minutes resulted in severe ulceration and tissue damage.

Acute administration of citric-acid to mice and rats by oral, subcutaneous, and intraperitoneal administration resulted in ataxia, other motor changes, and death caused by respiratory or cardiac failure. Median lethal dose values for citric-acid were similar to those for commercial citric-acid. Subacute administration resulted in normal behavior and no significant hematological changes. Increased protein was seen in the urine and a decrease was seen in plasma protein, albumin, and cholesterol. Histopathology revealed no specific deleterious effects of citric-acid on the organs and tissues studied.

Citric acid and malic acid caused 71% and 43% fall in Mean Arterial Blood Pressure (MABP) of rats at the doses of 15 mg/kg and 30 mg/kg respectively while pyridine hydrochloride produced 34% rise in the MABP of rats at the dose of 30 mg/kg. LD50 and LD100 of citric acid in mice have been determined as 545 mg/kg and 1000 mg/kg, respectively.

Citric acid has a low acute toxicity by oral application in both rat and mouse. General effects comprised physiological disturbances (acidosis and calcium deficiency), while "high" doses caused nervous system effects as well as severe damage to the stomach mucosa.

Citric acid tested on rabbit eyes as single drop of 2% to 5% solution in water caused little or no injury ... Irrigation for 30 min with 0.5% to 2% solution causes severe injury; the 0.5% solution causes permanent cloudiness of cornea, and the 2% solution causes severe dense opacification.

Application of 500 mg citric acid to rabbit skin produced moderate irritation in 24 hr, whereas 750 mg caused severe irritation in the rabbit eye.

Laboratory animals: Subchronic or Prechronic Exposure
Citric acid (sodium salt) at 7.7% (equivalent to 5% free acid) in the diet of rabbits for 150 days produced no gross or histopathological changes or differences in growth or survival.

In dogs, a daily oral dose of 1380 mg/kg for 112 to 120 days produced no evidence of renal damage.

Groups of 10 male rats being fed up to 4.8% citric acid in feed (corresponding to approximately 4.67 g/kg/d) for 6 weeks showed slight growth reduction and, in the highest-dose group, mild blood and urine parameter changes and slight degeneration of the thymus gland and spleen.

In guinea pigs fed 1-5% citric acid (approx. 0.4- 2 g/kg/d) for 60 days, a reduced packed cell volume in the blood was the only effect noted.

Laboratory animals: Chronic Exposure or Carcinogenicity
A 2-year chronic oral study in rats being given 5% or 3% citric acid in feed (approx. 2 resp. 1.2 g/kg/d) found slightly decreased growth in the higher dosage group but no tissue abnormalities in the major organs. From the lower dosage a NOAEL of 1200 mg/kg/d results. Similarly, NOAELs of 1500 mg/kg/d (rabbit) and of 1400 mg/kg/d (dog) have been determined.

Laboratory animals: Developmental or Reproductive Toxicity
The effect of citric acid on the survival time of immature and sexually mature male mice and on the survival time and reproductive capacity of rats and mice /was studied/. Citric acid (5% in the diet) did not depress food intake but caused a loss in body weight gain and reduced survival time in mice and a slightly greater influence on mature animals. No effect was detected on the litter size or survival up to weaning of young mice or rats. The effects on body weight gain and survival time may have resulted from the chelating ability of citric acid, which could impair absorption of calcium and iron.

In a two-generation 90 days study with male and female rats fed 1.2 % citric acid no adverse effect on reproductive parameters nor any teratogenicity of dietary citric acid was seen. There were no indications of teratogenic or other adverse effects in three shorterterm reproductive studies in rats with dietary dosage of either 5% citric acid (approx. 2.5 g/kg/d) previous, during and after mating (NOEL = 2500 mg/kg/d), or 295 mg/kg/d (route unspecified) during days 6-15 of pregnancy.

Findings of no effects were reported for two reproductive and teratogenicity studies in mice receiving either 5 % citric acid (approx. 7.5 g/kg/d; in the range of acute LD50) previous, during and after mating (NOEL = 7500 mg/kg/d) or 241 mg/kg/d during days 6-15 of pregnancy.

There were no indications of teratogenicity or other adverse effects in female hamsters receiving 272 mg citric acid/kg during days 6-10 of pregnancy nor in female rabbits receiving up to 425 mg/kg/d during days 6-18 (NOEL = 425 mg/kg/d).

Genotoxicity
A dominant lethal assay with male rats being treated with up to 3 g/kg/d for 5 days was negative; no chromosomal damage occurred in the bone marrow cell of these male rats.

Citric acid did not induce mutations at concentrations up to 5 mg/plate in Salmonella typhimurium strains TA 92, TA 94, TA 98, TA 100, or TA 1535, and TA 1537 with or without a liver homogenate from rats pretreated with the polychlorinated biphenyl KC-400, and no clastogenic effects were seen in Chinese hamster fibroblast cells at concentrations up to 1 mg/mL.

To assess the ex vivo cytotoxicity of EDTA and citric acid solutions on macrophages. The cytotoxicity of 17% EDTA and 15% citric acid was evaluated on murine macrophage cultures using MTT-Tetrazolium method [3-(4,5-dimethylthiazol-2-yl)-2,5 diphenyl tetrazolium bromide]. A total of 5 x 10(5) cells were plated in medium culture with 17% EDTA or 15% citric acid. Fresh medium was used as a control. Toxicity values were analysed statistically by anova and Tukey's test ($P<0.05$) at short (0, 6, 12, 24 hr) and medium periods (1, 3, 5, 7 days), using ELISA absorbance. On the short term, both EDTA (0.253 nm) and citric acid (0.260 nm) exhibited cytotoxic effects on macrophage cultures ($P<0.05$). On the medium term, statistical differences were observed ($P<0.05$) between the groups. EDTA (0.158 nm) and citric acid (0.219 nm) were cytotoxic when compared with the control group; EDTA-reduced macrophage viability significantly more than citric acid ($P<0.05$). Both EDTA and citric acid had effects on macrophages cells ex vivo, but citric acid was less toxic in periods from 1 to 7 days of use.

Delayed and immediate effects and also chronic effects from short and long term exposure

Refer section 11.1 "Toxicological (health) effects" above.

Numerical measures of toxicity (such as acute toxicity estimates)

Acute Toxicity – Oral
Effect levels

Sex:	male/female
Dose descriptor:	LD50
Effect level:	5 400 mg/kg bw
95% CL:	4 500 - 6 400
Remarks on result:	other: observation limited to 10 days
Mortality:	No deaths at 3 g/kg bw, death of all exposed within 24 h at 8.5 g/kg bw, and above. See

table 1.

Clinical signs:	Clinical observations were limited to 2 h and 24 h after treatment; the descriptions given are very limited. See table 1.
Body weight:	No data on body weight following treatment.
Gross pathology:	Not examined.
Other findings:	None.

Acute Toxicity – Dermal

Effect levels

Sex:	male/female
Dose descriptor:	LD50
Effect level:	> 2 000 mg/kg bw
Mortality:	There were no deaths.
Clinical signs:	There were no signs of systemic toxicity.
Body weight:	All animals showed expected bodyweight gains during the study period.
Gross pathology:	No abnormalities were noted at necropsy.
Other findings:	None reported.

Acute Toxicity – Inhalation

Test animals

Species:	guinea pig

Administration / exposure

Route of administration:	inhalation: aerosol
Type of inhalation exposure:	whole body
Duration of exposure:	3 min

Results and discussion

Citric acid aerosol, 0.93M, 75 mg-ml-1 produced 90 +/-1.9 coughs during 3 minute exposure.

Acute Toxicity – Other Routes

Acute intraperitoneal LD50 values of 940 in mice and 725 mg/kg in rats (males only) were determined in a reliable study conducted according to an appropriate test protocol. The study was not conducted according to GLP.

Interactive effects

Citric acid aerosol inhalation caused decreases in dynamic respiratory compliance and forced expiratory volume in 0.1 s (FEV0.1). This airway constriction was significantly attenuated by MK-886, mepyramine, cromolyn sodium, and compound 48/80, but not by either methysergide or indomethacin. Both LTC4 and histamine infusion significantly increased the magnitude of citric acid-induced airway constriction in compound 48/80-pretreated guinea pigs. Citric acid inhalation caused significant increase in histamine level in the bronchoalveolar lavage sample, which was significantly suppressed by compound 48/80.

The relative efficacy of citric, malic, malonic, oxalic and succinic acids, and deferoxamine mesylate on the toxicity, distribution and excretion in mice exposed to aluminum were compared. Chelating agents were administered ip at a dose equal to one-fourth of their respective LD50. To determine the effect of the various chelators on the toxicity of aluminum, various doses of aluminum nitrate (938-3188 mg/kg) were administered ip, followed by one of the chelators. Survival was recorded at the end of 14 days. Malic acid and deferoxamine mesylate were the most effective in increasing the urinary excretion of aluminum. Citric acid was the most effective in increasing the fecal excretion of aluminum. Malonic, oxalic and succinic acids had no overall beneficial effects. Citric acid would appear to be the most effective agent of those tested in the prevention of acute aluminium intoxication.

When aluminum hydroxide and citric acid (133 mg Al/kg and 62 mg/kg, respectively) were simultaneously given orally to mice, fetal skeletal development defects resulted.

The primary purpose of this study was to determine the relative usefulness of various measures to monitor body aluminum burden in weanling rats fed various amounts of aluminum (0.39 umol aluminum/g diet for 29 days, approximately 40 umol aluminum/g diet with or without citrate for 29 days and approximately 100 umol aluminum/g diet with citrate for 12 or 29 days) or injected ip with graded doses of aluminum (0.01, 4.6, 11.8, 23.5 or 94 umol aluminum). Twenty four hours prior to sacrifice, all rats were injected ip with either desferrioxamine (75 mg) or buffer. All seven indices of aluminum exposure monitored (eg: tibia, liver, kidney and serum aluminum concn; changes in serum aluminum

concn in response to desferrioxamine; urinary aluminum excretion with and without desferrioxamine treatment) were highly (p< 0.001) correlated to parenteral aluminum exposure. Ingestion of citrate had small but significant effects on aluminum retention.

A high dietary intake of citric acid was without effect on growth rate unless the animals were on a low-calcium diet, in which case a reduced body weight was observed.

Ingestion of citric acid may impair absorption of calcium and iron.

Citric acid increased the incidence of bladder carcinomas in F344 rats administered N-butyl-N-(4-hydroxybutyl)nitrosamine or N-ethyl-N-(4-hydroxybutyl)nitrosamine in drinking water, apparently due to a secondary effect of the acid, which increased water consumption and hence the dose of the test material.

Where specific chemical data are not available

No additional data.

Mixtures

No additional data.

Mixture versus ingredient information

No additional data.

Other information

No additional data.

12 Ecological information

Toxicity

Hazard for aquatic organisms

Freshwater

Hazard assessment conclusion:	PNEC aqua (freshwater)
PNEC value:	0.44 mg/L
Assessment factor:	1 000
Extrapolation method:	assessment factor

Marine water

Hazard assessment conclusion:	PNEC aqua (marine water)
PNEC value:	0.044 mg/L
Assessment factor:	10 000
Extrapolation method:	assessment factor

STP

Hazard assessment conclusion:	PNEC STP
PNEC value:	1 000 mg/L
Assessment factor:	10
Extrapolation method:	assessment factor

Sediment (freshwater)

Hazard assessment conclusion:	PNEC sediment (freshwater)
PNEC value:	34.6 mg/kg sediment dw
Assessment factor:	1
Extrapolation method:	equilibrium partitioning method

Sediment (marine water)

Hazard assessment conclusion:	PNEC sediment (marine water)
PNEC value:	3.46 mg/kg sediment dw
Assessment factor:	1
Extrapolation method:	equilibrium partitioning method

Hazard for air

Hazard for terrestrial organisms

Soil

Hazard assessment conclusion:	PNEC soil

PNEC value:	33.1 mg/kg soil dw
Extrapolation method:	equilibrium partitioning method

Short-term toxicity to fish
Two 48 h LC50 values corresponding to 440 mg/L and 760 mg/L have been determined for the effects of citric acid on mortality of the freshwater fish Leuciscus idus melanotus. It is not clear whether the test medium was neutralised; the OECD guideline followed suggests adjusting the pH to ca. 7 when needed but the OECD SIDS on citric acid (2001) states that the test media was not neutralised. In spite of this uncertainty the LC50 of 440 mg/L has been selected as the key value.

The other available data support the conclusion that citric acid has low toxicity to fish in short-term exposures – the other LC50 values all being >100 mg/L.

Long-term toxicity to fish
There are no long-term test data for fish. Testing is not considered necessary because: Short-term toxicity to aquatic organisms is low.

Risk characterisation ratios based on PNECaquatic calculated using the short-term data are <1.

The substance is naturally occurring in aquatic organisms.

In accordance with Column 2 of REACH Annex IX, the requirement for long-term toxicity data for fish (required in Section 9.1.5) is waived on the grounds that the chemical safety assessment, conducted according to Annex I, indicates that it is not necessary.

Short-term toxicity to aquatic invertebrates
Freshwater
A reliable 24 h LC50 value of 1535 mg/L has been determined for the effects of the test substance on immobility of Daphnia magna in a neutralised solution. Although reported as an LC50 value it can be considered as analogous to an EC50 value based on immobility; it is difficult to differentiate dead and immobile daphnids by visual inspection.
The study reflects the lowest value that is available for this endpoint and has been selected as key for the freshwater compartment.

Marine
A reliable 48 h EC50 value of >50 mg/L has been determined for the effects of citric acid on substrate reattachment abilities of the zebra mussel D. polymorpha. The study does not present a conclusive EC50 (> value), therefore it has not been selected as key for the marine environment.

Other reliability 4 studies available with short term toxicity to invertebrates indicate that citric acid is of low acute toxicity to invertebrates in neutralised solutions.

Long-term toxicity to aquatic invertebrates
There are no long-term test data for invertebrates. Testing is not considered necessary because: Short-term toxicity to aquatic organisms is low.

Risk characterisation ratios based on PNECaquaticcalculated using the short-term data are <1.

The substance is naturally occurring in aquatic organisms.
In accordance with Column 2 of REACH Annex IX, the requirement for long-term toxicity data for aquatic invertebrates (required in Section 9.1.5) is waived on the grounds that the chemical safety assessment, conducted according to Annex I, indicates that it is not necessary.

Toxicity to aquatic algae and cyanobacteria
In accordance with Section 2 of REACH Annex XI, the study does not need to be conducted because an assessment of toxicity to aquatic algae and cyanobacteria is technically not feasible because of the substance's capacity to complex essential nutrients present in the test media, rendering them unavailable for uptake.

Toxicity to aquatic plants other than algae
A 72-h IC50 value of 1.58 g/L has been determined for the effects of citric acid on germination (root length) of Lepidium

sativum through an aqueous exposure. The publication does not provide sufficient details on the test system and environmental conditions during the test.

A 72 h EC50 equivalent to 0.99 g/L has also been determined for the effects of citric acid on germination of the lettuce Lactuca sativa. The EC50 was determined at a pH of 2.2 and therefore the study cannot be considered reliable for the purposes of REACH. Although the germination study was conducted at an extremely low pH the results do still indicate the low toxicity of the test substance.

Toxicity to microorganisms
In accordance with Column 2 of REACH Annex VIII, the activated sludge respiration inhibition study (required in Section 9.1.4) does not need to be conducted as the substance is readily biodegradable and the applied test concentrations are in the range that can be expected in the influent to a sewage treatment plant.

However for the purpose of setting a PNEC the Bringmann and Kuhn (1980) study with Pseudomonas putida has been selected since no other study on the test substance is available (R.10 guidance).

Sediment toxicity
Testing is not considered necessary because: The substance is of low toxicity to aquatic organisms.

The substance is naturally occurring in sediment organisms.

The substance has a negative Log KOW value and is therefore not expected to be taken up and accumulated by sediment organisms.
Risk characterisation ratios obtained using a PNECsediment calculated from the PNECaquatic by the equilibrium partitioning method are <1.
In accordance with Column 2 of REACH Annex X, the requirement for long-term toxicity data for sediment-dwelling organisms (required in Section 9.5.1.iswaived on the grounds that the chemical safety assessment, conducted according to Annex I, indicates that it is not necessary.

Terrestrial toxicity
Toxicity to soil macroorganisms except arthropods
Limited data are available for the toxicity of citric acid to terrestrial organisms. However, further testing is not considered necessary because:
The substance is of low toxicity to aquatic organisms.

The substance is naturally occurring in terrestrial organisms.

Risk characterisation ratios obtained using PNECsoilcalculated from the PNECaquaticby the equilibrium partitioning method are <1.
The substance has a negative Log Kow value and therefore, partitioning to the terrestrial compartment is expected to be minimal.

Toxicity to birds
Results are only available from a short-term feeding study with Gallus domesticus. A 14-day NOEC value of > 4 g/kg body weight has been determined for the effects of the test substance on mortality. Although they do not relate to a REACH endpoint, the results do provides some indication to the low toxicity of citric acid to birds in dietary exposures.

Persistence and degradability

Stability
Hydrolysis
Citric acid is a readily biodegradable substance, therefore hydrolysis as a function of pH study which is required in REACH guidance, Annex VIII (Section 9.2.2.1) does not need to be conducted.

Biodegradation
Endpoint summary
Ready (OECD 301 B and OECD 301 E) and inherent (OECD 302 B) biodegration tests have been conducted for citric acid. Detailed information on individual test methods was not stated in the report, however, there were agreement among the test results obtained.

A biodegradation test results of 97% (28 days), 100% (19 days) and 85% (14 days) was obtained in a study conducted according toOECD 301 B, OECD 301 E and OECD 302 B respespectively. The results obtained are considred reliable and acceptable for assessment of the biodegradation of citric acid. The test substance is considered readily biodegradable.

Biodegradation in water: screening tests
OECD 301B (CO_2 evolution test): 97% in 28 days, OECD 301E(Modified OECD screening test): 100% in 19 days, OECD 302B (Inherent biodegradability: Zahn-Wellens test): 85% in 14 days
Citric acid is a readily biodegradable substance. Non of the available reliable study reports could be assigned as key study due to lack of sufficient information. However, the results are found consistent and reliable for the purpose of environmental assessment of the substance.

Biodegradation in water and sediment: simulation tests
The simulation test on ultimate degradation in surface water and sediment simulation test study required in REACH guidance, Annex IX (Sections 9.2.1.2 and 9.2.1.4) do not need to be conducted as the substance is readily biodegradable. The chemical safety assessment also indicates that identification of degradation products (required in section 9.2.3) is not necessary.

However, a simulation in water and sediment test value of 93 ± 5 % has been determined for Citric acid in a test conducted according to OECD 303 A. The observed removal could be due to adsorption and/or degradation.

Biodegradation in soil
The biodegradation in soil study required in REACH guidance, Annex IX (Sections 9.2.1.3) does not need to be conducted as the substance is readily biodegradable.

Bioaccumulative potential

Bioaccumulation: aquatic / sediment
Predicted log BCF of 0.5 = 3.2 L/kg wet weight

The bioaccumulation in aquatic species study required in REACH guidance, Annex IX (Section 9.3.2) does not need to be conducted as the substance and its hydrolysis products have low potential for bioaccumulation with a partition coefficient of less than zero (log Kow < 0).

However, an estimated BCF of 3.2 (log BCF = 0.5) was obtained for citric acid using BCFBAF program which forms part of the Syracuse EPIWEB suite. The estimated value of citric acid is well below the cut off value of BCF ≥ 500 that is considered to indicate potential for bioaccumulation; the result indicates that citric acid is not expected to bioaccumulate.

Citrate is found in all eukaryotic cells as an intermediate of the TCA cycle, which is part of the basic metabolic pathway that generates useable energy from carbohydrates, proteins and fats. Citric acid is formed and broken down in the course of this cycle at very high rates.The weight of evidence of the low estimated BCF, biodegradability and role in cell metabolism indicate that citric acid is extremely unlikely to bioaccumulate and testing is not considered necessary for this endpoint.

Mobility in soil

Transport and distribution
Adsorption / desorption
The adsorption/desorption study required in REACH guidance, Annex IX (Section 9.3.3) does not need to be conducted as the substance has low potential for adsorption with a partition coefficient of less than zero (log Kow <0).

However, the Chemical Safety Assessment (CSA) has shown that any adsorption/desorption associated with the Koc value of citric acid is insignificant.

Other adverse effects

No additional data.

13 Disposal considerations

Disposal methods

Waste disposal recommendations:
At the time of review, criteria for land treatment or burial (sanitary landfill) disposal practices are subject to significant revision. Prior to implementing land disposal of waste residue (including waste sludge), consult with environmental

regulatory agencies for guidance on acceptable disposal practices.

Dispose of waste and container in accordance with local and/or national regulations. Hazardous waste shall not be mixed together with other waste. Different types of hazardous waste shall not be mixed together if this may entail a risk of pollution or create problems for the further management of the waste. Hazardous waste shall be managed responsibly. All entities that store, transport or handle hazardous waste shall take the necessary measures to prevent risks of pollution or damage to people or animals. Recycle/reuse. Remove for physico-chemical/biological treatment. **DO NOT** discharge into drains or the environment.

Ecology - waste materials
Avoid release to the environment.

Packaging material/Empty container
Dispose of material in a safe manner as per local and/or national regulations.

14 Transport information

UN Number

Not regulated for transport.

UN Proper Shipping Name

Not regulated for transport.

Transport hazard class(es)

Not regulated for transport.

Packing group, if applicable

Not regulated for transport.

Environmental hazards

No additional data.

Special precautions for user

None except those in sections 4 to 8.

Transport in bulk according to Annex II of MARPOL 73/78 and the IBC Code

Not applicable.

15 Regulatory information

Safety, health and environmental regulations specific for the product in question

SA NATIONAL LEGISLATION
Hazardous Substances Act 15 of 1973 and Regulations.
Occupational Health and Safety Act 85 of 1993 and Regulations.

SA NATIONAL STANDARDS
SANS 10228 : 2006 : Identification and Classification of Dangerous Goods for Transport by Road and Rail.
SANS 10231 : 2018 : Transport of dangerous goods - Operational requirements for road vehicles.
SANS 10234 : 2008 : Globally Harmonized System of classification and labelling of chemicals (GHS).
SANS 10236-0 : 2009 : The warehousing of dangerous goods Part 0: General requirements
SANS 11014 : 2010 : Safety Data Sheets for chemical Products.

REACH Regulation (EC) No 1907/2006
This product contains only components that have been either pre-registered, registered, are exempt from registration, are regarded as registered or are not subject to registration according to Regulation (EC) No. 1907/2006 (REACH)., The aforementioned indications of the REACH registration status are provided in good faith and believed to be accurate as of the effective date shown above. However, no warranty, express or implied, is given. It is the buyer´s/user´s responsibility to ensure that his/her understanding of the regulatory status of this product is correct.

Seveso III: Directive 2012/18/EU
Listed in Regulation: Not applicable

Chemical safety assessment
Performed for this substance: YES

16 Other information

Other information

Full text of H & P - Statements referred to under section 2
Hazard statements
H319

Precautionary statements

P264	Wash thoroughly after handling.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	IF eye irritation persists: Get medical advice/attention.

Labelling REGULATION (EC) No 1272/2008
Signal Word
Warning
Pictograms Hazard to Human
GHS07 Health hazard
Pictogram Hazard during Transport
None

Training advice
Provide adequate information, instruction and training for operators.

Information sources
ECHA European Chemicals Agency

Compiled by Aquatrade Water Treatment Chemicals (Pty) Ltd, R. van Rooyen, SHEQ Co-ordinator and E. Le Sar, Director.

MANUFACTURER/SUPPLIER DISCLAIMER:

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Revision History

Revision	Date	Change
1.0	2019/05/24	Preparation of the safety data sheet according to SANS 11014:2010