

Rust Remover (HiChem) HiChem Paint Technologies Pty Ltd

Chemwatch Hazard Alert Code: 3

Chemwatch: **5206-16** Version No: **3.1.1.1**

Safety Data Sheet according to WHS and ADG requirements

Issue Date: **15/04/2016** Print Date: **12/12/2016** S.GHS.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	Rust Remover (HiChem)
Synonyms	Not Available
Proper shipping name	PHOSPHORIC ACID, SOLUTION
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	HiChem Paint Technologies Pty Ltd
Address	73 Hallam South Road Hallam VIC 3803 Australia
Telephone	+61 3 9796 3400
Fax	+61 3 9796 4500
Website	www.hichem.com.au
Email	info@hichem.com.au

Emergency telephone number

Association / Organisation	HiChem Paint Technologies
Emergency telephone numbers	In Australia: HiChem: +61 3 9796 3400
Other emergency telephone numbers	+800 2436 225

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
1800 039 008	1800 039 008	+612 9186 1132

Once connected and if the message is not in your prefered language then please dial 01

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS CHEMICAL. DANGEROUS GOODS. According to the WHS Regulations and the ADG Code.

CHEMWATCH HAZARD RATINGS

	Min	Max	
Flammability	0		
Toxicity	1		0 = Minimum
Body Contact	3		1 = Low 2 = Moderate
Reactivity	1		3 = High
Chronic	0	1	4 = Extreme

Poisons Schedule	S5
Classification ^[1]	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Chronic Aquatic Hazard Category 4
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

GHS label elements			
SIGNAL WORD	DANGER		
Hazard statement(s)			
H290	May be corrosive to metals.		
H314	Causes severe skin burns and eye damage.		
H318	Causes serious eye damage.		
H413	May cause long lasting harmful effects to aquatic life.		
Precautionary statement(s)	Precautionary statement(s) Prevention		
P260	Do not breathe dust/fume/gas/mist/vapours/spray.		
P280	Wear protective gloves/protective clothing/eye protection/face protection.		
P234	Keep only in original container.		
P273	Avoid release to the environment.		

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P310	Immediately call a POISON CENTER or doctor/physician.
P363	Wash contaminated clothing before reuse.
P390	Absorb spillage to prevent material damage.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P405 Store locked up.

Precautionary statement(s) Disposal

P501 Dispose of contents/container in accordance with local regulations.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
7664-38-2.	10-20	orthophosphoric acid
111-76-2	0-5	ethylene glycol monobutyl ether
9004-65-3	0-2	hydroxypropyl methylcellulose
	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: Immediately hold eyelids apart and flush the eye continuously with running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. Transport to hospital or doctor without delay. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	 If skin or hair contact occurs: Immediately flush body and clothes with large amounts of water, using safety shower if available. Quickly remove all contaminated clothing, including footwear. Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. Transport to hospital, or doctor.
Inhalation	 If furnes or combustion products are inhaled remove from contaminated area. Lay patient down. Keep warm and rested. Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. Transport to hospital, or doctor.

Inhalation of vapours or aerosols (mists, fumes) may cause lung oedema. Corrosive substances may cause lung damage (e.g. lung oedema, fluid in the lungs). + As this reaction may be delayed up to 24 hours after exposure, affected individuals need complete rest (preferably in semi-recumbent posture) and must be kept under medical observation even if no symptoms are (vet) manifested. • Before any such manifestation, the administration of a spray containing a dexamethasone derivative or becomethasone derivative may be considered. This must definitely be left to a doctor or person authorised by him/her. (ICSC13719) For advice, contact a Poisons Information Centre or a doctor at once. Urgent hospital treatment is likely to be needed. If swallowed do NOT induce vomiting F If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Ingestion Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

for phosphate salts intoxication:

- All treatments should be based on observed signs and symptoms of distress in the patient. Consideration should be given to the possibility that overexposure to materials other than this product may have occurred.
- Ingestion of large quantities of phosphate salts (over 1.0 grams for an adult) may cause an osmotic catharsis resulting in diarrhoea and probable abdominal cramps. Larger doses such as 4-8 grams will almost certainly cause these effects in everyone. In healthy individuals most of the ingested salt will be excreted in the faeces with the diarrhoea and, thus, not cause any systemic toxicity. Doses greater than 10 grams hypothetically may cause systemic toxicity.
- Treatment should take into consideration both anionic and cation portion of the molecule.
- · All phosphate salts, except calcium salts, have a hypothetical risk of hypocalcaemia, so calcium levels should be monitored.

Treat symptomatically.

For acute or short term repeated exposures to strong acids:

- Airway problems may arise from laryngeal edema and inhalation exposure. Treat with 100% oxygen initially.
- Respiratory distress may require cricothyroidotomy if endotracheal intubation is contraindicated by excessive swelling
- Intravenous lines should be established immediately in all cases where there is evidence of circulatory compromise.
- + Strong acids produce a coagulation necrosis characterised by formation of a coagulum (eschar) as a result of the dessicating action of the acid on proteins in specific tissues.

INGESTION:

- Immediate dilution (milk or water) within 30 minutes post ingestion is recommended.
- > DO NOT attempt to neutralise the acid since exothermic reaction may extend the corrosive injury.
- Be careful to avoid further vomit since re-exposure of the mucosa to the acid is harmful. Limit fluids to one or two glasses in an adult.
- Charcoal has no place in acid management.
- Some authors suggest the use of lavage within 1 hour of ingestion.

SKIN:

▶ Skin lesions require copious saline irrigation. Treat chemical burns as thermal burns with non-adherent gauze and wrapping.

Deep second-degree burns may benefit from topical silver sulfadiazine

EYE:

- Eye injuries require retraction of the eyelids to ensure thorough irrigation of the conjuctival cul-de-sacs. Irrigation should last at least 20-30 minutes. DO NOT use neutralising agents or any other additives. Several litres of saline are required.
- Cycloplegic drops, (1% cyclopentolate for short-term use or 5% homatropine for longer term use) antibiotic drops, vasoconstrictive agents or artificial tears may be indicated dependent on the severity of the injury.
- Steroid eye drops should only be administered with the approval of a consulting ophthalmologist).

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ Water spray or fog.
- Foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.

Special hazards arising from the substrate or mixture

Fire Incompatibility None known

Advice for firefighters		
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Use fire fighting procedures suitable for surrounding area. Do not approach containers suspected to be hot. Cool fire exposed containers with water spray from a protected location. If safe to do so, remove containers from path of fire. Equipment should be thoroughly decontaminated after use. 	
Fire/Explosion Hazard	 Non combustible. Not considered to be a significant fire risk. Acids may react with metals to produce hydrogen, a highly flammable and explosive gas. Heating may cause expansion or decomposition leading to violent rupture of containers. May emit corrosive, poisonous fumes. May emit acrid smoke. Decomposition may produce toxic fumes of: phosphorus oxides (POx) 	
HAZCHEM	2R	

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	 Environmental hazard - contain spillage. Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material. Check regularly for spills and leaks. Clean up all spills immediately. Avoid breathing vapours and contact with skin and eyes. Control personal contact with the substance, by using protective equipment. Contain and absorb spill with sand, earth, inert material or vermiculite. Wipe up. Place in a suitable, labelled container for waste disposal.
Major Spills	 Environmental hazard - contain spillage. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear full body protective clothing with breathing apparatus. Prevent, by any means available, spillage from entering drains or water course. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Neutralise/decontaminate residue (see Section 13 for specific agent). Collect solid residues and sea and prevent runoff into drains. After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. If contamination of drains or waterways occurs, advise emergency services.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Precautions for safe hand	ing
Safe handling	 DO NOT allow clothing wet with material to stay in contact with skin Avoid all personal contact, including inhalation. Wear protective clothing when risk of exposure occurs. Use in a well-ventilated area. Avoid contact with moisture. Avoid contact with incompatible materials. When handling, DO NOT eat, drink or smoke. Keep containers securely sealed when not in use. Avoid physical damage to containers. Always wash hands with scap and water after handling. Work clothes should be laundered separately. Launder contaminated clothing before re-use. Use good occupational work practice. Observe manufacturer's storage and handling recommendations contained within this SDS. Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	 Store in original containers. Keep containers securely sealed. Store in a cool, dry, well-ventilated area. Store away from incompatible materials and foodstuff containers. Protect containers against physical damage and check regularly for leaks. Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	 DO NOT use aluminium or galvanised containers Check regularly for spills and leaks Lined metal can, lined metal pail/ can. Plastic pail. Polyliner drum. Packing as recommended by manufacturer. Check all containers are clearly labelled and free from leaks. For low viscosity materials Drums and jerricans must be of the non-removable head type. Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): Removable head packaging; Cans with friction closures and low pressure tubes and cartridges may be used. Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	 Phosphoric acid: is a medium-strong acid which produces violent reaction with bases may produce violent react when water is added to the concentrated form reacts violently with solutions containing ammonia or bleach, azo compounds, epoxides and other polymerisable compounds reacts, possibly violently with amines, aldehydes, alkanolamines, alcohols, alkylene oxides, amides, ammonia, ammonia hydroxide, calcium oxide, cyanides,

 epichlorohydrin, esters, halogenated organics, isocyanates, ketones, oleum, organic anhydrides, sodium tetraborate, sulfides, sulfuric acid, strong oxidisers,
vinyl acetate
forms explosive mixtures with nitromethane
 at elevated temperatures attacks many metals producing hydrogen gas
at room temperature does not attack stainless steel, copper or its alloys
 attacks glass, ceramics, and some plastics, rubber and coatings
Inorganic acids are generally soluble in water with the release of hydrogen ions. The resulting solutions have pH's of less than 7.0.
Inorganic acids neutralise chemical bases (for example: amines and inorganic hydroxides) to form salts - neutralisation can generate dangerously large amounts of heat in small spaces.
The dissolution of inorganic acids in water or the dilution of their concentrated solutions with additional water may generate significant heat.
The addition of water to inorganic acids often generates sufficient heat in the small region of mixing to cause some of the water to boil explosively. The resulting "bumping" can spatter the acid.
Inorganic acids react with active metals, including such structural metals as aluminum and iron, to release hydrogen, a flammable gas.
Inorganic acids can initiate the polymerisation of certain classes of organic compounds.
Inorganic acids react with cyanide compounds to release gaseous hydrogen cyanide.
Inorganic acids generate flammable and/or toxic gases in contact with dithiocarbamates, isocyanates, mercaptans, nitrides, nitriles, sulfides, and strong reducing agents. Additional gas-generating reactions occur with sulfites, nitrites, thiosulfates (to give H2S and SO3), dithionites (SO2), and even carbonates.
 Acids often catalyse (increase the rate of) chemical reactions.
▶ Reacts vigorously with alkalis
Reacts with mild steel, galvanised steel / zinc producing hydrogen gas which may form an explosive mixture with air.
Phosphates are incompatible with oxidising and reducing agents.
Phosphates are susceptible to formation of highly toxic and flammable phosphine gas in the presence of strong reducing agents such as hydrides.
Partial oxidation of phosphates by oxidizing agents may result in the release of toxic phosphorus oxides.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	orthophosphoric acid	Phosphoric acid	1 mg/m3	3 mg/m3	Not Available	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m3 / 20 ppm	242 mg/m3 / 50 ppm	Not Available	Sk

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1		TEEL-2	TEEL-3
orthophosphoric acid	Phosphoric acid	Not Avai	lable	Not Available	Not Available
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm		120 ppm	700 ppm
Ingredient	Original IDLH		Revised IDLH		
orthophosphoric acid	10,000 mg/m3		1,000 mg/m3		
ethylene glycol monobutyl ether	700 ppm		700 [Unch] ppm		
hydroxypropyl methylcellulose	Not Available		Not Available		

Exposure controls

	Engineering controls are used to remove a hazard or place a barrier between the worker and the ha effective in protecting workers and will typically be independent of worker interactions to provide this I. The basic types of engineering controls are: Process controls which involve changing the way a job activity or process is done to reduce the risk. Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure. Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. C	high level of protection. the worker and ventilation that stra d properly. The design of a ventilation	tegically "adds" and on system must match		
	Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ens				
	An approved self contained breathing apparatus (SCBA) may be required in some situations.				
	Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the	workplace possess varying "esca	pe" velocities which, in		
	turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contra	aminant.			
	Type of Contaminant:		Air Speed:		
Appropriate engineering controls	solvent, vapours, degreasing etc., evaporating from tank (in still air).				
	aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)				
	direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)				
	grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).				
	Within each range the appropriate value depends on:				
	Lower end of the range Upper end of the range				
	1: Room air currents minimal or favourable to capture 1: Disturbing room air currents				
	2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity			

	3: Intermittent, low production.	3: High production, heavy use		
	4: Large hood or large air mass in motion 4: Small hood-local co			
	Simple theory shows that air velocity falls rapidly with distance away from the open of distance from the extraction point (in simple cases). Therefore the air speed at the distance from the contaminating source. The air velocity at the extraction fan, for explored in a tank 2 meters distant from the extraction point. Other mediapparatus, make it essential that theoretical air velocities are multiplied by factors of the test of test of test of the test of te	he extraction point should be adjusted, accordingly, after reference to kample, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of hanical considerations, producing performance deficits within the extraction		
Personal protection				
Eye and face protection	 Chemical goggles. Full face shield may be required for supplementary but never for primary prote Contact lenses may pose a special hazard; soft contact lenses may absorb at lenses or restrictions on use, should be created for each workplace or trask. T chemicals in use and an account of injury experience. Medical and first-aid per readily available. In the event of chemical exposure, begin eye irrigation immer at the first signs of eye redness or irritation - lens should be removed in a clear Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent] 	nd concentrate irritants. A written policy document, describing the wearing of his should include a review of lens absorption and adsorption for the class of ersonnel should be trained in their removal and suitable equipment should be diately and remove contact lens as soon as practicable. Lens should be removed and remove contact lens as soon as practicable.		
Skin protection	See Hand protection below			
Hands/feet protection	minutes according to EN 374, AS/NZS 2161.10.1 or national equivalen When only brief contact is expected, a glove with a protection cla EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. Some glove polymer types are less affected by movement and th Contaminated gloves should be replaced. For general applications, gloves with a thickness typically greater than 0.35 mm, a It should be emphasised that glove thickness is not necessarily a good predictor o glove will be dependent on the exact composition of the glove material. Therefore, requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type always be taken into account to ensure selection of the most appropriate glove for t Note: Depending on the activity being conducted, gloves of varying thickness may Thinner gloves (down to 0.1 mm or less) may be required where likely to give short duration protection and would normally be just for sir	further marks of quality which vary from manufacturer to manufacturer. Where aterial can not be calculated in advance and has therefore to be checked prior turer of the protective gloves and has to be observed when making a final in on clean hands. After using gloves, hands should be washed and dried the selection of gloves include: 25 2161.1 or national equivalent). we with a protection class of 5 or higher (breakthrough time greater than 240 t) is recommended. ass of 3 or higher (breakthrough time greater than 60 minutes according to is should be taken into account when considering gloves for long-term use. are recommended. of glove resistance to a specific chemical, as the permeation efficiency of the glove selection should also be based on consideration of the task e and the glove model. Therefore, the manufacturers' technical data should the task. be required for specific tasks. For example: a high degree of manual dexterity is needed. However, these gloves are only rgle use applications, then disposed of. the is a mechanical (as well as a chemical) risk i.e. where there is abrasion or		
Body protection	See Other protection below			
	Overalls.			
Other protection	 PVC Apron. PVC protective suit may be required if exposure severe. Eyewash unit. Ensure there is ready access to a safety shower. 			

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index". The effect(s) of the following substance(s) are taken into account in the *computer-generated* selection:

Rust Remover (HiChem)

Material	CPI
BUTYL	С
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NEOPRENE	С
NITRILE	С

Respiratory protection

Type A Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1	-	A-PAPR-AUS / Class 1
up to 25 x ES	Air-line*	A-2	A-PAPR-2
up to 50 x ES	-	A-3	-

PE/EVAL/PE	С
PVA	С
PVC	С
SARANEX-23	С
VITON	С

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Acidic liquid; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	32.4

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7	
Chemical stability	Contact with alkaline material liberates heat	
Possibility of hazardous reactions	See section 7	
Conditions to avoid	See section 7	
Incompatible materials	See section 7	
Hazardous decomposition products	See section 5	

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Corrosive acids can cause irritation of the respiratory tract, with coughing, choking and mucous membrane damage. There may be dizziness, headache, nausea and weakness.
Ingestion	The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. Accidental ingestion of the material may be damaging to the health of the individual. Ingestion of acidic corrosives may produce burns around and in the mouth, the throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident.
Skin Contact	The material can produce chemical burns following direct contact with the skin. Open cuts, abraded or irritated skin should not be exposed to this material Skin contact with acidic corrosives may result in pain and burns; these may be deep with distinct edges and may heal slowly with the formation of scar tissue. Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

Air-line** 50+ x ES ^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. If applied to the eyes, this material causes severe eye damage. Direct eye contact with acid corrosives may produce pain, tears, sensitivity to light and burns. Mild burns of the epithelia generally recover rapidly and completely.		
Chronic	Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure. Repeated or prolonged exposure to acids may result in the erosion of teeth, swelling and/or ulceration of mouth lining. Irritation of airways to lung, with cough, and inflammation of lung tissue often occurs.		
Rust Remover (HiChem)	TOXICITY Not Available	IRRITATION Not Available	
	ΤΟΧΙΟΙΤΥ	IRRITATION	
orthonhacnharia aaid	Dermal (rabbit) LD50: >1260 mg/kg ^[2]	Not Available	
orthophosphoric acid	Inhalation (rat) LC50: 0.0255 mg/L/4hr ^[2]		
	Oral (rat) LD50: 1.7 ml ^[1]		
	ΤΟΧΙΟΙΤΥ	IRRITATION	
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE	
ethylene glycol monobutyl ether	Inhalation (rat) LC50: 450 ppm/4hr ^[2]	Eye (rabbit): 100 mg/24h-moderate	
•			
	Oral (rat) LD50: 250 mg/kg ^[2]	Skin (rabbit): 500 mg, open; mild	
hydroxypropyl	TOXICITY	IRRITATION	
methylcellulose	Oral (rat) LD50: >10000 mg/kg ^[2]	Not Available	
Legend:	 Value obtained from Europe ECHA Registered Substances - Acute to extracted from RTECS - Register of Toxic Effect of chemical Substance 	xicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data s	
ORTHOPHOSPHORIC ACID	No significant acute toxicological data identified in literature search. for acid mists, aerosols, vapours Data from assays for genotoxic activity in vitro suggest that eukaryotic cells are susceptible to genetic damage when the pH falls to about 6.5. Cells from the respiratory tract have not been examined in this respect. Mucous secretion may protect the cells of the airways from direct exposure to inhaled acidic mists, just as mucous plays an important role in protecting the gastric epithelium from its auto-secreted hydrochloric acid. In considering whether pH itself induces genotoxic events in vivo in the respiratory system, comparison should be made with the human stomach, in which gastric juice may be at pH 1-2 under fasting or nocturnal conditions, and with the human urinary bladder, in which the pH of urine can range from <5 to > 7 and normally averages 6.2. Furthermore, exposures to low pH in vivo differ from exposures <i>in vitro</i> in that, <i>in vivo</i> , only a portion of the cell surface is subjected to the adverse conditions, so that perturbation of intracellular homeostasis may be maintained more readily than in vitro. Asthma-like symptoms may continue for months or even years after exposure to he material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characteris		
ETHYLENE GLYCOL MONOBUTYL ETHER	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are ostesmistiser in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in		

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGPE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of enythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects

in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma. 1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glycal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glycxylate; glycxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12-24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia. Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria , and uttimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

	Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol. NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS		
HYDROXYPROPYL METHYLCELLULOSE	Intraperitoneal (rat) 5200 mg/kg		
ORTHOPHOSPHORIC ACID & ETHYLENE GLYCOL MONOBUTYL ETHER	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.		
Acute Toxicity	\otimes	Carcinogenicity	\odot
Skin Irritation/Corrosion	×	Reproductivity	\otimes
Serious Eye Damage/Irritation	*	STOT - Single Exposure	\otimes
Respiratory or Skin sensitisation	\otimes	STOT - Repeated Exposure	\otimes
Mutagenicity	\odot	Aspiration Hazard	\otimes
			 Data available but does not fill the criteria for classification Data required to make classification available

🚫 - Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Legend:

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
orthophosphoric acid	LC50	96	Fish	75.1mg/L	2
orthophosphoric acid	EC50	48	Crustacea	>376mg/L	2
orthophosphoric acid	EC50	72	Algae or other aquatic plants	77.9mg/L	2
orthophosphoric acid	EC50	24	Crustacea	>376mg/L	2
orthophosphoric acid	NOEC	72	Algae or other aquatic plants	<7.5mg/L	2
ethylene glycol monobutyl ether	LC50	96	Fish	222.042mg/L	3
ethylene glycol monobutyl ether	EC50	48	Crustacea	>1000mg/L	4
ethylene glycol monobutyl ether	EC50	96	Algae or other aquatic plants	1081.644mg/L	3
ethylene glycol monobutyl ether	EC50	384	Crustacea	51.539mg/L	3
ethylene glycol monobutyl ether	NOEC	96	Crustacea	1000mg/L	4
	Extracted from 1. IU	CLID Toxicity Data 2. Europe ECH	A Registered Substances - Ecotoxicological I	Information - Aquatic Toxicity 3.	EPIWIN Suite V3.12

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 -Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) -Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

May cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites. Prevent, by any means available, spillage from entering drains or water courses.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
orthophosphoric acid	HIGH	HIGH
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)

Bioaccumulative potential

Ingredient	Bioaccumulation	
orthophosphoric acid	LOW (LogKOW = -0.7699)	
ethylene glycol monobutyl ether	LOW (BCF = 2.51)	

Mobility in soil

Ingredient	Mobility
orthophosphoric acid	HIGH (KOC = 1)
ethylene glycol monobutyl ether	HIGH (KOC = 1)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.
	A Hierarchy of Controls seems to be common - the user should investigate:
	► Reduction
	▶ Reuse
	▶ Recycling
	Disposal (if all else fails)
	This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be
	possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type.
	Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.
Product / Packaging	DO NOT allow wash water from cleaning or process equipment to enter drains.
disposal	It may be necessary to collect all wash water for treatment before disposal.
	In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.
	Where in doubt contact the responsible authority.
	Recycle wherever possible.
	Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility
	can be identified.
	Treat and neutralise at an approved treatment plant. Treatment should involve: Neutralisation with soda-ash or soda-lime followed by: burial in a land-fill
	specifically licenced to accept chemical and / or pharmaceutical wastes or Incineration in a licenced apparatus (after admixture with suitable combustible
	material).
	Decontaminate empty containers with 5% aqueous sodium hydroxide or soda ash, followed by water. Observe all label safeguards until containers are allocated and destrained.
	cleaned and destroyed.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	A CONTRACTOR OF A CONTRACTOR O	
Marine Pollutant	NO	
HAZCHEM	2R	
Land transport (ADG)		
UN number	1805	
UN proper shipping name	PHOSPHORIC ACID, SOLUTION	
Class 8 Subrisk Not Applicable		
Packing group	g group III	
Environmental hazard	d Not Applicable	
Special precautions for user	Special provisions 223 Limited quantity 5 L	

Air transport (ICAO-IATA / DGR)

UN number	1805	
UN proper shipping name	Phosphoric acid, solution	
Transport hazard class(es)	ICAO/IATA Class 8	
	ICAO / IATA Subrisk Not Applicable	
	ERG Code 8L	
Packing group	111	
Environmental hazard	Not Applicable	
Special precautions for user	Special provisions	A3A803
	Cargo Only Packing Instructions	856
		i
	Cargo Only Maximum Qty / Pack	60 L
	Passenger and Cargo Packing Instructions	852
	Passenger and Cargo Maximum Qty / Pack	5 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y841
	Passenger and Cargo Limited Maximum Qty / Pack	1L

Sea transport (IMDG-Code / GGVSee)

UN number

1805

UN proper shipping name	PHOSPHORIC ACID SOLUTION
Transport hazard class(es)	IMDG Class 8 IMDG Subrisk Not Applicable
Packing group	Ш
Environmental hazard	Not Applicable
Special precautions for user	EMS NumberF-A, S-BSpecial provisions223Limited Quantities5 L

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

SECTION 15 REGULATORY INFORMATION

Safety, health and environmental regulations / legislation specific for the substance or mixture

ORTHOPHOSPHORIC ACID(7664-38-2.) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS) International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

Australia Inventory of Chemical Substances (AICS)

HYDROXYPROPYL METHYLCELLULOSE(9004-65-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status	
Australia - AICS	Υ	
Canada - DSL	Υ	
Canada - NDSL	N (orthophosphoric acid; hydroxypropyl methylcellulose; ethylene glycol monobutyl ether)	
China - IECSC	Y	
Europe - EINEC / ELINCS / NLP	N (hydroxypropyl methylcellulose)	
Japan - ENCS	Y	
Korea - KECI	Y	
New Zealand - NZIoC	Y	
Philippines - PICCS	Y	
USA - TSCA	Y	
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)	

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
hydroxypropyl methylcellulose	9004-65-3, 8063-82-9

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC-TWA: Permissible Concentration-Time Weighted Average

PC-STEL: Permissible Concentration-Short Term Exposure Limit

IARC: International Agency for Research on Cancer

ACGIH: American Conference of Governmental Industrial Hygienists

STEL: Short Term Exposure Limit

TEEL: Temporary Emergency Exposure Limit $_{\circ}$ IDLH: Immediately Dangerous to Life or Health Concentrations

OSF: Odour Safety Factor

NOAEL :No Observed Adverse Effect Level

LOAEL: Lowest Observed Adverse Effect Level

TLV: Threshold Limit Value LOD: Limit Of Detection OTV: Odour Threshold Value BCF: BioConcentration Factors BEI: Biological Exposure Index

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