Auto Klene Solutions

Chemwatch: **5191-29**Version No: **5.1.1.1**

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 3

Issue Date: **01/11/2019**Print Date: **04/05/2020**L.GHS.AUS.EN

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

Product Identifier

Product name	Auto Klene FERRIS VEET			
Synonyms	Not Available			
Proper shipping name	ORROSIVE LIQUID, N.O.S. (contains potassium hydroxide)			
Other means of identification	Not Available			

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

Details of the supplier of the safety data sheet

Registered company name	Auto Klene Solutions			
Address	/83 Merrindale Drive Croydon VIC 3136 Australia			
Telephone	3 8761 1900			
Fax	61 3 8761 1955			
Website	http://www.autoklene.com/msds/			
Email	Not Available			

Emergency telephone number

Association / Organisation	Auto Klene Solutions		
Emergency telephone numbers	126 (Poisons Information Centre)		
Other emergency telephone numbers	0800 764 766 (New Zealand Poisons Information Centre)		

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	S6			
Classification [1]	Metal Corrosion Category 1, Skin Corrosion/Irritation Category 1B, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3			
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI			

Label elements

Hazard pictogram(s)





SIGNAL WORD DANGER

Hazard statement(s)

H290	May be corrosive to metals.	
H314	Causes severe skin burns and eye damage.	

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H317	May cause an allergic skin reaction.				
H412	Harmful to aquatic life with long lasting effects.				
Precautionary statement(s) Prevention					
P260	Do not breathe mist/vapours/spray.				
P280	Wear protective gloves/protective clothing/eye protection/face protection				

P280	Wear protective gloves/protective clothing/eye protection/face protection.			
P234	pep only in original container.			
P273	void release to the environment.			
P272	Contaminated work clothing should not be allowed out of the workplace.			

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.					
P321	Specific treatment (see advice on this label).					
P363	Wash contaminated clothing before reuse.					
P302+P352	IF ON SKIN: Wash with plenty of water.					
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.					
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 to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name			
61789-40-0	<10	cocamidopropylbetaine			
7758-29-4	<10	sodium tripolyphosphate			
1310-58-3	<10	potassium hydroxide			
111-76-2	<5	ethylene glycol monobutyl ether			
Not Available	>60	Ingredients determined not to be hazardous			

SECTION 4 FIRST AID MEASURES

Description of first aid measures

If this product comes in contact with the eyes: ▶ Immediately hold eyelids apart and flush the eye continuously with running water.

Eye Contact

- 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.

If skin or hair contact occurs:

Skin Contact

- 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.

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	► Transport to hospital, or doctor.
Inhalation	 If fumes 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 (yet) manifested. Before any such manifestation, the administration of a spray containing a dexamethasone derivative or beclomethasone derivative may be considered. This must definitely be left to a doctor or person authorised by him/her. (ICSC13719)
Ingestion	 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. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. 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

Treat symptomatically.

For acute or short term repeated exposures to ethylene glycol:

- Early treatment of ingestion is important. Ensure emesis is satisfactory.
- ▶ Test and correct for metabolic acidosis and hypocalcaemia.
- Apply sustained diuresis when possible with hypertonic mannitol.
- Evaluate renal status and begin haemodialysis if indicated. [I.L.O]
- Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- ► Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- ▶ Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days
- Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: Occupational & Environmental Medicine 1996; 53, 595-600

for corrosives:

BASIC TREATMENT

Establish a patent airway with suction where necessary.

- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- ► Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for pulmonary oedema.
- ► Monitor and treat, where necessary, for shock.
- Anticipate seizures.
- ▶ Where eyes have been exposed, flush immediately with water and continue to irrigate with normal saline during transport to hospital.
- ▶ DO NOT use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- ▶ Skin burns should be covered with dry, sterile bandages, following decontamination.
- ▶ DO NOT attempt neutralisation as exothermic reaction may occur.

ADVANCED TREATMENT

- ▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- ▶ Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- ▶ Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- ▶ Drug therapy should be considered for pulmonary oedema.
- ▶ Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- ► Treat seizures with diazepam.

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▶ Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime.

- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- ▶ Consider endoscopy to evaluate oral injury.
- Consult a toxicologist as necessary.

BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

- ▶ foam
- ▶ dry chemical powder.
- carbon dioxide.

Special hazards arising from the substrate or mixture

opecial nazards arising from the substrate or mixture							
Fire Incompatibility	None known.						
dvice 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	The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO2) nitrogen oxides (NOx) other pyrolysis products typical of burning organic material. May emit corrosive fumes.						
HAZCHEM	2X						

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

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.

Minor Spills

- · 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.

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- 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.
- ► Consider evacuation (or protect in place).

Environmental hazard - contain spillage.

- Major Spills

 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 seal in labelled drums for disposal.
 - Wash area 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

▶ 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.
- Safe handling

 Keep containers securely sealed when not in use.
 - Avoid physical damage to containers.
 - Always wash hands with soap 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

► 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.

Suitable container 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

Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

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Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	potassium hydroxide	Potassium hydroxide	Not Available	Not Available	2 mg/m3	Not Available
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
sodium tripolyphosphate	Sodium tripolyphosphate	0.61 mg/m3	6.8 mg/m3	620 mg/m3
potassium hydroxide	Potassium hydroxide	0.18 mg/m3	2 mg/m3	54 mg/m3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm

Ingredient	Original IDLH	Revised IDLH
cocamidopropylbetaine	Not Available	Not Available
sodium tripolyphosphate	Not Available	Not Available
potassium hydroxide	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
cocamidopropylbetaine	Е	≤ 0.1 ppm
sodium tripolyphosphate	Е	≤ 0.01 mg/m³
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

MATERIAL DATA

for potassium hydroxide:

The TLV-TWA is protective against respiratory tract irritation produced at higher concentrations

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL)

Exposure controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

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 the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

Appropriate engineering controls

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
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)	0.5-1 m/s (100-200 f/min.)

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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)

1-2.5 m/s
(200-500 f/min.)

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 control only

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection









Eye and face protection

► Chemical goggles.

- Full face shield may be required for supplementary but never for primary protection of eyes.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

Hands/feet protection

See Hand protection below

- Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber
- When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact
- chemical resistance of glove material,
- glove thickness and
- dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).

- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
- Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
- Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:

- Excellent when breakthrough time > 480 min
- Good when breakthrough time > 20 min
- Fair when breakthrough time < 20 min
- Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection

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should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. **Body protection** See Other protection below ▶ Overalls. PVC Apron. Other protection ▶ 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:

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Material	СРІ
BUTYL	A
NEOPRENE	В
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NATURAL+NEOPRENE	С
NITRILE	С
NITRILE+PVC	С
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.

Respiratory protection

Type AK-P 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 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - 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)

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Liquid with a characteristic odour; mixes with water.		
Physical state Liquid Relative density (Water = 1) Not Available			Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable

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	1		
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	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	 Unstable in the presence of incompatible materials. Product is considered stable. Hazardous polymerisation will not occur.
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	Not normally a hazard due to non-volatile nature of product Inhalation of alkaline corrosives may produce irritation of the respiratory tract with coughing, choking, pain and mucous membrane damage. Pulmonary oedema may develop in more severe cases; this may be immediate or in most cases following a latent period of 5-72 hours. Symptoms may include a tightness in the chest, dyspnoea, frothy sputum, cyanosis and dizziness. Findings may include hypotension, a weak and rapid pulse and moist rales.
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.
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 Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds 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.
Еуе	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

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TOXICITY

IRRITATION

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	Not Available	Not Available	
	TOXICITY	IRRITATION	
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]	
cocamidopropylbetaine	Oral (rat) LD50: 2700 mg/kg ^[2]	Eye: primary irritant *	
		Skin: adverse effect observed (irritating) ^[1]	
		Skin: primary irritant *	
	TOXICITY	IRRITATION	
sodium tripolyphosphate	Dermal (rabbit) LD50: >3160 mg/kg ^[2]	Not Available	
	Oral (rat) LD50: >2000 mg/kg ^[1]		
	TOXICITY	IRRITATION	
	Oral (rat) LD50: =214-324 mg/kg ^[2]	Eye (rabbit):1mg/24h rinse-moderate	
potassium hydroxide		Skin (human): 50 mg/24h SEVERE	
		Skin (rabbit): 50 mg/24h SEVERE	
	TOXICITY	IRRITATION	
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE	
	Inhalation (rat) LC50: 449.48655 mg/l/4H ^[2]	Eye (rabbit): 100 mg/24h-moderate	
ethylene glycol monobutyl ether	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]	
Cirici		Skin (rabbit): 500 mg, open; mild	
		Skin: adverse effect observed (irritating) ^[1]	
		Skin: no adverse effect observed (not irritating) ^[1]	
Legend:	Value obtained from Europe ECHA Registered Subst Unless otherwise specified data extracted from RTECS	ances - Acute toxicity 2.* Value obtained from manufacturer's SDS.	

The following information refers to contact allergens as a group and may not be specific to this product.

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

Possible cross-reactions to several fatty acid amidopropyl dimethylamines were observed in patients that were reported to have allergic contact dermatitis to a baby lotion that contained 0.3% oleamidopropyl dimethylamine.

Stearamidopropyl dimethylamine at 2% in hair conditioners was not a contact sensitiser when tested neat or diluted to 30%. However, irritation reactions were observed.

A 10-year retrospective study found that out of 46 patients with confirmed allergic eyelid dermatitis, 10.9% had relevant reactions to oleamidopropyl dimethylamine and 4.3% had relevant reactions to cocamidopropyl dimethylamine. Several cases of allergic contact dermatitis were reported in patients from the Netherlands that had used a particular type of body lotion that contained oleamidopropyl dimethylamine.

COCAMIDOPROPYLBETAINE

In 12 patients tested with their personal cosmetics, containing the fatty acid amidopropyl dimethylamine cocamidopropyl betaine (CAPB), 9 had positive reactions to at least one dilution and 5 had irritant reactions. All except 3 patients, who were not tested, had 2 or 3+ reaction to the 3,3-dimethylaminopropylamine (DMAPA, the reactant used in producing fatty acid amidopropyl dimethylamines) at concentrations as low as 0.05%. The presence of DMAPA was investigated via thin-layer chromatography in the personal cosmetics of 4 of the patients that had positive reactions. DMAPA was measured in the products at 50 - 150 ppm suggesting that the sensitising agent in CAPB-induced allergy is DMAPA, .

The sensitisation potential of a 4% aqueous liquid fabric softener formulation containing 0.5% stearyl/palmitylamidopropyl dimethylamine was investigated using. The test material caused some irritation in most volunteers. After a rest period of 2 weeks, the subjects received challenge patches with the same concentration of test material on both arms. Patch sites were graded 48 and 96 h after patching. Eight subjects reacted at challenge, and 7 of the eight submitted to rechallenge with 4% and 0.4% aqueous formulations. No reactions indicative of sensitisation occurred at rechallenge. The test formulation containing stearyl/palmitylamidopropyl dimethylamine had no significant sensitisation potential.subjects.

Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. For betaines:

Several sources revealing data on skin irritation, skin sensitisation and dermal absorption in humans are available for CAS

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683-10-3, the C12-alkyldimethyl betaine, which is the most frequently occurring betaine because it is one of the components of most of the substances of the alkyldimethyl betaine group, among those also Betaines, C12-14 (even numbered)alkyldimethyl. Therefore, read-across of exposure-related observations in humans from CAS 683-10-3 is justified. Data from several human closed patch tests demonstrate skin irritation in humans ranging from mild to strong under occlusive conditions even with concentrations as low as 1%. In contrast, exposure to 0.1% under open conditions did not induce any positive skin

Information on skin sensitisation in humans is available from a closed human patch test with CAS 683-10-3. After an induction period of 6 days with 0.1% of active ingredient, followed by a treatment-free period of 10 days, the volunteers were challenged under occlusive conditions for 24 hours. No reactions were observed immediately after challenge; during the next 4 days only irritation reactions were observed.

This finding was further supported by industrial medical monitoring data. Workers involved in the production of CAS 683-10-3 are routinely checked every 3 years for signs of skin sensitisation, respiratory irritation, skin irritation and eye irritation. During these examinations no signs of the aforementioned disorders were observed which were related to the test substance. Moreover, a study focusing on dermal uptake of C12-alkyldimethyl betaine) into human skin and the effects of surfactants on skin barrier function demonstrated that only up to 0.4% of the applied dose was absorbed within 30 minutes of exposure, with absorbance being dependent on the concentration applied. Tape stripping of the skin revealed that the administered test substance was primarily located in the outer stratum corneum layer

Test material does not demonstrate mutagenic or clastogenic effects in bacteria or mammalian cells in vitro

Amphoteric surfactants are easily absorbed in the intestine and are excreted partly unchanged via the faeces. Metabolisation to CO2 and short-chained fatty acids also occur. No tendency to accumulation in the organism or storage of betaines in certain organs has been detected. Betaines generally have a low acute toxicity. E.g., LD50 values for cocoamidopropylbetaine (30% solution) by oral administration have been determined to 4,910 mg/kg body weight in rats.

Betaines do not carry any net charge, and, therefore, they can only form hydrophobic bonds with proteins in the skin. This may be the explanation for the low protein denaturation potential of betaines as the ion-binding of other surfactants contributes to denaturation. In combination with anionic surfactants a positive synergistic effect with regard to skin compatibility is often found. Compared to a 20% solution of C12 alkyl sulfate (AS; sodium lauryl sulfate) alone, decreased erythema was observed for the combination of 20% C12 AS and 10% cocoamidopropyl betaine one hour after the removal of patches. The combination of cocoamidopropyl betaine and C12 AS also reduced swelling of the skin, and generally interactions between amphoterics and AS produce less swelling and result in milder skin reactions. Concentrated betaines are expected to be irritant to skin and eyes. Diluted solutions (3-10%) are not irritant to skin, but they are mildly irritant to the eyes (4.5%)

No evidence of delayed contact hypersensitivity was found in quinea pigs after topically administrated solutions of 10% cocoamidopropyl betaine by using the Magnusson-Kligman maximization test. Various instances of contact allergy to cocoamidopropyl betaine have been reported. In all of the reports it was concluded that the observed skin reactions were due to the presence of 3-dimethylaminopropylamine which is an impurity in cocoamidopropyl betaine. This impurity is an intermediate in the synthesis of alkylamidopropyldimethylamines that are intermediates in the synthesis of the corresponding alkylamido betaines.

Cocoamidopropyl betaine was proven to be non-mutagenic to Salmonella typhimurium in the Ames Salmonella/microsome reverse mutation assay. Short-term genotoxicity tests have shown negative results of mutagenicity for lauryl betaine in various strains of Salmonella typhimurium.

* [Van Waters and Rogers] ** [Canada Colors and Chemicals Ltd.] Toxicokinetics, metabolism and distribution. Absorption of the chemical across dermal and gastrointestinal membranes is possible based on the relatively low molecular weight of the chemical (500 Da) and given that it is a surfactant (EC, 2003). Acute toxicity. Acute oral toxicity studies in rats and mice indicated that the LD50 values of the chemical (at 30-35.61% concentration) ranged from 1800 mg/kg bw (male rats) up to 5000 mg/kg bw, with mortalities noted in most studies (CIR, 2010). Of note is an acute oral toxicity study conducted in Sprague-Dawley rats (5/sex) at a single dose of 1800 mg/kg bw (formulation containing 35.61% of the chemical), where no males but all five females died. Overall, the data suggests that mortality occurs following oral administration of the chemical and that it may be an acute oral toxicant. Therefore, based on these data the chemical may be harmful if swallowed. An acute dermal toxicity study in rats was conducted using 2000 mg/kg bw of a 31% formulation of the chemical (CIR, 2010). Irritation was observed, but there were no clinical signs of systemic toxicity or mortalities. The lack of effects in this study suggests that the chemical is likely to be of low acute dermal toxicity. Irritation. The chemical has a quaternary ammonium functional group, which is a structural alert for corrosion Numerous skin irritation studies, conducted with formulations containing 7.5-30% of the chemical, indicated that the chemical has irritant properties. The studies were, in-general, conducted under occlusive conditions, with exposure times of up to 24 hours (7.5-10%). Based on the information available, the chemical is likely to be a skin irritant. Eye irritation studies with the chemical showed that corrosive and necrotic effects occurred at 30% whereas less severe effects were observed at lower concentrations of 2.3-10% The chemical is classified with the risk phrase R36: Irritating to eyes, however, based on studies conducted on the chemical it may be a severe eye irritant. Sensitisation. The chemical has a quaternary ammonium functional group, which is a structural alert for sensitisation (Conflicting results have been obtained with the chemical in animal studies. Positive results were reported in an LLNA study (an EC3 value was not reported). In addition, positive results were obtained in two guinea pig maximisation studies conducted by a single laboratory, the first at 3% induction and 3% challenge, and the second at 0.15% induction and 0.015% challenge. However, there was no sensitisation in a guinea pig maximisation test when the chemical was tested at 6% induction and 1% challenge. In addition, no sensitisation was observed in another test in guinea pigs at 0.75% induction and 0.02% challenge. No evidence of sensitisation was reported in a HRIPT on a formulation containing the chemical at 0.6% concentration (a 10% dilution of a ~6% formulation) with 110 volunteers. In HRIPT studies on formulations containing the chemical, no evidence of sensitisation was reported at concentrations of 1.87% (88 subjects), 0.93% (93 subjects), 0.3% (100 subjects), 1.5-3.0% (141 subjects), 6.0% (210 subjects), 0.018% (27 subjects). However, positive results were observed in provocative studies conducted on formulations containing the chemical (at 0.3-1% concentration), conducted in subjects diagnosed with various forms of contact dermatitis, suggesting that the chemical may cause reactions in sensitive individuals In one study authors note that sensitisation effects of the chemical (and related compounds) are most likely due to the impurities, including DMAPA and amidopropyl dimethylamines, however, they do not exclude the possibility of the causing the sensitisation. The potential for skin sensit

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POTASSIUM HYDROXIDE

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

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

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. 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 not sensitisers 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 humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE in vitro than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

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 EGHE (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 erythrocytes 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

ETHYLENE GLYCOL MONOBUTYL ETHER

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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 glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate: glyoxylate 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 ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near 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

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

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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 *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

COCAMIDOPROPYLBETAINE & POTASSIUM HYDROXIDE

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

SODIUM TRIPOLYPHOSPHATE & POTASSIUM HYDROXIDE

Asthma-like symptoms may continue for months or even years after exposure to the 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 irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an 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 characterised by dyspnea, cough and mucus production.

Acute Toxicity	×	Carcinogenicity	×
Skin Irritation/Corrosion	✓	Reproductivity	×
Serious Eye Damage/Irritation	~	STOT - Single Exposure	×
Respiratory or Skin sensitisation	~	STOT - Repeated Exposure	×
Mutagenicity	×	Aspiration Hazard	×

Legend:

∠ − Data either not available or does not fill the criteria for classification

→ − Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Auto Klene FERRIS VEET	Not Available	Not Available	Not Available	Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	=1mg/L	1
cocamidopropylbetaine	EC50	48	Crustacea	6.4mg/L	2
	EC50	96	Algae or other aquatic plants	0.55mg/L	2
	NOEC	672	Fish	0.16mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
sodium tripolyphosphate	EC50	48	Crustacea	>100mg/L	2
	EC50	96	Algae or other aquatic plants	69.2mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish		4
potassium hydroxide	EC0	48 Crustacea		<1mg/L	2
	NOEC	24	Fish	28mg/L	2
ethylene glycol monobutyl ether	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca.1-800mg/L	2

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	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2
Legend:	3. EPIWIN Su	n 1. IUCLID Toxicity Data 2. Europe ECHA uite V3.12 (QSAR) - Aquatic Toxicity Data (uatic Hazard Assessment Data 6. NITE (Ja _l	Estimated) 4. US EPA, Ecotox database - A	Aquatic Toxicity D	ata 5.

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.

Harmful to aquatic organisms, 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.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)

Mobility in soil

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

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging

disposal

- ▶ Containers may still present a chemical hazard/ danger when empty.
- ▶ Return to supplier for reuse/ recycling if possible.

Otherwise:

- If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill.
- ▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.

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.

- 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.
- Freat and neutralise at an approved treatment plant. Treatment should involve: Neutralisation followed by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or Incineration in a licensed apparatus (after admixture with suitable combustible material)
- ▶ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 TRANSPORT INFORMATION

Labels Required

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Land transport (ADG)

UN number	1760		
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains potassium hydroxide)		
Transport hazard class(es)	Class 8 Subrisk Not Applicable		
Packing group	III		
Environmental hazard	Not Applicable		
Special precautions for user	Special provisions 223 274 Limited quantity 5 L		

Air transport (ICAO-IATA / DGR)

UN number	1760		
UN proper shipping name	Corrosive liquid, n.o.s. *	(contains potassium hydroxide)	
	ICAO/IATA Class	8	
Transport hazard class(es)	ICAO / IATA Subrisk	Not Applicable	
	ERG Code	8L	
Packing group	III		
Environmental hazard	Not Applicable		
	Special provisions		A3 A803
	Cargo Only Packing Instructions		856
	Cargo Only Maximum Qty / Pack		60 L
Special precautions for user	Passenger and Cargo Packing Instructions		852
usei	Passenger and Cargo Maximum Qty / Pack		5 L
	Passenger and Cargo Limited Quantity Packing Instructions		Y841
	Passenger and Cargo Limited Maximum Qty / Pack		1 L

Sea transport (IMDG-Code / GGVSee)

UN number	1760		
UN proper shipping name	CORROSIVE LIQUID, N.O.S. (contains potassium hydroxide)		
Transport hazard class(es)	IMDG Class 8 IMDG Subrisk Not Applicable		
Packing group	III		
Environmental hazard	Not Applicable		
Special precautions for user	EMS Number F-A , S-B Special provisions 223 274 Limited Quantities 5 L		

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

COCAMIDOPROPYLBETAINE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

SODIUM TRIPOLYPHOSPHATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

POTASSIUM HYDROXIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

ETHYLENE GLYCOL MONOBUTYL ETHER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (cocamidopropylbetaine; sodium tripolyphosphate; potassium hydroxide; ethylene glycol monobutyl ether)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	01/11/2019
Initial Date	28/08/2015

SDS Version Summary

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Version	Issue Date	Sections Updated
2.1.1.1	28/08/2015	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Classification, Disposal, Engineering Control, Environmental, Exposure Standard, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire fighting), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (inhaled), First Aid (skin), First Aid (swallowed), Handling Procedure, Ingredients, Instability Condition, Personal Protection (other), Personal Protection (Respirator), Personal Protection (eye), Personal Protection (hands/feet), Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container), Transport, Transport Information, Use

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01/11/2019 One-off system update. NOTE: This may or may not change the GHS classification 5.1.1.1

Other information

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.

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。

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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