



Cutek Proclean

Chemisys Manufacturing Pty Ltd

Chemwatch Hazard Alert Code: 2

Catalogue number: D490 Australia

Version No: 3.4

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 08/12/2016

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L.GHS.AUS.EN

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

Product Identifier

| | |
|-------------------------------|----------------|
| Product name | Cutek Proclean |
| Synonyms | Not Available |
| Other means of identification | Not Available |

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

| | |
|--------------------------|--------------|
| Relevant identified uses | Wood cleaner |
|--------------------------|--------------|

Details of the supplier of the safety data sheet

| | |
|-------------------------|--|
| Registered company name | Chemisys Manufacturing Pty Ltd |
| Address | 72 Chetwynd Street Loganholme Queensland Australia |
| Telephone | 1300 128835 0432955101 |
| Fax | Not Available |
| Website | www.chemisys.com.au |
| Email | admin@chemisys.com.au |

Emergency telephone number

| | |
|-----------------------------------|---------------|
| Association / Organisation | Not Available |
| Emergency telephone numbers | 0438923248 |
| Other emergency telephone numbers | 0432955101 |

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

| | Min | Max |
|--------------|-----|-----|
| Flammability | 0 | |
| Toxicity | 2 | |
| Body Contact | 2 | |
| Reactivity | 0 | |
| Chronic | 2 | |

0 = Minimum
1 = Low
2 = Moderate
3 = High
4 = Extreme

| | |
|------------------|---|
| Poisons Schedule | 6 |
|------------------|---|

Continued...

| | |
|--------------------------------------|--|
| Classification ^[1] | Skin Corrosion/Irritation Category 1A, Serious Eye Damage Category 1 |
| Legend: | 1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI |

Label elements

| | |
|---------------------------|---|
| GHS label elements |  |
|---------------------------|---|

| | |
|--------------------|---------------|
| SIGNAL WORD | DANGER |
|--------------------|---------------|

Hazard statement(s)

| | |
|-------------|--|
| H314 | Causes severe skin burns and eye damage. |
| H318 | Causes serious eye damage. |

Precautionary statement(s) Prevention

| | |
|-------------|--|
| P260 | Do not breathe dust/fume/gas/mist/vapours/spray. |
| P280 | Wear protective gloves/protective clothing/eye protection/face protection. |

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. |
| 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 |
|-----------|-----------|--|
| 6153-56-6 | <10 | <u>oxalic acid</u> |
| 111-76-2 | <10 | <u>ethylene glycol monobutyl ether</u> |
| 112-34-5 | <10 | <u>diethylene glycol monobutyl ether</u> |
| 151-21-3 | <10 | <u>sodium lauryl sulfate</u> |

SECTION 4 FIRST AID MEASURES

Description of first aid measures

| | |
|--------------------|---|
| Eye Contact | <p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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. |
|--------------------|---|

| | |
|---------------------|--|
| | <ul style="list-style-type: none"> ▶ 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 | <p>If skin or hair contact occurs:</p> <ul style="list-style-type: none"> ▶ 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 | <ul style="list-style-type: none"> ▶ 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, without delay. |
| Ingestion | <ul style="list-style-type: none"> ▶ 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

- ▶ Effective therapy against burns from oxalic acid involves replacement of calcium.
- ▶ Intravenous oxalic acid is substantially excreted (88% - 90%) in the urine within 36 hours.

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

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

| | |
|-----------------------------|-------------|
| Fire Incompatibility | None known. |
|-----------------------------|-------------|

Advice for firefighters

| | |
|----------------------|---|
| Fire Fighting | ▶ Alert Fire Brigade and tell them location and nature of hazard. |
|----------------------|---|

| | |
|------------------------------|--|
| | <ul style="list-style-type: none"> ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ 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 | <p>carbon dioxide (CO₂) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p> |
| HAZCHEM | Not Applicable |

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

| | |
|---------------------|--|
| Minor Spills | <ul style="list-style-type: none"> ▶ 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 | <p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ 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 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

| | |
|--------------------------|---|
| Safe handling | <ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ 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 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. ▶ DO NOT allow clothing wet with material to stay in contact with skin |
| Other information | |

Conditions for safe storage, including any incompatibilities

| | |
|--------------------------------|---|
| Suitable container | <ul style="list-style-type: none"> ▶ 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. |
| Storage incompatibility | <p>Oxalic acid (and its dihydrate):</p> <ul style="list-style-type: none"> ▶ react violently with strong oxidisers, bromine, furfuryl alcohol, hydrogen peroxide (90%), phosphorous trichloride, silver powders ▶ reacts explosively with chlorites and hypochlorites ▶ mixture with some silver compounds form explosive salts of silver oxalate ▶ is incompatible with caustics and alkalis, urea, alkaline metals and steel ▶ attacks polyvinyl alcohol and acetal plastics <p>None known</p> |



+ X + O + + +

X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

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 | oxalic acid | Oxalic acid | 1 mg/m3 | 2 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 |
|-----------------------------------|--|-----------|----------|-----------|
| oxalic acid | Oxalic acid, anhydrous; (Ethanedioic acid) | 2 mg/m3 | 20 mg/m3 | 500 mg/m3 |
| ethylene glycol monobutyl ether | Butoxyethanol, 2-; (Glycol ether EB) | 60 ppm | 120 ppm | 700 ppm |
| diethylene glycol monobutyl ether | Butoxyethoxy)ethanol, 2-(2-; (Diethylene glycol monobutyl ether) | 30 ppm | 33 ppm | 200 ppm |
| sodium lauryl sulfate | Sodium lauryl sulfate | 3.9 mg/m3 | 43 mg/m3 | 260 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|-----------------------------------|---------------|------------------|
| oxalic acid | 500 mg/m3 | 500 [Unch] mg/m3 |
| ethylene glycol monobutyl ether | 700 ppm | 700 [Unch] ppm |
| diethylene glycol monobutyl ether | Not Available | Not Available |
| sodium lauryl sulfate | Not Available | Not Available |

MATERIAL DATA

There is only scant data regarding the toxicology of industrial exposure to airborne oxalates. There is no data regarding potential systemic toxicity or bioavailability of inhaled oxalates. The TLV-TWA (corresponding to 0.27 ppm on a molecular basis) is comparable to that of sulfuric acid and phosphoric acid and is thought to provide protection against the risk of eye and skin burns and respiratory tract irritation.

The recommendation for a STEL is added to prevent irritation of skin and mucous membranes.

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)

For diethylene glycol monobutyl ether:

CEL TWA: 15.5 ppm, 100 mg/m³

(CEL = Chemwatch Exposure Limit)

In studies involving the inhalation toxicity of diethylene glycol monobutyl ether, exposure for 6 hours daily at 100 mg/m³ had no effect. This concentration is in the range of the saturated vapour concentration.

Local damage was produced following inhalation of concentrations higher than the saturated vapour concentrations, that is, during inhalation of the aerosol (350 mg/m³). Since the only potential effects of inhalation are restricted to local discomfort (in the aerosol concentration range) the substance is classified in category I for the limitation of exposure peaks.

Teratogenicity studies have not revealed prenatal toxic effects at high oral doses and this ether is classified in pregnancy risk group C.

Exposure controls

| Appropriate engineering controls | <p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.</p> <p>An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. 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.</p> | | | | | | | | | | | |
|---|---|----------------------|------------------------|--|---|---|--|--|----------------------------------|--|---|----------------------------------|
| | <table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> | 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.) | direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) | grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion). | 2.5-10 m/s (500-2000 f/min.) | |
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| | <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>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.</p> | | 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 |
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| 1: Room air currents minimal or favourable to capture | 1: Disturbing room air currents | | | | | | | | | | | |
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| 3: Intermittent, low production. | 3: High production, heavy use | | | | | | | | | | | |
| 4: Large hood or large air mass in motion | 4: Small hood-local control only | | | | | | | | | | | |
| Personal protection |  | | | | | | | | | | | |
| Eye and face protection | <ul style="list-style-type: none"> ▶ Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure. ▶ Chemical goggles whenever there is a danger of the material coming in contact with the eyes; goggles must be properly | | | | | | | | | | | |

| | |
|------------------------------|--|
| | <ul style="list-style-type: none"> ▶ fitted. ▶ Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection. ▶ Alternatively a gas mask may replace splash goggles and face shields. ▶ 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 | See Hand protection below |
| Hands/feet protection | <ul style="list-style-type: none"> ▶ Elbow length PVC gloves ▶ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. <p>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.</p> <p>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.</p> <p>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 moisturizer is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · 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. <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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 should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>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.</p> |
| Body protection | See Other protection below |
| Other protection | <ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C. apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash unit. |
| Thermal hazards | Not Available |

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:

Cutek Proclean

| Material | CPI |
|----------|-----|
|----------|-----|

Respiratory protection

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.

Continued...

| | |
|-------------------|---|
| BUTYL | A |
| NEOPRENE | B |
| NAT+NEOPR+NITRILE | C |
| NATURAL RUBBER | C |
| NATURAL+NEOPRENE | C |
| NEOPRENE/NATURAL | C |
| NITRILE | C |
| NITRILE+PVC | C |
| PE/EVAL/PE | C |
| PVA | C |
| PVC | C |
| SARANEX-23 | C |
| VITON | C |

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

| Required minimum protection factor | Maximum gas/vapour concentration present in air p.p.m. (by volume) | Half-face Respirator | Full-Face Respirator |
|------------------------------------|--|----------------------|----------------------|
| up to 10 | 1000 | A-AUS / Class 1 | - |
| up to 50 | 1000 | - | A-AUS / Class 1 |
| up to 50 | 5000 | Airline * | - |
| up to 100 | 5000 | - | A-2 |
| up to 100 | 10000 | - | A-3 |
| 100+ | | - | Airline** |

* - Continuous Flow

** - Continuous-flow or positive pressure demand.

A(All classes) = Organic vapours, B AUS or B1 = Acid gases, 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 deg C)

* 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 | Clear/white | | |
| Physical state | Liquid | Relative density (Water = 1) | 1.10 |
| Odour | Not Available | Partition coefficient n-octanol / water | Not Available |
| Odour threshold | Not Available | Auto-ignition temperature (°C) | Not Available |
| pH (as supplied) | 2 | 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 Available |
| 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 Available | Surface Tension (dyn/cm or mN/m) | Not Available |
| Lower Explosive Limit (%) | Not Available | 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 | Not Available |

SECTION 10 STABILITY AND REACTIVITY

| | |
|-------------------|---------------|
| Reactivity | See section 7 |
|-------------------|---------------|

| | |
|---|--|
| Chemical stability | <ul style="list-style-type: none"> ▶ 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 | <p>Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Acidic corrosives produce respiratory tract irritation with coughing, choking and mucous membrane damage. Symptoms of exposure may include dizziness, headache, nausea and weakness. In more severe exposures, pulmonary oedema may be evident either immediately or after a latent period of 5-72 hours. Symptoms of pulmonary oedema include a tightness in the chest, dyspnoea, frothy sputum and cyanosis. Examination may reveal hypotension, a weak and rapid pulse and moist rates. Death, due to anoxia, may occur several hours after onset of the pulmonary oedema.</p> <p>Inhalation of oxalic acid dusts or vapours can cause ulceration of the mucous membranes of the nose and throat, epistaxis (nosebleed), headache and nervousness. The airborne dust behaves as a strong acid producing severe local burns of the mucous membranes.</p> |
| Ingestion | <p>Ingestion of acidic corrosives may produce circumoral burns with a distinct discolouration of the mucous membranes of the mouth, throat and oesophagus. Immediate pain and difficulties in swallowing and speaking may also be evident. Oedema of the epiglottis may produce respiratory distress and possibly, asphyxia. Nausea, vomiting, diarrhoea and a pronounced thirst may occur. More severe exposures may produce a vomitus containing fresh or dark blood and large shreds of mucosa. Shock, with marked hypotension, weak and rapid pulse, shallow respiration and clammy skin may be symptomatic of the exposure. Circulatory collapse may, if left untreated, result in renal failure. Severe cases may show gastric and oesophageal perforation with peritonitis, fever and abdominal rigidity. Stricture of the oesophageal, gastric and pyloric sphincter may occur as within several weeks or may be delayed for years. Death may be rapid and often results from asphyxia, circulatory collapse or aspiration of even minute amounts. Delayed deaths may be due to peritonitis, severe nephritis or pneumonia. Coma and convulsions may be terminal.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Oxalic acid is a minor, normal body constituent occurring in blood at approximately 0.150 mg/100 ml and in kidney, muscle and liver at about 0.050 mg/100 ml dry weight, but higher concentrations are toxic.</p> <p>Ingestion of 5 grams has caused death within hours. It is a systemic poison which affects the central nervous system and kidney function. Low doses (i.e. excess in blood) may cause hypocalcemia (presence in the blood of an abnormally low concentration of calcium).</p> <p>Oxalic acid occurs naturally in the common weed Oxalis, 'sour sobs'.</p> |
| Skin Contact | <p>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.</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>Solutions of 5% to 10% oxalic acid are irritating to the skin after prolonged contact; early gangrene may occur after hand immersion in oxalate solutions.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> |
| Eye | <p>When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.</p> <p>Direct eye contact with acid corrosives may produce pain, lachrymation, photophobia and burns. Mild burns of the epithelia generally recover rapidly and completely. Severe burns produce long-lasting and possible irreversible damage. The appearance of the burn may not be apparent for several weeks after the initial contact. The cornea may ultimately become deeply vascularised and opaque resulting in blindness.</p> |
| Chronic | <p>Repeated or prolonged exposure to acids 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.</p> <p>The impact of inhaled acidic agents on the respiratory tract depends upon a number of interrelated factors. These include</p> |

physicochemical characteristics, e.g., gas versus aerosol; particle size (small particles can penetrate deeper into the lung); water solubility (more soluble agents are more likely to be removed in the nose and mouth). Given the general lack of information on the particle size of aerosols involved in occupational exposures to acids, it is difficult to identify their principal deposition site within the respiratory tract. Acid mists containing particles with a diameter of up to a few micrometers will be deposited in both the upper and lower airways. They are irritating to mucous epithelia, they cause dental erosion, and they produce acute effects in the lungs (symptoms and changes in pulmonary function). Asthmatics appear to be at particular risk for pulmonary effects.

Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

On the basis, primarily, of animal experiments, concern has been expressed 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.

| | | |
|-----------------------------------|---|---|
| Cutek Proclean | TOXICITY | IRRITATION |
| | #55rads#551alksulf ^[2] | Not Available |
| oxalic acid | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 2000 mg/kg ^[2] Oral (rat) LD50: 1.08 ml ^[1] | Not Available |
| ethylene glycol monobutyl ether | TOXICITY | IRRITATION |
| | dermal (rat) LD50: >2000 mg/kg ^[1] | Eye (rabbit): 100 mg SEVERE |
| | Inhalation (rat) LC50: 450 ppm/4hr ^[2] Oral (rat) LD50: 250 mg/kg ^[2] | Eye (rabbit): 100 mg/24h-moderate Skin (rabbit): 500 mg, open; mild |
| diethylene glycol monobutyl ether | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 2700 mg/kg ^[2] Oral (rat) LD50: 3306 mg/kg ^[1] | Eye (rabbit): 20 mg/24h moderate Eye (rabbit): 5 mg - SEVERE |
| sodium lauryl sulfate | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >500 mg/kg ^[1] Oral (rat) LD50: 977 mg/kg ^[1] | Eye (rabbit): 100 mg/24 hr-moderate Skin (human): 25 mg/24 hr - mild |
| Legend: | 1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances | |

ETHYLENE GLYCOL MONOBUTYL ETHER

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 sensitizers 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* cytogenetic 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 foetotoxicity 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 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 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 embryotoxicity 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.

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

DIETHYLENE GLYCOL MONOBUTYL ETHER

For diethylene glycol monoalkyl ethers and their acetates:

This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates.

Acute toxicity: There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative.

Repeat dose toxicity: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than

continuous exposure regimens.

Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in *S. typhimurium* strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in *E. coli* WP2uvrA, with and without metabolic activation. *In vitro* cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic.

Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGBE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA.

Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m³) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21. In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus.

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group. Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Acute toxicity: These substances are well absorbed after ingestion; penetration through the skin is however poor. After absorption, these chemicals are distributed mainly to the liver.

Acute oral LD50 values of alkyl sulfates in rats and/or mice were (in mg/kg):

C10-; 290-580

C10-16-, and C12-; 1000-2000

C12-14, C12-15, C12-16, C12-18 and C16-18-; >2000

C14-18, C16-18-; >5000

The clinical signs observed were non-specific (piloerection, lethargy, decreased motor activity and respiratory rate, diarrhoea). At necropsy the major findings were irritation of the gastrointestinal tract and anemia of inner organs.

Based on limited data, the acute oral LD50 values of alkane sulfonates and alpha-olefin sulfonates of comparable chain lengths are assumed to be in the same range.

The counter ion does not appear to influence the toxicity in a substantial way.

Acute dermal LD50 values of alkyl sulfates in rabbits (mg/ kg):

C12-; 200

C12-13 and C10-16-; >500

Apart from moderate to severe skin irritation, clinical signs included tremor, tonic-clonic convulsions, respiratory failure, and body weight loss in the study with the C12- alkyl sulfate and decreased body weights after administration of the C10-16- alkyl sulfates. No data are available for alkane sulfonates but due to a comparable metabolism and effect concentrations in long-term studies effect concentrations are expected to be in the same range as found for alkyl sulfates.

There are no data available for acute inhalation toxicity of alkyl sulfates, alkane sulfonates or alpha-olefin sulfonates.

In skin irritation tests using rabbits (aqueous solutions, OECD TG 404):

C8-14 and C8-16 (30%), C12-14 (90%), C14-18 (60%)- corrosive

Under occlusive conditions:

C12, and C12-14 (25%), C12-15-, C13-15 and C15-16 (5-7%) - moderate to strong irritants

Comparative studies investigating skin effects like transepidermal water loss, epidermal electrical conductance, skin swelling, extraction of amino acids and proteins or development of erythema in human volunteers consistently showed a maximum of effects with C12-alkyl sulfate, sodium; this salt is routinely used as a positive internal control giving borderline irritant reactions in skin irritation studies performed on humans. As the most irritant alkyl sulfate it can be concluded that in humans 20% is the threshold concentration for irritative effects of alkyl sulfates in general. No data were available with regard to the skin irritation potential of alkane sulfonates. Based on the similar chemical structure they are assumed to exhibit similar skin irritation properties as alkyl sulfates or alpha-olefin sulfonates of comparable chain lengths.

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant.

SODIUM LAURYL SULFATE

Concentrated C14-16- alpha-olefin sulfonates were severely irritating, but caused irreversible effects only if applied as undiluted powder. At concentrations below 10% mild to moderate, reversible effects, were found. No data were available for alkane sulfonates

Alkyl sulfates and C14-18 alpha-olefin sulfonates were not skin sensitizers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitization is expected.

However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitization resulting in hyperactive airway dysfunction and pulmonary allergy accompanied by fatigue, malaise and aching. Significant symptoms of exposure can persist for more than two years and can be activated by a variety of non-specific environmental stimuli such as a exhaust, perfumes and passive smoking.

Absorbed sulfonates are quickly distributed through living systems and are readily excreted. Toxic effects may result from the effects of binding to proteins and the ability of sulfonates to translocate potassium and nitrate (NO₃⁻) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies. Repeated skin contact with some sulfonated surfactants has produced sensitization dermatitis in predisposed individuals

Repeat dose toxicity: After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium). C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.

No data were available with regard to the repeated dose toxicity of alkane sulfonates. Based on the similarity of metabolic pathways between alkane sulfonates, alkyl sulfates and alkyl-olefin sulfonates, the repeated dose toxicity of alkane sulfonates is expected to be similar with NOAEL and LOAEL values in the same range as for alkyl sulfates and alpha-olefin sulfonates, i.e. 100 and 200-250 mg/kg/day, respectively, with the liver as potential target organ.

Genotoxicity: Alkyl sulfates of different chain lengths and with different counter ions were not mutagenic in standard bacterial and mammalian cell systems both in the absence and in the presence of metabolic activation. There was also no indication for a genotoxic potential of alkyl sulfates in various in vivo studies on mice (micronucleus assay, chromosome aberration test, and dominant lethal assay).

alpha-Olefin sulfonates were not mutagenic in the Ames test, and did not induce chromosome aberrations in vitro. No genotoxicity data were available for alkane sulfonates. Based on the overall negative results in the genotoxicity assays with alkyl sulfates and alpha-olefin sulfonates, the absence of structural elements indicating mutagenicity, and the overall database on different types of sulfonates, which were all tested negative in mutagenicity assays, a genotoxic potential of alkane sulfonates is not expected.

Carcinogenicity: Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day). alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure. No carcinogenicity studies were available for the alkane sulfonates.

Reproductive toxicity: No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates. The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

Developmental toxicity: In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death). The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day indicative of a delayed development. The lowest reliable NOAEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOAELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits.

For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity.

No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

Although the database for category members with C<12 is limited, the available data are indicating no risk as the substances have comparable toxicokinetic properties and metabolic pathways. In addition, longer-term studies gave no indication for adverse effects on reproductive organs with different alkyl sulfates

Alkyl sulfates (AS) anionic surfactants are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). An exception has been made for C12 AS which is classified as Harmful (Xn) with the risk phrases R22 (Harmful if swallowed) and R38 and R41 (CESIO 2000). AS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

AS are readily absorbed from the gastrointestinal tract after oral administration. Penetration of AS through intact skin appears to be minimal. AS are extensively metabolized in various species resulting in the formation of several metabolites. The primary metabolite is butyric acid-4-sulfate. The major site of metabolism is the liver. AS and their metabolites are primarily

eliminated via the urine and only minor amounts are eliminated via the faeces. In rats about 70-90% of the dose was eliminated via the urine within 48 hours after oral, intravenous or intraperitoneal administration of 1 mg of AS per rat. The acute toxicity of AS in animals is considered to be low after skin contact or oral intake.

For a homologous series of AS (C8 to C16), maximum swelling of stratum corneum (the outermost layer of epidermis) of the skin was produced by the C12 homologue. This is in accordance with the fact that the length of the hydrophobic alkyl chain influences the skin irritation potential. Other studies have shown that especially AS of chain lengths C11, C12 and C13 remove most amino acids and soluble proteins from the skin during washing.

Concentrated samples of AS are skin irritants in rabbits and guinea pigs. AS are non-irritant to laboratory animals at a 0.1% concentration. C12 AS is used in research laboratories as a standard substance to irritate skin and has been shown to induce an irritant eczema. AS were found, by many authors, to be the most irritating of the anionic surfactants, although others have judged the alkyl sulfates only as irritant as laurate (fatty acid soap).

A structure/effect relationship with regard to the length of the alkyl chain can also be observed on mucous membranes. The maximum eye irritation occurs at chain lengths of C10 to C14. In acute ocular tests, 10% C12 AS caused corneal damage to the rabbit eyes if not irrigated. Another study showed that a 1.0% aqueous C12 AS solution only had a slight effect on rabbit eyes, whereas 5% C12 AS caused temporary conjunctivitis, and 25% C12 AS resulted in corneal damage.

In a 13-week feeding study, rats were fed dietary levels of 0, 40, 200, 1,000 or 5,000 ppm of C12 AS. The only test material related effect observed was an increase in absolute organ weights in the rats fed with the highest concentration which was 5,000 ppm. The organ weights were not further specified and no other abnormalities were found.

In a mutagenicity study, rats were fed 1.13 and 0.56% C12 AS in the diet for 90 days. This treatment did not cause chromosomal aberrations in the bone marrow cells.

Mutagenicity studies with *Salmonella typhimurium* strains (Ames test) indicate no mutagenic effects of C12 AS. The available long-term studies in experimental animals (rats and mice) are inadequate to evaluate the carcinogenic potential of AS. However, in studies in which animals were administered AS in the diet at levels of up to 4% AS, there was no indication of increased risk of cancer after oral ingestion.

No specific teratogenic effects were observed in rabbits, rats or mice when pregnant animals were dosed with 0.2, 2.0, 300 and 600 mg C12 AS/kg body weight/day by gavage during the most important period of organogenesis (day 6 to 15 of pregnancy for mice and rats and day 6 to 18 of pregnancy for rabbits). Reduced litter size, high incidence of skeletal abnormalities and foetal loss were observed in mice at 600 mg C12 AS/kg/day, a dose level which also caused severe toxic effects in the parent animals in all three species. An aqueous solution of 2% AS was applied (0.1 ml) once daily to the dorsal skin (2 x 3 cm) of pregnant mice from day 1 to day 17 of gestation. A solution of 20% AS was tested likewise from day 1 to day 10 of gestation. The mice were killed on days 11 and 18, respectively. A significant decrease in the number of implantations was observed when mice were treated with 20% AS compared to a control group which was dosed with water. No evidence of teratogenic effects was noted.

When aqueous solutions of 2% and 20% AS (0.1 ml) were applied once per day to the dorsal skin (2 x 3 cm) of pregnant ICR/Jc1 mice from day 12 to day 17 of gestation no effects on pregnancy outcome were detected. Treatment with 20% AS resulted in growth retardation of suckling mice, but this effect disappeared after weaning. A 10% AS solution (0.1 ml) was applied twice daily to the dorsal skin (2 x 3 cm) of pregnant ICR/Jc1 mice during the preimplantation period (days 0-3 of gestation). A significant number of embryos collected on day 3 as severely deformed or remained at the morula stage. The number of embryos in the oviducts was significantly greater for the mice dosed with AS as compared to the control mice. No pathological changes were detected in the major organs of the dams

NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

Eye (None) None: None None rabbit None 250 ugSkin (rabbit):25 mg/24 hr-moderate Skin (None) None: None rabbit None 50 mg/24Eye (rabbit) 10: mg-

**OXALIC ACID &
SODIUM LAURYL
SULFATE**

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.

**ETHYLENE GLYCOL
MONOBUTYL ETHER &
DIETHYLENE 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 | ✘ | Carcinogenicity | ⊖ |
| Skin Irritation/Corrosion | ✔ | Reproductivity | ⊖ |
| Serious Eye Damage/Irritation | ✔ | STOT - Single Exposure | ⊖ |
| Respiratory or Skin sensitisation | ⊖ | STOT - Repeated Exposure | ⊖ |

Mutagenicity



Aspiration Hazard



Legend: ✘ – 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

| Ingredient | Endpoint | Test Duration (hr) | Species | Value | Source |
|-----------------------------------|----------|--------------------|-------------------------------|---------------|--------|
| oxalic acid | LC50 | 96 | Fish | 50.37564mg/L | 3 |
| oxalic acid | EC50 | 48 | Crustacea | 136.9mg/L | 4 |
| oxalic acid | EC50 | 96 | Algae or other aquatic plants | 91267.289mg/L | 3 |
| oxalic acid | EC0 | 192 | Algae or other aquatic plants | 80mg/L | 1 |
| oxalic acid | NOEC | 0.33 | Algae or other aquatic plants | 2.000mg/L | 4 |
| 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 |
| diethylene glycol monobutyl ether | LC50 | 96 | Fish | 488.016mg/L | 3 |
| diethylene glycol monobutyl ether | EC50 | 48 | Crustacea | >100mg/L | 1 |
| diethylene glycol monobutyl ether | EC50 | 96 | Algae or other aquatic plants | >100mg/L | 1 |
| diethylene glycol monobutyl ether | EC50 | 384 | Crustacea | 112.547mg/L | 3 |
| diethylene glycol monobutyl ether | NOEC | 96 | Algae or other aquatic plants | >=100mg/L | 1 |
| sodium lauryl sulfate | LC50 | 96 | Fish | 0.59mg/L | 4 |
| sodium lauryl sulfate | EC50 | 48 | Crustacea | 0.67mg/L | 4 |
| sodium lauryl sulfate | EC50 | 96 | Algae or other aquatic plants | 1.2mg/L | 4 |
| sodium lauryl sulfate | BCF | 1 | Fish | 0.85mg/L | 4 |
| sodium lauryl sulfate | EC50 | 96 | Crustacea | 0.12mg/L | 4 |
| sodium lauryl sulfate | NOEC | 0.08 | Fish | 0.0000013mg/L | 4 |

Legend:

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

DO NOT discharge into sewer or waterways.

Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|-----------------------------------|---------------------------|-----------------------------|
| oxalic acid | LOW | LOW |
| ethylene glycol monobutyl ether | LOW (Half-life = 56 days) | LOW (Half-life = 1.37 days) |
| diethylene glycol monobutyl ether | LOW | LOW |
| sodium lauryl sulfate | HIGH | HIGH |

Bioaccumulative potential

Continued...

| Ingredient | Bioaccumulation |
|-----------------------------------|------------------------|
| oxalic acid | LOW (LogKOW = -1.7365) |
| ethylene glycol monobutyl ether | LOW (BCF = 2.51) |
| diethylene glycol monobutyl ether | LOW (BCF = 0.46) |
| sodium lauryl sulfate | LOW (BCF = 7.15) |

Mobility in soil

| Ingredient | Mobility |
|-----------------------------------|--------------------|
| oxalic acid | HIGH (KOC = 1.895) |
| ethylene glycol monobutyl ether | HIGH (KOC = 1) |
| diethylene glycol monobutyl ether | LOW (KOC = 10) |
| sodium lauryl sulfate | LOW (KOC = 10220) |

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

| | |
|-------------------------------------|--|
| Product / Packaging disposal | <p>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.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>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.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ 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. ▶ Dispose of 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. Observe all label safeguards until containers are cleaned and destroyed. |
|-------------------------------------|--|

SECTION 14 TRANSPORT INFORMATION

Labels Required

| | |
|-------------------------|----------------|
| Marine Pollutant | NO |
| HAZCHEM | Not Applicable |

Land transport (ADG): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Air transport (ICAO-IATA / DGR): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

Sea transport (IMDG-Code / GGVSee): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

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

OXALIC ACID(6153-56-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

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 Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

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

DIETHYLENE GLYCOL MONOBUTYL ETHER(112-34-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

SODIUM LAURYL SULFATE(151-21-3) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

| National Inventory | Status |
|-------------------------------|--|
| Australia - AICS | Y |
| Canada - DSL | Y |
| Canada - NDSL | N (diethylene glycol monobutyl ether; oxalic acid; ethylene glycol monobutyl ether) |
| China - IECSC | Y |
| Europe - EINEC / ELINCS / NLP | Y |
| 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 |
|-----------------------|---|
| sodium lauryl sulfate | 151-21-3, 1335-72-4, 3088-31-1, 9004-82-4 |

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,
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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