Factory Equipped Wheel & Tyre Cleaner

Motor Active

Chemwatch Hazard Alert Code: **3**

Chemwatch: **4782-70**
Version No: **6.1**
Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Issue Date: **01/11/2019**
Print Date: **03/08/2022**
L.GHS.AUS.EN.E

SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Factory Equipped Wheel & Tyre Cleaner
Chemical Name	Not Applicable
Synonyms	Product Code: G9524
Proper shipping name	CORROSIVE LIQUID, BASIC, INORGANIC, N.O.S. (contains sodium metasilicate, anhydrous and sodium C14-16-olefin sulfonate)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	automotive; remove grime, grit and brake dust from all factory wheel surfaces Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Motor Active**
Address	35 Slough Business Park, Holker Street Silverwater NSW 2128 Australia
Telephone	+61 2 9737 9422|1800 350 622
Fax	+61 2 9737 9414
Website	www.motoractive.com.au
Email	andrews@motoractive.com.au

Emergency telephone number

Association / Organisation	**MotorActive**
Emergency telephone numbers	+61 2 9737 9422 (For General Information Monday to Friday 8:30am to 5:pm)
Other emergency telephone numbers	13 11 26 (In Case of Emergency contact: Poison Information Hotline)

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

		Min	Max
Flammability	1		
Toxicity	0		
Body Contact	3		
Reactivity	1		
Chronic	2		

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

Poisons Schedule	S5
Classification [1]	Skin Corrosion/Irritation Category 1A, Serious Eye Damage/Eye Irritation Category 1, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 2, Corrosive to Metals Category 1
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**

Page 1 continued...

Hazard statement(s)

H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H401	Toxic to aquatic life.
H290	May be corrosive to metals.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P234	Keep only in original packaging.
P273	Avoid 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): Take off immediately all contaminated clothing. Rinse skin with water [or 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/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P390	Absorb spillage to prevent material damage.
P304+P340	IF INHALED: Remove person to fresh air and keep 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.

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
2807-30-9	1-5	2-propoxyethanol
6834-92-0	1-5	sodium metasilicate, anhydrous
2605-79-0	1-5	N,N-dimethyldecylamine N-oxide
64-02-8	1-5	EDTA tetrasodium salt
68439-57-6	1-5	sodium C14-16-olefin sulfonate
Not Available	balance	Ingredients determined not to be hazardous
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available*	

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

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Skin Contact	If skin or hair contact occurs: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If 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

▸ 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.

For acute or short-term repeated exposures to highly alkaline materials:

▸ Respiratory stress is uncommon but present occasionally because of soft tissue edema.
▸ Unless endotracheal intubation can be accomplished under direct vision, cricothyroidotomy or tracheotomy may be necessary.
▸ Oxygen is given as indicated.
▸ The presence of shock suggests perforation and mandates an intravenous line and fluid administration.
▸ Damage due to alkaline corrosives occurs by liquefaction necrosis whereby the saponification of fats and solubilisation of proteins allow deep penetration into the tissue.

Alkalis continue to cause damage after exposure.

INGESTION:

▸ Milk and water are the preferred diluents

No more than 2 glasses of water should be given to an adult.

▸ Neutralising agents should never be given since exothermic heat reaction may compound injury.

* Catharsis and emesis are absolutely contra-indicated.
* Activated charcoal does not absorb alkali.
* Gastric lavage should not be used.

Supportive care involves the following:

▸ Withhold oral feedings initially.
▸ If endoscopy confirms transmucosal injury start steroids only within the first 48 hours.
▸ Carefully evaluate the amount of tissue necrosis before assessing the need for surgical intervention.
▸ Patients should be instructed to seek medical attention whenever they develop difficulty in swallowing (dysphagia).

SKIN AND EYE:

▸ Injury should be irrigated for 20-30 minutes.

Eye injuries require saline. [Ellenhorn & Barceloux: Medical Toxicology]

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **Do not** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include:

Continued...

	carbon dioxide (CO2) nitrogen oxides (NOx) sulfur oxides (SOx) 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

Minor Spills

- Drains for storage or use areas should have retention basins for pH adjustments and dilution of spills before discharge or disposal of material.
- Check regularly for spills and leaks.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact with the substance, by using protective equipment.
- Contain and absorb spill with sand, earth, inert material or vermiculite.
- Wipe up.
- Place in a suitable, labelled container for waste disposal.

Major Spills

Chemical Class: bases
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
LAND SPILL - SMALL				
cross-linked polymer - particulate	1	shovel	shovel	R,W,SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
foamed glass - pillow	2	throw	pitchfork	R, P, DGC, RT
expanded minerals - particulate	3	shovel	shovel	R, I, W, P, DGC
foamed glass - particulate	4	shovel	shovel	R, W, P, DGC,
LAND SPILL - MEDIUM				
cross-linked polymer -particulate	1	blower	skiploader	R,W, SS
sorbent clay - particulate	2	blower	skiploader	R, I, P
expanded mineral - particulate	3	blower	skiploader	R, I,W, P, DGC
cross-linked polymer - pillow	3	throw	skiploader	R, DGC, RT
foamed glass - particulate	4	blower	skiploader	R, W, P, DGC
foamed glass - pillow	4	throw	skiploader	R, P, DGC., RT

Legend
DGC: Not effective where ground cover is dense
R; Not reusable
I: Not incinerable
P: Effectiveness reduced when rainy
RT:Not effective where terrain is rugged
SS: Not for use within environmentally sensitive sites
W: Effectiveness reduced when windy
Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;
R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

- 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).
- 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

- **DO NOT** allow clothing wet with material to stay in contact with skin
- Avoid all personal contact, including inhalation.

Continued...

	‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Avoid contact with moisture. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with 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. ‣ **DO NOT** store near acids, or oxidising agents ‣ No smoking, naked lights, heat or ignition sources.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Lined metal can, lined metal pail/ can. ‣ Plastic pail. ‣ Polyliner drum. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks. For low viscosity materials ‣ Drums and jerricans must be of the non-removable head type. ‣ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ‣ Removable head packaging; ‣ Cans with friction closures and ‣ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, porcelain or stoneware, there must be sufficient inert cushioning material in contact with inner and outer packages unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	‣ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates. ‣ Avoid contact with copper, aluminium and their alloys. ‣ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
2-propoxyethanol	2.2 ppm	24 ppm	140 ppm
sodium metasilicate, anhydrous	3.8 mg/m3	42 mg/m3	250 mg/m3
EDTA tetrasodium salt	82 mg/m3	900 mg/m3	5,500 mg/m3
EDTA tetrasodium salt	75 mg/m3	830 mg/m3	5,000 mg/m3

Ingredient	Original IDLH	Revised IDLH
2-propoxyethanol	Not Available	Not Available
sodium metasilicate, anhydrous	Not Available	Not Available
N,N-dimethyldecylamine N-oxide	Not Available	Not Available
EDTA tetrasodium salt	Not Available	Not Available
sodium C14-16-olefin sulfonate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
2-propoxyethanol	E	≤ 0.1 ppm
sodium metasilicate, anhydrous	E	≤ 0.01 mg/m³
N,N-dimethyldecylamine N-oxide	E	≤ 0.1 ppm
EDTA tetrasodium salt	E	≤ 0.01 mg/m³
sodium C14-16-olefin sulfonate	E	≤ 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.*	

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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.

Exposure controls

Appropriate engineering 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.

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.

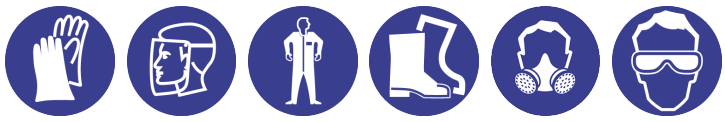
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
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.)

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

See Hand protection below

Hands/feet protection

- 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).

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	· 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 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
Other protection	‣ Overalls. ‣ PVC Apron. ‣ PVC protective suit may be required if exposure severe. ‣ Eyewash unit. ‣ Ensure there is ready access to a safety shower.

Respiratory protection

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

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	AK-AUS / Class1 P2	-
up to 50	1000	-	AK-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	AK-2 P2
up to 100	10000	-	AK-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand
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	Clear highly alkaline liquid with a mild odour; miscible with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.02-1.03
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	13.56	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	>=93	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>93	**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

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Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (Not Available%)**	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	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. 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. 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. 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). Oxalic acid occurs naturally in the common weed Oxalis, sour sobs'.
Skin Contact	The material can produce chemical burns following direct contact with the skin. 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. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	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. Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive. Substances than can cuase occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers Wherever it is reasonably practicable, exposure to substances that can cuase occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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. Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. .

Factory Equipped Wheel & Tyre Cleaner	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
2-propoxyethanol	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 960 mg/kg[2]	Eye (rabbit): 0.75 mg/24h SEVERE

Continued...

	Inhalation(Rat) LC50; >2300 ppm4h[1]	Eye (rabbit): 100 mg - SEVERE
	Oral (Rat) LD50; 3089 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg/24h -mild
		Skin: adverse effect observed (irritating)[1]

sodium metasilicate, anhydrous	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >5000 mg/kg[1]	Skin (human): 250 mg/24h SEVERE
	Inhalation(Rat) LC50; >2.06 mg/l4h[1]	Skin (rabbit): 250 mg/24h SEVERE
	Oral (Rat) LD50; 1153 mg/kg[2]	

N,N-dimethyldecylamine N-oxide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Not Available
	Oral (Rat) LD50; >300<2000 mg/kg[1]	

EDTA tetrasodium salt	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50; 630 mg/kg[2]	Eyes (rabbit): 1.9 mg
		Eyes (rabbit):100 mg/24h-moderate
		Skin (rabbit):500 mg/24h-moderate

sodium C14-16-olefin sulfonate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 578 mg/kg[1]	Eye: irritant **
	Inhalation(Rat) LC50; >51.5 mg/l4h[1]	Skin: irritant **
	Oral (Rat) LD50; >2000 mg/kg[2]	

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*

Factory Equipped Wheel & Tyre Cleaner	For glycolic acid: **Acute toxicity:** Glycolic acid (70% solution) is slightly toxic via the oral route, having an LD50 in rats of 193 8 mg/kg. It is moderately toxic via the inhalation route in male rats with a 4-hour LC50 of 3.6 mg/L. Glycolic acid is a skin and eye corrosive, but it is not a skin sensitiser in animals. However, numerous studies in humans with cosmetic products containing lower percentages of glycolic acid have shown some skin irritation potential, but no corrosivity. **Repeat dose toxicity:** Repeated exposures to glycolic acid via inhalation produced liver, spleen, thymus changes, and gastrointestinal tract alterations. Repeated administration of glycolic acid to rats by oral intubation caused decreases in body weight, body weight gain, food consumption, and food efficiency. In addition, toxicologically significant changes in haematologic measurements, clinical chemistry, and urinalysis parameters, as well as kidney lesions were observed. **Developmental and reproductive toxicity:** Maternal and developmental toxicity of crystalline, 99.6% pure, glycolic acid in the rat was seen at 300 and 600 mg/kg/day. The maternal and developmental NOEL was 150 mg kg/day, thus glycolic acid is not considered a unique developmental hazard to the conceptus. Glycolic acid did not affect reproductive performance in rats during a one-generation reproduction study following a 90-day feeding study. **Genotoxicity:** The compound was negative in the *in vitro* bacterial reverse mutation assay *(Salmonella* and E. *coli).* Glycolic acid produced a positive response in the *in* vitro mouse lymphoma assay only at excessively high concentrations under activated conditions, but was negative in the *in vivo* mouse micronucleus assay.
2-PROPOXYETHANOL	There have been no specific human studies, but the consistency of the animal experiments emphasizes that human exposure should be dramatically reduced. 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.

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	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).
SODIUM METASILICATE, ANHYDROUS	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.
N,N-DIMETHYLDECYLAMINE N-OXIDE	For amine oxides (AOs): Substantial data exist for mammalian toxicity by *in vitro* and *in vivo* testing. Amine oxides are produced, and transported in aqueous solutions that are 25-35% concentration and most tests were conducted with aqueous solutions in that concentration range. Sometimes aqueous formulations were tested where the AO was at lesser concentrations than 25-35%. Whatever concentration were tested, results are reported below for the active ingredient, amine oxide, in mg AO/kg bw for dermal and oral acute toxicity results and mg AO/kg bw/day for repeated dose studies. Toxicokinetic and metabolism studies indicate AOs are extensively metabolised and readily excreted after oral administration. Amine oxide was readily absorbed dermally by rats, mice and rabbits after 24 to 72 hours of exposure. After 8 hours of dermal exposure, humans absorbed <1%. **Acute toxicity:** In rat oral acute toxicity limit tests, no deaths occurred at single doses of 600 mg C10-16 AO/kg bw or less (for CAS No 70592-80-2). In multi-dose studies, acute oral LD50 values for rats ranged from 846 mg AO/kg bw to 3873 mg AO/kg bw (both values for CAS No 61788-90-7), with several other AOs having rat oral LD50s falling within this range. In single dose acute dermal toxicity limit tests, no deaths occurred at a dose of 520 mg AO/kg bw (CAS No 70592-80-2). This dose was equivalent to 2 mL/kg of a 30% formulation. There were no deaths observed in a rat acute inhalation study to aerosol droplets of a consumer product providing a dose of 0.016 mg AO/L. In a series of studies on rabbits, AOs of varying chain length showed consistent results and all ▸ were not irritating to the skin or eyes at low concentrations (1%), ▸ were moderately irritating at 5%, and ▸ more severely irritating when tested as produced (e.g., ~30% aqueous solutions). In studies that included rinsing, eye irritation effects diminished with rinsing after 30 seconds of exposure and were slight with rinsing after 4 seconds of exposure. In Draize rabbit eye irritation tests using ~30% AO solutions, rabbits experienced severe to moderate irritation. (The maximum concentration of AO is 10% active in consumer products.) Accidental eye exposure in manufacturing employee incidents and consumer incidents established that eye irritation effects of exposure during manufacturing and use of products containing AO and other surfactants are moderate, transient and reversible There is no indication of skin sensitisation for the AO category based on the available animal and human data. **Repeat dose toxicity:** In four repeated-dose studies with rats and mice exposed to AO via oral and dermal routes (all with CAS No 70592-80-2), three dermal studies were designed to assess the effect of repeated exposure on skin at maximum doses of 1.5 mg AO/kg-bw/day. Higher doses were tested in a 90-day dietary study with rabbits. No treatment related clinical chemistry, hematology and histopathological changes were observed. In these studies, LOAELs ranged from 87 to 150 mg AO/kg bw/day with the highest oral NOAEL below the lowest LOAEL as 80 mg AO/kg bw/day. Signs of toxicity observed in the oral study included suppressed mean body weight gain, lenticular opacities and diarrhea; in the dermal studies, local dermal irritation was evident. **Genetic toxicity:** In five *in vitro* bacterial (*Salmonella*) mutagenicity studies, AO shows no evidence of mutagenicity either with or without S9 metabolic activation at concentrations up to 250 ug/plate (higher concentrations caused cytotoxicity). Three *in vivo* studies investigated clastogenic effects on a close structural analog of the category, 1-(methyldodecyl)dimethylamine-N-oxide including: a mouse micronucleus, a Chinese hamster micronucleus and a Chinese hamster cytogenetics study. These studies were all negative showing no increase in micronuclei or chromosome aberrations. An *in vivo* mouse dominant lethal assay showed no evidence of heritable effects. Two AOs (CAS No 1643-20-5 and CAS No 3332-27-2) were negative in an *in vitro* cell transformation assay tested at concentrations up to 20 ug/ml. **Carcinogenicity:** The carcinogenic potential of amine oxides has been thoroughly investigated in three carcinogenicity studies in rats or mice by dermal, dietary, or drinking water routes. In all cases the substances demonstrated no evidence of a carcinogenic response. **Reproductive and developmental toxicity:** No evidence of reproductive toxicity or fertility effects was observed in a study in which rats were given dietary doses of AO in the diet over two generations (CAS No 1643-20-5). No macroscopic or histopathological changes were attributable to treatment with the test substance. The maternal NOAEL from this reproductive study was >40 mg AO/kg bw/day, which was the highest dose tested. At all treatment levels, the rate of bodyweight gain for the F1 and F2 offspring was reduced during the lactation period, however, this reduction was not greater than 10%. This effect appeared to be dose-related, but was not statistically significant until after weaning in the mid and high dose levels. This was not considered an adverse effect since the body weight change only reached statistical significance when the rat pups were getting the majority of their calories from solid food (Developmental NOAEL >40 mg/kg bw/day). In three developmental toxicity studies via gavage in rats and rabbits (with CAS No 1643-20-5 & 70592-80-2), effects such as decreased foetal weight or delayed ossification, were most often observed only at maternally toxic doses and were associated with the irritation effects of AO on the gastrointestinal tract. No decreases in litter size, no changes in litter parameters, no malformations or significant differences in skeletal defects were observed at oral doses up to 25 mg/kg bw/day in rats (based on decreased foetal weight at 100 mg/kg bw/day) and >160 mg/kg bw/day in rabbits (the highest dose tested).
EDTA TETRASODIUM SALT	* Sigma Aldrich - for the dihydrate For ethylenediaminetetraacetic acid (EDTA) and its salts: EDTA is a strong organic acid (approximately 1000 times stronger than acetic acid). It has a high affinity for alkaline-earth ions (for example, calcium and magnesium) and heavy-metal ions (for example, lead and mercury). This affinity generally results in the formation of highly stable and soluble hexadentate chelate complexes. EDTA s ability to complex is used commercially to either promote or inhibit chemical reactions, depending on application. EDTA and its salts are expected to be absorbed by the lungs and gastrointestinal tract; absorption through the skin is unlikely. In general, EDTA and its salts are mild skin irritants but considered severe eye irritants. The greatest risk in the human body will occur when the EDTA attempts to scavenge the trace metals used and required by the body. The binding of divalent and trivalent cations by EDTA can cause mineral deficiencies, which seem to be responsible for all of the known pharmacological effects. Sensitivity to the toxic effects of EDTA is, at least in part, related to the deficiency of zinc. Several short term studies, reported no adverse effects from administering doses up to 5% of EDTA and its salts to lab rodents daily and for several weeks. Only diarrhoea and lowered food consumption were reported in animals given 5% disodium EDTA. However, abnormal effects were seen in animals that were fed mineral deficient diets. Abnormal symptoms were observed in male and female rats fed a low mineral diet (0.54% Ca and 0.013%Fe) with the addition of 0%, 0.5%, or 1% disodium EDTA for 205 days. Rats fed a low percent of disodium EDTA in the diet for short term studies with adequate minerals showed no signs of toxicity. Rats fed 0.5% disodium EDTA for 44-52 weeks were without deleterious effects on weight gain, appetite, activity and appearance. Rats fed 1% disodium EDTA with adequate mineral diet for 220 days showed no evidence of dental erosion. EDTA and its salts are eliminated from the body, 95% via the kidneys and 5% by the bile, along with the metals and free ionic calcium which was

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	bound in transit through the circulatory system. Trisodium EDTA was tested in a bioassay for carcinogenicity by the National Cancer Institute. Trisodium EDTA administered to male and female rats at low (3,750 ppm) or high (7,500 ppm) concentrations for 103 weeks produced no compound-related signs of chemical toxicity, and tumor incidence was not related to treatment . EDTA and its salts should not pose a teratogenic concern based on previous studies in lab rodents. Study results indicate no teratogenic effects are likely in lab rodents at doses up to 1000 mg/kg. Adequate minerals in the diet and administration of tap water prevented possible teratogenic effects of EDTA during pregnancy. Teratogenic effects observed in lab rodents were likely due to animals maintained on deionised water and a semi-purified diet, and housed in nonmetallic caging. Infants and children will unlikely be exposed to high concentrations as in lab rodents. Rats given 1250 mg/kg or 1500 mg/kg by gavage exhibited more maternal toxicity than the diet group, but produced only 21% malformations in the offspring at the lower dose. The subcutaneously administration of 375 mg/kg was also maternally toxic, but did not result in malformations in the offspring. Differences in toxicity and teratogenicity are probably related to absorption differences and interaction with metals. Disodium EDTA ingested during pregnancy is teratogenic in rats at 2% in the diet and greater. The maximum human consumption of EDTA and its salts in foods was reported to be in the order of 0.4 mg/kg/day. Infants and children also generally drink tap water instead of deionised or distilled water. Even if young infants were to be fed some solid food, given the characteristics of EDTA and its salts, residues are not likely to be present at concentrations for potential sensitivity.
SODIUM C14-16-OLEFIN SULFONATE	* Van Waters and Rogers ** Albright & Wilson alpha-Olefine sulfonates (AOS) are classified as Irritant (Xi) with the risk phrases R38 and R41 for concentrations > 80% and R36/38 (Irritating to eyes and skin) for concentrations of 40-80% according to CESIO (CESIO 2000). AOS are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC. The absorption of AOS through intact skin is considered to be very low. Unchanged a -olefine sulfonate (AOS) and/or metabolites of AOS are primarily eliminated in the urine and, to a lesser extent, in the faeces within 24 hours of administration. The chemical structures of the metabolites have not yet been identified. AOS has a moderately low acute oral toxicity as indicated by LD50 values between 1,300 and 2,400 mg/kg body weight for rats and between 2,500 and 4,300 mg/kg body weight for mice. The toxic effects at high oral doses were reduced voluntary activity, diarrhoea and anaemia. AOS are mildly to moderately irritating to human skin depending on the concentration. In patch tests, human skin can tolerate contact to solutions containing up to 1% AOS for 24 hours resulting in only mild irritation. Instillation in the rabbit eye of 0.5% AOS caused no irritation after 24 hours, while 1% AOS caused a weak irritation The long-term toxicity and potential tumourigenic activity of AOS were assessed in a 2 year feeding study in rats at dietary levels of 0.1, 0.25 and 0.5%. No adverse clinical effects were observed, and survival rates were not affected by treatment with AOS. Histological examination of the tissues did not provide any evidence of toxicity or tumour induction In the Salmonella/microsome assay (Ames test) AOS were tested as negative showing a negligible potential to cause genetic damage. AOS were studied in rabbits, mice and rats for teratogenic potential. AOS were administered orally once a day by gavage on day 6-15 of pregnancy in mice and rats and on day 6-18 of pregnancy in rabbits. The doses were from 0.2?600 mg/kg body weight. The study showed no evidence of teratogenic potential. Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)
Factory Equipped Wheel & Tyre Cleaner & EDTA TETRASODIUM SALT	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.
Factory Equipped Wheel & Tyre Cleaner & N,N-DIMETHYLDECYLAMINE N-OXIDE	No significant acute toxicological data identified in literature search.
Factory Equipped Wheel & Tyre Cleaner & SODIUM C14-16-OLEFIN SULFONATE	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.

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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.
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 sensitisers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitisation is expected.
However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitisation 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 sensitisation 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

Factory Equipped Wheel & Tyre Cleaner & SODIUM METASILICATE, ANHYDROUS & EDTA TETRASODIUM SALT	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. 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. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

Acute Toxicity	✗	**Carcinogenicity**	✗
Skin Irritation/Corrosion	✓	**Reproductivity**	✗

Continued...

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

Factory Equipped Wheel & Tyre Cleaner

Endpoint	Test Duration (hr)	Species	Value	Source
Not Available	Not Available	Not Available	Not Available	Not Available

2-propoxyethanol

Endpoint	Test Duration (hr)	Species	Value	Source
LC50	96h	Fish	>91.3mg/l	Not Available
EC50	72h	Algae or other aquatic plants	>100mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	>=100mg/l	2

sodium metasilicate, anhydrous

Endpoint	Test Duration (hr)	Species	Value	Source
EC50(ECx)	48h	Crustacea	22.94-49.01mg/l	4
EC50	72h	Algae or other aquatic plants	207mg/l	2
EC50	48h	Crustacea	22.94-49.01mg/l	4
LC50	96h	Fish	180mg/l	1

N,N-dimethyldecylamine N-oxide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	0.015mg/l	2
EC50	48h	Crustacea	2.9mg/l	2
EC10(ECx)	72h	Algae or other aquatic plants	0.002mg/l	2
LC50	96h	Fish	2.4mg/l	2

EDTA tetrasodium salt

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	0.39mg/l	1
EC50	72h	Algae or other aquatic plants	1.01mg/l	1
EC50	48h	Crustacea	140mg/l	2
LC50	96h	Fish	>500mg/l	Not Available

sodium C14-16-olefin sulfonate

Endpoint	Test Duration (hr)	Species	Value	Source
NOEC(ECx)	72h	Algae or other aquatic plants	3.2mg/l	2
EC50	72h	Algae or other aquatic plants	5.2mg/l	2
EC50	48h	Crustacea	4.14-4.95mg/l	4
LC50	96h	Fish	1mg/l	1

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 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*

Toxic to aquatic organisms.
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.
for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates:

Environmental fate:
The close structural similarities result in physico-chemical properties and environmental fate characteristic which follow a regular pattern.
The most important common structural feature of the category members is the presence of a predominantly linear aliphatic hydrocarbon chain with a polar sulfate or sulfonate group, neutralised with a counter ion (i.e., Na+, K+, NH4+, or an alkanolamine cation).
The hydrophobic hydrocarbon chain (with a length typically between C8 and C18) and the polar sulfate or sulfonate groups confer surfactant properties and enable the commercial use of these substances as anionic surfactants
The structural similarities result in the same mode of ecotoxic action. Within each subcategory the most important parameter influencing ecotoxicity is the varying length of the alkyl chain. Although the counter ion may also influence the physico-chemical behaviour of these chemicals, the chemical reactivity and classification for the purpose of this assessment is not expected to be affected by the difference in counter ion.
As ionic substances, all members of this category have extremely low vapor pressures. Calculated values are in the ranges 10-11 to 10-15 hPa (C8-18 alkyl sulfates), 4.3.10-11 to 9.10-15 hPa (C8-18 alkane sulfonates), 2.1.10-13 to 6.9.10-15 hPa (C14-18 alkene sulfonates) and 3.3.10-17 to 5.8.10-19 hPa (C14-18 hydroxy alkane sulfonates). Therefore, they decompose before reaching their theoretical boiling points.
Measured water solubilities are available only for alkyl sulfates; they are in the range 196 000 mg/l (C12) to 300 mg/l (C16) and by factors of 50 to 300 higher than calculated values (C12: 617 mg/l, C16: 5 mg/l).
As surfactants have a tendency to concentrate at hydrophilic/hydrophobic boundaries rather than to equilibrate between phases log Kow is not a good descriptor of surfactant hydrophobicity and only of limited predictive value for the partitioning of these compounds in the environment.
All calculated physico-chemical properties of surfactants should be treated with caution, because the estimation models do not take into account surfactant properties. In addition, the

Continued...

results are doubtful for ionic substances.
Deduced from physico-chemical and surfactancy properties the target compartment for the substances of this category is the hydrosphere. Based on the ionic structure partitioning into the atmosphere can be excluded. In water, the compounds are stable to hydrolysis under environmental conditions.
Taking into account the low BCF factors (<73) that were determined for (up to) C16-alkyl sulfates, any significant bioaccumulation is not expected.
Soil sorption increases with chain length. Strong sorption on soils would be expected for chain length C14 upwards. Sediment concentrations were between 0.0035 and 0.021 mg/kg dw indicating that accumulation in sediments is low. Under certain conditions of reduced moisture in soil, i.e. in arid or semi-arid regions, accumulation in soil cannot be excluded.
The substances of this category are readily biodegradable. Significant biodegradation of alkyl sulfates in the raw sewage, i.e. in the sewer system before reaching the (waste-water treatment plant (WWTPs) is very likely. The substances of this category are quantitatively removed in WWTPs, mainly by biodegradation. Because of the anaerobic degradation of alkyl sulfates in sewage sludge, exposure of agricultural soils due to application of sludge as fertiliser is not expected. However, for alkane sulfonates and alpha-olefin sulfonates this exposure pathway cannot be excluded due to their recalcitrant or limited anaerobic degradability.
For alkyl sulfates: The biological degradation of AS is initiated by a hydrolytic cleavage of the sulfate ester bond catalysed by alkylsulfatases. The cleavage leaves inorganic sulfate and fatty alcohol which undergo oxidation by dehydrogenases to produce fatty acids via fatty aldehydes. The fatty acids are degraded by beta-oxidation and finally totally mineralised or incorporated into biomass. The biodegradation pathway for secondary AS differs from that of the primary AS by the formation of a ketone instead of an aldehyde. The biological degradation of AS is initiated by a hydrolytic cleavage of the sulfate ester bond catalysed by alkylsulfatases. The cleavage leaves inorganic sulfate and fatty alcohol which undergo oxidation by dehydrogenases to produce fatty acids via fatty aldehydes. The fatty acids are degraded by beta-oxidation and finally totally mineralised or incorporated into biomass. The biodegradation pathway for secondary AS differs from that of the primary AS by the formation of a ketone instead of an aldehyde. Biodegradation under anoxic conditions is anticipated to follow the same pathway as for the aerobic degradation.
Primary and secondary AS generally undergo complete primary biodegradation within a few days followed by a rapid ultimate biodegradation. Branched AS are also degraded quite rapidly, but multiple branchings of the alkyl chain considerably reduce the rate and extent of primary biodegradation. There are numerous studies confirming the aerobic biodegradability of AS, and linear primary AS exceeds all other anionic surfactants in the rate of primary and ultimate biodegradation. Also secondary AS are normally readily biodegradable as, e.g., the oxygen uptake from biodegradation of a linear secondary C10-13 AS corresponded to 77% ThOD in 22 days. Some highly branched AS being poorly primary biodegradable may also resist ultimate biodegradation.
Both linear and 2-alkyl-branched primary AS are degraded to a high extent under anaerobic conditions.
AS are generally considered to have a low potential for bioconcentration in aquatic organisms
For alkane sulfonates: Alkane sulfonate anionics (SAS) undergo rapid primary biodegradation with Methylene Blue Active Substance (MBAS) removal higher than 90% within a few days. Removal of 96% were seen in the OECD screening test for primary biodegradation. In activated sludge simulation tests, 96% of C10-18 SAS was removed, while the parent C13-18 SAS was removed by 83-96%.
Alkyl sulfonates are not degraded under anoxic conditions
For alpha-olefin sulfonates: alpha-Olefine sulfonates (AOS) AOS undergo rapid primary biodegradability with methylene blue active substances (MBAS) removal between 95 and 100% in 2 to 8 days in river water and inoculated media. The ultimate biodegradability of AOS exceeds the pass requirements in OECD 301 tests for ready biodegradability. report 85% DOC removal in the modified OECD screening test, 85% ThOD in the closed bottle test, and 65-80% $ThCO_2$ in the Sturm test. In activated sludge simulation tests, AOS was removed by 100% MBAS and 88% DOC. The alkene sulfonates and hydroxyalkane sulfonates in commercial AOS are both ultimately biodegraded as approximately 84% $ThCO_2$ was obtained during degradation of C14, C16, and C18 within 27 days, whereas the corresponding 3-hydroxyalkane sulfonates were degraded by approximately 86% under the same conditions.
AOS are not readily degradable under anaerobic conditions Reports indicate a range of 31% to 43% MBAS removal under anoxic conditions indicating primary biodegradation
Ecotoxicity:
The aquatic toxicity is influenced by a number of parameters, the length of the alkyl chain being most important. The pH and temperature of water bodies can affect the EC/LC50 values for compounds that contain ammonium ions.
The most sensitive trophic level in tests on the toxicity of alkyl sulfates were invertebrates, followed by fish. Algae proved to be less sensitive. The key study for the aquatic hazard assessment is a chronic test on Ceriodaphnia dubia, which covers a range of the alkyl chain length from C12 to C18. A parabolic response was observed with the C14 chain length being the most toxic (NOEC = 0.045 mg/l).
For alkyl sulfates: Fish LC50 (96 h): fathead minnow - fry 10.2 mg/l; juvenile 17 mg/l; adult 22.5 mg/l; rainbow trout 4.6 mg/l (static)
The aquatic toxicity of AS seems to increase with increasing alkyl chain length. This has been shown for daphnids and for some fish species. An overall comparison of the acute toxicity between the primary and secondary AS shows only minor differences in the toxicity, although only a few studies for comparison are available.
The available data describing the toxicity of AS towards algae indicate that the lowest EC50 values range between 1 and 10 mg/l for C12 AS
The toxicity of AS towards invertebrates has mainly been examined in tests with Daphnia magna. The acute toxicity of AS to Daphnia magna increased with increasing alkyl chain length. It has been shown that during degradation of C12 AS, the toxicity first increased to a maximum after 30 hours and then fell to almost a negligible value. The increase in toxicity was explained by the formation of the more toxic dodecanoic acid which is rapidly transformed to other and less toxic metabolites.
Studies showed that the 24 h-LC50 values for killifish in distilled water decreased by a factor of about 10 when the alkyl chain was increased by two carbon atoms. C16 was 10 times more toxic than C14, which was about 10 times more toxic than C12 AS.
The toxicity of AS to fish has been demonstrated to increase with increasing alkyl chain length as also seen in studies with Daphnia magna. The acute toxicity on Daphnia magna has been determined for chain length C8-C14. Results were comparable to alkyl sulfates in the range between C8 and C10, while C12 and C14 are significantly less toxic. Chronic data obtained for C12 alkane sulfonate sodium and C12-alkyl sulfate sodium with the rotifer *Brachionus calicyflorus* similarly show that alkane sulfonates might be less toxic than alkyl sulfates. C16 and C18 alkane sulfonates are assumed to exhibit the same toxicity than alkyl sulfates of comparable chain lengths. No data are available concerning the toxicity of alkane sulfonates on fish and algae. However, a similar toxicity might be assumed because of structural and physico-chemical similarities between the three subcategories
Whereas most correlations between AS structure and toxicity show an increasing toxicity with increasing alkyl chain length, the budding in Hydra attenuata was apparently more affected by C10 AS than by C12, C14, and C16 AS. The authors suggested that the decrease in toxicity with increasing alkyl chain length was attributable to reduced solubility in water
Tests on the toxicity to microorganisms were only conducted with alkyl sulfates as test substances. A test on the inhibition of respiration of activated sludge resulted in an 3 h-EC50 of 135 mg/l (nominally). The lowest effect value for protozoa was obtained from a test on *Uronema parduczi* using C12-alkyl sulfate sodium - the 20 h-EC5 was 0.75 mg/l.
Experimental test results on benthic organisms in a water-sediment system are not available. However, due to sediment-water partitioning coefficients Kd < 350, no significant risk for organisms in this compartment is to be expected.
Data indicate that toxic effects on soil organisms might only be expected at high concentrations for alkyl sulfates. Toxicity of alkane sulfonates and alpha-olefin sulfonates can not be assessed because test results for terrestrial organisms are not available.
For alpha-olefin sulfonates, reliable short-term tests on fish, invertebrates and algae are available. The results indicate that toxicity is increasing as the alkyl chain length increases. The lowest available effect value is the 96 h-LC50 = 0.5 mg/l, determined in tests on *Oryzias latipes, Rasbora heteromorpha* and *Salmo trutta*
Algae show toxic effects to growth when exposed 10-100 mg/l for C14-18 AOS.
EC50 values for *Daphnia magna* have been determined within the range 5-50 mg/l for C14-18 AOS . Another study with *Daphnia magna*, showed EC50 values of 16.6 mg/l for C14-16 AOS and 7.7 mg/l for C16-18 AOS.
Studies performed with fish show that the higher homologues of AOS are more toxic than the lower ones. This has been illustrated for different fish species (LC50 (96 h) range 0.5-5.3 mg/l)
For alkane sulfonates: The toxicity of various SAS homologues was determined in tests with *Chlamydomonas variabilis***.** After 24 hours of exposure at 20 C, there was a tendency to an increased toxicity with increasing chain length. The EC50 values were 125 mg/l for C10.3, 74.9 mg/l for C11.2, 32.4 mg/l for C14, 15.8 mg/l for C15, 9.42 mg/l for C16, 3.93 mg/l for C17, 3.71 mg/l for C18.9, and 8.47 mg/l for C20.7.

SIDS Initial Assessment Profile
Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency
Prevent, by any means available, spillage from entering drains or water courses.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
2-propoxyethanol	LOW	LOW
N,N-dimethyldecylamine N-oxide	LOW	LOW

Continued...

Bioaccumulative potential

Ingredient	Bioaccumulation
2-propoxyethanol	LOW (LogKOW = 0.0755)
N,N-dimethyldecylamine N-oxide	LOW (LogKOW = 3.6908)

Mobility in soil

Ingredient	Mobility
2-propoxyethanol	HIGH (KOC = 1)
N,N-dimethyldecylamine N-oxide	LOW (KOC = 5487)

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. ▸ **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. ▸ Treat and neutralise at an approved treatment plant. ▸ Treatment should involve: Neutralisation with suitable dilute acid 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

	
Marine Pollutant	NO
HAZCHEM	2X

Land transport (ADG)

UN number	3266
UN proper shipping name	CORROSIVE LIQUID, BASIC, INORGANIC, N.O.S. (contains sodium metasilicate, anhydrous and sodium C14-16-olefin sulfonate)
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	3266
UN proper shipping name	Corrosive liquid, basic, inorganic, n.o.s. * (contains sodium metasilicate, anhydrous and sodium C14-16-olefin sulfonate)

Continued...

Transport hazard class(es)	ICAO/IATA Class: 8 ICAO / IATA Subrisk: Not Applicable ERG Code: 8L
Packing group	III
Environmental hazard	Not Applicable
Special precautions for user	Special provisions: A3 A803 Cargo Only Packing Instructions: 856 Cargo Only Maximum Qty / Pack: 60 L Passenger and Cargo Packing Instructions: 852 Passenger and Cargo Maximum Qty / Pack: 5 L Passenger and Cargo Limited Quantity Packing Instructions: Y841 Passenger and Cargo Limited Maximum Qty / Pack: 1 L

Sea transport (IMDG-Code / GGVSee)

UN number	3266
UN proper shipping name	CORROSIVE LIQUID, BASIC, INORGANIC, N.O.S. (contains sodium metasilicate, anhydrous and sodium C14-16-olefin sulfonate)
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

Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
2-propoxyethanol	Not Available
sodium metasilicate, anhydrous	Not Available
N,N-dimethyldecylamine N-oxide	Not Available
EDTA tetrasodium salt	Not Available
sodium C14-16-olefin sulfonate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
2-propoxyethanol	Not Available
sodium metasilicate, anhydrous	Not Available
N,N-dimethyldecylamine N-oxide	Not Available
EDTA tetrasodium salt	Not Available
sodium C14-16-olefin sulfonate	Not Available

SECTION 15 Regulatory information

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

2-propoxyethanol is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

sodium metasilicate, anhydrous is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

N,N-dimethyldecylamine N-oxide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

EDTA tetrasodium salt is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Continued...

sodium C14-16-olefin sulfonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals | Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (2-propoxyethanol; sodium metasilicate, anhydrous; N,N-dimethyldecylamine N-oxide; EDTA tetrasodium salt; sodium C14-16-olefin sulfonate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (N,N-dimethyldecylamine N-oxide)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (2-propoxyethanol; N,N-dimethyldecylamine N-oxide; sodium C14-16-olefin sulfonate)
Vietnam - NCI	Yes
Russia - FBEPH	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. These ingredients may be exempt or will require registration.*

SECTION 16 Other information

Revision Date	01/11/2019
Initial Date	30/05/2012

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	13/08/2018	Name
6.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

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

Continued...

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
ES: Exposure Standard
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
AIIC: Australian Inventory of Industrial Chemicals
DSL: Domestic Substances List
NDSL: Non-Domestic Substances List
IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances