MS TopFoam Power

Stockyard Industries PTY. LTD

Chemwatch Hazard Alert Code: 4

Chemwatch: **5575-54**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **09/03/2023**
Print Date: **29/04/2024**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	MS TopFoam Power
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	CAUSTIC ALKALI LIQUID, N.O.S. (contains sodium hydroxide)
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	Use according to manufacturer's directions.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Stockyard Industries PTY. LTD**
Address	54 King Street Clifton Queensland 4361 Australia
Telephone	+61 7 4697 3344
Fax	+61 7 4697 3352
Website	www.JHswash.com
Email	sales@stockradindustries.com.au

Emergency telephone number

Association / Organisation	**Poisons Information Centre**
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Corrosive to Metals Category 1, Skin Corrosion/Irritation Category 1A, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 2
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

H290	May be corrosive to metals.
H314	Causes severe skin burns and eye damage.
H401	Toxic to aquatic life.

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.

Precautionary statement(s) Response

P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.

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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.
P363	Wash contaminated clothing 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.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
1310-73-2	0-9	sodium hydroxide
112-34-5	0-7	diethylene glycol monobutyl ether
1300-72-7	0-6	sodium xylenesulfonate
68891-38-3	0-5	sodium lauryl ether sulfate
137-16-6	0-3	lauroylsarcosine, sodium salt
68155-07-7	0-1	caprylic acid diethanolamide
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.
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, without delay. ▸ 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

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.

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* 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: carbon dioxide (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material. May emit corrosive fumes.
HAZCHEM	2R

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	‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Neutralise/decontaminate residue (see Section 13 for specific agent). ‣ Collect solid residues and 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

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Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ **WARNING:** To avoid violent reaction, **ALWAYS** add material to water and **NEVER** water to material. ▸ Avoid smoking, naked lights or ignition sources. ▸ 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

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	sodium hydroxide	Sodium hydroxide	Not Available	Not Available	2 mg/m3	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
sodium hydroxide	Not Available	Not Available	Not Available
diethylene glycol monobutyl ether	30 ppm	33 ppm	200 ppm

Ingredient	Original IDLH	Revised IDLH
sodium hydroxide	10 mg/m3	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available
sodium xylenesulfonate	Not Available	Not Available
sodium lauryl ether sulfate	Not Available	Not Available
lauroylsarcosine, sodium salt	Not Available	Not Available
caprylic acid diethanolamide	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
diethylene glycol monobutyl ether	E	≤ 0.1 ppm
sodium xylenesulfonate	E	≤ 0.01 mg/m³
sodium lauryl ether sulfate	E	≤ 0.01 mg/m³
lauroylsarcosine, sodium salt	E	≤ 0.01 mg/m³
caprylic acid diethanolamide	E	≤ 0.1 ppm
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.***	

Continued...

MATERIAL DATA

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.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.
An approved self contained breathing apparatus (SCBA) may be required in some situations.
Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "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.

Individual protection measures, such as personal protective equipment

Eye and face protection

- Safety glasses with unperforated side shields may be used where continuous eye protection is desirable, as in laboratories; spectacles are not sufficient where complete eye protection is needed such as when handling bulk-quantities, where there is a danger of splashing, or if the material may be under pressure.
- Chemical goggles. Whenever there is a danger of the material coming in contact with the eyes; goggles must be properly fitted. [AS/NZS 1337.1, EN166 or national equivalent]
- Full face shield (20 cm, 8 in minimum) may be required for supplementary but never for primary protection of eyes; these afford face protection.
- Alternatively a gas mask may replace splash goggles and face shields.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

Hands/feet protection

- Elbow length PVC gloves
- When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots.

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity
Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.

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	· 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.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
MS TopFoam Power

Material	CPI
BUTYL	A
NAT+NEOPR+NITRILE	A
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
NITRILE+PVC	A
PE	A
PE/EVAL/PE	A
PVC	A
SARANEX-23	A
SARANEX-23 2-PLY	A
TEFLON	A
VITON/CHLOROBUTYL	A

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

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

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

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SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Alkaline liquid with characteristic odour; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.158 @20C
Odour	Characteristic	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	200
pH (as supplied)	13.2	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	0	**Viscosity (cSt)**	16 @40C
Initial boiling point and boiling range (°C)	100	**Molecular weight (g/mol)**	Not Applicable

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Flash point (°C)	Not Available	**Taste**	Not Available
Evaporation rate	0.3 BuAC = 1	**Explosive properties**	Not Available
Flammability	Not Available	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	24.6	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	0.85	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	2.332 @20C	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Applicable	**VOC g/L**	73.649

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	Evidence shows, or practical experience predicts, that the material produces irritation of the respiratory system, in a substantial number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system. 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. Severe acute sodium hydroxide dust inhalation exposure may be fatal due to spasm, inflammation and oedema of the larynx and bronchi, chemical pneumonitis and severe pulmonary oedema. Symptoms of overexposure include burning sensation, coughing, wheezing, laryngitis, shortness of breath, headache, nausea and vomiting. One case report describes kidney and liver damage in two people working in a closed room with paint containing diethylene glycol monobutyl ether and at the same time consuming large quantities of alcoholic beverages. It has as yet not been established whether the glycol ether and alcohol have synergistic effects but it is possible that oxidation and elimination of both substances probably involves alcohol dehydrogenases; competitive inhibition would be the result.
Ingestion	The material can produce severe chemical burns within the oral cavity and gastrointestinal tract following ingestion. Ingestion of alkaline corrosives may produce immediate pain, and circumoral burns. Mucous membrane corrosive damage is characterised by a white appearance and soapy feel; this may then become brown, oedematous and ulcerated. Profuse salivation with an inability to swallow or speak may also result. Even where there is limited or no evidence of chemical burns, both the oesophagus and stomach may experience a burning pain; vomiting and diarrhoea may follow. The vomitus may be thick and may be slimy (mucous) and may eventually contain blood and shreds of mucosa. Epiglottal oedema may result in respiratory distress and asphyxia. Marked hypotension is symptomatic of shock; a weak and rapid pulse, shallow respiration and clammy skin may also be evident. Circulatory collapse may occur and, if uncorrected, may produce renal failure. Severe exposures may result in oesophageal or gastric perforation accompanied by mediastinitis, substernal pain, peritonitis, abdominal rigidity and fever. Although oesophageal, gastric or pyloric stricture may be evident initially, these may occur after weeks or even months and years. Death may be quick and results from asphyxia, circulatory collapse or aspiration of even minute amounts. Death may also be delayed as a result of perforation, pneumonia or the effects of stricture formation. Accidental ingestion of the material may be damaging to the health of the individual. At sufficiently high doses the material may be nephrotoxic (i.e. poisonous to the kidney). Ingestion of anionic surfactants/ hydrotropes may produce diarrhoea, intestinal distension and occasional vomiting. Lethal doses in animals range from 1 to 5 gm/kg. Cyanosis, rapid breathing and heart beat, low blood pressure, muscle tenderness and unconsciousness may follow ingestion of diethylene glycol monobutyl ether. Swallowing large or repeated doses may affect kidney function.
Skin Contact	The material can produce severe chemical burns following direct contact with the skin. Because the substance contains a polar sulfonate group, poor dermal absorption is expected. Skin contact with alkaline corrosives may produce severe pain and burns; brownish stains may develop. The corroded area may be soft, gelatinous and necrotic; tissue destruction may be deep. Anionic surfactants/ hydrotropes generally produce skin reactions following the removal of natural oils. The skin may appear red and may become sore. Papular dermatitis may also develop. Sensitive individuals may exhibit cracking, scaling and blistering. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected. There are indications that diethylene glycol monobutyl ether is absorbed through intact skin. Toxic effects only occur at very high doses.
Eye	The material can produce severe 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. Direct contact with alkaline corrosives may produce pain and burns. Oedema, destruction of the epithelium, corneal opacification and iritis may occur. In less severe cases these symptoms tend to resolve. In severe injuries the full extent of the damage may not be immediately apparent with late complications comprising a persistent oedema, vascularisation and corneal scarring, permanent opacity, staphyloma, cataract, symblepharon and loss of sight. Direct eye contact with some concentrated anionic surfactants/ hydrotropes produces corneal damage, in some cases severe. Low concentrations may produce immediate discomfort, conjunctival hyperaemia, and oedema of the corneal epithelium. Healing may take several days. Temporary clouding of the cornea may occur.

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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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Long-term exposure to respiratory irritants may result in disease of the airways involving difficult breathing and related systemic problems. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. 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 exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. 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. Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

MS TopFoam Power	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
sodium hydroxide	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1350 mg/kg[2]	Eye (rabbit): 0.05 mg/24h SEVERE
	Oral (Rabbit) LD50; 325 mg/kg[1]	Eye (rabbit):1 mg/24h SEVERE
		Eye (rabbit):1 mg/30s rinsed-SEVERE
		Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg/24h SEVERE
		Skin: adverse effect observed (corrosive)[1]
diethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 4120 mg/kg[2]	Eye (rabbit): 20 mg/24h moderate
	Oral (Rat) LD50: 5660 mg/kg[2]	Eye (rabbit): 5 mg - SEVERE
sodium xylenesulfonate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >10 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]
sodium lauryl ether sulfate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 1600 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit):25 mg/24 hr moderate
		Skin: adverse effect observed (irritating)[1]
lauroylsarcosine, sodium salt	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: >0.05<0.5 mg/l4h[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >5000 mg/kg[1]	Skin: adverse effect observed (irritating)[1]
caprylic acid diethanolamide	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50: >2000 mg/kg[1]	
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***	

SODIUM HYDROXIDE	The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.
DIETHYLENE GLYCOL MONOBUTYL ETHER	For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates. **Acute toxicity:** There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative.

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Repeat dose toxicity: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens.
Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in ***S. typhimurium*** strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in E. coli WP2uvrA, with and without metabolic activation. ***In vitro*** cytogenicity and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and ***in vivo*** micronucleus or cytogenicity tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic.
Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA.
Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m3) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21 In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus

SODIUM XYLENESULFONATE

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group.
Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.
Acute toxicity: These substances are well absorbed after ingestion; penetration through the skin is however poor. After absorption, these chemicals are distributed mainly to the liver.

Acute oral LD50 values of alkyl sulfates in rats and/or mice were (in mg/kg):
C10-; 290-580
C10-16-, and C12-; 1000-2000
C12-14, C12-15, C12-16, C12-18 and C16-18-; >2000
C14-18, C16-18-; >5000
The clinical signs observed were non-specific (piloerection, lethargy, decreased motor activity and respiratory rate, diarrhoea). At necropsy the major findings were irritation of the gastrointestinal tract and anemia of inner organs.
Based on limited data, the acute oral LD50 values of alkane sulfonates and alpha-olefin sulfonates of comparable chain lengths are assumed to be in the same range.
The counter ion does not appear to influence the toxicity in a substantial way.

Acute dermal LD50 values of alkyl sulfates in rabbits (mg/ kg):
C12-; 200
C12-13 and C10-16-;>500

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

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

In skin irritation tests using rabbits (aqueous solutions, OECD TG 404):
C8-14 and C8-16 (30%), C12-14 (90%), C14-18 (60%)- corrosive
Under occlusive conditions:
C12, and C12-14 (25%), C12-15-, C13-15 and C15-16 (5-7%) - moderate to strong irritants

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

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant.
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 (NO_3^-) 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

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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

SODIUM LAURYL ETHER SULFATE

* [CESIO]
Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.
Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .
On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.
Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers.
Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69
Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.
PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations.
Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used
Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology
http://doi.org/10.5487/TR.2015.31.2.105
Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) (syn: AAASD ,alkyl alcohol alkoxylate sulfates, SLES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3E0) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes).
AES are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC.
In assessing this family the Cosmetic Ingredient Review (CIR) Expert Panel recognized that most of the acute oral toxicity, dermal irritation and sensitization, subchronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies have been conducted on ammonium laureth sulfate and sodium laureth sulfate. Sodium and ammonium laureth sulfate have not evoked adverse responses in any toxicological testing, including acute oral toxicity, sub-chronic and chronic oral toxicity, reproductive and develop-mental toxicity, carcinogenicity, and photosensitization studies. These data, however, are considered a sufficient basis for concluding that the other ingredients are safe in the practices of use and concentration described in the safety assessment because of the fundamental chemical similarities between them and because they all are chemically similar salts(salts are expected to be dissociated in any product formulation independent of whether the salt is sodium, ammonium, magnesium, or zinc) of sulfated ethoxylated alcohols, and they all function as surfactants in cosmetic formulations. Based on these considerations, safety test data on one ingredient may be extrapolated to all of them. The panel noted that sodium laureth sulfate and ammonium laureth sulfate can produce eye and/or skin irritation in experimental animals and in some human test subjects; irritation may occur in some users of cosmetic formulations containing these ingredients. The irritant effects, however, are similar to those produced by other detergents, and the severity of the irritation appears to increase directly with concentration
Acute toxicity: AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact sensitiser and AES is not expected to be irritating to the skin at in-use concentrations. The available repeated dose toxicity data demonstrate the low toxicity of AES. Also, they are not considered to be

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mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. The consumer aggregate exposure from direct and indirect skin contact as well as from the oral route via dishware residues results in an estimated total body burden of 29 ug /kg bw/day.
AES are easily absorbed in the intestine in rats and humans after oral administration. Radiolabelled C11 AE3S and C12 AE3S were extensively metabolized in rats and most of the 14C-activity was eliminated via the urine and expired air independently of the route of administration (oral, intraperitoneal or intravenous). The main urinary metabolite from C11 AE3S is propionic acid-3-(3EO)-sulfate. For C12 and C16 AE3S, the main metabolite is acetic acid-2-(3EO)-sulfate. The alkyl chain appears to be oxidised to CO2 which is expired. The EO-chain seems to be resistant to metabolism.
AES are better tolerated on the skin than, e.g., alkyl sulfates and it is generally agreed that the irritancy of AES is lower than that of other anionic surfactants. Alkyl chain lengths of 12 carbon atoms are considered to be more irritating to the skin compared to other chain lengths. The skin irritating properties of AES normally decrease with increasing level of ethoxylation. Undiluted AES should in general be considered strongly irritating. Even at concentrations of 10% moderate to strong effects can be expected. However, only mild to slight irritation was observed when a non-specified AES was applied at 1% to the skin.
Subchronic toxicity: A 90-day subchronic feeding study in rats with 1% of AE3S or AE6S with alkyl chain lengths of C12-14 showed only an increased liver/body weight ratio. In a chronic oral study with a duration of 2 years, doses of C12-AE3S of 0.005 - 0.05% in the diet or drinking water had no effects on rats. The concentration of 0.5% sometimes resulted in increased kidney or liver weight.
Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.
Reproductive and developmental toxicity: No evidence of reproductive and teratogenic effects was seen in a two-generation study in rats fed with a mixture (55:45) of AES and linear alkylbenzene sulfonates. Dietary levels of 0.1, 0.5, and 1% were administered to the rats either continuously or during the period of major organogenesis during six pregnancies. No changes in reproductive or embryogenic parameters were observed.
Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives.
Carcinogenicity: Chronic dietary studies conducted with rats showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).
NOTE: Some products containing AES/ SLES have been found to also contain traces (up to 279 ppm) of 1,4-dioxane; this is formed as a by-product during the ethoxylation step of its synthesis. The U.S. Food and Drug Administration recommends that these levels be monitored.The U.S. Environmental Protection Agency classifies 1,4-dioxane to be a probable human carcinogen (not observed in epidemiological studies of workers using the compound, but resulting in more cancer cases in controlled animal studies), and a known irritant with a no-observed-adverse-effects level of 400 milligrams per cubic meter at concentrations significantly higher than those found in commercial products. Under Proposition 65, 1,4-dioxane is classified in the U.S. state of California to cause cancer. The FDA encourages manufacturers to remove 1,4-dioxane, though it is not required by federal law.
Sensitising potential: Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .
On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing
Toxicokinetics:
Following oral exposure, AES is readily absorbed in the gastrointestinal tract in human and rat and excreted principally via the urine or faeces depending on the length of the ethoxylate chain but independently of the route of administration. Once absorbed, AES is extensively metabolized by beta- or omega oxidation. The alkyl chain appears to be oxidized to CO2 which is expired. The EO-chain seems to be resistant to metabolism. Regarding the different anions, it is expected that the salts will be converted to the acid form in the stomach. This means that for all types of parent chemical the same compound structure eventually enters the small intestine. Hence, the situation will be similar for compounds originating from different salts and therefore no differences in uptake are anticipated.
The length of the ethoxylate portion in an AES molecule seems to have an important impact on the biokinetics of AES in humans and in the rat. Alcohol ethoxysulfates with longer ethoxylate chains (>7-9 EO units) are excreted at a higher proportion in the faeces. This is however not of interest for the AES within this category as their ethoxylation grade is 1 to 2.5.
Dermal absorption
There are two reliable and relevant studies available assessing the dermal absorption rate of AES. The study with AES (C12 -14; 2 EO) Na (CAS 68891-38-3) was performed according to OECD guideline 428 with human skin of the abdomen region (3 donors, n=2). The test substance was applied at a concentration of 10% for 24 h
The mean amount removed from the skin surface (skin wash) ranged from 87.16% to 94.56% of the dose applied. The amounts in the receptor could not be quantified, since it was below the analytical limit of quantification (LOQ). The mean recovery in the two first tape strips was 1.48% during all performed experiments. In the further 18 tape strips a mean recovery of 2.86% was documented. The recovery values for the cryocuts have accounted 0.56% in mean.
The mean absorbed dose, sum of the amounts found in the viable epidermis, dermis and receptor medium was 0.56%. The mean recovery values have varied from 90.90% to 100.21%, which complies with the acceptance criteria of 100 ± 15%.
There is also an in vivo study according to OECD guideline 427 for AES (C12 -14; 2 EO) Na (CAS 68891-38-3) available (Aulmann, 1996). Wistar rats were exposed to 1% aqueous solutions of the test item for 15 min and 48 h under semi-occlusive conditions. The mean amount of AES (C12-14; 2 EO) Na (CAS 68891-38-3) removed from the skin surface after the 15 min exposure period (via washing) ranged from 92.8% to 97.2% of the dose and from 91.6% to 98.4% after 48 h when the skin was not washed until sacrifice. The amounts in faeces and skin could not always be quantified, since it was below the analytical limit of quantification (LOQ).
The mean absorbed dose, sum of the amounts found in urine, faeces and skin in the experiment with washing was about 0.1% and 0.9% without washing.
The mean recovery values varied from 98.6% to 103%.
Taking the results of both studies together the dermal absorption is very low. The in vitro study with human skin indicated the dermal absorption to be 0.56% within 24 h and the in vivo study indicated the dermal absorption to be 0.9% within 48 h. The mean recovery rates on the skin are greater than 87%. These data demonstrate that the test substance remains on the skin surface. Thus, the value of 0.9% dermal absorption is taken for the dermal absorption.
References:
Danish EPA - Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products (2001). Environmental Project No. 615, pp. 24-28
HERA (2003). Human & Environmental Risk Assessment on ingredients of European household cleaning products Alcohol Ethoxysulphates, Human Health Risk Assessment Draft, 2003. http: //www. heraproject. com.
Final Report of the Amended Safety Assessment of Sodium Laureth Sulfate and Related Salts of Sulfated Ethoxylated Alcohols: (nternational Journal of Toxicology 29 (Supplement 3) 151S-161S: 2010
http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151
The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

LAUROYLSARCOSINE, SODIUM SALT

* Dow Chemical

Continued...

The amino acids alkyl amides most likely dissociate into amino acids and fatty acids in the presence of water. Because most of these amino acids and fatty acids are found in the foods we consume daily, oral toxicity is not expected.
In turn, dermal toxicity would not be expected to be different from oral exposure.
Data from the previous safety assessments on alpha-amino acids and fatty acids support that these ingredients would not likely be irritants or sensitisers.
No irritation was observed in in vitro studies with disodium capryloyl glutamate.Acetyl proline was a mild irritant in another in vitro study. In human studies, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were not dermal irritants.
No ocular irritation was observed in in vitro studies of acetyl tyrosinamide, disodium capryloyl glutamate, and sodium lauroyl glutamate. No adverse effects were observed during in-use studies of acetyl hydroxyproline and acetyl tyrosinamide in human subject.
Severe irritation was observed in 1 study of sodium cocoyl glutamate at 5%, but was not irritating in another study with an unknown concentration. Sodium cocoyl glutamate and glycinate (fatty acids, C8-14 -even numbered are generally classified as R41/ H318 - Causes severe eye damage - by their suppliers, in spite of contrasting evidence.
No sensitisation was observed in human studies with acetyl hydroxyproline, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate.
Acetyl tyrosinamide, sodium cocoyl glutamate and sodium lauroyl glutamate were not phototoxic in human studies
In in vitro studies, acetyl glutamic acid, acetyl proline, acetyl tyrosinamide, disodium capryloyl glutamate, sodium cocoyl glutamate, and sodium lauroyl glutamate were negative for genotoxicity. Acetyl glutamic acid was negative in an in vivo study.
The analogue chemicals, acyl sarcosines. raised concern about the possible formation of potentially carcinogenic nitrosated derivatives. For the analogue, the reactive material is likely to be the precursor sarcosine. This chemical varies in that the precursor amine glycine is a primary amine, whereas the precursor amine sarcosine in the analogue material is a secondary amine. Secondary amines are of more concern for nitrosamine formation than primary or tertiary amines. Whereas the nitrogen in chemicals fatty acid glycinates and glutamates is secondary, its functional group is an amide rather than an amine and has different chemical properties. Free amine is not present therefore the possibility of nitrosamine formation is considered to be low.

CAPRYLIC ACID DIETHANOLAMIDE

Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure.

Fatty acid diethanolamides (C8-C18) are classified by Comite Europeen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41

Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common.

Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978).

Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in ***Salmonella typhimurium*** strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of ***Salmonella typhimurium*** when tested with or without metabolic activation

Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljoministeriet (Danish Environmental Protection Agency)

For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides)
The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented.
The Fatty nitrogen-derived amides (FND amides) comprise four categories:
Subcategory I: Substituted Amides
Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)
Subcategory III: Imidazole Derivatives
Subcategory IV: FND Amphoterics
Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies.
Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II.
Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories.
Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories.
Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II.
In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole.
Some typical applications of FND Amides are:
masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers.
The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging;

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	irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals. Coconut oil diethanolamine condensate is possibly carcinogenic to humans (IARC Group 2B) In a study of the dermal application in mice, coconut oil diethanolamine condensate increased the incidence of hepatocellular carcinoma and hepatocellular adenoma in males and females, and of hepatoblastoma in males. The incidence of renal tubule adenoma and carcinoma combined was also increased in males. In a study of dermal application in rats, no increase in tumour incidence was observed. Tumours of the kidney and hepatoblastoma are rare spontaneous neoplasms in experimental animals. The amide linkage between diethanolamine and of the fatty acid moiety is resistant to metabolic hydrolysis. The carcinogenic effects of the coconut diethanolamine condensate used in the cancer bioassay may be due to the levels of diethanolamine (18.2%) in the solutions tested. Mechanistic data are very weak to evaluate the carcinogenic potential of coconut oil diethanolamine condensate per se. A test material composed primarily of diethanolamides of coconut oil acids, with unreacted diethanolamine, alkanolamides of unsaturated acids, and amine salts of the acids, was evaluated. The polar nitrosamine, N-nitrosodiethanolamine, was detected at a concentration of 219 ppb Under test conditions, there was no evidence of carcinogenic activity of the test material in male rats administered 50 or 100 mg/kg bw. There was an equivocal evidence of carcinogenic activity in female rats based on a marginal increase in the incidences of renal tubule neoplasms. However, the absence of an increase in neoplasms in the 100 mg/kg group in the presence of increased hyperplasia makes the association with chemical exposure uncertain. Coconut oil fatty acid diethanolamine condensates (Cocamide DEA) have not been experimentally determined to be carcinogenic Metabolic fate: Lauramide diethanolamine, a major component of coconut oil diethanolamine condensate, in the presence of rat liver and kidney microsomes, is rapidly converted into 11 and 12 hydroxy derivatives Toxicity: Both Oral and Dermal LD50s for rats exceeded 2000 mg/kg for cocamide monoisopropanolamide (CAS RN: 1335203-30-9) Repeat dose toxicity: The 28d NOAEL of coconut fatty acid ethanolamide (Comperlan 100) to rat is considered to be 1500 mg/kg bw/d. Groups of 10 male and 10 female rats were orally gavaged with the test substance diluted in olive oil 5 d/wk for 28 d. Clinical signs, bodyweight, hematology, clinical chemistry, urinalysis, gross and microscopic pathology were recorded. Additional groups of 5 male and 5 female rats were kept for a 4 month recovery period. No treatment-related adverse effects were observed at any of the doses A study was conducted to evaluate the subchronic toxic effects of amides, C8 -18 and C18 -unsatd., N, N-bis(hydroxyethyl) when administered by dermal route in B6C3F1 mice. Under the test conditions, the No Observed Adverse Effect Level (NOAEL) of cocamide monoisopropanolamide was considered to be 50 mg/kg body weight. Genetic toxicity: Under the test conditions, test substance was considered to be non-clastogenic in cultured human lymphocytes in vitro. However, the test material may have the potential to disturb mitotic processes and to induce numerical chromosome aberrations.
SODIUM HYDROXIDE & SODIUM XYLENESULFONATE	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.
SODIUM HYDROXIDE & DIETHYLENE GLYCOL MONOBUTYL ETHER	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
SODIUM XYLENESULFONATE & SODIUM LAURYL ETHER SULFATE	No significant acute toxicological data identified in literature search.
SODIUM XYLENESULFONATE & LAUROYLSARCOSINE, SODIUM SALT	Toxicological data are available and well documented for representative toluenesulfonates, xylenesulfonates and cumenesulfonates (including sodium, potassium, ammonium and calcium salts). These data demonstrate that hydrotropes have a low order of acute toxicity by all relevant routes (LC50s range from 100s to 1000s mg/kg), are not genotoxic ***in vitro*** or ***in vivo***, show no evidence of a carcinogenic response (or any other systemic toxicity) in 2-year dermal exposure studies, and failed to induce developmental, teratogenic or fertility (sex organ) effects. Adverse effects after repeated long term dosing of hydrotropes to animals included epidermal hyperplasia at the site of application in dermal studies, and decreased relative spleen weight in females in oral studies. The critical adverse effect and corresponding systemic NOAEL is 763 mg a.i./kg bw based upon decreased relative spleen weight in female rats in a 90-day oral study. The NOAEL for local effects, based on epidermal hyperplasia at the site of application, was 440 mg a.i./kg bw for mice in 90-day dermal studies. Hydrotropes can be classified as a negligible-to-slight irritant to skin and a slight-to-moderate irritant to eyes. The irritation potential of aqueous solutions of hydrotropes depends on concentration, and the irritation is lessened with rinsing. Hydrotropes are not considered to be skin sensitisers. HERA Report (Hydrotropes) September 2005 Hydrotropes in this category were assessed for mutagen/ genotoxic potential in a variety of assays including the mouse micronucleus, Ames, mouse lymphoma, sister chromatid exchange and chromosome aberration assays. No positive results were seen in vitro or in vivo in any of the studies. For both mice and rats exposed dermally for two years, there was no evidence of carcinogenic potential. Examination of the sex organs (such as prostate, testes or ovaries) from animals in 90-day feeding studies and 90-day and two year dermal studies yielded no evidence to suggest that these chemicals have an adverse affect on the reproductive organs.

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

Legend: ✗ ***– Data either not available or does not fill the criteria for classification***
✓ ***– Data available to make classification***

SECTION 12 Ecological information

Continued...

Toxicity

MS TopFoam Power	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

sodium hydroxide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	144-267mg/l	4
	EC50	48h	Crustacea	34.59-47.13mg/l	4
	EC50(ECx)	48h	Crustacea	34.59-47.13mg/l	4

diethylene glycol monobutyl ether	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	1300mg/l	2
	EC50	48h	Crustacea	>100mg/l	1
	EC50	72h	Algae or other aquatic plants	1101mg/l	2
	NOEC(ECx)	96h	Algae or other aquatic plants	>=100mg/l	1
	EC50	96h	Algae or other aquatic plants	>100mg/l	1

sodium xylenesulfonate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	~252mg/l	2
	EC50	48h	Crustacea	>400mg/l	1
	NOEC(ECx)	72h	Algae or other aquatic plants	40mg/l	2
	EC50	96h	Algae or other aquatic plants	>=230mg/l	2

sodium lauryl ether sulfate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	48h	Fish	0.26mg/L	5
	EC50	48h	Crustacea	2.43-4.01mg/l	4

lauroylsarcosine, sodium salt	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	32.1mg/l	2
	EC50	72h	Algae or other aquatic plants	39mg/l	2
	EC50	48h	Crustacea	8.91mg/l	2
	NOEC(ECx)	48h	Crustacea	5mg/l	2

caprylic acid diethanolamide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	~2.4mg/l	2
	NOEC(ECx)	504h	Crustacea	~0.1mg/l	2
	EC50	72h	Algae or other aquatic plants	~2.1mg/l	2
	EC50	48h	Crustacea	~3.2mg/l	2

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.
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
sodium hydroxide	LOW	LOW
diethylene glycol monobutyl ether	LOW	LOW
lauroylsarcosine, sodium salt	LOW	LOW
caprylic acid diethanolamide	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
sodium hydroxide	LOW (LogKOW = -3.8796)
diethylene glycol monobutyl ether	LOW (BCF = 0.46)
lauroylsarcosine, sodium salt	MEDIUM (LogKOW = 4.0996)
caprylic acid diethanolamide	LOW (LogKOW = 0.9206)

Mobility in soil

Ingredient	Mobility
sodium hydroxide	LOW (Log KOC = 14.3)

Continued...

Ingredient	Mobility
diethylene glycol monobutyl ether	LOW (Log KOC = 10)
lauroylsarcosine, sodium salt	LOW (Log KOC = 434.3)
caprylic acid diethanolamide	LOW (Log KOC = 10)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	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	2R

Land transport (ADG)

14.1. **UN number or ID number**	1719
14.2. **UN proper shipping name**	CAUSTIC ALKALI LIQUID, N.O.S. (contains sodium hydroxide)
14.3. **Transport hazard class(es)**	Class: 8 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: 223 274 Limited quantity: 1 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	1719
14.2. **UN proper shipping name**	Caustic alkali liquid, n.o.s. * (contains sodium hydroxide)
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 8 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 8L
14.4. **Packing group**	II
14.5. **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	Special provisions: A3 A803 Cargo Only Packing Instructions: 855 Cargo Only Maximum Qty / Pack: 30 L Passenger and Cargo Packing Instructions: 851 Passenger and Cargo Maximum Qty / Pack: 1 L Passenger and Cargo Limited Quantity Packing Instructions: Y840

Continued...

	Passenger and Cargo Limited Maximum Qty / Pack	0.5 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	1719
14.2. **UN proper shipping name**	CAUSTIC ALKALI LIQUID, N.O.S. (contains sodium hydroxide)
14.3. **Transport hazard class(es)**	IMDG Class: 8 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	II
14.5 **Environmental hazard**	Not Applicable
14.6. **Special precautions for user**	EMS Number: F-A , S-B Special provisions: 274 Limited Quantities: 1 L

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

Not Applicable

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

Product name	Group
sodium hydroxide	Not Available
diethylene glycol monobutyl ether	Not Available
sodium xylenesulfonate	Not Available
sodium lauryl ether sulfate	Not Available
lauroylsarcosine, sodium salt	Not Available
caprylic acid diethanolamide	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium hydroxide	Not Available
diethylene glycol monobutyl ether	Not Available
sodium xylenesulfonate	Not Available
sodium lauryl ether sulfate	Not Available
lauroylsarcosine, sodium salt	Not Available
caprylic acid diethanolamide	Not Available

SECTION 15 Regulatory information

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

sodium hydroxide is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 10 / Appendix C
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

diethylene glycol monobutyl ether is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australian Inventory of Industrial Chemicals (AIIC)

sodium xylenesulfonate is found on the following regulatory lists

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

sodium lauryl ether sulfate is found on the following regulatory lists

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

lauroylsarcosine, sodium salt is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

caprylic acid diethanolamide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

Continued...

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium hydroxide; diethylene glycol monobutyl ether; sodium xylenesulfonate; sodium lauryl ether sulfate; lauroylsarcosine, sodium salt)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	No (caprylic acid diethanolamide)
Philippines - PICCS	No (caprylic acid diethanolamide)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium lauryl ether sulfate; caprylic acid diethanolamide)
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	09/03/2023
Initial Date	09/03/2023

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	09/03/2023	Toxicological information - Acute Health (eye), Toxicological information - Acute Health (skin), Toxicological information - Acute Health (swallowed), Physical and chemical properties - Appearance, Hazards identification - Classification, Ecological Information - Environmental, Exposure controls / personal protection - Exposure Standard, Accidental release measures - Spills (major), Handling and storage - Storage (storage incompatibility), Toxicological information - Toxicity and Irritation (Other)

Other information

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- 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
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration

- 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

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end of SDS