Sunshine Cleanstar Grease Remover
Sunrise Chemical Enterprise

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

Product Identifier

Product name	Sunshine Cleanstar Grease Remover
Chemical Name	Not Applicable
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	Grease removed. Use according to manufacturer's directions.

Details of the supplier of the safety data sheet

Registered company name	**Sunrise Chemical Enterprise**
Address	9002 Tampines Street 93 #03-28 528836 Singapore
Telephone	67862355
Fax	67861612
Website	http://www.sunrisechemical.com.sg
Email	info@sunrisechemical.com.sg

Emergency telephone number

Association / Organisation	**Not Available**
Emergency telephone numbers	**Not Available**
Other emergency telephone numbers	**Not Available**

SECTION 2 Hazards identification

Classification of the substance or mixture

Classification	Skin Corrosion/Irritation Category 2, Skin Sensitizer Category 1, Serious Eye Damage/Eye Irritation Category 1, Carcinogenicity Category 2, Reproductive Toxicity Category 2, Specific target organ toxicity - repeated exposure Category 2

Label elements

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

Hazard statement(s)

H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H351	Suspected of causing cancer.
H361	Suspected of damaging fertility or the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

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Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P310	Immediately call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

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
68891-38-3	1-10	sodium lauryl ether sulfate
9016-45-9	1-10	nonylphenol ethoxylates
111-76-2	1-5	ethylene glycol monobutyl ether
7758-29-4	1-5	sodium tripolyphosphate
1310-73-2	1-5	sodium hydroxide
68603-42-9	1-5	cocamide diethanolamide.
532-32-1	<1	sodium benzoate
Not Available	balance	Ingredients determined not to be hazardous

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Immediately hold eyelids apart and flush the eye continuously with running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▸ Transport to hospital or doctor without delay. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▸ Other measures are usually unnecessary.
Ingestion	▸ 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. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 Firefighting measures

Extinguishing media

The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.
Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.
In such an event consider:

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.

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Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves in the event of a fire. ▸ Prevent, by any means available, spillage from entering drains or water courses. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **DO NOT** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ The material is not readily combustible under normal conditions. ▸ However, it will break down under fire conditions and the organic component may burn. ▸ Not considered to be a significant fire risk. ▸ Heat may cause expansion or decomposition with violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. Decomposes on heating and produces toxic fumes of: carbon dioxide (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) metal oxides other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.

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	▸ 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. Slippery when spilt.
Major Spills	Moderate hazard. ▸ Clear area of personnel and move upwind. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear breathing apparatus plus protective gloves. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services. Slippery when spilt.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ **DO NOT** allow clothing wet with material to stay in contact with skin ▸ Avoid all personal contact, including inhalation. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ Prevent concentration in hollows and sumps. ▸ **DO NOT** enter confined spaces until atmosphere has been checked. ▸ **DO NOT** allow material to contact humans, exposed food or food utensils. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
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.

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	▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	None known

+	x	+	x	+	+	+

X — *Must not be stored together*
0 — *May be stored together with specific preventions*
+ — *May be stored together*

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Singapore Permissible Exposure Limits of Toxic Substances	ethylene glycol monobutyl ether	2-Butoxyethanol (EGBE)	25 ppm / 121 mg/m3	Not Available	Not Available	Not Available
Singapore Permissible Exposure Limits of Toxic Substances	sodium hydroxide	Sodium hydroxide	Not Available	2 mg/m3	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
nonylphenol ethoxylates	43 mg/m3	470 mg/m3	5,400 mg/m3
ethylene glycol monobutyl ether	60 ppm	120 ppm	700 ppm
sodium tripolyphosphate	0.61 mg/m3	6.8 mg/m3	620 mg/m3
sodium hydroxide	Not Available	Not Available	Not Available
sodium benzoate	61 mg/m3	680 mg/m3	810 mg/m3

Ingredient	Original IDLH	Revised IDLH
sodium lauryl ether sulfate	Not Available	Not Available
nonylphenol ethoxylates	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available
sodium tripolyphosphate	Not Available	Not Available
sodium hydroxide	10 mg/m3	Not Available
cocamide diethanolamide.	Not Available	Not Available
sodium benzoate	Not Available	Not Available

Occupational Exposure Banding

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

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.

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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.

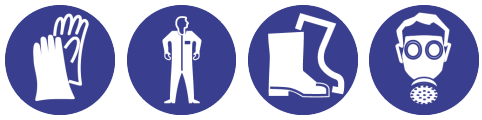
Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at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

Personal protection



Eye and face protection

- Safety glasses with side shields.
- Chemical goggles.
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]

Skin protection

See Hand protection below

Hands/feet protection

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

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

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.
Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:
· frequency and duration of contact,
· chemical resistance of glove material,
· glove thickness and
· dexterity

Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
· When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
· Contaminated gloves should be replaced.

As defined in ASTM F-739-96 in any application, gloves are rated as:
· Excellent when breakthrough time > 480 min
· Good when breakthrough time > 20 min
· Fair when breakthrough time < 20 min
· Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.
It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection 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:

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	· 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. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

Respiratory protection

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Yellow liquid with lemon fragrance; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	10	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting. Not normally a hazard due to non-volatile nature of product
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.

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Skin Contact	This material can cause inflammation of the skin on contact in some persons. The material may accentuate any pre-existing dermatitis condition Non-ionic surfactants cause less irritation than other surfactants as they have less ability to denature protein in the skin. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions 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.
Eye	If applied to the eyes, this material causes severe eye damage. Non-ionic surfactants can cause numbing of the cornea, which masks discomfort normally caused by other agents and leads to corneal injury. Irritation varies depending on the duration of contact, the nature and concentration of the surfactant.
Chronic	There has been concern that this material can cause cancer or mutations, but there is not enough data to make an assessment. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. Skin contact with the material is more likely to cause a sensitisation reaction in some persons compared to the general population. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. This material can cause serious damage if one is exposed to it for long periods. It can be assumed that it contains a substance which can produce severe defects. Ample evidence from experiments exists that there is a suspicion this material directly reduces fertility. Based on experience with animal studies, exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother.

Sunshine Cleanstar Grease Remover	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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]

nonylphenol ethoxylates	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 1851.2 mg/kg[2]	Eye (rabbit): 5 mg SEVERE
	Oral(Rat) LD50; 1310 mg/kg[2]	Skin (human): 15 mg/3D mild

ethylene glycol monobutyl ether	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: 667 mg/kg[1]	Eye (rabbit): 100 mg SEVERE
	Inhalation(Rat) LC50; 2.21 mg/l4h[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral(Guinea) LD50; 1414 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
		Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating)[1]
		Skin: no adverse effect observed (not irritating)[1]

sodium tripolyphosphate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >3160 mg/kg[2]	Not Available
	Inhalation(Rat) LC50; >0.39 mg/l4h[1]	
	Oral(Rat) LD50; >2000 mg/kg[1]	

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]

cocamide diethanolamide.	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available
	Oral(Rat) LD50; >2000 mg/kg[1]	

sodium benzoate	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Not Available
	Inhalation(Rat) LC50; >12.2 mg/L4h[1]	
	Oral(Mouse) LD50; 1600 mg/kg[2]	

Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*

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SODIUM LAURYL ETHER SULFATE	* [CESIO] No significant acute toxicological data identified in literature search. Alcohol ethoxysulfates (AES) are of low acute toxicity. Neat AES are irritant to the skin and eyes. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
NONYLPHENOL ETHOXYLATES	For nonylphenol and its compounds: Alkylphenols like nonylphenol and bisphenol A have estrogenic effects in the body. They are known as xenoestrogens. Estrogenic substances and other endocrine disruptors are compounds that have hormone-like effects in both wildlife and humans. Xenoestrogens usually function by binding to estrogen receptors and acting competitively against natural estrogens. Nonylphenol has been found to act as an agonist of GPER (G protein-coupled estrogen receptor),. Nonylphenol has been shown to mimic the natural hormone 17beta-estradiol, and it competes with the endogeous hormone for binding with the estrogen receptors ERalpha and ERbeta. Effects in pregnant women. Subcutaneous injections of nonylphenol in late pregnancy causes the expression of certain placental and uterine proteins, namely CaBP-9k, which suggest it can be transferred through the placenta to the fetus. It has also been shown to have a higher potency on the first trimester placenta than the endogenous estrogen 17beta-estradiol. In addition, early prenatal exposure to low doses of nonylphenol cause an increase in apoptosis (programmed cell death) in placental cells. These “low doses” ranged from 10-13-10-9 M, which is lower than what is generally found in the environment. Nonylphenol has also been shown to affect cytokine signaling molecule secretions in the human placenta. In vitro cell cultures of human placenta during the first trimester were treated with nonylphenol, which increase the secretion of cytokines including interferon gamma, interleukin 4, and interleukin 10, and reduced the secretion of tumor necrosis factor alpha. This unbalanced cytokine profile at this part of pregnancy has been documented to result in implantation failure, pregnancy loss, and other complications. Effects on metabolism Nonylphenol has been shown to act as an obesity enhancing chemical or obesogen, though it has paradoxically been shown to have anti-obesity properties. Growing embryos and newborns are particularly vulnerable when exposed to nonylphenol because low-doses can disrupt sensitive processes that occur during these important developmental periods. Prenatal and perinatal exposure to nonylphenol has been linked with developmental abnormalities in adipose tissue and therefore in metabolic hormone synthesis and release. Specifically, by acting as an estrogen mimic, nonylphenol has generally been shown to interfere with hypothalamic appetite control. The hypothalamus responds to the hormone leptin, which signals the feeling of fullness after eating, and nonylphenol has been shown to both increase and decrease eating behavior by interfering with leptin signaling in the midbrain. Nonylphenol has been shown mimic the action of leptin on neuropeptide Y and anorectic POMC neurons, which has an anti-obesity effect by decreasing eating behavior. This was seen when estrogen or estrogen mimics were injected into the ventromedial hypothalamus. On the other hand, nonylphenol has been shown to increase food intake and have obesity enhancing properties by lowering the expression of these anorexigenic neurons in the brain. Additionally, nonylphenol affects the expression of ghrelin: an enzyme produced by the stomach that stimulates appetite. Ghrelin expression is positively regulated by estrogen signaling in the stomach, and it is also important in guiding the differentiation of stem cells into adipocytes (fat cells). Thus, acting as an estrogen mimic, prenatal and perinatal exposure to nonylphenol has been shown to increase appetite and encourage the body to store fat later in life. Finally, long-term exposure to nonylphenol has been shown to affect insulin signaling in the liver of adult male rats. Cancer Nonylphenol exposure has also been associated with breast cancer. It has been shown to promote the proliferation of breast cancer cells, due to its agonistic activity on ERalpha (estrogen receptor alpha) in estrogen-dependent and estrogen-independent breast cancer cells. Some argue that nonylphenol's suggested estrogenic effect coupled with its widespread human exposure could potentially influence hormone-dependent breast cancer disease Humans have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents and other cleaning products. Exposure to these chemicals can occur through swallowing, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that relatively high volumes would have to occur to produce any toxic response. No death due to poisoning with alcohol ethoxylates has ever been reported. Studies show that alcohol ethoxylates have low toxicity through swallowing and skin contact. Animal studies show these chemicals may produce gastrointestinal irritation, stomach ulcers, hair standing up, diarrhea and lethargy. Slight to severe irritation occurred when undiluted alcohol ethyoxylates were applied to the skin and eyes of animals. These chemicals show no indication of genetic toxicity or potential to cause mutations and cancers. Toxicity is thought to be substantially lower than that of nonylphenol ethoxylates. Some of the oxidation products of this group of substances may have sensitizing properties. As they cause less irritation, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their tendency to auto-oxidise also increases their irritation. Due to their irritating effect it is difficult to diagnose allergic contact dermatitis (ACD) by patch testing. Both laboratory and animal testing has shown that there is no evidence for alcohol ethoxylates (AEs) causing genetic damage, mutations or cancer. No adverse reproductive or developmental effects were observed. Tri-ethylene glycol ethers undergo enzymatic oxidation to toxic alkoxy acids. They may irritate the skin and the eyes. At high oral doses, they may cause depressed reflexes, flaccid muscle tone, breathing difficulty and coma. Death may result in experimental animal. However, repeated exposure may cause dose dependent damage to the kidneys as well as reproductive and developmental defects. For nonylphenol: Animal testing suggests that repeated exposure to nonylphenol may cause liver changes and kidney dysfunction. Nonylphenol was not found to cause mutations or chromosomal aberrations. Oral (rat) TDLo: 150 mg/kg/3D-I Skin (rabbit): 500 mg mild
ETHYLENE GLYCOL MONOBUTYL ETHER	NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers. **Acute Toxicity:** Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats. **Repeat dose toxicity:** The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*. **Mutagenicity:** In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without

Continued...

	metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic. **Carcinogenicity:** In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity **Reproductive and developmental toxicity**. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE). Animal testing showed that exposure to ethylene glycol monobutyl ether resulted in toxicity to both the mother and the embryo. Reproductive effects were thought to be less than that of other monoalkyl ethers of ethylene glycol. Chronic exposure may cause anaemia, with enlargement and fragility of red blood cells. It is thought that in animals butoxyethanol may cause generalized clotting and bone infarction. In animals, 2-butoxyethanol also increased the rate of some cancers, including liver cancer. For ethylene glycol: Ethylene glycol is quickly and extensively absorbed throughout the gastrointestinal tract. Limited information suggests that it is also absorbed through the airways; absorption through skin is apparently slow. Following absorption, it is distributed throughout the body. In humans, it is initially metabolized by alcohol dehydrogenase to form glycoaldehyde, which is rapidly converted to glycolic acid and glyoxal. These breakdown products are oxidized to glyoxylate, which may be further metabolized to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate carbon dioxide, which is one of the major elimination products of ethylene glycol. In addition to exhaled carbon dioxide, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination is rapid and occurs within a few hours. Respiratory effects: Respiratory system involvement occurs 12-24 hours after swallowing sufficient amounts of ethylene glycol. Symptoms include hyperventilation, shallow rapid breathing, and generalized swelling of the lungs with calcium oxalate deposits occasionally appearing in the lungs. Respiratory system involvement appears to be dose-dependent and occurs at the same time as cardiovascular changes. Later, there may be other changes compatible with adult respiratory distress syndrome (ARDS). Swelling of the lung can be a result of heart failure, ARDS, or aspiration of stomach contents. Symptoms related to acidosis such as fast or excessive breathing are frequently observed; however, major symptoms such as swelling of the lung and inflammation of the bronchi and lungs are relatively rare, and are usually seen only in extreme poisoning. Cardiovascular effects: Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of ethylene glycol poisoning by swallowing, which is 12-24 hours after acute exposure. The symptoms of poisoning involving the heart include increased heart rate, heart enlargement and ventricular gallop. There may also be high or low blood pressure, which may progress to cardiogenic shock. In lethal cases, inflammation of the heart muscle has been observed at autopsy. Cardiovascular involvement appears to be rare and usually seen after swallowing higher doses of ethylene glycol. In summary, acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown. Gastrointestinal effects: Common early acute effects of swallowing ethylene glycol include nausea, vomiting with or without blood, heartburn and abdominal cramping and pain. One patient showed intermittent diarrhea and pain, and after surgery, deposition of oxalate crystals was shown to have occurred. Musculoskeletal effects: Reported musculoskeletal effects in cases of acute ethylene glycol poisoning include diffuse muscle tenderness and pain, associated with high levels of creatinine in the blood, and jerks and contractions associated with low calcium. Liver effects: Autopsies carried out on people who died following acute ethylene glycol poisoning showed deposition of calcium oxalate in the liver as well as hydropic and fatty degeneration and cell death (necrosis) of the liver. Kidney effects: Adverse kidney effects are seen during the third stage of ethylene glycol poisoning, 2-3 days after acute exposure. Calcium oxalate crystals are deposited in the tubules and are seen in the urine. There may also be degeneration and death of tubule cells, and inflammation of the tubule interstitium. If untreated, the degree of kidney damage progresses and leads to blood and protein in the urine, decreased kidney function, reduction in urine output and ultimately, kidney failure. With adequate supportive therapy, kidney function can return to normal or near normal. Metabolic effects: Metabolic changes can occur within 12 hours of exposure to ethylene glycol. There may be metabolic acidosis, caused by accumulation of glycolic acid in the blood and therefore a reduction in blood pH. The anion gap is increased, due to increased unmeasured anions (mainly glycolate). Effects on the nervous system: Adverse reactions involving the nervous system are among the first symptoms to appear in humans after ethylene glycol is swallowed. These early effects are also the only symptoms caused by unmetabolised ethylene glycol. Together with metabolic effects (see above), they occur from 0.5-12 hours after exposure and are considered to be part of the first stage in ethylene glycol poisoning. Inco-ordination, slurred speech, confusion and sleepiness are common in the early stages, as are irritation, restlessness and disorientation. Later, there may be effects on cranial nerves (which may be reversible over many months). Swelling of the brain (cerebrum) and crystal deposits of calcium oxalate in the walls of the small blood vessels of the brain were found at autopsy in people who died after acute ethylene glycol poisoning. Reproductive effects: Animal testing showed that ethylene glycol may affect fertility, survival of fetuses and the male reproductive organs. Effects on development: Animal studies indicate that birth defects may occur after exposure in pregnancy; there may also be reduction in foetal weight. Cancer: No studies are known regarding cancer effects in humans or animal, after skin exposure to ethylene glycol. Genetic toxicity: No human studies available, but animal testing results are consistently negative.
SODIUM HYDROXIDE	The material may cause severe skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin. Repeated exposures may produce severe ulceration.
COCAMIDE DIETHANOLAMIDE.	*Stephan SDS Ninol 49-CE Laboratory testing shows that the fatty acid amide, cocoamide DEA, causes occupational allergic contact dermatitis, and that allergy to this substance is becoming more common. Alkanolamides are manufactured by condensation of diethanolamine and the methyl ester of long chain fatty acids. The chemicals in the Fatty Nitrogen Derived (FND) Amides are generally similar in terms of physical and chemical properties, environmental fate and toxicity. Its low acute oral toxicity is well established across all subcategories by the available data and show no apparent organ specific toxicity, mutation, reproductive or developmental defects. 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.

Continued...

	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. DEA has low acute toxicity if ingested orally or applied on the skin. It can cause moderate skin irritation and severe eye irritation. It may affect sperm production, cause anaemia and damage the liver and kidney. It has not been shown to cause cancer in humans; though there is evidence that it may cause cancer in mice, and damage to the foetus at levels toxic to the mother. **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
SODIUM BENZOATE	NOTE: Oral doses of 8-10g may cause nausea and vomiting, though tolerance in human is 50 g/day. Use in food limited to 0.1%. [ICI] For benzoates: Benzyl alcohol, benzoic acid and its sodium and potassium salt have a common metabolic and excretion pathway. All but benzyl alcohol are considered to be unharmful and of low acute toxicity. They may cause slight irritation by oral, dermal or inhalation exposure except sodium benzoate which doesn't irritate the skin. Studies showed increased mortality, reduced weight gain, liver and kidney effects at higher doses, also, lesions of the brains, thymus and skeletal muscles may occur with benzyl alcohol. However, they do not cause cancer, genetic or reproductive toxicity. Developmental toxicity may occur but only at maternal toxic level.
SODIUM LAURYL ETHER SULFATE & NONYLPHENOL ETHOXYLATES	Polyethers (such as ethoxylated surfactants and polyethylene glycols) are highly susceptible to being oxidized in the air. They then form complex mixtures of oxidation products. Animal testing reveals that whole the pure, non-oxidised surfactant is non-sensitizing, many of the oxidation products are sensitisers. The oxidization products also cause irritation.
NONYLPHENOL ETHOXYLATES & SODIUM TRIPOLYPHOSPHATE & SODIUM HYDROXIDE & COCAMIDE DIETHANOLAMIDE.	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.
ETHYLENE GLYCOL MONOBUTYL ETHER & SODIUM HYDROXIDE & COCAMIDE DIETHANOLAMIDE.	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLENE GLYCOL MONOBUTYL ETHER & COCAMIDE DIETHANOLAMIDE.	The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
COCAMIDE DIETHANOLAMIDE. & SODIUM BENZOATE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

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

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

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species	Value	Source
Sunshine Cleanstar Grease Remover	Not Available	Not Available	Not Available	Not Available	Not Available

Continued...

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
nonylphenol ethoxylates	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	BCF	1008h	Fish	<0.2	7
	EC50(ECx)	120h	Crustacea	0.08-0.29mg/l	4
	EC50	96h	Algae or other aquatic plants	12mg/l	4
	EC50	48h	Crustacea	13-16mg/l	4
ethylene glycol monobutyl ether	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96h	Fish	1250mg/l	2
	EC50	72h	Algae or other aquatic plants	623mg/l	2
	EC50	48h	Crustacea	164mg/l	2
	EC10(ECx)	48h	Crustacea	7.2mg/l	2
	EC50	96h	Algae or other aquatic plants	720mg/l	2
sodium tripolyphosphate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50(ECx)	96h	Algae or other aquatic plants	69.2mg/l	2
	EC50	48h	Crustacea	>70.7<101.3mg/l	2
	EC50	96h	Algae or other aquatic plants	69.2mg/l	2
sodium hydroxide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	34.59-47.13mg/l	4
	LC50	96h	Fish	144-267mg/l	4
	EC50(ECx)	48h	Crustacea	34.59-47.13mg/l	4
cocamide diethanolamide.	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	504h	Crustacea	0.07mg/l	2
	LC50	96h	Fish	2.4mg/l	2
	EC50	48h	Crustacea	~3.2mg/l	2
sodium benzoate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	NOEC(ECx)	72h	Algae or other aquatic plants	0.09mg/l	2
	EC50	72h	Algae or other aquatic plants	>30.5mg/l	2
	LC50	96h	Fish	>100mg/l	2
	EC50	48h	Crustacea	<650mg/l	1
Legend:	*Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*				

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
nonylphenol ethoxylates	LOW (BCF = 1.4)
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
sodium hydroxide	LOW (LogKOW = -3.8796)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
sodium hydroxide	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

Continued...

Product / Packaging disposal	**DO NOT** allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible. Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. Dispose of by: burial in a land-fill specifically 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

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

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

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

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

Not Applicable

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

Product name	Group
sodium lauryl ether sulfate	Not Available
nonylphenol ethoxylates	Not Available
ethylene glycol monobutyl ether	Not Available
sodium tripolyphosphate	Not Available
sodium hydroxide	Not Available
cocamide diethanolamide.	Not Available
sodium benzoate	Not Available

Transport in bulk in accordance with the ICG Code

Product name	Ship Type
sodium lauryl ether sulfate	Not Available
nonylphenol ethoxylates	Not Available
ethylene glycol monobutyl ether	Not Available
sodium tripolyphosphate	Not Available
sodium hydroxide	Not Available
cocamide diethanolamide.	Not Available
sodium benzoate	Not Available

SECTION 15 Regulatory information

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

sodium lauryl ether sulfate is found on the following regulatory lists

Not Applicable

nonylphenol ethoxylates is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

ethylene glycol monobutyl ether is found on the following regulatory lists

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

Singapore Permissible Exposure Limits of Toxic Substances

sodium tripolyphosphate is found on the following regulatory lists

Not Applicable

sodium hydroxide is found on the following regulatory lists

Singapore Permissible Exposure Limits of Toxic Substances

cocamide diethanolamide. is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List

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

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B: Possibly carcinogenic to humans

sodium benzoate is found on the following regulatory lists

Not Applicable

National Inventory Status

Continued...

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium lauryl ether sulfate; nonylphenol ethoxylates; ethylene glycol monobutyl ether; sodium tripolyphosphate; sodium hydroxide; cocamide diethanolamide.; sodium benzoate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium lauryl ether sulfate)
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 and are not exempt from listing(see specific ingredients in brackets)*

SECTION 16 Other information

Revision Date	22/07/2021
Initial Date	22/07/2021

Other information

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.

end of SDS