MICROSHIELD HANDWASH

Schulke Australia Pty Ltd

Chemwatch Hazard Alert Code: **1**

Chemwatch: **60-3463**
Version No: **8.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Issue Date: **31/08/2023**
Print Date: **14/11/2024**
L.GHS.AUS.EN.E

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

Product Identifier

Product name	MICROSHIELD HANDWASH
Chemical Name	Not Applicable
Synonyms	schulke codes: 70000373, 70000362, 70000348
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	Liquid hand, face and body washing. SDS are intended for use in the workplace ONLY. For domestic-use products, refer to consumer labels.

Details of the manufacturer or supplier of the safety data sheet

Registered company name	**Schulke Australia Pty Ltd**
Address	2-4 Lyonpark Road Macquarie Park NSW 2113 Australia
Telephone	+61 2 8875 9300
Fax	+61 2 8875 9301
Website	www.schuelke.com.au
Email	customerservice.au@schuelke.com

Emergency telephone number

Association / Organisation	**Poisons information Centre**
Emergency telephone number(s)	**13 11 26**
Other emergency telephone number(s)	**Not Available**

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Non hazardous
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)	Not Applicable
Signal word	**Not Applicable**

Hazard statement(s)

Not Applicable

Precautionary statement(s) Prevention

Not Applicable

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

SECTION 3 Composition / information on ingredients

Substances

Page 1 continued...

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
9004-82-4	0-10	sodium lauryl ether sulfate
68603-42-9	0-10	coconut diethanolamide
7647-14-5	0-10	sodium chloride
111-60-4	0-10	ethylene glycol monostearate
78491-02-8	0-10	diazolidinyl urea
99-76-3	0-10	methyl paraben
94-13-3	0-10	propyl paraben
Not Available	0-10	citric acid monohydrate for pH adjustment
7732-18-5	>30	water
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: ▸ Wash out immediately with fresh 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. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	No adverse effects anticipated from normal use. If skin or hair contact occurs: ▸ 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

▸ There is no restriction on the type of extinguisher which may be used.
▸ Use extinguishing media suitable for surrounding area.

Special hazards arising from the substrate or mixture

Fire Incompatibility	Avoid contamination with strong oxidising agents as ignition may result

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	▸ Non combustible. ▸ Not considered to be a significant fire risk. ▸ Expansion or decomposition on heating may lead to violent rupture of containers. ▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. Other decomposition products include: carbon dioxide (CO_2) nitrogen oxides (NOx)
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

Continued...

See section 12

Methods and material for containment and cleaning up

Minor Spills	Slippery when spilt. Clean up all spills immediately. Wipe up. Place in clean drum then flush area with water.
Major Spills	Slippery when spilt. Minor hazard. ▸ Clear area of personnel. ▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Control personal contact with the substance, by using protective equipment as required. ▸ Prevent spillage from entering drains or water ways. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal. ▸ Wash area and prevent runoff into drains or waterways. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	▸ Limit all unnecessary personal contact. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ When handling **DO NOT** eat, drink or smoke. ▸ Always wash hands with soap and water after handling. ▸ Avoid physical damage to containers. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.
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. Keep cool. Store below 25 deg.C

Conditions for safe storage, including any incompatibilities

Suitable container	Plastic container ▸ Packaging as recommended by manufacturer.
Storage incompatibility	Avoid storage with oxidisers

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	ethylene glycol monostearate	Stearates	10 mg/m3	Not Available	Not Available	(a) This value is for inhalable dust containing no asbestos and < 1% crystalline silica.

Ingredient	Original IDLH	Revised IDLH
sodium lauryl ether sulfate	Not Available	Not Available
coconut diethanolamide	Not Available	Not Available
sodium chloride	Not Available	Not Available
ethylene glycol monostearate	Not Available	Not Available
diazolidinyl urea	Not Available	Not Available
methyl paraben	Not Available	Not Available
propyl paraben	Not Available	Not Available
water	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
coconut diethanolamide	E	≤ 0.1 ppm
sodium chloride	E	≤ 0.01 mg/m³
diazolidinyl urea	D	> 0.01 to ≤ 0.1 mg/m³
methyl paraben	E	≤ 0.01 mg/m³
propyl paraben	E	≤ 0.01 mg/m³
Notes:	***Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.***	

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MATERIAL DATA

None assigned. Refer to individual constituents.

Exposure controls

Appropriate engineering controls	None under normal operating conditions. Provide adequate ventilation in warehouse or closed storage areas.
Individual protection measures, such as personal protective equipment	
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. OTHERWISE: ▸ Safety glasses with side shields. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	▸ Bare skin is cleaned with this material. ▸ Application of hand cream / barrier cream after use is recommended.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities **OTHERWISE:** ▸ Overalls ▸ Eyewash unit.

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:
MICROSHIELD HANDWASH

Material	CPI
BUTYL	A
NEOPRENE	A
VITON	A
NATURAL RUBBER	C
PVA	C

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

Respiratory protection

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face
A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Milky, viscous liquid; miscible in water.

Physical state	Liquid	**Relative density (Water = 1)**	1.015
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	6.7-7.5	**Decomposition temperature (°C)**	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

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Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	Not normally a hazard due to non-volatile nature of product
Ingestion	Considered to be non toxic Ingestion may result in nausea, abdominal irritation, pain and vomiting
Skin Contact	Not considered to cause discomfort through normal use. Discontinue use if irritation occurs
Eye	The liquid may produce eye discomfort causing transient smarting, blinking
Chronic	No adverse effects anticipated from normal use. Principal hazards are accidental eye contact and cleaner overuse. Overuse or obsessive cleaner use may lead to defatting of the skin and may cause irritation, drying, cracking, leading to dermatitis.

MICROSHIELD HANDWASH	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
sodium lauryl ether sulfate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 1600 mg/kg[2]	Eye (Rodent - rabbit): 100uL/24H - Severe
		Eye (Rodent - rabbit): 20mg/24H - Moderate
		Skin (Rodent - guinea pig): 5%/9H (intermittent)
		Skin (Rodent - rabbit): 25mg/24H - Moderate
		Skin (Rodent - rabbit): 500mg/24H - Severe
coconut diethanolamide	**TOXICITY**	**IRRITATION**
	Inhalation (Rat) LC50: 44 ppm4h[2]	Eye (Rodent - rabbit): 100uL - Severe
	Oral (Rat) LD50: 2700 mg/kg[2]	Skin (Rodent - rabbit): 300uL - Moderate
sodium chloride	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >10000 mg/kg[1]	Eye (Rodent - rabbit): 100mg/24H - Moderate
	Inhalation (Rat) LC50: >10.5 mg/l4h[1]	Eye (Rodent - rabbit): 10mg - Moderate
	Oral (Rat) LD50: 3000 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
		Skin (Rodent - rabbit): 500mg/24H - Mild
		Skin: no adverse effect observed (not irritating)[1]
ethylene glycol monostearate	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: 12100 mg/kg[2]	Skin (Rodent - rabbit): 500mg/24H - Mild
diazolidinyl urea	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irritating)[1]
	Oral (Rat) LD50: >2000 mg/kg[2]	Skin: no adverse effect observed (not irritating)[1]
methyl paraben	**TOXICITY**	**IRRITATION**
	Oral (Mouse) LD50; 2100 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human): 10%/5D(intermittent)
		Skin (Rodent - rabbit): 0.1mL/24H - Mild
		Skin (Rodent - rabbit): 0.5mL/21D (intermittent) - Moderate

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		Skin: no adverse effect observed (not irritating)[1]
propyl paraben	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >5000 mg/kg[1]	Eye: no adverse effect observed (not irritating)[1]
		Skin (Human - man): 15%/2D
		Skin: no adverse effect observed (not irritating)[1]
water	**TOXICITY**	**IRRITATION**
	Oral (Rat) LD50: >90000 mg/kg[2]	Not Available

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 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
https://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 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

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	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 https://journals.sagepub.com/doi/pdf/10.1177/1091581810373151
COCONUT DIETHANOLAMIDE	*Ethoquad C/12 SDS In a study of dermal application in mice, coconut oil diethanolamine condensate (coconut diethanolamide) 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 carcinogenic effects of the coconut oil 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 According to IARC: Coconut oil diethanolamine condensate is possibly carcinogenic to humans (Group 2B) 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.

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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; 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.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
for diethanolamine (DEA):
In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.
Subchronic toxicity: The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.
Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL =32 mg/g) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone marrow. Haematological parameters were not evaluated in mice.
Developmental toxicity: In a developmental toxicity study conducted via the oral route, effects of concern were observed only in the presence of maternal toxicity. In a developmental toxicity study conducted via the dermal route using two species of mammals, developmental toxicity was observed only in one species and only at doses causing significant maternal toxicity. Metabolically, DEA is excreted largely unchanged in the urine.
Carcinogenicity: A two-year dermal cancer study bioassay results on DEA and three fatty acid condensates of DEA indicated that liver tumours occurred in male and female mice exposed to DEA and two of the condensates. In addition kidney tumours occurred in male mice exposed to DEA and one of the condensates. Compelling evidence suggested that the toxicity observed in mice and rats treated with the DEA condensates was associated with free DEA and not with other components of the condensates. A weight of evidence analysis of data relevant to the assessment of the liver and kidney tumours in mice resulted in the conclusion that these tumours are not relevant to humans under the expected conditions of exposure and that liver and kidney toxicity should be evaluated on a threshold basis. This conclusion is based on the following:

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- DEA is not genotoxic
- tumour development occurred at doses also associated with chronic hyperplasia
- there was no dose-related increase in malignancy, multiplicity of tumours or decrease in latency period
- tumours occurred late in life
- tumour response was species-specific (only mice were affected, not rats)
- tumour response was sex-specific (only male mice were affected, not females)
- tumour development was site-specific, with only liver and kidney affected, both sites of DEA accumulation;
- there was no tumour response in skin, despite evidence of chronic dermal toxicity
- there is a plausible mechanism, supported by various data, to explain the renal toxicity of DEA
- data support threshold mechanisms of renal carcinogenesis for a number of non-genotoxic chemicals
- the exposure regime used in the mouse study (***i.e.***, lifetime continuous exposure to DEA in ethanol vehicle at doses causing chronic dermal toxicity) is not relevant to human exposure (exposure through cosmetic vehicles with daily removal, under non-irritating conditions).

In considering the aggregate data on a DEA basis from the four studies using DEA and related condensates, the NOEL for kidney toxicity was 19 mg/kg/d, which resulted from a dose of 100 mg/kg/d of cocamide DEA containing 19% free DEA.
Anaemia: Rats exposed to DEA condensates developed anaemia. This was considered to be of to be relevant for humans since anaemia in rodents and humans share common etiologies. The proposed mechanism by which DEA could cause anemia involves disruption of phospholipid metabolism leading to membrane perturbation and functional change to erythrocytes. Some doubt about the relevance of the findings arises because ethanol was used as the vehicle in the dermal studies, and ethanol is known to cause anaemia in rodents through a mechanism involving membrane disruption. The possibility of a synergistic or additive role for DEA and ethanol in combination cannot be ruled out.
In considering the aggregate data on a DEA basis from the four 13-week dermal studies using DEA and related condensates, the NOEL for microcytic anemia was 9.5 mg/kg/d, which resulted from a dose of 50 mg/kg/d of cocamide DEA containing 19% free DEA.
The NOELs for mice and rats derived in this hazard assessment were as follows:
Anaemia in rats: 9.5 mg/kg/d (based on microcytic anemia)
Organ toxicity in mice: 2.2 mg/kg/d (based on liver toxicity)
In extrapolating among species for the purposes of risk assessment, the prime consideration with respect to dermally applied DEA was differential dermal absorption. Evidence indicates that dermal penetration of
DEA is greatest in mice and lower in rats and humans. Interspecies extrapolation was accomplished in this assessment by converting applied doses to bioavailable doses (***i.e.***, internal doses) using dermal bioavailability determined in studies with rats and mice ***in vivo***, so as to be able to compare these with internal doses expected to be experienced by humans through use of personal care products.
Based on measured bioavailability in mice and rats, the bioavailable NOELs corresponding to the foregoing were:
Anaemia in rats: 0.8 mg/kg/d (based on microcytic anemia)
Organ toxicity in mice: 0.55 mg/kg/d (based on liver toxicity)
Kidney toxicity: Effects on the kidney were observed in rats treated with DEA in drinking water or by dermal exposure after as little as 2 weeks of exposure. Effects included renal tubule hyperplasia, renal tubular epithelial necrosis, renal tubule mineralization and increased relative organ weight. Similar changes were observed after 13 weeks of exposure of rats to DEA in drinking water and by dermal administration. The NOEL in male rats was 250 mg/kg/d in the dermal study, while in female rats renal tubule mineralisation was observed at the lowest dose of 32 mg/kg/d. After 2 years of dermal exposure there were no histopathological changes in the kidneys of male rats given doses of up to 64 mg/kg/d. In females, there were no significant increases in the incidences of renal tubule epithelial necrosis, hyperplasia or mineralisation as was observed after 13 weeks of exposure, however, there was an increase in the severity and incidence of nephropathy. This was the result of a treatment-related exacerbation of a previously existing lesion, since the incidence in controls was 80%, increasing to 94-96% in treated groups. There was no significant increase in the incidence of kidney tumours in rats treated with DEA or any of the condensates in 2-year dermal studies.
Liver toxicity: Effects on liver, including increases in relative organ weight and histopathological changes were observed in male and female mice in the 2 week drinking water study with DEA. Increases in liver weight were observed in the two week dermal study, but were not associated with histopathological changes. After 13 weeks of exposure, relative liver weights were increased compared to controls in male and female rats, with no associated histopathology. There is some doubt about whether these changes in liver weights were of toxicological significance, since there was no associated histopathology, the dose-response was not consistent and there were no effects on liver in the 2 year study in rats.
In the study with coconut diethanolamide (CDEA) (100 and 200 mg/kg/d) in which 19% of the applied dose was DEA, there were no liver effects in rats after 13 weeks or 2 years of dermal exposure. No liver toxicity in rats was observed in the 2 year dermal studies of lauramide or oleamide DEA

WARNING: This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.

ETHYLENE GLYCOL MONOSTEARATE

Non-comedogenic * [Manufacturer]
For glycol and diol aliphatic esters:(group C)
According to a classification scheme described by the American Chemistry Council' Aliphatic Esters Panel, Group C substances are comprised of a monocarboxylic acid (generally natural fatty acids, e.g., oleic, stearic, C6-C10 fatty acids) and a dihydroxy alcohol (glycol or diol such as ethylene glycol, polyethylene glycol, propylene glycol, 2,2-dimethyl-1,3-propanediol). These esters are often referred to as "glycol or diol esters" or as "alkylidene or alkanediyl esters".
The rationale for grouping the glycol or diol esters is that they represent structurally similar ethylene/ propylene glycol esters in which the hydroxyl groups in the glycol are functionalised with fatty acids as ester derivatives. Esterification of the glycol with fatty acids such as stearic and oleic acid can provide glycol diesters in the 38 to 41 carbon number range, which typically make them relatively non-volatile and high boiling liquids with limited water solubility and with sufficient polar characteristics to make them useful as lubricants and solvents. In the case of the tri- and tetraethylene glycol diesters, the ether linkage in the polyalkylene portion of the glycol also imparts additional polar character to these glycol esters.
Metabolism of these glycol esters in animals would be expected to occur initially via enzymatic hydrolysis leading to the corresponding free fatty acids and free glycol alcohols (e.g., ethylene glycol, propylene glycol, 2,2-dimethyl-1,3-propanediol, polyethylene glycol). These free fatty acids and glycols can be further metabolised or conjugated (e.g., glucuronides, sulfates, etc.) to polar products that are excreted in the urine. The fatty acids, especially the natural occurring ones such as stearic and oleic acids, have low degrees of toxicity . The toxicity of the alkylidene or alkanediyl glycols has been extensively reviewed, especially in case of ethylene glycol and propylene glycol
Acute toxicity: Overall, the acute oral LD50 values for these substances is greater than the 2000 mg/kg, indicating a very low order of toxicity for the glycol esters. Acute dermal toxicity studies have also been carried out and reported for the various propylene glycol fatty acid esters and polyethylene glycol fatty acid esters, particularly those used in cosmetic applications . Overall, the glycol fatty acids exhibit very low degrees of acute oral and dermal toxicity.
Repeat dose toxicity: Studies have also been carried out for various propylene glycol fatty acid esters and polyethylene glycol esters . Data suggests that members of the glycol esters category would be expected to exhibit a low order of toxicity following repeated oral administration. Additional support data that glycol esters are likely to have low orders of repeated-dose toxicity are based on a number of feeding studies conducted in rats, dogs, mice, rabbits and monkeys for PEG-8 stearate .An expert panel has reviewed these studies and has reported that polyethylene glycol-8 stearate (PEG-8 stearate) produced no significant changes in growth mortality rates, histopathological observations or haematology values in long-term feeding studies in rats (i.e., 8-week feeding study at 2% in diet; 9-week feeding study at 4% in diet and 2-year 3-generation feeding studies at 4% in the diet) Repeated-dose toxicity studies carried out with PEG-40 stearate and PEG-100 stearate also have been reported to demonstrate low degrees of toxicity
Reproductive toxicity: Although no adequate reproductive toxicity studies were located on members of the glycol esters category, numerous regulatory bodies have determined that these substances do not pose a reproductive hazard. These hazard and/or risk assessments are based on the fact that glycol esters would be metabolised (hydrolysed) in vivo to the corresponding fatty acids and free glycol alcohols (e.g., ethylene glycol, propylene glycol) [WHO (2003)]. The free fatty acids and glycols can undergo further metabolism or conjugation to polar products that are either excreted or can be used as nutrients. In most cases, the parent fatty acids derived from the glycol esters are comprised of natural fatty acids that are typical of those (e.g., oleic, stearic acid) found in edible oils and fats.
Additional supporting data that glycol esters are unlikely to be reproductive toxicants are based on a multiple generation feeding of PEG-8 stearate. Animals receiving 4% PEG-8 stearate in their diet for three successive generations did not affect growth or fecundity. In another three-generation study in rats receiving diets containing 5%, 10%, or 20% PEG-8 stearate, reproduction and lactation responses were no

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	different from controls at the 5% dose level. Newborn litter survival times were diminished most likely due to maternal neglect at the 10% and 20% dose levels. The overall level of reproductive performance (e.g., greater mortality rate of nurslings, impairment of lactation efficiency) was lower in animals fed the 20% PEG-8 stearate diet Results from these studies showed a low order of reproductive/developmental toxicity. PEG stearates (including PEG-8 stearate) have been approved by the FDA for use in the bakery and pharmaceutical industries. Although adequate reproductive and developmental studies have not been reported for ethylene glycol stearates or other ethylene glycol fatty acid esters, numerous studies have been conducted to evaluate reproductive and developmental effects of the parent glycol alcohol, namely, ethylene glycol (EG). EG itself is considered to have a relatively low order of toxicity; however, it is oxidized to more toxic metabolites such as glycolic acid, glycolaldehyde, glyoxalic acid, and oxalic acid. Accumulation of these C2 acid products leads to metabolic acidosis which is the underlying cause of EG systemic toxicity. **Developmental Toxicity/Teratogenicity;** Although no adequate developmental toxicity studies are available on members of the glycol esters category, numerous regulatory bodies have determined that these substances do not pose a reproductive/developmental hazard. This is based on the previously discussed reproductive effects of related substances Propylene glycol (PG) was found not to be teratogenic in female mice given single oral doses of 10,000 ppm PG during gestation days 8-12. Fertility rates and all other parameters measured in mice given PG were not significantly different from controls. From these findings, it appears unlikely that glycol esters, as a category would pose developmental toxicity concerns **Genotoxicity**: Tests on several glycol esters were shown to be negative for mutagenic activity, with and without metabolic activation. These findings indicate that the glycol esters are not expected to cause point mutations. Substances tested using in vitro cytogenetics assays for chromosomal aberration show negative results. This is consistent with the chemistry of the glycol esters, which does not suggest the likelihood that these substances, or their constituent glycols or fatty acids, are electrophilic or reactive in nature. Therefore, the likelihood that the glycol esters may cause chromosomal aberration is expected to be very low.
DIAZOLIDINYL UREA	*REACh Dossier For imidazolidinyl urea and diazolidinyl urea: Imidazolidinyl urea releases formaldehyde into cosmetics at temperatures above 10 ºC. A 1974 study found formaldehyde release occurs at the non-physiological conditions of 60 ºC and a pH of 6. In water-containing cosmetics like shampoos, formaldehyde release increases with a rise in pH and temperature of the solution as well as a longer storage period **Acute toxicity:** The prevalence of positive reactions to imidazolidinyl urea was 1.9 percent and 3.2percent in patients with contact dermatitis in two independent studies. Concomitant positive reactions have also been reported for imidazolidinyl urea and formaldehyde as well as imidazolidinyl urea and ***N***-(3-chloroallyl)hexaminium chloride (Quaternium-15) ./ A British study conducted between 1982-1993 showed that the frequency of preservative allergy to imidazolidinyl urea in 5,167 patients with contact dermatitis was 0.99%. Furthermore, the face and the hands were the sites of allergy for 69% and 19% of patients, respectively . Curiously imidazolidinyl urea did not induce contact sensitivity in several reports. In a study, 200 subjects were given repeated insult patch tests with a 10% aqueous solution of imidazolidinyl urea three times per week for five weeks. These subjects were challenged for 24 hours after treatment and no evidence of primary irritation or sensitization occurred Imidazolidinyl urea sensitised 60-70% of female Dunkin Hartley guinea pigs in a dose-dependent manner. The animals were patch tested with 1, 5, and 10% imidazolidinyl urea in petrolatum and read after 48 hours Imidazolidinyl urea was found to be a sensitiser after topical applications of 25 ul of 10, 25, or 50 percent to CBA/Ca mice daily for three days induced significant radiolabelled thymidine incorporation into local lymph nodes four days after the last treatment with imidazolidinyl urea **Skin irritation**: Imidazolidinyl urea was non-irritating after an application of 0.1 ml of a solution containing 5, 10, or 20 percent of this compound in the right eye of albino rabbits. In another study, imidazolidinyl urea did not produce any irritation after application to the shaved backs of albino rabbits at concentrations of 0, 1, 2.5, and 5 percent Imidazolidinyl urea was described as not phototoxic in female Hartley guinea pigs after intradermal injections of 1-5% doses into the shaved backs and subsequent irradiation with FL20E and FL20BLB light for 30 minutes. The animals were again injected and irradiated 24 and 48 hours after the initial injection with no reaction **Subchronic Toxicity:** Imidazolidinyl urea was applied in powder form at concentrations of 20, 45, 90, and 200 mg/kg/day to the shaved backs of five male and female albino rabbits for 6 hr/day, 5 d/wk, for three weeks. The only treatment-related effects reported were a slight to mild inflammatory and focal ulcerative effect. Seven male and female rats that were fed 6, 28, 130, or 600 mg/kg of imidazolidinyl urea daily for 90 days showed no differences in the haematology, urinalysis, and pathology profiles when compared to controls. However, imidazolidinyl urea induced a decrease in weight gain in males fed diets over 28 mg/kg/day **Reproductive and/or Developmental Effects:** Imidazolidinyl urea induced a slight increase in the number of resorptions and/or foetal deaths ***in utero*** on day 17 in female albino mice that were orally intubated with 30, 95, and 300 mg/kg from day 6 to 15 of gestation. However, no different abnormalities in soft or skeletal tissue with respect to controls were found. This compound was found to be slightly foetotoxic but not teratogenic in mice **Genetic toxicity:** Imidazolidinyl urea was mutagenic in ***Salmonella typhimurium*** strains TA98 and TA100 at concentrations up to 1,500 µg/plate in the presence or absence of rodent liver S-9 fraction Diazolidinyl urea was mutagenic in ***Salmonella typhimurium*** strains TA98, TA100, and TA102 with and without metabolic activation. This compound induced micronuclei in Chinese hamster V79 cells with and without metabolic activation. Diazolidinyl urea also inhibited the formation of microtubuli at 3 mmol/l . In contrast, the Cosmetic Ingredients Review Expert Panel (1990) found that diazolidinyl urea was not mutagenic in ***S. typhimurium*** and that this compound did not induce micronuclei **Cytotoxicity:** Imidazolidinyl urea induced a significant dose- and time-dependent decrease in cell viability of HL60 cells after 3, 6, or 24 hours of incubation at a concentration range of 0.01-1%. Apoptotic markers of cell death, DNA subdiploid content, internucleosomal DNA fragmentation, and caspase activation were observed in HL60 cells treated with low concentrations of imidazolidinyl urea (0.01% and 0.1%). However, at higher concentrations (0.5-1%), the mechanism of cell death was necrosis . Imidazolidinyl urea was not cytotoxic to normal human fibroblasts after incubation with 1- 30% solutions for up to 30 minutes 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. The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic. Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers. A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation. A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015. It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms). Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped. Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators. Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"),

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	There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin. One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs).The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, ***All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.*** Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism.
SODIUM LAURYL ETHER SULFATE & COCONUT DIETHANOLAMIDE & PROPYL PARABEN & WATER	No significant acute toxicological data identified in literature search.
SODIUM LAURYL ETHER SULFATE & COCONUT DIETHANOLAMIDE & SODIUM CHLORIDE	The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
COCONUT DIETHANOLAMIDE & SODIUM CHLORIDE & ETHYLENE GLYCOL MONOSTEARATE & METHYL PARABEN & PROPYL PARABEN	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 CHLORIDE & ETHYLENE GLYCOL MONOSTEARATE	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
METHYL PARABEN & PROPYL PARABEN	For benzoates: **Acute toxicity:** Benzyl alcohol, benzoic acid and its sodium and potassium salt can be considered as a single category regarding human health, as they are all rapidly metabolised and excreted via a common pathway within 24 hrs. Systemic toxic effects of similar nature (e.g. liver, kidney) were observed. However with benzoic acid and its salts toxic effects are seen at higher doses than with benzyl alcohol. The compounds exhibit low acute toxicity as for the oral and dermal route. The LD50 values are > 2000 mg/kg bw except for benzyl alcohol which needs to be considered as harmful by the oral route in view of an oral LD50 of 1610 mg/kg bw. The 4 hrs inhalation exposure of benzyl alcohol or benzoic acid at 4 and 12 mg/l as aerosol/dust respectively gave no mortality, showing low acute toxicity by inhalation for these compounds. Benzoic acid and benzyl alcohol are slightly irritating to the skin, while sodium benzoate was not skin irritating. No data are available for potassium benzoate but it is also expected not to be skin irritating. Benzoic acid and benzyl alcohol are irritating to the eye and sodium benzoate was only slightly irritating to the eye. No data are available for potassium benzoate but it is expected also to be only slightly irritating to the eye. **Sensitisation:** The available studies for benzoic acid gave no indication for a sensitising effect in animals, however occasionally very low positive reactions were recorded with humans (dermatological patients) in patch tests. The same occurs for sodium benzoate. It has been suggested that the very low positive reactions are non-immunologic contact urticaria. Benzyl alcohol gave positive and negative results in animals. Benzyl alcohol also demonstrated a maximum incidence of sensitization of only 1% in human patch testing. Over several decades no sensitization with these compounds has been seen among workers. **Repeat dose toxicity:** For benzoic acid repeated dose oral toxicity studies give a NOAEL of 800 mg/kg/day. For the salts values > 1000 mg/kg/day are obtained. At higher doses increased mortality, reduced weight gain, liver and kidney effects were observed. For benzyl alcohol the long-term studies indicate a NOAEL > 400 mg/kg bw/d for rats and > 200 mg/kg bw/d for mice. At higher doses effects on bodyweights, lesions in the brains, thymus, skeletal muscle and kidney were observed. It should be taken into account that administration in these studies was by gavage route, at which saturation of metabolic pathways is likely to occur. **Mutagenicity:** All chemicals showed no mutagenic activity in ***in vitro*** Ames tests. Various results were obtained with other ***in vitro*** genotoxicity assays. Sodium benzoate and benzyl alcohol showed no genotoxicity ***in vivo.*** While some mixed and/or equivocal ***in vitro*** chromosomal/chromatid responses have been observed, no genotoxicity was observed in the ***in vivo*** cytogenetic, micronucleus, or other assays. The weight of the evidence of the ***in vitro*** and ***in vivo*** genotoxicity data indicates that these chemicals are not mutagenic or clastogenic. They also are not carcinogenic in long-term carcinogenicity studies. In a 4-generation study with benzoic acid no effects on reproduction were seen (NOAEL: 750 mg/kg). No compound related effects on reproductive organs (gross and histopathology examination) could be found in the (sub) chronic studies in rats and mice with benzyl acetate, benzyl alcohol, benzaldehyde, sodium benzoate and supports a non-reprotoxic potential of these compounds. In addition, data from reprotoxicity studies on benzyl acetate (NOAEL >2000 mg/kg bw/d; rats and mice) and benzaldehyde (tested only up to 5 mg/kg bw; rats) support the non-reprotoxicity of benzyl alcohol and benzoic acid and its salts. **Developmental toxicity:** In rats for sodium benzoate dosed via food during the entire gestation developmental effects occurred only in the presence of marked maternal toxicity (reduced food intake and decreased body weight) (NOAEL = 1400 mg/kg bw). For hamster (NOEL: 300 mg/kg bw), rabbit (NOEL: 250 mg/kg bw) and mice (CD-1 mice, NOEL: 175 mg/kg bw) no higher doses (all by gavage) were tested and no maternal toxicity was observed. For benzyl alcohol: NOAEL= 550 mg/kg bw (gavage; CD-1 mice). LOAEL = 750 mg/kg bw (gavage mice). In this study maternal toxicity was observed e.g. increased mortality, reduced body weight and clinical toxicology. Benzyl acetate: NOEL = 500 mg/kg bw (gavage rats). No maternal toxicity was observed.

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

MICROSHIELD HANDWASH

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

sodium lauryl ether sulfate

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

coconut diethanolamide

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	96h	Algae or other aquatic plants	2.2mg/l	1
EC50	72h	Algae or other aquatic plants	2.2mg/l	1
NOEC(ECx)	504h	Crustacea	0.07mg/l	1
LC50	96h	Fish	2.52mg/l	1
EC50	48h	Crustacea	2.25mg/l	1

sodium chloride

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	20.76-36.17mg/L	4
EC50	96h	Algae or other aquatic plants	1110.36mg/L	4
NOEC(ECx)	6h	Fish	0.001mg/L	4
EC50	48h	Crustacea	0.004-0.006mg/L	4
LC50	96h	Fish	1000mg/L	4

ethylene glycol monostearate

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

diazolidinyl urea

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	5.78mg/l	2
NOEC(ECx)	72h	Algae or other aquatic plants	1.6mg/l	2
EC50	48h	Crustacea	34.9mg/l	2
LC50	96h	Fish	>67mg/l	2

methyl paraben

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	15-16mg/l	4
LC50	96h	Fish	59.5mg/l	2
EC50	48h	Crustacea	5.73-22mg/l	4
NOEC(ECx)	504h	Crustacea	0.2mg/l	2

propyl paraben

Endpoint	Test Duration (hr)	Species	Value	Source
EC50	72h	Algae or other aquatic plants	7.6mg/l	2
LC50	96h	Fish	6.4mg/l	2
EC50	48h	Crustacea	7mg/l	4
EC10(ECx)	48h	Algae or other aquatic plants	0.1-0.13mg/l	4

water

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

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***

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
sodium chloride	LOW	LOW
methyl paraben	LOW	LOW
propyl paraben	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Continued...

Ingredient	Bioaccumulation
sodium chloride	LOW (LogKOW = 0.5392)
methyl paraben	LOW (LogKOW = 1.96)
propyl paraben	LOW (LogKOW = 3.04)

Mobility in soil

Ingredient	Mobility
sodium chloride	LOW (Log KOC = 14.3)
methyl paraben	LOW (Log KOC = 125.6)
propyl paraben	LOW (Log KOC = 427.2)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Management Authority for disposal. ▸ Bury residue in an authorised landfill. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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 lauryl ether sulfate	Not Available
coconut diethanolamide	Not Available
sodium chloride	Not Available
ethylene glycol monostearate	Not Available
diazolidinyl urea	Not Available
methyl paraben	Not Available
propyl paraben	Not Available
water	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
sodium lauryl ether sulfate	Not Available
coconut diethanolamide	Not Available
sodium chloride	Not Available
ethylene glycol monostearate	Not Available
diazolidinyl urea	Not Available
methyl paraben	Not Available
propyl paraben	Not Available
water	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

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

coconut diethanolamide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

Continued...

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

sodium chloride is found on the following regulatory lists

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

ethylene glycol monostearate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

diazolidinyl urea is found on the following regulatory lists

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

methyl paraben is found on the following regulatory lists

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

propyl paraben is found on the following regulatory lists

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Australian Inventory of Industrial Chemicals (AIIC)
International WHO List of Proposed Occupational Exposure Limit (OEL) Values for Manufactured Nanomaterials (MNMS)

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium lauryl ether sulfate; coconut diethanolamide; sodium chloride; ethylene glycol monostearate; diazolidinyl urea; methyl paraben; propyl paraben; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (diazolidinyl urea)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium lauryl ether sulfate; ethylene glycol monostearate)
Vietnam - NCI	Yes
Russia - FBEPH	No (ethylene glycol monostearate; diazolidinyl urea; methyl paraben)
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	31/08/2023
Initial Date	05/10/2015

SDS Version Summary

Version	Date of Update	Sections Updated
7.1	22/08/2023	Composition / information on ingredients - Ingredients
8.1	31/08/2023	Physical and chemical properties - Appearance

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

Continued...

- 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
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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

end of SDS