

Iron Removing Spray Clay

Motor Active

Chemwatch: 5415-29
Version No: 2.1.1.1
Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 30/07/2020
Print Date: 03/08/2020
L.GHS.AUS.EN

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

Product Identifier

Product name	Iron Removing Spray Clay
Synonyms	DRTU200232 (32oz/946ml)
Other means of identification	Not Available

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

Relevant identified uses	Automotive.
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Details of the supplier of the safety data sheet

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

Emergency telephone number

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

SECTION 2 Hazards identification

Classification of the substance or mixture

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

ChemWatch Hazard Ratings

	Min	Max	
Flammability	0		
Toxicity	2		0 = Minimum
Body Contact	2		1 = Low
Reactivity	0		2 = Moderate
Chronic	2		3 = High
			4 = Extreme

Poisons Schedule	Not Applicable
Classification [1]	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Acute Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

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

H302	Harmful if swallowed.
H315	Causes skin irritation.
H318	Causes serious eye damage.
H317	May cause an allergic skin reaction.

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H401 Toxic to aquatic life.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

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.
P310	Immediately call a POISON CENTER or doctor/physician.
P321	Specific treatment (see advice on this label).
P362	Take off contaminated clothing and wash before reuse.
P302+P352	IF ON SKIN: Wash with plenty of water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P301+P312	IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

Not Applicable

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
367-51-1	5-10	<u>sodium thioglycolate</u>
68439-46-3	1-5	<u>alcohols C9-11 ethoxylated</u>
1300-72-7	1-5	<u>sodium xylenesulfonate</u>
68585-34-2	<3	<u>sodium lauryl ether sulfate</u>
7732-18-5	>80	<u>water</u>

SECTION 4 First aid measures

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold eyelids apart and flush the eye continuously with running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY. ▶ For advice, contact a Poisons Information Centre or a doctor. ▶ Urgent hospital treatment is likely to be needed. ▶ In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. ▶ If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS

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- should be provided. Further action will be the responsibility of the medical specialist.
- ▶ If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS.

Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:

- ▶ **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.

NOTE: Wear a protective glove when inducing vomiting by mechanical means.

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	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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. <p>Other decomposition products include: carbon dioxide (CO₂) sulfur oxides (SO_x) other pyrolysis products typical of burning organic material. May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Control personal contact with the substance, by using protective equipment. ▶ Contain and absorb spill with sand, earth, inert material or vermiculite. ▶ Wipe up. ▶ Place in a suitable, labelled container for waste disposal.
Major Spills	<p>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ 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.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	strong acids

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
Iron Removing Spray Clay	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
sodium thioglycolate	Not Available	Not Available
alcohols C9-11 ethoxylated	Not Available	Not Available
sodium xylenesulfonate	Not Available	Not Available
sodium lauryl ether sulfate	Not Available	Not Available
water	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium thioglycolate	E	≤ 0.01 mg/m ³
alcohols C9-11 ethoxylated	E	≤ 0.1 ppm
sodium xylenesulfonate	E	≤ 0.01 mg/m ³
sodium lauryl ether sulfate	E	≤ 0.01 mg/m ³

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.

MATERIAL DATA

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate</p>
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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:
- 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

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	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	<ul style="list-style-type: none"> ▶ Overalls. ▶ P.V.C apron. ▶ Barrier cream. ▶ Skin cleansing cream. ▶ Eye wash 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:

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

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

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

* - Continuous Flow ** - Continuous-flow or positive pressure demand

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

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

SECTION 9 Physical and chemical properties**Information on basic physical and chemical properties**

Appearance	Transparent colorless liquid with citrus odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.02
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable
pH (as supplied)	6	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	19.61-78.43
Initial boiling point and boiling range (°C)	100	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>93	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	90 (%wt)
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	368

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SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information**Information on toxicological effects**

Inhaled	<p>Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Thiols (particularly ethyl mercaptan) produce lethargy or sleepiness (deep sedation may be produced by maximal sublethal intraperitoneal doses). The mercaptans generally produce central nervous system depression (although C-8 and isomeric C-8 members possess analeptic and psychomimetic action). Exposure to high levels may result in headache, dizziness, nausea, vomiting, restlessness, muscular incoordination, skeletal muscle paralysis, severe or mild cyanosis, respiratory depression, coma and death. Odour threshold should not be used as a warning threshold since some materials are detected way below the measurable concentration. Odour fatigue may occur.</p> <p>Prolonged exposure may cause headache, nausea and ultimately loss of consciousness.</p>
Ingestion	<p>Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual.</p> <p>Thioglycolate salts and derivatives may produce hypoglycaemia, central nervous system depression, dyspnea, and convulsions in experimental animals.</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Fatalities were produced by topical application of a 10% solution of thioglycolic acid to guinea pigs at less than 5 ml/kg. Signs of intoxication included weakness, gasping and convulsions. Professional hair-dressers exposed to thioglycolate products show skin irritation and skin sensitisation.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p>
Eye	When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>Chronic exposure to thioglycolate salts (in occupational settings) have produce dermatoses and allergic reactions characterised by oedema, burning of the skin, papular rash, eczematous dermatitis of the scalp or hands, erythema and subcutaneous haemorrhage. Experimental animals have exhibited thyroid hyperplasia.</p> <p>Chronic exposure to mercaptans may result in damage to the lungs, kidneys and liver.</p>

Iron Removing Spray Clay	TOXICITY	IRRITATION
	Dermal (None) LD50: >5000 mg/kg ^[2]	Not Available
	Oral (None) LD50: >300 mg/kg ^[2]	
sodium thioglycolate	TOXICITY	IRRITATION
	dermal (rat) LD50: >798-1596 mg/kg ^[1]	Not Available
	Oral (rat) LD50: 25-200 mg/kg ^[1]	
alcohols C9-11 ethoxylated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye (human): SEVERE
	Oral (rat) LD50: 1378 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
		Skin: SEVERE
sodium xylenesulfonate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >=2000 mg/kg ^[1]	Eye: adverse effect observed (irritating) ^[1]
	Oral (rat) LD50: >10 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
sodium lauryl ether sulfate	TOXICITY	IRRITATION
	Oral (rat) LD50: 1600 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]

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		Skin (rabbit):25 mg/24 hr moderate
		Skin: adverse effect observed (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 THIOGLYCOLATE	<p>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.</p> <p>Ammonium and glyceryl thioglycolate and thioglycolic acid are used predominantly in cosmetic permanent waving lotions at concentrations up to 15.4% (as thioglycolic acid). At use concentrations, these cosmetic ingredients are only slightly toxic in acute single oral and dermal exposures. In repeated dermal tests for extended periods of exposure, these ingredients were toxic. Commercial permanent wave products produced transient conjunctival redness to both rinsed and unrinsed eyes.</p> <p>The results of skin testing for irritation and sensitisation of these thioglycolates depends on the type of test system used. Under occlusive patch testing, the data indicate that these ingredients are cumulative irritants and possibly weak sensitisers, but not under semi-occlusive test conditions. In clinical patients, mainly hairdressers, glyceryl thioglycolate elicited allergic reactions at concentrations down to 0.25%. Administration of ammonium thioglycolate to animals has produced hypoglycaemia, and thyroid effects.</p> <p>The acute inhalation toxicity of a liquid droplet aerosol containing aqueous ammonium thioglycolate (as 60% thioglycolic acid) was evaluated using rats (number and strain not stated). Animals were exposed to the aerosol for 1 hr and then observed for 14 days. None of the animals died. Few animals experienced respiratory distress, and signs were not observed beyond 24 hr post-exposure. At necropsy, minor pulmonary abnormalities were observed.</p> <p>Hair-waving solutions containing thioglycolates may produce keratitis; they may also produce irritation, burning sensations, conjunctival inflammation, corneal epithelial erosion, turbidity of the cornea, mydriasis, cycloplegia, loss of convergence and disturbances of vision. A mother and daughter developed bilateral optic neuritis after the use of a cold wave lotion containing ammonium thioglycolate. Oedema of the discs and retina and centrocecal scotomas horizontal oval defect in the field of vision) were seen. Oedema subsided in six months but the scotomas persisted.</p> <p>Sodium thioglycolate, which has widespread occupational and consumer exposure to women from cosmetics and hair-care products, was evaluated for developmental toxicity by topical exposure during the embryonic and fetal periods of pregnancy. In rats, maternal topical exposure to sodium thioglycolate, at 200 mg/kg/day (the highest dose tested) on gestational days (GD) 6-19, resulted in maternal toxicity, including reduced body weights and weight gain, increased relative water consumption and one death. Treatment-related increases in feed consumption and changes at the application site occurred at all doses, in the absence of increased body weights or body weight change. Foetal body weights/litter were decreased at 200 mg/kg/day, with no other embryo/foetal toxicity and no treatment-related teratogenicity in any group. In rabbits, maternal topical exposure to sodium thioglycolate on GD 6-29 resulted in maternal dose-related toxicity at the dosing site in all groups; no maternal systemic toxicity, embryo/fetal toxicity, or treatment-related teratogenicity were observed in any group. A no observed adverse effect level (NOAEL) was not identified for maternal toxicity in either species with the dosages tested. The developmental toxicity NOAEL was 100 mg/kg/day (rats) and >65 mg/kg/day (rabbits; the highest dose tested). The clinical relevance of these study results is uncertain because no data were available for levels, frequency, or duration of exposures in female workers or end users.</p> <p>Skin (human): 7%/24h</p>
ALCOHOLS C9-11 ETHOXYLATED	<p>Somnolence, ataxia, diarrhoea recorded.</p> <p>Human beings 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 ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.</p> <p>Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.</p> <p>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.</p> <p>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.</p> <p>On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.</p> <p>Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units: EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41 EO > 15-20 gives Harmful (Xn) with R22-41 >20 EO is not classified (CESIO 2000) Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin). AE are not included in Annex 1 of the list of dangerous substances of the Council Directive 67/548/EEC</p> <p>In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD50 values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.</p> <p>The ability of nonionic surfactants to cause a swelling of the stratum corneum of guinea pig skin has been studied. The swelling mechanism of the skin involves a combination of ionic binding of the hydrophilic group as well as hydrophobic interactions of the alkyl chain with the substrate. One of the mechanisms of skin irritation caused by surfactants is considered to be denaturation of the proteins of skin. It has also been</p>

established that there is a connection between the potential of surfactants to denature protein *in vitro* and their effect on the skin. Nonionic surfactants do not carry any net charge and, therefore, they can only form hydrophobic bonds with proteins. For this reason, proteins are not deactivated by nonionic surfactants, and proteins with poor solubility are not solubilized by nonionic surfactants. A substantial amount of toxicological data and information *in vivo* and *in vitro* demonstrates that there is no evidence for alcohol ethoxylates (AEs) being genotoxic, mutagenic or carcinogenic. No adverse reproductive or developmental effects were observed. The majority of available toxicity studies revealed NOAELs in excess of 100 mg/kg bw/d but the lowest NOAEL for an individual AE was established to be 50 mg/kg bw/day. This value was subsequently considered as a conservative, representative value in the risk assessment of AE. The effects were restricted to changes in organ weights with no histopathological organ changes with the exception of liver hypertrophy (indicative of an adaptive response to metabolism rather than a toxic effect). It is noteworthy that there was practically no difference in the NOAEL in oral studies of 90-day or 2 years of duration in rats. A comparison of the aggregate consumer exposure and the systemic NOAEL (taking into account an oral absorption value of 75%) results in a Margin of Exposure of 5,800. Taking into account the conservatism in the exposure assessment and the assigned systemic NOAEL, this margin of exposure is considered more than adequate to account for the inherent uncertainty and variability of the hazard database and inter and intra-species extrapolations.

AEs are not contact sensitizers. Neat AE are irritating to eyes and skin. The irritation potential of aqueous solutions of AEs depends on concentrations. Local dermal effects due to direct or indirect skin contact in certain use scenarios where the products are diluted are not of concern as AEs are not expected to be irritating to the skin at in-use concentrations. Potential irritation of the respiratory tract is not a concern given the very low levels of airborne AE generated as a consequence of spray cleaner aerosols or laundry powder detergent dust.

In summary, the human health risk assessment has demonstrated that the use of AE in household laundry and cleaning detergents is safe and does not cause concern with regard to consumer use.

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that

of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes

in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

Mutagenicity: Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGME is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).

Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.

The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

The material may produce severe skin irritation after prolonged or repeated exposure, and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) thickening of the epidermis.

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is

unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

Most chemicals of this category are not defined substances, but mixtures of homologues with different alkyl chain lengths. Alpha-olefin sulfonates are mixtures of alkene sulfonate and hydroxyl alkane sulfonates with the sulfonate group in the terminal position and the double bond, or hydroxyl group, located at a position in the vicinity of the sulfonate group.

Common physical and/or biological pathways result in structurally similar breakdown products, and are, together with the surfactant properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

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

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

C10-; 290-580

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

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

C14-18, C16-18-; >5000

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

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

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

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

C12-; 200

C12-13 and C10-16-;>500

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

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

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

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

Under occlusive conditions:

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

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

SODIUM XYLENESULFONATE

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

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

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

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

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

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

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

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

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

Carcinogenicity: Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day).

alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure.

No carcinogenicity studies were available for the alkane sulfonates.

Reproductive toxicity: No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates.

The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

Iron Removing Spray Clay

Developmental toxicity: In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death).

The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day indicative of a delayed development. The lowest reliable NOEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits.

For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOEL was 600 mg/kg/day both for maternal and developmental toxicity. No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

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

Toxicological data are available and well documented for representative toluenesulfonates, xylenesulfonates and cumenesulfonates (including sodium, potassium, ammonium and calcium salts). These data demonstrate that hydrotropes have a low order of acute toxicity by all relevant routes (LC50s range from 100s to 1000s mg/kg), are not genotoxic *in vitro* or *in vivo*, show no evidence of a carcinogenic response (or any other systemic toxicity) in 2-year dermal exposure studies, and failed to induce developmental, teratogenic or fertility (sex organ) effects.

Adverse effects after repeated long term dosing of hydrotropes to animals included epidermal hyperplasia at the site of application in dermal studies, and decreased relative spleen weight in females in oral studies. The critical adverse effect and corresponding systemic NOEL is 763 mg a.i./kg bw based upon decreased relative spleen weight in female rats in a 90-day oral study. The NOEL for local effects, based on epidermal hyperplasia at the site of application, was 440 mg a.i./kg bw for mice in 90-day dermal studies.

Hydrotropes can be classified as a negligible-to-slight irritant to skin and a slight-to-moderate irritant to eyes. The irritation potential of aqueous solutions of hydrotropes depends on concentration, and the irritation is lessened with rinsing. Hydrotropes are not considered to be skin sensitizers.

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Hydrotropes in this category were assessed for mutagen/ genotoxic potential in a variety of assays including the mouse micronucleus, Ames, mouse lymphoma, sister chromatid exchange and chromosome aberration assays. No positive results were seen *in vitro* or *in vivo* in any of the studies. For both mice and rats exposed dermally for two years, there was no evidence of carcinogenic potential.

Examination of the sex organs (such as prostate, testes or ovaries) from animals in 90-day feeding studies and 90-day and two year dermal studies yielded no evidence to suggest that these chemicals have an adverse effect on the reproductive organs.

* [CESIO]

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 (2-3EO) 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 developmental 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 NOEL from a 90-day rat gavage study with NaC12-14 POE n=2 (CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOEL 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 (NOEL) 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 NOEL 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

SODIUM LAURYL ETHER SULFATE

Iron Removing Spray Clay

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: (International Journal of Toxicology 29 (Supplement 3) 151S-161S: 2010 <http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151>

The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

SODIUM THIOGLYCOLATE & SODIUM XYLENESULFONATE

Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

ALCOHOLS C9-11 ETHOXYLATED & SODIUM LAURYL ETHER SULFATE

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 Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology <http://doi.org/10.5487/TR.2015.31.2.105>

SODIUM XYLENESULFONATE & SODIUM LAURYL ETHER SULFATE & WATER

No significant acute toxicological data identified in literature search.

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
Iron Removing Spray Clay	Not Available	Not Available	Not Available	Not Available	Not Available
sodium thioglycolate	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	38mg/L	2
	EC50	72	Algae or other aquatic plants	6.3mg/L	2
	EC10	72	Algae or other aquatic plants	2.1mg/L	2
	NOEC	72	Algae or other aquatic plants	0.81mg/L	2
alcohols C9-11 ethoxylated	LC50	96	Fish	8.5mg/L	4
	EC50	48	Crustacea	2.5mg/L	2
	EC50	96	Algae or other aquatic plants	1.4mg/L	2
	EC20	72	Algae or other aquatic plants	0.711mg/L	2
	NOEC	240	Fish	0.16mg/L	2
sodium xylenesulfonate	LC50	96	Fish	>1-mg/L	2
	EC50	48	Crustacea	>1-20mg/L	2
	EC50	96	Algae or other aquatic plants	>=230mg/L	2
	NOEC	504	Crustacea	<30mg/L	2
sodium lauryl ether sulfate	NOEC	48	Fish	0.26mg/L	5
water	LC50	96	Fish	897.520mg/L	3
	EC50	96	Algae or other aquatic plants	8768.874mg/L	3

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

Toxic to aquatic organisms.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Iron Removing Spray Clay

Ingredient	Persistence: Water/Soil	Persistence: Air
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
water	LOW (KOC = 14.3)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Recycle wherever possible. ▶ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▶ Dispose of by: burial in a land-fill specifically 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.
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SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 Regulatory information

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

sodium thioglycolate is found on the following regulatory lists

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

Australian Inventory of Industrial Chemicals (AIIC)

alcohols C9-11 ethoxylated is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

sodium xylenesulfonate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

sodium lauryl ether sulfate is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australian Inventory of Industrial Chemicals (AIIC)

water is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

National Inventory Status

National Inventory	Status
Australia - AIIC	Yes
Australia - Non-Industrial Use	No (sodium thioglycolate; alcohols C9-11 ethoxylated; sodium xylenesulfonate; sodium lauryl ether sulfate; water)
Canada - DSL	Yes
Canada - NDSL	No (sodium thioglycolate; alcohols C9-11 ethoxylated; sodium xylenesulfonate; sodium lauryl ether sulfate; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (alcohols C9-11 ethoxylated)
Japan - ENCS	No (alcohols C9-11 ethoxylated)
Korea - KECI	Yes

Iron Removing Spray Clay

National Inventory	Status
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium thioglycolate; sodium lauryl ether sulfate)
Vietnam - NCI	Yes
Russia - ARIPS	No (sodium thioglycolate; alcohols C9-11 ethoxylated)
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	30/07/2020
Initial Date	30/07/2020

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	30/07/2020	Acute Health (inhaled), Advice to Doctor, Classification, Disposal, Fire Fighter (fire/explosion hazard), Physical Properties, Spills (minor)

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
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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TEL (+61 3) 9572 4700.