# Meguiar's A25 - Soft Wash Gel

**Motor Active**

Chemwatch Hazard Alert Code: **3**

Chemwatch: **4910-77**
Version No: **6.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **04/09/2019**
Print Date: **18/09/2019**
L.GHS.AUS.EN

## SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

### Product Identifier

| | |
|---|---|
| **Product name** | Meguiar's A25 - Soft Wash Gel |
| **Synonyms** | Product Code: A25 |
| **Other means of identification** | Not Available |

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

| | |
|---|---|
| **Relevant identified uses** | Car shampoo. |

### 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 | 0 | |
| Body Contact | 3 | |
| Reactivity | 0 | |
| Chronic | 2 | |

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

| | |
|---|---|
| **Poisons Schedule** | Not Applicable |
| **Classification [1]** | Skin Corrosion/Irritation Category 2, Serious Eye Damage Category 1, Skin Sensitizer Category 1, Germ cell mutagenicity Category 2, Carcinogenicity Category 2, Specific target organ toxicity - repeated exposure Category 2, 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)**

| | |
|---|---|
| **SIGNAL WORD** | **DANGER** |

### Hazard statement(s)

| | |
|---|---|
| **H315** | Causes skin irritation. |
| **H318** | Causes serious eye damage. |
| **H317** | May cause an allergic skin reaction. |
| **H341** | Suspected of causing genetic defects. |
| **H351** | Suspected of causing cancer. |

**Continued...**

| | |
|---|---|
| **H373** | May cause damage to organs through prolonged or repeated exposure. |
| **H401** | Toxic to aquatic life. |

### Supplementary statement(s)

Not Applicable

### CLP classification (additional)

Not Applicable

### Precautionary statement(s) Prevention

| | |
|---|---|
| **P201** | Obtain special instructions before use. |
| **P260** | Do not breathe dust/fume/gas/mist/vapours/spray. |
| **P280** | Wear protective gloves/protective clothing/eye protection/face protection. |
| **P281** | Use personal protective equipment as required. |
| **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. |
| **P308+P313** | IF exposed or concerned: Get medical advice/attention. |
| **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 soap and water. |
| **P333+P313** | If skin irritation or rash occurs: Get medical advice/attention. |

### Precautionary statement(s) Storage

| | |
|---|---|
| **P405** | Store locked up. |

### Precautionary statement(s) Disposal

| | |
|---|---|
| **P501** | Dispose of contents/container in accordance with local regulations. |

## SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

### Substances

See section below for composition of Mixtures

### Mixtures

| CAS No | %[weight] | Name |
|---|---|---|
| 68439-57-6 | 10-20 | sodium C14-16-olefin sulfonate |
| 61790-63-4 | 1-5 | coconut oil diethanolamide |
| Not Available | 0.5-2 | conditioners proprietary |
| 131-57-7 | 0.1-1 | oxybenzone |
| Not Available | <0.1 | distyryl biphenyl derivative proprietary |
| 111-42-2 | 0.5 max | diethanolamine |
| 7732-18-5 | 65-85 | water |

## SECTION 4 FIRST AID MEASURES

### Description of first aid measures

| | |
|---|---|
| **Eye Contact** | If this product comes in contact with the eyes:<br>▸ Immediately hold eyelids apart and flush the eye continuously with running water.<br>▸ 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.<br>▸ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes.<br>▸ Transport to hospital or doctor without delay.<br>▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel. |
| **Skin Contact** | If skin contact occurs:<br>▸ Immediately remove all contaminated clothing, including footwear.<br>▸ Flush skin and hair with running water (and soap if available).<br>▸ Seek medical attention in event of irritation. |
| **Inhalation** | ▸ If fumes, aerosols or combustion products are inhaled remove from contaminated area.<br>▸ Other measures are usually unnecessary. |
| **Ingestion** | ▸ Immediately give a glass of water.<br>▸ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. |

### Indication of any immediate medical attention and special treatment needed

**Continued...**

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. |

### Advice for firefighters

| | |
|---|---|
| **Fire Fighting** | ▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear breathing apparatus plus protective gloves in the event of a fire.<br>▸ Prevent, by any means available, spillage from entering drains or water courses.<br>▸ Use fire fighting procedures suitable for surrounding area.<br>▸ **DO NOT** approach containers suspected to be hot.<br>▸ Cool fire exposed containers with water spray from a protected location.<br>▸ If safe to do so, remove containers from path of fire.<br>▸ Equipment should be thoroughly decontaminated after use. |
| **Fire/Explosion Hazard** | ▸ The material is not readily combustible under normal conditions.<br>▸ However, it will break down under fire conditions and the organic component may burn.<br>▸ Not considered to be a significant fire risk.<br>▸ Heat may cause expansion or decomposition with violent rupture of containers.<br>▸ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).<br>▸ May emit acrid smoke.<br><br>Decomposes on heating and produces toxic fumes of:<br>carbon dioxide ($CO_2$)<br>nitrogen oxides (NOx)<br>sulfur oxides (SOx)<br>other pyrolysis products typical of burning organic material.<br>May emit poisonous fumes. |
| **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** | ▸ Clean up all spills immediately.<br>▸ Avoid breathing vapours and contact with skin and eyes.<br>▸ Control personal contact with the substance, by using protective equipment.<br>▸ Contain and absorb spill with sand, earth, inert material or vermiculite.<br>▸ Wipe up.<br>▸ Place in a suitable, labelled container for waste disposal. |
| **Major Spills** | Moderate hazard.<br>▸ Clear area of personnel and move upwind.<br>▸ Alert Fire Brigade and tell them location and nature of hazard.<br>▸ Wear breathing apparatus plus protective gloves.<br>▸ Prevent, by any means available, spillage from entering drains or water course.<br>▸ Stop leak if safe to do so.<br>▸ Contain spill with sand, earth or vermiculite.<br>▸ Collect recoverable product into labelled containers for recycling.<br>▸ Neutralise/decontaminate residue (see Section 13 for specific agent).<br>▸ Collect solid residues and seal in labelled drums for disposal.<br>▸ Wash area and prevent runoff into drains.<br>▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.<br>▸ If contamination of drains or waterways occurs, advise emergency services. |

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

## SECTION 7 HANDLING AND STORAGE

### Precautions for safe handling

| | |
|---|---|
| **Safe handling** | ▸ **DO NOT** allow clothing wet with material to stay in contact with skin<br>▸ Avoid all personal contact, including inhalation.<br>▸ Wear protective clothing when risk of exposure occurs. |

**Continued...**

| | |
|---|---|
| | ▸ Use in a well-ventilated area.<br>▸ Prevent concentration in hollows and sumps.<br>▸ **DO NOT** enter confined spaces until atmosphere has been checked.<br>▸ **DO NOT** allow material to contact humans, exposed food or food utensils.<br>▸ Avoid contact with incompatible materials.<br>▸ When handling, **DO NOT** eat, drink or smoke.<br>▸ Keep containers securely sealed when not in use.<br>▸ Avoid physical damage to containers.<br>▸ Always wash hands with soap and water after handling.<br>▸ Work clothes should be laundered separately. Launder contaminated clothing before re-use.<br>▸ Use good occupational work practice.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS.<br>▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. |
| **Other information** | ▸ Store in original containers.<br>▸ Keep containers securely sealed.<br>▸ Store in a cool, dry, well-ventilated area.<br>▸ Store away from incompatible materials and foodstuff containers.<br>▸ Protect containers against physical damage and check regularly for leaks.<br>▸ Observe manufacturer's storage and handling recommendations contained within this SDS. |

### Conditions for safe storage, including any incompatibilities

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

## 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 | diethanolamine | Diethanolamine | 3 ppm / 13 mg/m3 | Not Available | Not Available | Not Available |

**EMERGENCY LIMITS**

| Ingredient | Material name | TEEL-1 | TEEL-2 | TEEL-3 |
|---|---|---|---|---|
| diethanolamine | Diethanolamine | 3 mg/m3 | 28 mg/m3 | 130 mg/m3 |

| Ingredient | Original IDLH | Revised IDLH |
|---|---|---|
| sodium C14-16-olefin sulfonate | Not Available | Not Available |
| coconut oil diethanolamide | Not Available | Not Available |
| oxybenzone | Not Available | Not Available |
| diethanolamine | Not Available | Not Available |
| water | Not Available | Not Available |

**MATERIAL DATA**

for diethanolamine:
Odour Threshold: 2.6 ppm
The TLV-TWA is thought to be protective against the significant risk of eye damage and skin irritation.
Odour Safety Factor (OSF)
OSF=1.7 (DIETHANOLAMINE)

### Exposure controls

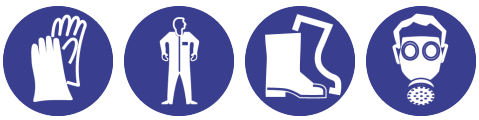
**Appropriate engineering controls**

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

General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

| Type of Contaminant: | Air Speed: |
|---|---|
| solvent, vapours, degreasing etc., evaporating from tank (in still air). | 0.25-0.5 m/s (50-100 f/min) |
| aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation) | 0.5-1 m/s (100-200 f/min.) |
| direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion) | 1-2.5 m/s (200-500 f/min.) |

**Continued...**

| | |
|---|---|
| | 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.)<br>Within each range the appropriate value depends on:<br>Lower end of the range: 1: Room air currents minimal or favourable to capture; 2: Contaminants of low toxicity or of nuisance value only.; 3: Intermittent, low production.; 4: Large hood or large air mass in motion<br>Upper end of the range: 1: Disturbing room air currents; 2: Contaminants of high toxicity; 3: High production, heavy use; 4: Small hood-local control only<br>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.<br>▸ Chemical goggles.<br>▸ 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.<br>▸ Wear safety footwear or safety gumboots, e.g. Rubber<br>**NOTE:**<br>▸ 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.<br>▸ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.<br>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.<br>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.<br>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.<br>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:<br>· frequency and duration of contact,<br>· chemical resistance of glove material,<br>· glove thickness and<br>· dexterity<br>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).<br>· 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.<br>· 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.<br>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.<br>· Contaminated gloves should be replaced.<br>As defined in ASTM F-739-96 in any application, gloves are rated as:<br>· Excellent when breakthrough time > 480 min<br>· Good when breakthrough time > 20 min<br>· Fair when breakthrough time < 20 min<br>· Poor when glove material degrades<br>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.<br>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.<br>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.<br>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:<br>· Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.<br>· 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<br>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. |
| **Body protection** | See Other protection below |
| **Other protection** | ▸ Overalls.<br>▸ P.V.C. apron.<br>▸ Barrier cream.<br>▸ Skin cleansing cream.<br>▸ Eye wash unit. |

**Recommended material(s)**

**GLOVE SELECTION INDEX**

**Respiratory protection**

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

**Continued...**

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:
Meguiar's A25 - Soft Wash Gel

| Material | CPI |
|---|---|
| BUTYL | A |
| NEOPRENE | A |
| VITON | A |
| NATURAL RUBBER | C |
| NATURAL+NEOPRENE | C |
| NITRILE | C |
| PVA | C |
| PVC | C |
| TEFLON | 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.

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)

- 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** | Pink liquid with pleasant odour; mixes with water. |

| | | | |
|---|---|---|---|
| **Physical state** | Liquid | **Relative density (Water = 1)** | 1 |
| **Odour** | Not Available | **Partition coefficient n-octanol / water** | Not Available |
| **Odour threshold** | Not Available | **Auto-ignition temperature (°C)** | Not Applicable |
| **pH (as supplied)** | 8.5 | **Decomposition temperature** | Not Available |
| **Melting point / freezing point (°C)** | Not Available | **Viscosity (cSt)** | Not Available |
| **Initial boiling point and boiling range (°C)** | 100 | **Molecular weight (g/mol)** | Not Applicable |
| **Flash point (°C)** | Not Applicable | **Taste** | Not Available |
| **Evaporation rate** | 1 (water=1) | **Explosive properties** | Not Available |
| **Flammability** | Not Applicable | **Oxidising properties** | Not Available |
| **Upper Explosive Limit (%)** | Not Applicable | **Surface Tension (dyn/cm or mN/m)** | Not Available |
| **Lower Explosive Limit (%)** | Not Applicable | **Volatile Component (%vol)** | Not Available |
| **Vapour pressure (kPa)** | 2.3 @25C | **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 |

## SECTION 10 STABILITY AND REACTIVITY

| | |
|---|---|
| **Reactivity** | See section 7 |
| **Chemical stability** | ▸ Unstable in the presence of incompatible materials.<br>▸ Product is considered stable.<br>▸ 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

**Continued...**

| | |
|---|---|
| **Inhaled** | The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.<br>Not normally a hazard due to non-volatile nature of product |
| **Ingestion** | The material has **NOT** been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern. |
| **Skin Contact** | 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.<br>The material may accentuate any pre-existing dermatitis condition<br><br>Anionic surfactants/ hydrotropes generally produce skin reactions following the removal of natural oils. The skin may appear red and may become sore. Papular dermatitis may also develop. Sensitive individuals may exhibit cracking, scaling and blistering.<br>Open cuts, abraded or irritated skin should not be exposed to this material |
| **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.<br>Direct eye contact with some concentrated anionic surfactants/ hydrotropes produces corneal damage, in some cases severe. Low concentrations may produce immediate discomfort, conjunctival hyperaemia, and oedema of the corneal epithelium. Healing may take several days. Temporary clouding of the cornea may occur. |
| **Chronic** | Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.<br>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.<br>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.<br>Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population.<br>Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking.<br>Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.<br>Repeated skin contact with some sulfonated surfactants has produced sensitisation dermatitis in predisposed individuals. |

| | TOXICITY | IRRITATION |
|---|---|---|
| **Meguiar's A25 - Soft Wash Gel** | Not Available | Not Available |
| **sodium C14-16-olefin sulfonate** | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 6300-13500 mg/kg[2] | Eye: irritant ** |
| | Oral (rat) LD50: >2000 mg/kg[2] | Skin: irritant ** |
| **coconut oil diethanolamide** | TOXICITY | IRRITATION |
| | Not Available | Not Available |
| **oxybenzone** | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: >16000 mg/kg[2] | Not Available |
| | Oral (rat) LD50: 7400 mg/kg[2] | |
| **diethanolamine** | TOXICITY | IRRITATION |
| | Dermal (rabbit) LD50: 8342.88 mg/kg[2] | Eye (rabbit): 5500 mg - SEVERE |
| | Oral (rat) LD50: 677.04 mg/kg[2] | Eye (rabbit):0.75 mg/24 hr SEVERE |
| | | Eye: adverse effect observed (irritating)[1] |
| | | Skin (rabbit): 50 mg (open)-mild |
| | | Skin (rabbit): 500 mg/24 hr-mild |
| | | Skin: adverse effect observed (irritating)[1] |
| **water** | TOXICITY | IRRITATION |
| | Oral (rat) LD50: >90000 mg/kg[2] | Not Available |

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

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| **SODIUM C14-16-OLEFIN SULFONATE** | for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates<br><br>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.<br>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. |

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

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

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

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

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

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

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

In eye irritation tests, using rabbits, C12-containing alkyl sulfates (>10% concentration) were severely irritating and produced irreversible corneal effects. With increasing alkyl chain length, the irritating potential decreases, and C16-18 alkyl sulfate sodium, at a concentration of 25%, was only a mild irritant. Concentrated C14-16- alpha-olefin sulfonates were severely irritating, but caused irreversible effects only if applied as undiluted powder. At concentrations below 10% mild to moderate, reversible effects, were found. No data were available for alkane sulfonates

Alkyl sulfates and C14-18 alpha-olefin sulfonates were not skin sensitisers in animal studies. No reliable data were available for alkane sulfonates. Based on the similar chemical structure, no sensitisation is expected.
However anecdotal evidence suggests that sodium lauryl sulfate causes pulmonary sensitisation resulting in hyperactive airway dysfunction and pulmonary allergy accompanied by fatigue, malaise and aching. Significant symptoms of exposure can persist for more than two years and can be activated by a variety of non-specific environmental stimuli such as a exhaust, perfumes and passive smoking.
Absorbed sulfonates are quickly distributed through living systems and are readily excreted. Toxic effects may result from the effects of binding to proteins and the ability of sulfonates to translocate potassium and nitrate (NO3-) ions from cellular to interstitial fluids. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies. Repeated skin contact with some sulfonated surfactants has produced sensitisation dermatitis in predisposed individuals

**Repeat dose toxicity:** After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium).
C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.
No data were available with regard to the repeated dose toxicity of alkane sulfonates. Based on the similarity of metabolic pathways between alkane sulfonates, alkyl sulfates and alkyl-olefin sulfonates, the repeated dose toxicity of alkane sulfonates is expected to be similar with NOAEL and LOAEL values in the same range as for alkyl sulfates and alpha-olefin sulfonates, i.e. 100 and 200-250 mg/kg/day, respectively, with the liver as potential target organ.

**Genotoxicity:** Alkyl sulfates of different chain lengths and with different counter ions were not mutagenic in standard bacterial and mammalian cell systems both in the absence and in the presence of metabolic activation. There was also no indication for a genotoxic potential of alkyl sulfates in various in vivo studies on mice (micronucleus assay, chromosome aberration test, and dominant lethal assay).
alpha-Olefin sulfonates were not mutagenic in the Ames test, and did not induce chromosome aberrations in vitro. No genotoxicity data were available for alkane sulfonates. Based on the overall negative results in the genotoxicity assays with alkyl sulfates and alpha-olefin sulfonates, the absence of structural elements indicating mutagenicity, and the overall database on different types of sulfonates, which were all tested negative in mutagenicity assays, a genotoxic potential of alkane sulfonates is not expected.

**Carcinogenicity:** Alkyl sulfates were not carcinogenic in feeding studies with male and female Wistar rats fed diets with C12-15 alkyl sulfate sodium for two years (corresponding to doses of up to 1125 mg/kg/day).
alpha-Olefin sulfonates were not carcinogenic in mice and rats after dermal application, and in rats after oral exposure.
No carcinogenicity studies were available for the alkane sulfonates.

**Reproductive toxicity**: No indication for adverse effects on reproductive organs was found in various oral studies with different alkyl sulfates. The NOAEL for male fertility was 1000 mg/kg/day for sodium dodecyl sulfate. In a study using alpha-olefin sulfonates in male and female rats, no adverse effects were identified up to 5000 ppm.

**Developmental toxicity:** In studies with various alkyl sulfates (C12 up to C16-18- alkyl) in rats, rabbits and mice, effects on litter parameters were restricted to doses that caused significant maternal toxicity (anorexia, weight loss, and death).
The principal effects were higher foetal loss and increased incidences of total litter losses. The incidences of malformations and visceral and skeletal anomalies were unaffected apart from a higher incidence of delayed ossification or skeletal variation in mice at > 500 mg/kg bw/day indicative of a delayed development. The lowest reliable NOAEL for maternal toxicity was about 200 mg/kg/day in rats, while the lowest NOAELs in offspring were 250 mg/kg/day in rats and 300 mg/kg/day for mice and rabbits.
For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity.
No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

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| | 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<br><br>alpha-Olefine sulfonates (AOS) are classified as Irritant (Xi) with the risk phrases R38 and R41 for concentrations > 80% and R36/38 (Irritating to eyes and skin) for concentrations of 40-80% according to CESIO (CESIO 2000). AOS are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC.<br><br>The absorption of AOS through intact skin is considered to be very low. Unchanged a -olefine sulfonate (AOS) and/or metabolites of AOS are primarily eliminated in the urine and, to a lesser extent, in the faeces within 24 hours of administration. The chemical structures of the metabolites have not yet been identified.<br><br>AOS has a moderately low acute oral toxicity as indicated by LD50 values between 1,300 and 2,400 mg/kg body weight for rats and between 2,500 and 4,300 mg/kg body weight for mice. The toxic effects at high oral doses were reduced voluntary activity, diarrhoea and anaemia.<br><br>AOS are mildly to moderately irritating to human skin depending on the concentration. In patch tests, human skin can tolerate contact to solutions containing up to 1% AOS for 24 hours resulting in only mild irritation. Instillation in the rabbit eye of 0.5% AOS caused no irritation after 24 hours, while 1% AOS caused a weak irritation<br><br>The long-term toxicity and potential tumourigenic activity of AOS were assessed in a 2 year feeding study in rats at dietary levels of 0.1, 0.25 and 0.5%. No adverse clinical effects were observed, and survival rates were not affected by treatment with AOS. Histological examination of the tissues did not provide any evidence of toxicity or tumour induction In the Salmonella/microsome assay (Ames test) AOS were tested as negative showing a negligible potential to cause genetic damage.<br><br>AOS were studied in rabbits, mice and rats for teratogenic potential. AOS were administered orally once a day by gavage on day 6-15 of pregnancy in mice and rats and on day 6-18 of pregnancy in rabbits. The doses were from 0.2?600 mg/kg body weight. The study showed no evidence of teratogenic potential.<br><br>Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Torben Madsen et al: Miljoministeriet (Danish Environmental Protection Agency)<br><br>* Van Waters and Rogers ** Albright & Wilson |
| **COCONUT OIL DIETHANOLAMIDE** | For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides)<br>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.<br>The Fatty nitrogen-derived amides (FND amides) comprise four categories:<br>Subcategory I: Substituted Amides<br>Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components)<br>Subcategory III: Imidazole Derivatives<br>Subcategory IV: FND Amphoterics<br>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.<br>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.<br>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.<br>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.<br>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.<br>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.<br>Some typical applications of FND Amides are:<br>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.<br>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.<br>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.<br><br>The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.<br><br>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.<br><br>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 |

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| | 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.<br><br>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).<br><br>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<br><br>Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljoministeriet (Danish Environmental Protection Agency)<br><br>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.<br>Tumours of the kidney and hepatoblastoma are rare spontaneous neoplasms in experimental animals.<br>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.<br>Mechanistic data are very weak to evaluate the carcinogenic potential of coconut oil diethanolamine condensate per se<br>According to IARC:<br>Coconut oil diethanolamine condensate is possibly carcinogenic to humans (Group 2B) |
| **OXYBENZONE** | *Symrise SDS Neo Heliopan BB |
| **DIETHANOLAMINE** | While it is difficult to generalise about the full range of potential health effects posed by exposure to the many different amine compounds, characterised by those used in the manufacture of polyurethane and polyisocyanurate foams, it is agreed that overexposure to the majority of these materials may cause adverse health effects.<br>▸ Many amine-based compounds can induce histamine liberation, which, in turn, can trigger allergic and other physiological effects, including bronchoconstriction or bronchial asthma and rhinitis.<br>▸ Systemic symptoms include headache, nausea, faintness, anxiety, a decrease in blood pressure, tachycardia (rapid heartbeat), itching, erythema (reddening of the skin), urticaria (hives), and facial edema (swelling). Systemic effects (those affecting the body) that are related to the pharmacological action of amines are usually transient.<br>Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion.<br>**Inhalation:**<br>Inhalation of vapors may, depending upon the physical and chemical properties of the specific product and the degree and length of exposure, result in moderate to severe irritation of the tissues of the nose and throat and can irritate the lungs.<br>Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure.<br>Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains.<br>Chronic exposure via inhalation may cause headache, nausea, vomiting, drowsiness, sore throat, bronchopneumonia, and possible lung damage. Also, repeated and/or prolonged exposure to some amines may result in liver disorders, jaundice, and liver enlargement. Some amines have been shown to cause kidney, blood, and central nervous system disorders in laboratory animal studies.<br>While most polyurethane amine catalysts are not sensitisers, some certain individuals may also become sensitized to amines and may experience respiratory distress, including asthma-like attacks, whenever they are subsequently exposed to even very small amounts of vapor. Once sensitised, these individuals must avoid any further exposure to amines. Although chronic or repeated inhalation of vapor concentrations below hazardous or recommended exposure limits should not ordinarily affect healthy individuals, chronic overexposure may lead to permanent pulmonary injury, including a reduction in lung function, breathlessness, chronic bronchitis, and immunologic lung disease.<br>Inhalation hazards are increased when exposure to amine catalysts occurs in situations that produce aerosols, mists, or heated vapors. Such situations include leaks in fitting or transfer lines. Medical conditions generally aggravated by inhalation exposure include asthma, bronchitis, and emphysema.<br>**Skin Contact:**<br>Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis.<br>Skin contact with some amines may result in allergic sensitisation. Sensitised persons should avoid all contact with amine catalysts. Systemic effects resulting from the absorption of the amines through skin exposure may include headaches, nausea, faintness, anxiety, decrease in blood pressure, reddening of the skin, hives, and facial swelling. These symptoms may be related to the pharmacological action of the amines, and they are usually transient.<br>**Eye Contact:**<br>Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations.<br>Direct contact with the liquid amine may cause severe irritation and tissue injury, and the “burning” may lead to blindness. (Contact with solid products may result in mechanical irritation, pain, and corneal injury.)<br>Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling.<br>The corneal swelling may manifest itself in visual disturbances such as blurred or “foggy” vision with a blue tint (“blue haze”) and sometimes a halo phenomenon around lights. These symptoms are transient and usually disappear when exposure ceases.<br>Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation.<br>**Ingestion:**<br>The oral toxicity of amine catalysts varies from moderately to very toxic.<br>Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract.<br>Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs.<br>Affected persons also may experience pain in the chest or abdomen, nausea, bleeding of the throat and the gastrointestinal tract, diarrhea, dizziness, drowsiness, thirst, circulatory collapse, coma, and even death.<br>**Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000**<br>**Alliance for Polyurethanes Industry**<br><br>**WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans. |
| **Meguiar's A25 - Soft Wash Gel & OXYBENZONE** | The following information refers to contact allergens as a group and may not be specific to this product.<br>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 |

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| | 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. |
| **Meguiar's A25 - Soft Wash Gel & WATER** | No significant acute toxicological data identified in literature search. |
| **COCONUT OIL DIETHANOLAMIDE & OXYBENZONE & DIETHANOLAMINE** | 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. |
| **COCONUT OIL DIETHANOLAMIDE & DIETHANOLAMINE** | 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.<br>for diethanolamine (DEA):<br>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.<br>**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.<br>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.<br>**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.<br>**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:<br>▸ DEA is not genotoxic<br>▸ tumour development occurred at doses also associated with chronic hyperplasia<br>▸ there was no dose-related increase in malignancy, multiplicity of tumours or decrease in latency period<br>▸ tumours occurred late in life<br>▸ tumour response was species-specific (only mice were affected, not rats)<br>▸ tumour response was sex-specific (only male mice were affected, not females)<br>▸ tumour development was site-specific, with only liver and kidney affected, both sites of DEA accumulation;<br>▸ there was no tumour response in skin, despite evidence of chronic dermal toxicity<br>▸ there is a plausible mechanism, supported by various data, to explain the renal toxicity of DEA<br>▸ data support threshold mechanisms of renal carcinogenesis for a number of non-genotoxic chemicals<br>▸ 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).<br>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.<br>**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.<br>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.<br>The NOELs for mice and rats derived in this hazard assessment were as follows:<br>Anaemia in rats: 9.5 mg/kg/d (based on microcytic anemia)<br>Organ toxicity in mice: 2.2 mg/kg/d (based on liver toxicity)<br>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<br>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.<br>Based on measured bioavailability in mice and rats, the bioavailable NOELs corresponding to the foregoing were:<br>Anaemia in rats: 0.8 mg/kg/d (based on microcytic anemia)<br>Organ toxicity in mice: 0.55 mg/kg/d (based on liver toxicity)<br>**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.<br>**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 |

**Continued...**

| | |
|---|---|
| | 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.<br>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 |

| | | | |
|---|---|---|---|
| **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

**Meguiar's A25 - Soft Wash Gel**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**sodium C14-16-olefin sulfonate**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 0.7mg/L | 2 |
| EC50 | 48 | Crustacea | 4.53mg/L | 2 |
| EC50 | 72 | Algae or other aquatic plants | 5.2mg/L | 2 |
| EC10 | 72 | Algae or other aquatic plants | 3.9mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | 3.2mg/L | 2 |

**coconut oil diethanolamide**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| Not Available | Not Available | Not Available | Not Available | Not Available |

**oxybenzone**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 3.8mg/L | 2 |
| EC50 | 48 | Crustacea | 1.87mg/L | 2 |
| EC50 | 72 | Algae or other aquatic plants | 0.41mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | 0.08mg/L | 2 |

**diethanolamine**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| LC50 | 96 | Fish | 1-480mg/L | 2 |
| EC50 | 48 | Crustacea | =28.8mg/L | 1 |
| EC50 | 96 | Algae or other aquatic plants | =2.1-2.3mg/L | 1 |
| EC10 | 72 | Algae or other aquatic plants | 0.7mg/L | 2 |
| NOEC | 72 | Algae or other aquatic plants | 0.6mg/L | 2 |

**water**

| ENDPOINT | TEST DURATION (HR) | SPECIES | VALUE | SOURCE |
|---|---|---|---|---|
| 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 allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for diethanolamine (DEA):
log Kow : -1.43
Koc : 4
Half-life (hr) air : 4
Henry's atm m3 /mol: 5.35E-14
BOD 5: 0.03-0.1,0.9%
BOD 28: 57 mg/gm
COD : 1590 mg/gm
TPC 470 mg/gm
ThOD : 2.13
BCF : <1
Based on its physicochemical properties and biodegradation characteristics , DEA is not expected to pose a high risk to drinking water, and its potential for bioconcentration in aquatic organisms is low. DEA is categorized as "practically nontoxic" on an acute basis to freshwater invertebrates, estuarine/marine invertebrates, and freshwater plants

**Continued...**

**Environmental fate:**
In soil and water, DEA is expected to biodegrade fairly rapidly following acclimation (half-life on the order of days to weeks). In soil, DEA should leach. In the atmosphere, DEA is expected to exist almost entirely in the vapor phase. Reaction with photochemically generated hydroxyl radicals is expected to be the dominant removal mechanism (half-life, four hours). This compound may also be removed from the atmosphere in precipitation. The Henry's Law constant for DEA is 3.87x10-11atm.m3/mol which suggests that DEA is essentially nonvolatile from water. The half-life for DEA vapour reacting with photochemically generated hydroxyl radicals in the atmosphere has been estimated to be four hours based on an estimated reaction rate constant of 8.9x10-11 cm3/molecules/sec at 25°C and an average ambient hydroxyl concentration of 5x10+5 molecules/cm3.
DEA, in the presence of nitrites, can form N-nitrosodiethanolamine (NDELA). In air, NDELA is expected to exist solely as a vapor based on a vapor pressure of 2.78 x 10-4 mmHg at 25 C . Vapor-phase NDELA will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with an estimated half-life of 13 hours. NDELA is stable to direct photolysis. In soil, an estimated Koc of 4.8 suggests that this compound is expected to have very high mobility; it is expected to biodegrade slowly in soil. In summary, it appears that DEA is relatively short lived and that it does not present a high risk to contaminate drinking waters. NDELA, a potential formation product, is persistent to biotic and abiotic processes, and mobile. The amounts formed are uncertain (it is only indicated that the half-life is in the order of days to weeks). The water quality criteria (WQC) for nitrosamines is 0.0008 ug/L (U.S. Clean Water Act)
DEA's potential for bioconcentration in aquatic organisms is low
At very low concentrations (about 10 ppm) diethanolamine can be degraded in biological wastewater treatment plants.
**Ecotoxicity:**
Fish LC50 (96 h): Fathead minnow 100 mg/l; (48 h): Bluegill sunfish 1850 mg/l
Daphnia magna LC50 (48 h): 109 mg/l
DEA is categorized as ranging from moderately toxic to practically nontoxic to freshwater invertebrates based on EC50 values ranging from 2.15 to 306 mg/L.
DEA is categorized as "practically nontoxic" to estuarine/ marine invertebrates. EC50 values for estuarine/ marine invertebrates (shrimps and mollusks) exposed to DEA ranged from >100 to 2,800 mg/L.
DEA is categorised as practically nontoxic to freshwater plants on an acute basis based on EC50 values ranging from 103 to 523 mg/L.
For surfactants:
**Environmental fate:**
Octanol/water partition coefficients cannot easily be determined for surfactants because one part of the molecule is hydrophilic and the other part is hydrophobic. Consequently they tend to accumulate at the interface and are not extracted into one or other of the liquid phases. As a result surfactants are expected to transfer slowly, for example, from water into the flesh of fish. During this process, readily biodegradable surfactants are expected to be metabolised rapidly during the process of bioaccumulation. This was emphasised by the OECD Expert Group stating that chemicals are not to be considered to show bioaccumulation potential if they are readily biodegradable.
Several anionic and nonionic surfactants have been investigated to evaluate their potential to bioconcentrate in fish. BCF values (BCF - bioconcentration factor) ranging from 1 to 350 were found. These are absolute maximum values, resulting from the radiolabelling technique used. In all these studies, substantial oxidative metabolism was found resulting in the highest radioactivity in the gall bladder. This indicates liver transformation of the parent compound and biliary excretion of the metabolised compounds, so that "real" bioconcentration is overstated. After correction it can be expected that "real" parent BCF values are one order of magnitude less than those indicated above, i.e. "real" BCF is <100. Therefore the usual data used for classification by EU directives to determine whether a substance is "Dangerous to the "Environment" has little bearing on whether the use of the surfactant is environmentally acceptable.
**Ecotoxicity:**
Surfactant should be considered to be toxic (EC50 and LC50 values of < 10 mg/L) to aquatic species under conditions that allow contact of the chemicals with the organisms. The water solubility of the chemicals does not impact the toxicity except as it relates to the ability to conduct tests appropriately to obtain exposure of the test species. The acute aquatic toxicity generally is considered to be related to the effects of the surfactant properties on the organism and not to direct chemical toxicity
alpha-Olefine sulfonates (AOS) AOS undergo rapid primary biodegradability with methylene blue active substances ( MBAS) removal between 95 and 100% in 2 to 8 days in river water and inoculated media. The ultimate biodegradability of AOS exceeds the pass requirements in OECD 301 tests for ready biodegradability. report 85% DOC removal in the modified OECD screening test, 85% ThOD in the closed bottle test, and 65-80% ThCO2 in the Sturm test. In activated sludge simulation tests, AOS was removed by 100% MBAS and 88% DOC. The alkene sulfonates and hydroxyalkane sulfonates in commercial AOS are both ultimately biodegraded as approximately 84% ThCO2 was obtained during degradation of C14, C16, and C18 within 27 days, whereas the corresponding 3-hydroxyalkane sulfonates were degraded by approximately 86% under the same conditions.

AOS are not readily degradable under anaerobic conditions Reports indicate a range of 31% to 43% MBAS removal under anoxic conditions indicating primary biodegradation

Algae show toxic effects to growth when exposed 10-100 mg/l for C14-18 AOS.

EC50 values for *Daphnia magna* have been determined within the range 5-50 mg/l for C14-18 AOS . Another study with *Daphnia magna*, showed EC50 values of 16.6 mg/l for C14-16 AOS and 7.7 mg/l for C16-18 AOS.

Studies performed with fish show that the higher homologues of AOS are more toxic than the lower ones. This has been illustrated for different fish species (LC50 (96 h) range 0.5-5.3 mg/l)

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

**DO NOT** discharge into sewer or waterways.

### Persistence and degradability

| Ingredient | Persistence: Water/Soil | Persistence: Air |
|---|---|---|
| oxybenzone | HIGH | HIGH |
| diethanolamine | LOW (Half-life = 14 days) | LOW (Half-life = 0.3 days) |
| water | LOW | LOW |

### Bioaccumulative potential

| Ingredient | Bioaccumulation |
|---|---|
| oxybenzone | LOW (BCF = 160) |
| diethanolamine | LOW (BCF = 1) |
| water | LOW (LogKOW = -1.38) |

### Mobility in soil

| Ingredient | Mobility |
|---|---|
| oxybenzone | LOW (KOC = 1268) |
| diethanolamine | HIGH (KOC = 1) |
| water | LOW (KOC = 14.3) |

## SECTION 13 DISPOSAL CONSIDERATIONS

### Waste treatment methods

**Continued...**

| | |
|---|---|
| **Product / Packaging disposal** | Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.<br>A Hierarchy of Controls seems to be common - the user should investigate:<br>▸ Reduction<br>▸ Reuse<br>▸ Recycling<br>▸ Disposal (if all else fails)<br>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.<br>▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains.<br>▸ It may be necessary to collect all wash water for treatment before disposal.<br>▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.<br>▸ Where in doubt contact the responsible authority.<br>▸ Recycle wherever possible.<br>▸ 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.<br>▸ 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).<br>▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed. |

## 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 C14-16-OLEFIN SULFONATE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
GESAMP/EHS Composite List - GESAMP Hazard Profiles

**COCONUT OIL DIETHANOLAMIDE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)

**OXYBENZONE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Dangerous Goods Code (ADG Code) - Dangerous Goods List
Australia Dangerous Goods Code (ADG Code) - List of Emergency Action Codes
Australia Inventory of Chemical Substances (AICS)
International Air Transport Association (IATA) Dangerous Goods Regulations
International Maritime Dangerous Goods Requirements (IMDG Code)
United Nations Recommendations on the Transport of Dangerous Goods Model Regulations

**DIETHANOLAMINE IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
GESAMP/EHS Composite List - GESAMP Hazard Profiles
IMO IBC Code Chapter 17: Summary of minimum requirements
IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in Bulk
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances
IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named) mixtures containing at least 99% by weight of components already assessed by IMO, presenting safety hazards
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International FOSFA List of Banned Immediate Previous Cargoes

**WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Inventory of Chemical Substances (AICS)
IMO IBC Code Chapter 18: List of products to which the Code does not apply

### National Inventory Status

| National Inventory | Status |
|---|---|
| Australia - AICS | Yes |
| Canada - DSL | Yes |
| Canada - NDSL | No (coconut oil diethanolamide; diethanolamine; water; oxybenzone; sodium C14-16-olefin sulfonate) |
| China - IECSC | Yes |
| Europe - EINEC / ELINCS / NLP | Yes |
| Japan - ENCS | No (coconut oil diethanolamide; sodium C14-16-olefin sulfonate) |
| Korea - KECI | Yes |
| New Zealand - NZIoC | Yes |

**Continued...**

| | |
|---|---|
| Philippines - PICCS | Yes |
| USA - TSCA | Yes |
| Taiwan - TCSI | Yes |
| Mexico - INSQ | No (coconut oil diethanolamide; sodium C14-16-olefin sulfonate) |
| Vietnam - NCI | Yes |
| Russia - ARIPS | No (coconut oil diethanolamide) |
| ***Legend:*** | *Yes = All CAS declared ingredients are on the inventory*<br>*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** | 04/09/2019 |
| **Initial Date** | 01/11/2001 |

### SDS Version Summary

| Version | Issue Date | Sections Updated |
|---|---|---|
| 4.1.1.1 | 06/03/2015 | Classification |
| 6.1.1.1 | 04/09/2019 | Appearance, Physical Properties |

### 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



**end of SDS**