Meguiar's G177 - Ultimate Wash & Wax
Motor Active

Chemwatch Hazard Alert Code: **2**

Chemwatch: **23-5522**
Version No: **5.1.1.1**
Safety Data Sheet according to WHS and ADG requirements

Issue Date: **01/11/2019**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Meguiar's G177 - Ultimate Wash & Wax
Synonyms	Product Code: 21-47C, G17748, G177475, G17716EU
Other means of identification	Not Available

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

Relevant identified uses	Surface cleaner. Use according to manufacturer's directions.

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	2	
Reactivity	1	
Chronic	2	

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

Poisons Schedule	Not Applicable
Classification [1]	Skin Corrosion/Irritation Category 2, Eye Irritation Category 2A, Skin Sensitizer Category 1, Carcinogenicity Category 2, Acute Aquatic Hazard Category 2, Chronic Aquatic Hazard Category 3
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	**Warning**

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.

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H351	Suspected of causing cancer.
H401	Toxic to aquatic life.
H412	Harmful to aquatic life with long lasting effects.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P281	Use personal protective equipment as required.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
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.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
61789-40-0	5-10	cocamidopropylbetaine
68585-34-2	5-10	sodium (C10-16)pareth sulfate
61790-63-4	0.5-3	coconut oil diethanolamide
111-42-2	0-0.5	diethanolamine

SECTION 4 First aid measures

Description of first aid measures

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

Treat symptomatically.

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SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves in the event of a fire. ‣ Prevent, by any means available, spillage from entering drains or water courses. ‣ Use fire fighting procedures suitable for surrounding area. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	‣ Non combustible. ‣ Not considered a significant fire risk, however containers may burn. Combustion products include: carbon dioxide (CO_2) nitrogen oxides (NOx) sulfur oxides (SOx) other pyrolysis products typical of burning organic material. 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. ‣ 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	Moderate hazard. ‣ Clear area of personnel and move upwind. ‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear breathing apparatus plus protective gloves. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Stop leak if safe to do so. ‣ Contain spill with sand, earth or vermiculite. ‣ Collect recoverable product into labelled containers for recycling. ‣ Neutralise/decontaminate residue (see Section 13 for specific agent). ‣ Collect solid residues and seal in labelled drums for disposal. ‣ Wash area and prevent runoff into drains. ‣ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ‣ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ **DO NOT** allow clothing wet with material to stay in contact with skin ‣ Avoid all personal contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Avoid contact with moisture. ‣ 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.

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Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Polyethylene or polypropylene container. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	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
cocamidopropylbetaine	Not Available	Not Available
sodium (C10-16)pareth sulfate	Not Available	Not Available
coconut oil diethanolamide	Not Available	Not Available
diethanolamine	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
cocamidopropylbetaine	E	≤ 0.1 ppm
sodium (C10-16)pareth sulfate	E	≤ 0.1 ppm
coconut oil diethanolamide	E	≤ 0.1 ppm
Notes:	*Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.*	

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 special circumstances. If risk of overexposure exists, wear approved respirator. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. Provide adequate ventilation in warehouses and enclosed 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.)
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:

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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 only likely to give short duration protection and would normally be just for single use applications, then disposed of.
· Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

See Other protection below

Other protection

- Overalls.
- P.V.C apron.
- Barrier cream.
- Skin cleansing cream.
- Eye wash unit.

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".

Respiratory protection

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

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The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
Meguiar's G177 - Ultimate Wash & Wax

Material	CPI
BUTYL	A
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NITRILE	A
PVC	A
TEFLON	A
VITON	A

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

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	Yellow green liquid with a sweet citrus odour; miscible with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.00
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	9.0	**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	as for water	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Available	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Available	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available

SECTION 10 Stability and reactivity

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

SECTION 11 Toxicological information

Information on toxicological effects

Inhaled	The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal

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	models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
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	Limited 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. Anionic surfactants/ hydrotropes generally produce skin reactions following the removal of natural oils. The skin may appear red and may become sore. Papular dermatitis may also develop. Sensitive individuals may exhibit cracking, scaling and blistering. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	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. Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems. There exists limited evidence that shows that skin contact with the material is capable either of inducing a sensitisation reaction in a significant number of individuals, and/or of producing positive response in experimental animals. Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and inflammatory or fibrotic pulmonary disease. Results of repeated exposure tests with diethanolamine (DEA) in laboratory animals include anaemia (rats) and effects on the kidneys (rats and mice) and liver (mice). DEA produces nervous system injury in dogs and rats. Heart and salivary gland lesions have also been seen in mice treated cutaneously with DEA and in mice receiving DEA in drinking water. Rats given high doses of DEA developed anaemia and testicular lesions. Exaggerated doses of DEA produced heart and nervous system effects in other animals. Changes in other organs were judged to be secondary due to the poor health of animals subjected to extremely high doses of DEA. Rats, rabbits and guinea pigs exposed to high vapour concentrations of volatile monoethanolamine (MEA) (up to 1250 ppm) for periods of up to 5 weeks developed pulmonary, hepatic and renal lesions. Dogs, rats and guinea pigs exposed to 100 ppm MEA for 30 days, became apathetic and developed poor appetites. Animal tests also indicate that inhalation exposure to MEA may result in nervous system injury. All species exposed to airborne MEA experienced dermal effects, varying from ulceration to hair loss probably resulting from contact with the cage. An increased incidence of skeletal variations, suggestive of a slight developmental delay was seen in the foetuses of rats given 1500 mg/kg/day DEA cutaneously; this also produced significant maternal toxicity. No foetal malformations, however, were seen in rats nor in rabbits receiving identical treatment. The foetus of rats given high doses of MEA by gavage, showed an increased rate of embryofoetal death, growth retardation, and some malformations including hydronephrosis and hydroureter. The high doses required to produce these effects bring into question the relevance of this finding to humans. There is some evidence that embryofoetotoxicity and teratogenicity does not occur in rats when MEA is administered by dermal application to the mother. The National Toxicology Program (NTP) concluded that there is clear evidence of liver tumours and some evidence of kidney tumours in mice exposed dermally to DEA over their lifetime. Chronic skin painting studies in mice of both sexes produced liver tumours and an increased incidence of kidney tumours in male mice. The significance of these findings to humans is unclear as DEA is neither genotoxic, mutagenic nor clastogenic, and did not induce tumours in rats or transgenic mice similarly treated. Alkanolamines (especially those containing a secondary amine moiety) may react with nitrites or other nitrosating agents to form carcinogenic N-nitrosamines. Alkanolamines are metabolised by biosynthetic routes to ethanolamine and choline and incorporated into phospholipids. They are excreted predominantly unchanged with a half-life of approximately one week. In the absence of sodium nitrite, no conversion to carcinogenic N-nitrosamines was observed. Diethanolamine competitively inhibits the cellular uptake of choline, in vitro, and hepatic changes in choline homeostasis, consistent with choline deficiency, are observed in vivo. Many amines are potent skin and respiratory sensitisers and certain individuals especially those described as "atopic" (i.e. those predisposed to asthma and other allergic responses) may show allergic reactions when chronically exposed to alkanolamines. In a study with coconut diethanolamide, the National Toxicology Program (Technical Report Series 479), showed clear evidence of carcinogenic activity in male B6C3F1 mice based on increased incidences of hepatic and renal tubule neoplasms and in female B6C3F1 mice based on increased incidences of hepatic neoplasms. There was equivocal evidence of carcinogenic activity in female F344/N rats based on a marginal increase in the incidence of renal tube neoplasms. These increases were associated with the concentration of free diethanolamine present as a contaminant in the diethanolamine condensate. Exposure to rats to coconut oil diethanolamine condensate by dermal application in ethanol for 2 years resulted in epidermal hyperplasia, sebaceous gland hyperplasia, hyperkeratosis and parakeratosis in males and females and ulcer in females at the site of application. There were increases in the incidences of chronic inflammation, epithelial hyperplasia, and epithelial ulcer in the forestomach of female rats. The severity of nephropathy in dosed female rats were increased. Exposure of mice to coconut oil diethanolamine condensate by dermal application for 2 years resulted in increased incidences of eosinophilic foci of the liver in males. Increased incidences of epidermal hyperplasia, sebaceous gland hyperplasia, and hyperkeratosis in males and females, ulcer in males, and parakeratosis and inflammation in females at the site of application and of follicular cell hyperplasia in the thyroid gland of males and females, were chemical related.

Meguiar's G177 - Ultimate Wash & Wax	**TOXICITY**	**IRRITATION**
	Not Available	Not Available

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cocamidopropylbetaine	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: =4900 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (rat) LD50: 2700 mg/kg[2]	Eye: primary irritant *
		Skin: adverse effect observed (irritating)[1]
		Skin: primary irritant *
sodium (C10-16)pareth sulfate	**TOXICITY**	**IRRITATION**
	Oral (rat) LD50: 1600 mg/kg[2]	Skin (rabbit):25 mg/24 hr moderate
coconut oil diethanolamide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
diethanolamine	**TOXICITY**	**IRRITATION**
	Oral (guinea pig) LD50: ~2000 mg/kg[2]	Eye (rabbit): 5500 mg - SEVERE
	Oral (mouse) LD50: =3300 mg/kg[2]	Eye (rabbit):0.75 mg/24 hr SEVERE
	Oral (rat) LD50: ~1600 mg/kg[2]	Eye: adverse effect observed (irritating)[1]
	Oral (rat) LD50: ~2000 mg/kg[2]	Skin (rabbit): 50 mg (open)-mild
	Oral (rat) LD50: =1410 mg/kg[2]	Skin (rabbit): 500 mg/24 hr-mild
	Oral (rat) LD50: =3460 mg/kg[2]	Skin: adverse effect observed (irritating)[1]
	Oral (rat) LD50: =3540 mg/kg[2]	
	Oral (rat) LD50: =710 mg/kg[2]	
	Oral (rat) LD50: 1820 mg/kg[2]	
	Oral (rat) LD50: 620 mg/kg[2]	
Legend:	*1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances*	

COCAMIDOPROPYLBETAINE

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.
Possible cross-reactions to several fatty acid amidopropyl dimethylamines were observed in patients that were reported to have allergic contact dermatitis to a baby lotion that contained 0.3% oleamidopropyl dimethylamine.
Stearamidopropyl dimethylamine at 2% in hair conditioners was not a contact sensitiser when tested neat or diluted to 30%. However, irritation reactions were observed.
A 10-year retrospective study found that out of 46 patients with confirmed allergic eyelid dermatitis, 10.9% had relevant reactions to oleamidopropyl dimethylamine and 4.3% had relevant reactions to cocamidopropyl dimethylamine.
Several cases of allergic contact dermatitis were reported in patients from the Netherlands that had used a particular type of body lotion that contained oleamidopropyl dimethylamine.
In 12 patients tested with their personal cosmetics, containing the fatty acid amidopropyl dimethylamine cocamidopropyl betaine (CAPB), 9 had positive reactions to at least one dilution and 5 had irritant reactions. All except 3 patients, who were not tested, had 2 or 3+ reaction to the 3,3-dimethylaminopropylamine (DMAPA, the reactant used in producing fatty acid amidopropyl dimethylamines) at concentrations as low as 0.05%. The presence of DMAPA was investigated via thin-layer chromatography in the personal cosmetics of 4 of the patients that had positive reactions. DMAPA was measured in the products at 50 - 150 ppm suggesting that the sensitising agent in CAPB-induced allergy is DMAPA, .
The sensitisation potential of a 4% aqueous liquid fabric softener formulation containing 0.5% stearyl/palmitylamidopropyl dimethylamine was investigated using. The test material caused some irritation in most volunteers. After a rest period of 2 weeks, the subjects received challenge patches with the same concentration of test material on both arms. Patch sites were graded 48 and 96 h after patching. Eight subjects reacted at challenge, and 7 of the eight submitted to rechallenge with 4% and 0.4% aqueous formulations. No reactions indicative of sensitisation occurred at rechallenge. The test formulation containing stearyl/palmitylamidopropyl dimethylamine had no significant sensitisation potential.subjects.
Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41.
The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
For betaines:
Several sources revealing data on skin irritation, skin sensitisation and dermal absorption in humans are available for CAS 683-10-3, the C12-alkyldimethyl betaine, which is the most frequently occurring betaine because it is one of the components of most of the substances of the alkyldimethyl betaine group, among those also Betaines, C12-14 (even numbered)-alkyldimethyl. Therefore, read-across of exposure-related observations in humans from CAS 683-10-3 is justified. Data from several human closed patch tests demonstrate skin irritation in humans ranging from mild to strong under occlusive conditions even with concentrations as low as 1%. In contrast, exposure to 0.1% under open conditions did not induce any positive skin reactions.
Information on skin sensitisation in humans is available from a closed human patch test with CAS 683-10-3. After an induction period of 6 days with 0.1% of active ingredient, followed by a treatment-free period of 10 days, the volunteers were challenged under occlusive conditions for 24 hours. No reactions were observed immediately after challenge; during the next 4 days only irritation reactions were observed.
This finding was further supported by industrial medical monitoring data. Workers involved in the production of CAS 683-10-3 are routinely checked every 3 years for signs of skin sensitisation, respiratory irritation, skin irritation and eye irritation. During these examinations no signs of the aforementioned disorders were observed which were related to the test substance.
Moreover, a study focusing on dermal uptake of C12-alkyldimethyl betaine) into human skin and the effects of surfactants on skin barrier function demonstrated that only up to 0.4% of the applied dose was absorbed within 30 minutes of exposure, with absorbance being dependent on the concentration applied. Tape stripping of the skin revealed that the administered test substance was primarily located in the outer stratum corneum layer
Test material does not demonstrate mutagenic or clastogenic effects in bacteria or mammalian cells in vitro
" REACH Dossier

Continued...

	Amphoteric surfactants are easily absorbed in the intestine and are excreted partly unchanged via the faeces. Metabolisation to CO2 and short-chained fatty acids also occur. No tendency to accumulation in the organism or storage of betaines in certain organs has been detected. Betaines generally have a low acute toxicity. E.g., LD50 values for cocoamidopropylbetaine (30% solution) by oral administration have been determined to 4,910 mg/kg body weight in rats. Betaines do not carry any net charge, and, therefore, they can only form hydrophobic bonds with proteins in the skin. This may be the explanation for the low protein denaturation potential of betaines as the ion-binding of other surfactants contributes to denaturation. In combination with anionic surfactants a positive synergistic effect with regard to skin compatibility is often found. Compared to a 20% solution of C12 alkyl sulfate (AS; sodium lauryl sulfate) alone, decreased erythema was observed for the combination of 20% C12 AS and 10% cocoamidopropyl betaine one hour after the removal of patches. The combination of cocoamidopropyl betaine and C12 AS also reduced swelling of the skin, and generally interactions between amphoterics and AS produce less swelling and result in milder skin reactions. Concentrated betaines are expected to be irritant to skin and eyes. Diluted solutions (3-10%) are not irritant to skin, but they are mildly irritant to the eyes (4.5%) No evidence of delayed contact hypersensitivity was found in guinea pigs after topically administrated solutions of 10% cocoamidopropyl betaine by using the Magnusson-Kligman maximization test. Various instances of contact allergy to cocoamidopropyl betaine have been reported. In all of the reports it was concluded that the observed skin reactions were due to the presence of 3-dimethylaminopropylamine which is an impurity in cocoamidopropyl betaine. This impurity is an intermediate in the synthesis of alkylamidopropyldimethylamines that are intermediates in the synthesis of the corresponding alkylamido betaines. Cocoamidopropyl betaine was proven to be non-mutagenic to Salmonella typhimurium in the Ames Salmonella/microsome reverse mutation assay. Short-term genotoxicity tests have shown negative results of mutagenicity for lauryl betaine in various strains of Salmonella typhimurium. * [Van Waters and Rogers] ** [Canada Colors and Chemicals Ltd.] Toxicokinetics, metabolism and distribution. Absorption of the chemical across dermal and gastrointestinal membranes is possible based on the relatively low molecular weight of the chemical (500 Da) and given that it is a surfactant (EC, 2003). Acute toxicity. Acute oral toxicity studies in rats and mice indicated that the LD50 values of the chemical (at 30-35.61% concentration) ranged from 1800 mg/kg bw (male rats) up to 5000 mg/kg bw, with mortalities noted in most studies (CIR, 2010). Of note is an acute oral toxicity study conducted in Sprague-Dawley rats (5/sex) at a single dose of 1800 mg/kg bw (formulation containing 35.61% of the chemical), where no males but all five females died. Overall, the data suggests that mortality occurs following oral administration of the chemical and that it may be an acute oral toxicant. Therefore, based on these data the chemical may be harmful if swallowed. An acute dermal toxicity study in rats was conducted using 2000 mg/kg bw of a 31% formulation of the chemical (CIR, 2010). Irritation was observed, but there were no clinical signs of systemic toxicity or mortalities. The lack of effects in this study suggests that the chemical is likely to be of low acute dermal toxicity. Irritation. The chemical has a quaternary ammonium functional group, which is a structural alert for corrosion Numerous skin irritation studies, conducted with formulations containing 7.5-30% of the chemical, indicated that the chemical has irritant properties. The studies were, in-general, conducted under occlusive conditions, with exposure times of up to 24 hours (7.5-10%). Based on the information available, the chemical is likely to be a skin irritant. Eye irritation studies with the chemical showed that corrosive and necrotic effects occurred at 30% whereas less severe effects were observed at lower concentrations of 2.3-10% The chemical is classified with the risk phrase R36: Irritating to eyes, however, based on studies conducted on the chemical it may be a severe eye irritant. Sensitisation. The chemical has a quaternary ammonium functional group, which is a structural alert for sensitisation (Conflicting results have been obtained with the chemical in animal studies. Positive results were reported in an LLNA study (an EC3 value was not reported). In addition, positive results were obtained in two guinea pig maximisation studies conducted by a single laboratory, the first at 3% induction and 3% challenge, and the second at 0.15% induction and 0.015% challenge. However, there was no sensitisation in a guinea pig maximisation test when the chemical was tested at 6% induction and 1% challenge. In addition, no sensitisation was observed in another test in guinea pigs at 0.75% induction and 0.02% challenge. No evidence of sensitisation was reported in a HRIPT on a formulation containing the chemical at 0.6% concentration (a 10% dilution of a ~6% formulation) with 110 volunteers. In HRIPT studies on formulations containing the chemical, no evidence of sensitisation was reported at concentrations of 1.87% (88 subjects), 0.93% (93 subjects), 0.3% (100 subjects), 1.5-3.0% (141 subjects), 6.0% (210 subjects), 0.018% (27 subjects). However, positive results were observed in provocative studies conducted on formulations containing the chemical (at 0.3-1% concentration), conducted in subjects diagnosed with various forms of contact dermatitis, suggesting that the chemical may cause reactions in sensitive individuals In one study authors note that sensitisation effects of the chemical (and related compounds) are most likely due to the impurities, including DMAPA and amidopropyl dimethylamines, however, they do not exclude the possibility of the causing the sensitisation. The potential for skin sensit
SODIUM (C10-16)PARETH SULFATE	No significant acute toxicological data identified in literature search. Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air. Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture . On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing. Allergic Contact Dermatitis—Formation, Structural Requirements,and Reactivity of Skin Sensitizers. Ann-Therese Karlberg et al; Chem. Res. Toxicol.2008,21,53-69 Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners. PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below 20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used Safety Evaluation of Polyethyene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology http://doi.org/10.5487/TR.2015.31.2.105 Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) (syn: AAASD ,alkyl alcohol alkoxylate sulfates, SLES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3E0) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes). AES are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC. In assessing this family the Cosmetic Ingredient Review (CIR) Expert Panel recognized that most of the acute oral toxicity, dermal irritation and sensitization, subchronic and chronic oral toxicity, reproductive and developmental toxicity, carcinogenicity, and photosensitization studies have been conducted on ammonium laureth sulfate and sodium laureth sulfate. Sodium and ammonium laureth sulfate have not evoked adverse responses in any toxicological testing, including acute oral toxicity, sub-chronic and chronic oral toxicity, reproductive and develop-mental toxicity, carcinogenicity, and photosensitization studies. These data, however, are considered a sufficient basis for concluding that the other ingredients are safe in the practices of use and concentration described in the safety assessment because of the fundamental chemical

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similarities between them and because they all are chemically similar salts(salts are expected to be dissociated in any product formulation independent of whether the salt is sodium, ammonium, magnesium, or zinc) of sulfated ethoxylated alcohols, and they all function as surfactants in cosmetic formulations. Based on these considerations, safety test data on one ingredient may be extrapolated to all of them. The panel noted that sodium laureth sulfate and ammonium laureth sulfate can produce eye and/or skin irritation in experimental animals and in some human test subjects; irritation may occur in some users of cosmetic formulations containing these ingredients. The irritant effects, however, are similar to those produced by other detergents, and the severity of the irritation appears to increase directly with concentration

Acute toxicity: AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact sensitiser and AES is not expected to be irritating to the skin at in-use concentrations. The available repeated dose toxicity data demonstrate the low toxicity of AES. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. The consumer aggregate exposure from direct and indirect skin contact as well as from the oral route via dishware residues results in an estimated total body burden of 29 ug /kg bw/day.

AES are easily absorbed in the intestine in rats and humans after oral administration. Radiolabelled C11 AE3S and C12 AE3S were extensively metabolized in rats and most of the 14C-activity was eliminated via the urine and expired air independently of the route of administration (oral, intraperitoneal or intravenous). The main urinary metabolite from C11 AE3S is propionic acid-3-(3EO)-sulfate. For C12 and C16 AE3S, the main metabolite is acetic acid-2-(3EO)-sulfate. The alkyl chain appears to be oxidised to CO2 which is expired. The EO-chain seems to be resistant to metabolism.

AES are better tolerated on the skin than, e.g., alkyl sulfates and it is generally agreed that the irritancy of AES is lower than that of other anionic surfactants. Alkyl chain lengths of 12 carbon atoms are considered to be more irritating to the skin compared to other chain lengths. The skin irritating properties of AES normally decrease with increasing level of ethoxylation. Undiluted AES should in general be considered strongly irritating. Even at concentrations of 10% moderate to strong effects can be expected. However, only mild to slight irritation was observed when a non-specified AES was applied at 1% to the skin.

Subchronic toxicity: A 90-day subchronic feeding study in rats with 1% of AE3S or AE6S with alkyl chain lengths of C12-14 showed only an increased liver/body weight ratio. In a chronic oral study with a duration of 2 years, doses of C12-AE3S of 0.005 - 0.05% in the diet or drinking water had no effects on rats. The concentration of 0.5% sometimes resulted in increased kidney or liver weight.

Subchronic 21-day repeat dose dietary studies showed low toxicity of compounds with carbon lengths of C12-15, C12-14 and C13-15 with sodium or ammonium alkyl ethoxylates with POE (polyoxyethylene) n=3. One study indicated that C16-18 POE n=18 had comparable low toxicity. No-observed-adverse-effect levels (NOAELs) range from 120 to 468 mg/kg/day, similar to a NOAEL from a 90-day rat gavage study with NaC12-14 POE n=2(CAS RN 68891-38-3), which was reported to be 225 mg/kg/day. In addition, another 90-day repeat dose dietary study with NaC12-15 POE n=3 (CAS RN 68424-50-0) resulted in low toxicity, with a NOAEL of greater than approximately 50 mg/kg/day (calculated based on dose of 1000 ppm in diet). Effects were usually related to hepatic hypertrophy, increased liver weight, and related increases in haematological endpoints related to liver enzyme induction.

Reproductive and developmental toxicity: No evidence of reproductive and teratogenic effects was seen in a two-generation study in rats fed with a mixture (55:45) of AES and linear alkylbenzene sulfonates. Dietary levels of 0.1, 0.5, and 1% were administered to the rats either continuously or during the period of major organogenesis during six pregnancies. No changes in reproductive or embryogenic parameters were observed.

Based on this study an overall no-observed-adverse-effect level (NOAEL) for systemic effects was 0.1%, which was 86.6 mg/kg/day for the F0 generation, and 149.5 mg/kg/day for the F1 generation. The NOAEL of 86.6 mg/kg/day was selected as the toxicology endpoint for the chronic risk assessment for the sulfate derivatives.

Carcinogenicity: Chronic dietary studies conducted with rats showed no incidence of cancer and no effects at the concentrations tested (lowest dose tested was ca 75 mg/kg/day).

NOTE: Some products containing AES/ SLES have been found to also contain traces (up to 279 ppm) of 1,4-dioxane; this is formed as a by-product during the ethoxylation step of its synthesis. The U.S. Food and Drug Administration recommends that these levels be monitored.The U.S. Environmental Protection Agency classifies 1,4-dioxane to be a probable human carcinogen (not observed in epidemiological studies of workers using the compound, but resulting in more cancer cases in controlled animal studies), and a known irritant with a no-observed-adverse-effects level of 400 milligrams per cubic meter at concentrations significantly higher than those found in commercial products. Under Proposition 65, 1,4-dioxane is classified in the U.S. state of California to cause cancer. The FDA encourages manufacturers to remove 1,4-dioxane, though it is not required by federal law.

Sensitising potential: Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

Sensitization studies in guinea pigs revealed that the pure nonoxidized surfactant itself is nonsensitizing but that many of the investigated oxidation products are sensitizers. Two hydroperoxides were identified in the oxidation mixture, but only one (16-hydroperoxy-3,6,9,12,15-pentaoxaheptacosan-1-ol) was stable enough to be isolated. It was found to be a strong sensitizer in LLNA (local lymph node assay for detection of sensitization capacity). The formation of other hydroperoxides was indicated by the detection of their corresponding aldehydes in the oxidation mixture .

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing

Toxicokinetics:

Following oral exposure, AES is readily absorbed in the gastrointestinal tract in human and rat and excreted principally via the urine or faeces depending on the length of the ethoxylate chain but independently of the route of administration. Once absorbed, AES is extensively metabolized by beta- or omega oxidation. The alkyl chain appears to be oxidized to CO2 which is expired. The EO-chain seems to be resistant to metabolism. Regarding the different anions, it is expected that the salts will be converted to the acid form in the stomach. This means that for all types of parent chemical the same compound structure eventually enters the small intestine. Hence, the situation will be similar for compounds originating from different salts and therefore no differences in uptake are anticipated.

The length of the ethoxylate portion in an AES molecule seems to have an important impact on the biokinetics of AES in humans and in the rat. Alcohol ethoxysulfates with longer ethoxylate chains (>7-9 EO units) are excreted at a higher proportion in the faeces. This is however not of interest for the AES within this category as their ethoxylation grade is 1 to 2.5.

Dermal absorption

There are two reliable and relevant studies available assessing the dermal absorption rate of AES. The study with AES (C12 -14; 2 EO) Na (CAS 68891-38-3) was performed according to OECD guideline 428 with human skin of the abdomen region (3 donors, n=2). The test substance was applied at a concentration of 10% for 24 h

The mean amount removed from the skin surface (skin wash) ranged from 87.16% to 94.56% of the dose applied. The amounts in the receptor could not be quantified, since it was below the analytical limit of quantification (LOQ). The mean recovery in the two first tape strips was 1.48% during all performed experiments. In the further 18 tape strips a mean recovery of 2.86% was documented. The recovery values for the cryocuts have accounted 0.56% in mean.

The mean absorbed dose, sum of the amounts found in the viable epidermis, dermis and receptor medium was 0.56%. The mean recovery values have varied from 90.90% to 100.21%, which complies with the acceptance criteria of 100 ± 15%.

There is also an in vivo study according to OECD guideline 427 for AES (C12 -14; 2 EO) Na (CAS 68891-38-3) available (Aulmann, 1996). Wistar rats were exposed to 1% aqueous solutions of the test item for 15 min and 48 h under semi-occlusive conditions. The mean amount of AES (C12-14; 2 EO) Na (CAS 68891-38-3) removed from the skin surface after the 15 min exposure period (via washing) ranged from 92.8% to 97.2% of the dose and from 91.6% to 98.4% after 48 h when the skin was not washed until sacrifice. The amounts in faeces and skin could not always be quantified, since it was below the analytical limit of quantification (LOQ).

The mean absorbed dose, sum of the amounts found in urine, faeces and skin in the experiment with washing was about 0.1% and 0.9% without washing.

The mean recovery values varied from 98.6% to 103%.

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	Taking the results of both studies together the dermal absorption is very low. The in vitro study with human skin indicated the dermal absorption to be 0.56% within 24 h and the in vivo study indicated the dermal absorption to be 0.9% within 48 h. The mean recovery rates on the skin are greater than 87%. These data demonstrate that the test substance remains on the skin surface. Thus, the value of 0.9% dermal absorption is taken for the dermal absorption. References: Danish EPA - Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products (2001). Environmental Project No. 615, pp. 24-28 HERA (2003). Human & Environmental Risk Assessment on ingredients of European household cleaning products Alcohol Ethoxysulphates, Human Health Risk Assessment Draft, 2003. http: //www. heraproject. com. Final Report of the Amended Safety Assessment of Sodium Laureth Sulfate and Related Salts of Sulfated Ethoxylated Alcohols: (nternational Journal of Toxicology 29 (Supplement 3) 151S-161S: 2010 http://journals.sagepub.com/doi/pdf/10.1177/1091581810373151 for similar product (sodium lauryl ether sulfate)
COCONUT OIL DIETHANOLAMIDE	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. Fatty acid amides (FAA) are ubiquitous in household and commercial environments. The most common of these are based on coconut oil fatty acids alkanolamides. These are the most widely studied in terms of human exposure. Fatty acid diethanolamides (C8-C18) are classified by Comite Europeen des Agents de Surface et de leurs Intermediaires Organiques (CESIO) as Irritating (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes). Fatty acid monoethanolamides are classified as Irritant (Xi) with the risk phrases R41 Several studies of the sensitization potential of cocoamide diethanolamide (DEA) indicate that this FAA induces occupational allergic contact dermatitis and a number of reports on skin allergy patch testing of cocoamide DEA have been published. These tests indicate that allergy to cocoamide DEA is becoming more common. Alkanolamides are manufactured by condensation of diethanolamine and the methylester of long chain fatty acids. Several alkanolamides (especially secondary alkanolamides) are susceptible to nitrosamine formation which constitutes a potential health problem. Nitrosamine contamination is possible either from pre-existing contamination of the diethanolamine used to manufacture cocoamide DEA, or from nitrosamine formation by nitrosating agents in formulations containing cocoamide DEA. According to the Cosmetic Directive (2000) cocoamide DEA must not be used in products with nitrosating agents because of the risk of formation of N-nitrosamines. The maximum content allowed in cosmetics is 5% fatty acid dialkanolamides, and the maximum content of N-nitrosodialkanolamines is 50 mg/kg. The preservative 2-bromo-2-nitropropane-1,3-diol is a known nitrosating agent for secondary and tertiary amines or amides. Model assays have indicated that 2-bromo-2-nitropropane-1,3-diol may lead to the N-nitrosation of diethanolamine forming the carcinogenic compound, N-nitrosodiethanolamine which is a potent liver carcinogen in rats (IARC 1978). Several FAAs have been tested in short-term genotoxicity assays. No indication of any potential to cause genetic damage was seen Lauramide DEA was tested in mutagenicity assays and did not show mutagenic activity in *Salmonella typhimurium* strains or in hamster embryo cells. Cocoamide DEA was not mutagenic in strains of *Salmonella typhimurium* when tested with or without metabolic activation Environmental and Health Assessment of Substances in Household Detergents and Cosmetic Detergent Products, Environment Project, 615, 2001. Miljoministeriet (Danish Environmental Protection Agency) In a study of dermal application in mice, coconut oil diethanolamine condensate (coconut diethanolamide) increased the incidence of hepatocellular carcinoma and hepatocellular adenoma in males and females, and of hepatoblastoma in males. The incidence of renal tubule adenoma and carcinoma combined was also increased in males. In a study of dermal application in rats, no increase in tumour incidence was observed. Tumours of the kidney and hepatoblastoma are rare spontaneous neoplasms in experimental animals. The carcinogenic effects of the coconut oil diethanolamine condensate used in the cancer bioassay may be due to the levels of diethanolamine (18.2%) in the solutions tested. Mechanistic data are very weak to evaluate the carcinogenic potential of coconut oil diethanolamine condensate per se According to IARC: Coconut oil diethanolamine condensate is possibly carcinogenic to humans (Group 2B)
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. ▸ 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. ▸ 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. Typically, there are four routes of possible or potential exposure: inhalation, skin contact, eye contact, and ingestion. **Inhalation:** 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. Products with higher vapour pressures have a greater potential for higher airborne concentrations. This increases the probability of worker exposure. Higher concentrations of certain amines can produce severe respiratory irritation, characterised by nasal discharge, coughing, difficulty in breathing, and chest pains. 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. 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. 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. **Skin Contact:** Skin contact with amine catalysts poses a number of concerns. Direct skin contact can cause moderate to severe irritation and injury-i.e., from

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	simple redness and swelling to painful blistering, ulceration, and chemical burns. Repeated or prolonged exposure may also result in severe cumulative dermatitis. 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. **Eye Contact:** Amine catalysts are alkaline in nature and their vapours are irritating to the eyes, even at low concentrations. 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.) Exposed persons may experience excessive tearing, burning, conjunctivitis, and corneal swelling. 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. Some individuals may experience this effect even when exposed to concentrations below doses that ordinarily cause respiratory irritation. **Ingestion:** The oral toxicity of amine catalysts varies from moderately to very toxic. Some amines can cause severe irritation, ulceration, or burns of the mouth, throat, esophagus,and gastrointestinal tract. Material aspirated (due to vomiting) can damage the bronchial tubes and the lungs. 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. **Polyurethane Amine Catalysts: Guidelines for Safe Handling and Disposal; Technical Bulletin June 2000** **Alliance for Polyurethanes Industry** **WARNING:** This substance has been classified by the IARC as Group 2B: Possibly Carcinogenic to Humans.
Meguiar's G177 - Ultimate Wash & Wax & COCONUT OIL DIETHANOLAMIDE	For Fatty Nitrogen Derived (FND) Amides (including several high molecular weight alkyl amino acid amides) The chemicals in the Fatty Nitrogen Derived (FND) Amides of surfactants are similar to the class in general as to physical/chemical properties, environmental fate and toxicity. Human exposure to these chemicals is substantially documented. The Fatty nitrogen-derived amides (FND amides) comprise four categories: Subcategory I: Substituted Amides Subcategory II: Fatty Acid Reaction Products with Amino Compounds (Note: Subcategory II chemicals, in many cases, contain Subcategory I chemicals as major components) Subcategory III: Imidazole Derivatives Subcategory IV: FND Amphoterics Acute Toxicity: The low acute oral toxicity of the FND Amides is well established across all Subcategories by the available data. The limited acute toxicity of these chemicals is also confirmed by four acute dermal and two acute inhalation studies. Repeated Dose and Reproductive Toxicity: Two subchronic toxicity studies demonstrating low toxicity are available for Subcategory I chemicals. In addition, a 5-day repeated dose study for a third chemical confirmed the minimal toxicity of these chemicals. Since the Subcategory I chemicals are major components of many Subcategory II chemicals, and based on the low repeat-dose toxicity of the amino compounds (e.g. diethanolamine, triethanolamine) used for producing the Subcategory II derivatives, the Subcategory I repeat-dose toxicity studies adequately support Subcategory II. Two subchronic toxicity studies in Subcategory III confirmed the low order of repeat dose toxicity for the FND Amides Imidazole derivatives. For Subcategory IV, two subchronic toxicity studies for one of the chemicals indicated a low order of repeat-dose toxicity for the FND amphoteric salts similar to that seen in the other categories. Genetic Toxicity in vitro: Based on the lack of effect of one or more chemicals in each subcategory, adequate data for mutagenic activity as measured by the Salmonella reverse mutation assay exist for all of the subcategories. Developmental Toxicity: A developmental toxicity study in Subcategory I and in Subcategory IV and a third study for a chemical in Subcategory III are available. The studies indicate these chemicals are not developmental toxicants, as expected based on their structures, molecular weights, physical properties and knowledge of similar chemicals. As above for repeat-dose toxicity, the data for Subcategory I are adequate to support Subcategory II. In evaluating potential toxicity of the FND Amides chemicals, it is also useful to review the available data for the related FND Cationic and FND Amines Category chemicals. Acute oral toxicity studies (approximately 80 studies for 40 chemicals in the three categories) provide LD50 values from approximately 400 to 10,000 mg/kg with no apparent organ specific toxicity. Similarly, repeated dose toxicity studies (approximately 35 studies for 15 chemicals) provide NOAELs between 10 and 100 mg/kg/day for rats and slightly lower for dogs. More than 60 genetic toxicity studies (in vitro bacterial and mammalian cells as well as in vivo studies) indicated no mutagenic activity among more than 30 chemicals tested. For reproductive evaluations, 14 studies evaluated reproductive endpoints and/or reproductive organs for 11 chemicals, and 15 studies evaluated developmental toxicity for 13 chemicals indicating no reproductive or developmental effects for the FND group as a whole. Some typical applications of FND Amides are: masonry cement additive; curing agent for epoxy resins; closed hydrocarbon systems in oil field production, refineries and chemical plants; and slip and antiblocking additives for polymers. The safety of the FND Amides to humans is recognised by the U.S. FDA, which has approved stearamide, oleamide and/or erucamide for adhesives; coatings for articles in food contact; coatings for polyolefin films; defoaming agents for manufacture of paper and paperboard; animal glue (defoamer in food packaging); in EVA copolymers for food packaging; lubricants for manufacture of metallic food packaging; irradiation of prepared foods; release agents in manufacture of food packaging materials, food contact surface of paper and paperboard; cellophane in food packaging; closure sealing gaskets; and release agents in polymeric resins and petroleum wax. The low order of toxicity indicates that the use of FND Amides does not pose a significant hazard to human health. The differences in chain length, degree of saturation of the carbon chains, source of the natural oils, or addition of an amino group in the chain would not be expected to have an impact on the toxicity profile. This conclusion is supported by a number of studies in the FND family of chemicals (amines, cationics, and amides as separate categories) that show no differences in the length or degree of saturation of the alkyl substituents and is also supported by the limited toxicity of these long-chain substituted chemicals.
COCAMIDOPROPYLBETAINE & SODIUM (C10-16)PARETH SULFATE	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 the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
COCONUT OIL DIETHANOLAMIDE & 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. 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. for diethanolamine (DEA):

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

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

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

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

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Toxicity

Meguiar's G177 - Ultimate Wash & Wax	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
cocamidopropylbetaine	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	=1mg/L	1
	EC50	48	Crustacea	6.4mg/L	2
	EC50	96	Algae or other aquatic plants	0.55mg/L	2
	NOEC	672	Fish	0.16mg/L	2
sodium (C10-16)pareth sulfate	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
coconut oil diethanolamide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
diethanolamine	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	LC50	96	Fish	1-664mg/L	2
	EC50	48	Crustacea	30.1mg/L	2
	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
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*				

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
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 Fatty Nitrogen-Derived Amides (FND Amides)
Environmental fate:
As expected for molecules of this size, model predictions for the chemicals with definable structures indicate they are nonvolatile. Predicted or measured Kow values are of limited practical use for the FND Amides. An inherent property of surfactants is that they tend to accumulate at the interface between hydrophobic and hydrophilic phases rather than equilibrate between the two phases. Therefore, the accurate measurement of the Kow of any surfactant is notoriously difficult. The measured values for water solubility of the FND Amides indicate that they are insoluble. The model predictions, however, range from insoluble to moderately soluble. The physical/chemical properties of surfactants often make water solubility data of little practical value in the determination of environmental fate and effects.
Due to the low volatility of the FND Amides atmospheric photodegradation estimates are of no practical use. However, photodegradation was predicted that could be modeled. These predictions indicate that these chemicals would be expected to degrade relatively rapidly upon exposure to light (t1/2 values ranging from approximately 2.2 to 9.5 hours).
Due to the surfactant properties and solubility of the FND Amides, hydrolytic stability is of minimal value for determining
environmental fate or effects.
Biodegradability: There are adequate measured data across Subcategories I, II and IV to allow the conclusion that the these chemicals are readily or inherently biodegradable. Further, the model predictions provide reasonably close estimates to these measured values. Minimal degradability of the one chemical,[CAS RN 68122-86-1], from Subcategory III indicates these chemicals are slowly degraded. The slower degradation of these materials is likely the result of limited water solubility and behavior of the chemicals in aqueous solution. Longer single alkyl group substitutions and/or multiple long-chain alkyl substituents result in slower “inherent” biodegradability.
Ecotoxicity:
The reliable data for acute toxicity to fish and daphnid indicate that the FND Amides like surfactants in general, may adversely affect aquatic organisms (LC50 and EC50 values ranging from 0.2 to 59 mg/l). Many of the ECOSAR model estimates for the acute toxicity endpoints indicate the chemicals are “not toxic at solubility”. However, for surfactants such as the FND Amides 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.
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.
Surfactants show a complex solubility behaviour due to aggregation. The monomer concentration, and hence the thermodynamic activity, reaches a limiting value at the critical micelle concentration (CMC). It remains approximately constant as the total concentration is further increased. For ecotoxicological models requiring a solubility value, the critical micelle concentration is therefore the appropriate parameter describing water solubility of surface active materials.
Surfactants can form dispersions or emulsions in which the bioavailablity for aquatic toxicity studies is difficult to ascertain, even with careful solution preparation. Micelle formation can result in an overestimation of the bioavailable fraction even when “solutions” are apparently formed. This presents significant problems of interpretation of aquatic toxicity test results for surface active materials. The so-called the critical micelle concentration (CMC) is is related to surface tension produced by the substance and is the key value for actual water solubility of the substance .
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.
DO NOT discharge into sewer or waterways.

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Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
diethanolamine	LOW (BCF = 1)

Mobility in soil

Ingredient	Mobility
diethanolamine	HIGH (KOC = 1)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate. ▸ **DO NOT** allow wash water from cleaning or process equipment to enter drains. ▸ It may be necessary to collect all wash water for treatment before disposal. ▸ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▸ Where in doubt contact the responsible authority. ▸ Recycle wherever possible. ▸ Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. ▸ Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). ▸ Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
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

cocamidopropylbetaine 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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)

sodium (C10-16)pareth sulfate is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

coconut oil diethanolamide is found on the following regulatory lists

Australian Inventory of Industrial Chemicals (AIIC)

diethanolamine 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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Group 2B : Possibly carcinogenic to humans

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National Inventory Status

National Inventory	Status
Australia - AIIC	Yes
Australia - Non-Industrial Use	No (cocamidopropylbetaine; sodium (C10-16)pareth sulfate; coconut oil diethanolamide; diethanolamine)
Canada - DSL	Yes
Canada - NDSL	No (cocamidopropylbetaine; sodium (C10-16)pareth sulfate; coconut oil diethanolamide; diethanolamine)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (sodium (C10-16)pareth sulfate; coconut oil diethanolamide)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (sodium (C10-16)pareth sulfate; coconut oil diethanolamide)
Vietnam - NCI	Yes
Russia - ARIPS	No (coconut oil diethanolamide)
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	01/11/2019
Initial Date	01/11/2009

SDS Version Summary

Version	Issue Date	Sections Updated
3.1.1.1	06/03/2015	Classification, Fire Fighter (fire/explosion hazard)
5.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

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