

# MAXI VEHICLE WASH

**Hazard Alert Code:**  
**MODERATE**

Chemwatch Material Safety Data Sheet

Revision No: 2.0

Chemwatch 21-9638

Issue Date: 23-Nov-2009

CD 2009/3

## Section 1 - CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

### PRODUCT NAME

Maxi Vehicle Wash

### PRODUCT USE

Linear alkylbenzene sulfonates (LAS) are, by volume, the most important group of synthetic anionic surfactant today. LAS are mainly used in laundry detergents and cleaning agents. LAS are frequently used as the sodium salts as the sole surfactant in a formulation or in conjunction with other anionic, nonionic or cationic surfactants. LAS consist of an alkyl chain attached to a benzene ring in the para position to the sulfonate group. Sometimes toluene, xylene and naphthalene are used in place of benzene. The homologue distribution in commercial products covers alkyl chain lengths from C10 to C13 with an average chain length of C11.6. LAS raw materials are derived from linear alkyl benzenes in which the ring is attached to a C-atom which is itself attached to two other C-atoms. The benzene ring may be attached to any of the C atoms from C2 to C6 but not to C1. Structures in which the benzene ring may be attached to different C atoms are described as isomers. E.g., the structure with a C12 alkyl chain and the benzene ring attached at the second alkyl carbon is designated as the C12-2-isomer and abbreviated C12-2. LAS can be represented structurally as:  $H_3C(CH_2)_xCH_2(C_6H_4S)_3Na$  or  $CH_2(CH_2)_yCH_3$  -  $x+y=9$ . Used according to manufacturer's directions. Heavy duty vehicle washing concentrate

### SUPPLIER

Company: GSB Chemical Co. Pty Ltd

Address:

84 Camp Road

Broadmeadows

VIC, 3047

AUS

Telephone: +61 3 9457 1125

Fax: +61 3 9459 7978

Email: info@gsbchem.com.au

### HAZARD RATINGS

	Min	Max	
Flammability:	0	1	
Toxicity:	2	4	
Body Contact:	2	4	
Reactivity:	1	2	
Chronic:	2	4	

Min/Nil=0  
Low=1  
Moderate=2  
High=3  
Extreme=4

## Section 2 - HAZARDS IDENTIFICATION

### STATEMENT OF HAZARDOUS NATURE

**NON-HAZARDOUS SUBSTANCE. NON-DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.**

### POISONS SCHEDULE

None

### RISK

- Toxic to aquatic organisms.
- May cause long-term adverse effects in the environment.
- Ingestion may produce health damage\*.
- Cumulative effects may result following exposure\*.
- May produce discomfort of the eyes respiratory tract and skin\*.
- Limited evidence of a carcinogenic effect\*.
- Possible respiratory and skin sensitiser\*.
- Repeated exposure potentially causes skin dryness and cracking\*.

\* (limited evidence).

### SAFETY

- Do not breathe gas/ fumes/ vapour/ spray.
- Avoid contact with skin.
- Wear eye/ face protection.
- In case of contact with eyes rinse with plenty of water and contact Doctor or Poisons Information Centre.

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## Section 3 - COMPOSITION / INFORMATION ON INGREDIENTS

NAME	CAS RN	%
diethanolamine dodecylbenzenesulfonate	26545-53-9	<10
coconut diethanolamide	68603-42-9	<10
sodium (C10-16)alkyl ether sulfate	68585-34-2	<10
sodium tripolyphosphate	7758-29-4	<10
dye		1-10
water	7732-18-5	>60

## Section 4 - FIRST AID MEASURES

### SWALLOWED

- 
- If swallowed do NOT induce vomiting.
- If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration.
- Observe the patient carefully.
- Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- Seek medical advice.

### EYE

- If this product comes in contact with the eyes:
- Wash out immediately with fresh running water.
- Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.
- If pain persists or recurs seek medical attention.
- Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

### SKIN

- 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.

### INHALED

- 
- If fumes or combustion products are inhaled remove from contaminated area.
- Other measures are usually unnecessary.

### NOTES TO PHYSICIAN

- Treat symptomatically.

## Section 5 - FIRE FIGHTING MEASURES

### EXTINGUISHING MEDIA

- The product contains a substantial proportion of water, therefore there are no restrictions on the type of extinguishing media which may be used. Choice of extinguishing media should take into account surrounding areas.

Though the material is non-combustible, evaporation of water from the mixture, caused by the heat of nearby fire, may produce floating layers of combustible substances.

In such an event consider:

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

### FIRE FIGHTING

- 
- Alert Fire Brigade and tell them location and nature of hazard.
- Wear breathing apparatus plus protective gloves for fire only.
- 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

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■ The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn.  
Decomposes on heating and produces toxic fumes of: carbon dioxide (CO<sub>2</sub>), nitrogen oxides (NO<sub>x</sub>), phosphorus oxides (PO<sub>x</sub>), sulfur oxides (SO<sub>x</sub>), metal oxides, other pyrolysis products typical of burning organic material.  
May emit poisonous fumes.

## FIRE INCOMPATIBILITY

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

## HAZCHEM

None

## Section 6 - ACCIDENTAL RELEASE MEASURES

### EMERGENCY PROCEDURES

#### MINOR SPILLS

- Environmental hazard - contain spillage.
- Clean up all spills immediately.
- Avoid breathing vapours and contact with skin and eyes.
- Control personal contact 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

- Environmental hazard - contain spillage.
- 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.
- 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 MSDS.**

## Section 7 - HANDLING AND STORAGE

### PROCEDURE FOR HANDLING

- Alkanolamines and iron may produced unstable complexes. Monoethanolamine (MEA) and iron form a trisethanolamino-iron complex. This material may spontaneously decompose at temperatures between 130 and 160 degrees C. and is suspected of causing a fire in a nearly empty storage tank containing a "heel" of MEA in contact with carbon steel coils. If steam coil heating is used, low pressure steam in stainless steel coils should be considered. Drum heating should also be reviewed and, where possible, temperatures should be maintained below 130 degrees C.
- DO NOT allow clothing wet with material to stay in contact with skin
- Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- DO NOT enter confined spaces until atmosphere has been checked.
- DO NOT allow material to contact humans, exposed food or food utensils.
- Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- Use good occupational work practice.
- Observe manufacturer's storing and handling recommendations.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

### SUITABLE CONTAINER

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- Do not use aluminium, zinc or galvanized iron container.

## STORAGE INCOMPATIBILITY

- 
- Avoid reaction with oxidising agents
- Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.

## STORAGE REQUIREMENTS

- 
- 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 storing and handling recommendations.

## SAFE STORAGE WITH OTHER CLASSIFIED CHEMICALS



X: Must not be stored together  
O: May be stored together with specific preventions  
+: May be stored together

## Section 8 - EXPOSURE CONTROLS / PERSONAL PROTECTION

### EXPOSURE CONTROLS

The following materials had no OELs on our records

- diethanolamine dodecylbenzenesulfonate: CAS:26545-53-9
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- sodium tripolyphosphate: CAS:7758-29-4 CAS:15091-98-2
- water: CAS:7732-18-5

### MATERIAL DATA

MAXI VEHICLE WASH:

■ Sensory irritants are chemicals that produce temporary and undesirable side-effects on the eyes, nose or throat. Historically occupational exposure standards for these irritants have been based on observation of workers' responses to various airborne concentrations. Present day expectations require that nearly every individual should be protected against even minor sensory irritation and exposure standards are established using uncertainty factors or safety factors of 5 to 10 or more. On occasion animal no-observable-effect-levels (NOEL) are used to determine these limits where human results are unavailable. An additional approach, typically used by the TLV committee (USA) in determining respiratory standards for this group of chemicals, has been to assign ceiling values (TLV C) to rapidly acting irritants and to assign short-term exposure limits (TLV STELs) when the weight of evidence from irritation, bioaccumulation and other endpoints combine to warrant such a limit. In contrast the MAK Commission (Germany) uses a five-category system based on intensive odour, local irritation, and elimination half-life. However this system is being replaced to be consistent with the European Union (EU) Scientific Committee for Occupational Exposure Limits (SCOEL); this is more closely allied to that of the USA.

OSHA (USA) concluded that exposure to sensory irritants can:

- cause inflammation
- cause increased susceptibility to other irritants and infectious agents
- lead to permanent injury or dysfunction
- permit greater absorption of hazardous substances and
- acclimate the worker to the irritant warning properties of these substances thus increasing the risk of overexposure.

DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ It is the goal of the ACGIH (and other Agencies) to recommend TLVs (or their equivalent) for all substances for which there is evidence of health effects at airborne concentrations encountered in the workplace.

At this time no TLV has been established, even though this material may produce adverse health effects (as evidenced in animal experiments or clinical experience). Airborne concentrations must be maintained as low as is practically possible and occupational exposure must be kept to a minimum.

NOTE: The ACGIH occupational exposure standard for Particles Not Otherwise Specified (P.N.O.S) does NOT apply.

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COCONUT DIETHANOLAMIDE:

■ 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).

SODIUM (C10-16)ALKYL ETHER SULFATE:

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WATER:

■ No exposure limits set by NOHSC or ACGIH.

## PERSONAL PROTECTION



## EYE

■

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- 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 lens 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]

## HANDS/FEET

- 
- Wear chemical protective gloves, eg. PVC.
- Wear safety footwear or safety gumboots, eg. 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.

Suitability and durability of glove type is dependent on usage. Factors such as:

- frequency and duration of contact,
- chemical resistance of glove material,
- glove thickness and
- dexterity,

are important in the selection of gloves.

## OTHER

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

## RESPIRATOR

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

Breathing Zone Level ppm (volume)	Maximum Protection Factor	Half-face Respirator	Full-Face Respirator
1000	10	AK-AUS P	-
1000	50	-	AK-AUS P
5000	50	Airline *	-
5000	100	-	AK-2 P
10000	100	-	AK-3 P
	100+		Airline**

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

The local concentration of material, quantity and conditions of use determine the type of personal protective equipment required. For further information consult site specific CHEMWATCH data (if available), or your Occupational Health and Safety Advisor.

## ENGINEERING CONTROLS

■ Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

An approved self contained breathing apparatus (SCBA) may be required in some situations.

Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Type of Contaminant:	Air Speed:
solvent, vapours, degreasing etc., evaporating from tank (in still air).	0.25-0.5 m/s (50-100 f/min.)
aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)	0.5-1 m/s (100-200 f/min.)
direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)	1-2.5 m/s (200-500 f/min.)
grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).	2.5-10 m/s (500-2000 f/min.)

Within each range the appropriate value depends on:

Lower end of the range	Upper end of the range
1: Room air currents minimal or favourable to capture	1: Disturbing room air currents
2: Contaminants of low toxicity or of nuisance value only.	2: Contaminants of high toxicity
3: Intermittent, low production.	3: High production, heavy use
4: Large hood or large air mass in motion	4: Small hood-local control only

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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.

## Section 9 - PHYSICAL AND CHEMICAL PROPERTIES

### APPEARANCE

Blue viscous liquid with a characteristic odour; miscible with water.

### PHYSICAL PROPERTIES

Liquid.

Mixes with water.

Molecular Weight: Not Available	Boiling Range (°C): 100	Melting Range (°C): Not Available
Specific Gravity (water=1): 1.1	Solubility in water (g/L): Miscible	pH (as supplied): Not Available
pH (1% solution): 11.2	Vapour Pressure (kPa): Not Available	Volatile Component (%vol): >90
Evaporation Rate: Not Available	Relative Vapour Density (air=1): Not Available	Flash Point (°C): Not Applicable
Lower Explosive Limit (%): Not Applicable	Upper Explosive Limit (%): Not Applicable	Autoignition Temp (°C): Not Applicable
Decomposition Temp (°C): Not Available	State: Liquid	Viscosity: Not Available

Material	Value
log Kow	-1.43

## Section 10 - CHEMICAL STABILITY

### CONDITIONS CONTRIBUTING TO INSTABILITY

- 
- Presence of incompatible materials.
- Product is considered stable.
- Hazardous polymerisation will not occur.

For incompatible materials - refer to Section 7 - Handling and Storage.

## Section 11 - TOXICOLOGICAL INFORMATION

### POTENTIAL HEALTH EFFECTS

#### ACUTE HEALTH EFFECTS

##### SWALLOWED

- Accidental ingestion of the material may be damaging to the health of the individual.
- Inorganic polyphosphates are used extensively in domestic and industrial products. Experiments on rats showed kidney damage, growth retardation, and tetany due to low calcium.
- Ingestion of anionic surfactants may produce diarrhoea, bloated stomach, and occasional vomiting.

##### EYE

- There is some evidence to suggest that this material can cause eye irritation and damage in some persons.
- Non-ionic surfactants can cause numbing of the cornea, which masks discomfort normally caused by other agents and leads to corneal injury. Irritation varies depending on the duration of contact, the nature and concentration of the surfactant.
- Direct eye contact with some anionic surfactants in high concentration can cause severe damage to the cornea. Low concentrations can cause discomfort, excess blood flow, and corneal clouding and swelling. Recovery may take several days.

##### SKIN

- Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.
- There is some evidence to suggest that this material can cause inflammation of the skin on contact in some persons.
- Anionic surfactants can cause skin redness and pain, as well as a rash. Cracking, scaling and blistering can occur.
- Open cuts, abraded or irritated skin should not be exposed to this material.
- Entry into the blood-stream, through, for example, cuts, abrasions or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

##### INHALED

- There is some evidence to suggest that the material can cause respiratory irritation in some persons. The body's response to such irritation can cause further lung damage.
- Not normally a hazard due to non-volatile nature of product.

### CHRONIC HEALTH EFFECTS

- There has been some concern that this material can cause cancer or mutations but there is not enough data to make an

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assessment.

Substance accumulation, in the human body, may occur and may cause some concern following repeated or long-term occupational exposure.

There is some evidence that inhaling this product is more likely to cause a sensitisation reaction in some persons compared to the general population.

There is limited evidence that, skin contact with this product is more likely to cause a sensitisation reaction in some persons compared to the general population.

In long-term animal studies, inorganic polyphosphates produced growth inhibition, increased kidney weights, bone decalcification, enlargement of the parathyroid gland, inorganic phosphate in the urine, focal necrosis of the kidney and alterations of muscle fibre size. Inorganic phosphates have not been shown to cause cancer, genetic damage or reproductive or developmental damage in animal tests.

Prolonged or repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

Prolonged or chronic exposure to alkanolamines may result in liver, kidney or nervous system injury. Repeated inhalation may aggravate asthma and lung disease involving inflammation or scarring.

Results of animal testing with diethanolamine (DEA) and monoethanolamine (MEA) has shown a wide range of possible effects, including induction of tumours, developmental abnormalities and injury to the foetus and mother.

Many amines greatly sensitise the skin and respiratory system, and certain individuals, especially those predisposed to asthma and other allergic responses, may show allergic reactions when chronically exposed to alkanolamines.

Exposure to sulfonates can cause an imbalance in cellular salts and therefore cellular function. Airborne sulfonates may be responsible for respiratory allergies and, in some instances, minor dermal allergies.

## TOXICITY AND IRRITATION

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

■ Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin (IPCS 1996). The bulk is metabolized in the liver to sulfophenylic carboxyl acids. The metabolites are excreted primarily via the urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.

No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.

LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation.

Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.

A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.

The mutagenic potential of LAS was tested using Salmonella typhimurium strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.

In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.

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

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.

## DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

### ■ for diethanolamine (DEA):

In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.

Subchronic toxicity: The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.

Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility. Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL = 32 mg/g) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone

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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.

Linear alkylbenzene sulfonates (LAS) are classified as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R41 (Risk of serious damage to eyes) according to CESIO (CESIO 2000). LAS are not included in Annex 1 of list of dangerous substances of Council Directive 67/548/EEC.

LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin (IPCS 1996). The bulk is metabolized in the liver to sulfophenylic carboxyl acids. The metabolites are excreted primarily via the

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urine and faeces. The main urinary metabolites in rats are sulfophenyl butanoic acid and sulfophenyl pentanoic acid. Accumulation of LAS or its main metabolites has not been established in any organ after repeated oral ingestion.

No serious injuries or fatalities in man have been reported following accidental ingestion of LAS-containing detergent. The main clinical signs observed after oral administration to rats of doses near or greater than the LD50 values consisted of reduced voluntary activity, diarrhoea, weakness etc. Death usually occurred within 24 hours of administration. Rats appear to be more sensitive to LAS than mice.

LAS and branched alkylbenzene sulfonates may cause irritation of the eyes, skin and mucous membranes. LAS are relatively more irritating to the skin than the corresponding branched alkylbenzene sulfonates. The potential of LAS to irritate the skin depends on the concentration applied. LAS have been classified as irritating to skin at concentrations above 20% according to EU-criteria. Human skin can tolerate contact with solution of up to 1% LAS for 24 hours resulting in only mild irritation. Application of > 5% LAS to the eyes of rabbits produced irritation. Concentration of < 0.1% LAS produced mild to no irritation.

Skin sensitization was not seen in 2,294 volunteers exposed to LAS or in 17,887 exposed to formulations of LAS.

A feeding study indicated that LAS, when administered for 2 years at extremely high levels (0.5%) in the diets to rats, produced no adverse effects on growth, health or feed efficiency.

The mutagenic potential of LAS was tested using Salmonella typhimurium strains, using Ames test. In these studies, LAS was not mutagenic. The available long-term studies are inadequate for evaluating the carcinogenic potential of LAS in laboratory animals. The studies available (oral administration to rats and mice) do not show any evidence of carcinogenicity.

In general no specific effect of LAS on reproductive processes has been seen, although dosages causing maternal toxicity may also induce some effects on reproduction. No teratogenic effects attributed to LAS exposure have been observed.

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

No significant acute toxicological data identified in literature search.

**COCONUT DIETHANOLAMIDE:**

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

**TOXICITY**

Oral (rat) LD50: 2700 mg/kg

**IRRITATION**

Nil reported.

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

for diethanolamine (DEA):

In animal studies, DEA has low acute toxicity via the oral and dermal routes with moderate skin irritation and severe eye irritation. In subchronic toxicity testing conducted via the oral route in rats and mice, the main effects observed were increased organ weights and histopathology of the kidney and/or liver, with the majority of other tissue effects noted only at relatively high dosages. In subchronic studies conducted via the dermal route, skin irritation was noted as well as systemic effects similar to those observed in the oral studies. DEA has not been shown to be mutagenic or carcinogenic in rats; however, there is evidence of its carcinogenicity in mice.

Subchronic toxicity: The subchronic toxicity of DEA has been studied in F344 rats and B6C3F1 mice by exposure through drinking water or dermal administration, in 2 week and 13 week studies.

Target organs for toxicity included blood, kidney, brain and spinal cord, seminiferous tubules and dermal application site in rats and liver, kidney, heart, salivary gland and dermal application site in mice. Effects on seminiferous tubules were accompanied by reductions in sperm count and reduced sperm motility. Hematological evaluations indicated normochromic, microcytic anemia in the dermal study in male rats (NOEL = 32 mg/g) and females (LOEL = 32 mg/kg). Anemia was also observed in rats in the drinking water study with a LOEL of 14 mg/kg/d in females and a LOEL of 48 mg/kg/d in males for altered hematological parameters. These findings were similar to those observed in the 2 week studies, but the magnitude of the changes was greater in the 13 week studies. Hematological parameters were normal in controls. No associated histopathological changes were noted in femoral bone marrow. Haematological parameters were not evaluated in mice.

Developmental toxicity: In a developmental toxicity study conducted via the oral route, effects of concern were observed only in the presence of maternal toxicity. In a developmental toxicity study conducted via the dermal route using two species of mammals, developmental toxicity was observed only in one species and only at doses causing significant maternal toxicity. Metabolically, DEA is excreted largely unchanged in the urine.

Carcinogenicity: A two-year dermal cancer study bioassay results on DEA and three fatty acid condensates of DEA indicated that liver tumours occurred in male and female mice exposed to DEA and two of the condensates. In addition kidney tumours occurred in male mice exposed to DEA and one of the condensates. Compelling evidence suggested that the toxicity observed in mice and rats treated with the DEA condensates was associated with free DEA and not with other components of the condensates. A weight of evidence analysis of data relevant to the assessment of the liver and kidney tumours in mice resulted in the conclusion that these tumours are not relevant to humans under the expected conditions of exposure and that liver and kidney toxicity should be evaluated on a threshold basis. This conclusion is based on the following:

- DEA is not genotoxic
- tumour development occurred at doses also associated with chronic hyperplasia

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- 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.

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 Comité Européen des Agents de Surface et de leurs Intermediaires

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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. Miljøministeriet (Danish Environmental Protection Agency).

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

**SODIUM (C10-16)ALKYL ETHER SULFATE:**

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

Oral (rat) LD50: 1600 mg/kg

Skin (rabbit):25 mg/24 hr Moderate

■ The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.

Alkyl ether sulfates (alcohol or alkyl ethoxysulfates) (AES) are generally classified according to Comité Européen des Agents de Surface et leurs Intermédiaires Organiques (CESIO) as Irritant (Xi) with the risk phrases R38 (Irritating to skin) and R36 (Irritating to eyes). An exception has been made for AES (2-3EO) in a concentration of 70-75% where R36 is substituted with R41 (Risk of serious damage to eyes).

AES are not included in Annex 1 of the list of dangerous substances of Council Directive 67/548/EEC.

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 sensitizer 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 µg/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 oxidized to CO<sub>2</sub> 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.

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

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

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for similar product (sodium lauryl ether sulfate)

**SODIUM TRIPOLYPHOSPHATE:**

■ unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances.

Oral (Rat) LD50: 5190 mg/kg

Nil Reported

Dermal (Rabbit) LD50: 3160 mg/kg \*

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■ 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.

**WATER:**

■ No significant acute toxicological data identified in literature search.

**SENSITISER**

coconut	Australia Final Report on Hazard Classification of Common	Recommended for Hazard	
diethanolamide	Skin Sensitisers	Classification (R43)	No

## Section 12 - ECOLOGICAL INFORMATION

Refer to data for ingredients, which follows:

MAXI VEHICLE WASH:

SODIUM TRIPOLYPHOSPHATE:

■ On the basis of available evidence concerning either toxicity, persistence, potential to accumulate and or observed environmental fate and behaviour, the material may present a danger, immediate or long-term and /or delayed, to the structure and/ or functioning of natural ecosystems.

MAXI VEHICLE WASH:

SODIUM TRIPOLYPHOSPHATE:

DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ 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.

MAXI VEHICLE WASH:

SODIUM TRIPOLYPHOSPHATE:

SODIUM (C10-16)ALKYL ETHER SULFATE:

COCONUT DIETHANOLAMIDE:

DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ DO NOT discharge into sewer or waterways.

MAXI VEHICLE WASH:

SODIUM (C10-16)ALKYL ETHER SULFATE:

COCONUT DIETHANOLAMIDE:

■ Octanol/water partition coefficients cannot easily be determined for surfactants because one part of the molecule is hydrophilic and the other part is hydrophobic. Consequently they tend to accumulate at the interface and are not extracted into one or other of the liquid phases. As a result surfactants are expected to transfer slowly, for example, from water into the flesh of fish. During this process, readily biodegradable surfactants are expected to be metabolised rapidly during the process of bioaccumulation. This was emphasised by the OECD Expert Group stating that chemicals are not to be considered to show bioaccumulation potential if they are readily biodegradable.

Several anionic and nonionic surfactants have been investigated to evaluate their potential to bioconcentrate in fish. BCF values (BCF - bioconcentration factor) ranging from 1 to 350 were found. These are absolute maximum values, resulting from the radiolabelling technique used. In all these studies, substantial oxidative metabolism was found resulting in the highest radioactivity in the gall bladder. This indicates liver transformation of the parent compound and biliary excretion of the metabolised compounds, so that "real" bioconcentration is overstated. After correction it can be expected that "real" parent BCF values are one order of magnitude less than those indicated above, i.e. "real" BCF is <100. Therefore the usual data used for classification by EU directives to determine whether a substance is "Dangerous to the Environment" has little bearing on whether the use of the surfactant is environmentally acceptable.

MAXI VEHICLE WASH:

COCONUT DIETHANOLAMIDE:

DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ for diethanolamine (DEA):

log Kow : -1.43

Koc : 4

Half-life (hr) air : 4

Henry's atm m<sup>3</sup>/mol: 5.35E-14

BOD 5: 0.03-0.1, 0.9%

BOD 28: 57 mg/gm

COD : 1590 mg/gm

TPC 470 mg/gm

ThOD : 2.13

BCF : &lt;1

Based on its physicochemical properties and biodegradation characteristics, DEA is not expected to pose a high risk to drinking water, and its potential for bioconcentration in aquatic organisms is low. DEA is categorized as "practically nontoxic" on an acute basis to freshwater invertebrates, estuarine/marine invertebrates, and freshwater plants

Environmental fate:

In soil and water, DEA is expected to biodegrade fairly rapidly following acclimation (half-life on the order of days to weeks). In soil, DEA should leach. In the atmosphere, DEA is expected to exist almost entirely in the vapor phase. Reaction with photochemically

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generated hydroxyl radicals is expected to be the dominant removal mechanism (half-life, four hours). This compound may also be removed from the atmosphere in precipitation. The Henry's Law constant for DEA is  $3.87 \times 10^{-11} \text{ atm} \cdot \text{m}^3/\text{mol}$  which suggests that DEA is essentially nonvolatile from water. The half-life for DEA vapour reacting with photochemically generated hydroxyl radicals in the atmosphere has been estimated to be four hours based on an estimated reaction rate constant of  $8.9 \times 10^{-11} \text{ cm}^3/\text{molecules} \cdot \text{sec}$  at  $25^\circ\text{C}$  and an average ambient hydroxyl concentration of  $5 \times 10^5 \text{ molecules}/\text{cm}^3$ .

DEA, in the presence of nitrites, can form N-nitrosodiethanolamine (NDELA). In air, NDELA is expected to exist solely as a vapor based on a vapor pressure of  $2.78 \times 10^{-4} \text{ mmHg}$  at  $25^\circ\text{C}$ . Vapor-phase NDELA will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals with an estimated half-life of 13 hours. NDELA is stable to direct photolysis. In soil, an estimated Koc of 4.8 suggests that this compound is expected to have very high mobility; it is expected to biodegrade slowly in soil. In summary, it appears that DEA is relatively short lived and that it does not present a high risk to contaminate drinking waters. NDELA, a potential formation product, is persistent to biotic and abiotic processes, and mobile. The amounts formed are uncertain (it is only indicated that the half-life is in the order of days to weeks). The water quality criteria (WQC) for nitrosamines is  $0.0008 \text{ ug/L}$  (U.S. Clean Water Act)

DEA's potential for bioconcentration in aquatic organisms is low

At very low concentrations (about 10 ppm) diethanolamine can be degraded in biological wastewater treatment plants.

Ecotoxicity:

Fish LC50 (96 h): Fathead minnow 100 mg/l; (48 h): Bluegill sunfish 1850 mg/l

Daphnia magna LC50 (48 h): 109 mg/l

DEA is categorized as ranging from moderately toxic to practically nontoxic to freshwater invertebrates based on EC50 values ranging from 2.15 to 306 mg/L.

DEA is categorized as "practically nontoxic" to estuarine/ marine invertebrates. EC50 values for estuarine/ marine invertebrates (shrimps and mollusks) exposed to DEA ranged from  $>100$  to  $2,800 \text{ mg/L}$ .

DEA is categorised as practically nontoxic to freshwater plants on an acute basis based on EC50 values ranging from 103 to 523 mg/L.

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DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ Toxic to aquatic organisms.

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DIETHANOLAMINE DODECYLBENZENESULFONATE:

■ Linear alkylbenzene sulfonates (LABS) are generally biodegradable.

The initial step in the biodegradation of LABS under aerobic conditions is an omega -oxidation of the terminal methyl group of the alkyl chain to form a carboxylic acid. Further degradation proceeds by a stepwise shortening of the alkyl chain by beta -oxidation leaving a short-chain sulfophenyl carboxylic acid. In the presence of molecular oxygen the aromatic ring structure hydrolyses to form a dihydroxy-benzene structure which is opened before desulfonation of the formed sulfonated dicarboxylic acid. The final degradation steps have not been investigated in details but are likely to occur by general bacterial metabolic routes involving a total mineralisation and assimilation into biomass. Both the initial omega -oxidation and the hydroxylation of the ring structure of LABS require molecular oxygen, and they are not expected to take place under anoxic conditions.

The BioConcentration Factor (BCF) tends to increase with increasing alkyl chain length but also the position of the aryl sulfonate moiety was important. A higher BCF was seen for linear alkyl benzenesulfonate isomers with the aryl sulfonate attached.

Numerous studies have been performed to determine the effects of LABS towards aquatic organisms. The aquatic effect concentrations that were observed in these studies are highly variable. This variation is partly related to the testing of different isomers and homologues, but it may also be due to the specific test conditions and species. The length of the alkyl chain is an important factor determining the aquatic toxicity. In general, the homologues with the highest number of carbons in the alkyl chain are more toxic than are those with shorter alkyl chains. Today, commercial LABS have a homologue distribution between C10 and C13 with a typical average alkyl chain length of C11.6. The widest range in the toxicity of LABS towards species belonging to the same group is found for algae. Approximately 90% of the data found in the literature fall between 0.1 and 100 mg/l. Typical ranges of EC50 values are 1 to 100 mg/l for fresh water species and  $< 1$  to 10 mg/l for marine species. A very low EC100 value of 0.025 mg/l was determined for *Gymnodium breve*. Previous studies in which *Gymnodium breve* was exposed with AES confirm that this species is highly sensitive to surfactants, and occasionally available data for *Gymnodium breve* should therefore not be used for comparison of the aquatic toxicity between various surfactants.

LC50 values have been found in the range of 1 to 10 mg/l when *Daphnia magna* were exposed with LABS homologues between C10 and C13. The acute toxicity of LABS to *Daphnia magna* generally increases with increasing alkyl chain length. A study with the marine crustacean *Acartia tonsa* indicated that a C10-13 LAS affected the survival at 0.54 mg/l (LC50) and the development rate at 0.51 mg/l (EC50) after 8 days of exposure. The 48 h-LC50 that was obtained in the same study with *Acartia tonsa* was 2.1 mg/l.

Metabolites from biotransformation of LABS are reported to have a much lower toxicity to invertebrates compared to the toxicity of the intact surfactant.

The toxicity of LABS to fish generally increases with increasing alkyl chain length, and approximately a 10-fold difference in toxicity between homologues separated by two carbon atoms has been observed. As also noted for invertebrates, fish are less susceptible to metabolites from biotransformation of LABS. LC50 values below 1 mg/l were found for C11.9 (0.71 mg/l), C13 and C14 (both 0.4 mg/l) in studies with fathead minnow.

LABS sorb to sediment with partition coefficients of 50 to 1,000. The toxicity of LABS bound to sediment is relatively low compared to LABS in solution. NOEC and LOEC values were as high as 319 and 993 mg LABS/kg, respectively, for the sediment-living *Chironomus riparius*. The corresponding NOEC for LABS in solution was as low as 2.4 mg/l indicating that only a small fraction of the sorbed LABS was bioavailable. LABS dissolved in water may also cause chronic effects like reduction of the growth rate of the marine mussel *Mytilus galloprovincialis*. LABS sorbed to sediments did not have similar effects.

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

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DIETHANOLAMINE DODECYLBENZENESULFONATE:

COCONUT DIETHANOLAMIDE:

■ Fatty acid amides (FAA) are nonionics used in hair shampoo, liquid soaps, shaving creams and other personal care products. FAA consist of a fatty acid, usually derived from coconut oil, which is linked to an amide group by a C-N bond. The amide may be typically either be monoethanolamide (MEA), diethanolamide (DEA), or monoisopropanolamide (MIPA).

Most fatty acid amides (FAA), such as the widely used cocodiethanolamide (cocoamide DEA) and cocomonethanolamide (cocoamide MEA), are ultimately degraded in the OECD tests for ready biodegradability under aerobic conditions. The available data describing the aerobic biodegradability of the ethoxylated FAA are contradictory. Certain data indicates that these surfactants do not pass the criteria for ready biodegradability, whereas the opposite is the case for data obtained from Akzo Nobel

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The anaerobic biodegradability of FAA has been examined for cocoamide MEA by using the ECETOC screening test. Ultimate anaerobic biodegradability of cocoamide MEA reached 79% of the theoretical gas production, ThGP, during incubation of diluted digested sludge for 42 days at 35 degree C. By use of the ISO 11734 screening test, which corresponds to the ECETOC method, the ultimate anaerobic biodegradability of cocoamide MEA attained 81% during 56 days

No experimental data describing the bioaccumulation potential of fatty acid amides were found in the literature

The aquatic toxicity of FAA has been determined for species representing the three trophic levels algae, invertebrates, and fish. Cocoamide DEA appears to be more toxic to aquatic organism than cocoamide MEA.

An exceptionally high toxicity of cocoamide MEA was reported for two tests with the green alga *Scenedesmus subspicatus* as the 96 h-EC50 were 1.0 and 1.1 mg/l. More recent tests with a pure cocoamide ME - purity about 95.5% C12-18 gave EC50 values of 16.6 mg/l for *Scenedesmus subspicatus* and 17.8 mg/l for *Pseudokirchneriella subcapitata* (formerly *Selenastrum capricornutum*). The latter data indicate that the toxicity of cocoamide MEA to algae are not markedly higher than the toxicity to daphnids and fish, and EC50 values above 10 mg/l are probably more representative for the toxicity towards algae. The ethoxylated FAA show the same level of aquatic toxicity as the non-ethoxylated FAA

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

**SODIUM (C10-16)ALKYL ETHER SULFATE:**

■ A large environmental data set is available for alcohol ethoxysulfates (AES). On the environmental fate side, this includes standard biodegradation studies, advanced simulation studies of removal in treatment systems, and field monitoring data. On the environmental effects side, acute as well as chronic single-species data are available, as well as advanced studies in micro- and mesocosm systems.

By means of these higher tier exposure and effects data, it could be shown that the use of AES in household detergents and cleaning products results in risk characterization ratios less than one, indicating no concern, for all environmental compartments.

The most frequent initial step in the biodegradation of AES is the cleavage of an ether bond. The cleavage may take place at any ether bond producing a fatty alcohol or an alcohol ethoxylate and ethylene glycol sulfates of various lengths. The length of the alkyl chain and the number of EO units apparently do not affect the degree of aerobic biodegradation, but branching of the alkyl chain may hinder the primary biodegradation of AES. AES are degraded readily and completely under aerobic conditions. E.g., for C12-14 AE3S, a rapid primary degradation of 90-100% is reported to take place within a period of 1 to 5 days. In activated sludge simulation tests 67-99% DOC was removed by degradation of C12-14 AE2S and C12-15 AE3S. The ultimate biodegradation of AES has been confirmed in OECD 301 tests for ready biodegradability.

Based on at least one study, AES are not considered to bioconcentrate in aquatic organisms.

The chemical structure of AES highly influences the effect on aquatic organisms. The relations between alkyl chain length, number of EO groups and toxicity are complex and not yet resolved, but in general, changes in EO numbers affects toxicity more than changes in the alkyl chain length. In AES with alkyl chains of less than C16, the toxicity tended to decrease with increasing numbers of EO, but this was reversed for alkyl chain lengths above C16. The toxicity of AES thus seems to peak at alkyl chain lengths of C16. In a study of the acute toxicity of various AES (C8 to C19.6 and 1-3 EO) to bluegill sunfish (*Lepomis macrochirus*), the LC50 fell from > 250 mg/l for C8 and 375 mg/l for C10 to 24 mg/l for C13, 4-7 mg/l for C14, 2 mg/l for C15 and 0.3 mg/l for C16, and then increased to 10.8 mg/l for C17.9 and 17 mg/l for C19.6. Reported ranges for EC50 for the acute toxicity of AES to daphnids between 1 and 50 mg/l. However, an EC50 of 0.37 mg/l was observed in a 21-day reproduction test with *Daphnia magna*. The LC50 values for fish are in the range between 0.39 to 450 mg/l. A LOEC value of 0.22 mg/l has been reported for a chronic life cycle test with a duration of 1 year. The toxicity of AES towards fish seems to increase with increasing alkyl chain length for AES with up to 16 carbons.

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

**SODIUM TRIPOLYPHOSPHATE:**

■ May cause long-term adverse effects in the aquatic environment.

■ The principal problems of phosphate contamination of the environment relates to eutrophication processes in lakes and ponds. Phosphorus is an essential plant nutrient and is usually the limiting nutrient for blue-green algae. A lake undergoing eutrophication shows a rapid growth of algae in surface waters. Planktonic algae cause turbidity and flotation films. Shore algae cause ugly muddying, films and damage to reeds. Decay of these algae causes oxygen depletion in the deep water and shallow water near the shore. The process is self-perpetuating because anoxic conditions at the sediment/water interface causes the release of more adsorbed phosphates from the sediment. The growth of algae produces undesirable effects on the treatment of water for drinking purposes, on fisheries, and on the use of lakes for recreational purposes.

**WATER:****Ecotoxicity**

Ingredient	Persistence: Water/Soil	Persistence: Air	Bioaccumulation	Mobility
Maxi Vehicle Wash		No data		
diethanolamine		No data		
dodecylbenzenesulfonate		No data		
coconut		No data		
diethanolamide		No data		
sodium (C10-16)		No data		
alkyl ether sulfate		No data		
sodium tripolyphosphate		No data		
water	LOW	No data	LOW	HIGH

**Section 13 - DISPOSAL CONSIDERATIONS**

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■ 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 licenced land-fill or incineration in a licenced apparatus (after admixture with suitable combustible material).
- Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.

## Section 14 - TRANSPORTATION INFORMATION

HAZCHEM: None (ADG7)

NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS: ADG7, UN, IATA, IMDG

## Section 15 - REGULATORY INFORMATION

### POISONS SCHEDULE

None

### REGULATIONS

Regulations for ingredients

**diethanolamine dodecylbenzenesulfonate (CAS: 26545-53-9) is found on the following regulatory lists;**

"Australia Inventory of Chemical Substances (AICS)"

**coconut diethanolamide (CAS: 68603-42-9,61791-31-9) is found on the following regulatory lists;**

"Australia Final Report on Hazard Classification of Common Skin Sensitisers", "Australia Inventory of Chemical Substances (AICS)", "International Council of Chemical Associations (ICCA) - High Production Volume List", "OECD Representative List of High Production Volume (HPV) Chemicals"

**sodium (C10-16)alkyl ether sulfate (CAS: 68585-34-2) is found on the following regulatory lists;**

"Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "OECD Representative List of High Production Volume (HPV) Chemicals"

**sodium tripolyphosphate (CAS: 7758-29-4,15091-98-2) is found on the following regulatory lists;**

"Australia High Volume Industrial Chemical List (HVICL)", "Australia Inventory of Chemical Substances (AICS)", "OECD Representative List of High Production Volume (HPV) Chemicals"

**water (CAS: 7732-18-5) is found on the following regulatory lists;**

"Australia Inventory of Chemical Substances (AICS)", "GESAMP/EHS Composite List of Hazard Profiles - Hazard evaluation of substances transported by ships", "IMO IBC Code Chapter 18: List of products to which the Code does not apply", "OECD Representative List of High Production Volume (HPV) Chemicals"

**No data for Maxi Vehicle Wash (CW: 21-9638)**

## Section 16 - OTHER INFORMATION

### Ingredients with multiple CAS Nos

Ingredient Name	CAS
coconut diethanolamide	68603-42-9, 61791-31-9
sodium tripolyphosphate	7758-29-4, 15091-98-2

■ 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.

A list of reference resources used to assist the committee may be found at:  
[www.chemwatch.net/references](http://www.chemwatch.net/references).

■ The (M)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.

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