

Puma Aqua Degreaser

Puma Energy Australia

Chemwatch: 85-7537 Version No: 3.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: 01/11/2019 Print Date: 14/07/2020

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

Product Identifier

Product name	Puma Aqua Degreaser
Synonyms	Degreaser
Other means of identification	Not Available

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

Relevant identified uses	Degreaser. Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	Puma Energy Australia
Address	365 Macarthur Avenue Hamilton QLD 4007 Australia
Telephone	1300 723 706
Fax	1300 723 321
Website	www.Pumaenergy.com
Email	PumaAu-Safety@pumaenergy.com

Emergency telephone number

Association / Organisation	Chemwatch Emergency Line 24/7	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	1800 24 88 66 (Puma Energy Bitumen Technical Helpline)	+61 1800 951 288
Other emergency telephone numbers	1800 24 88 66 (Puma Energy Bitumen Technical Helpline)	+61 2 9186 1132

Once connected and if the message is not in your prefered language then please dial 01

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

CHEMWATCH HAZARD RATINGS

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

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

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SIGNAL WORD

. WORD | WARNIN

Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.
H411	Toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P321	Specific treatment (see advice on this label).	
P362	Take off contaminated clothing and wash before reuse.	
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.	
P337+P313	If eye irritation persists: Get medical advice/attention.	
P391	Collect spillage.	
P302+P352	IF ON SKIN: Wash with plenty of water.	
P332+P313	If skin irritation occurs: Get medical advice/attention.	

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
25155-30-0	<5	sodium dodecylbenzenesulfonate
111-76-2	<5	ethylene glycol monobutyl ether
6834-92-0	<5	sodium metasilicate, anhydrous
Not Available	<5	Ingredients determined not to be hazardous
7732-18-5	>60	<u>water</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	 If this product comes in contact with the eyes: Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. Seek medical attention without delay; if pain persists or recurs seek medical attention. Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	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	 If swallowed do NOT induce vomiting. If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. Observe the patient carefully. Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.				
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	The emulsion is not combustible under normal conditions. However, it will break down under fire conditions and the hydrocarbon component will burn. May emit poisonous fumes. May emit corrosive 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.

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• 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

- ▶ DO NOT allow clothing wet with material to stay in contact with skin
- ▶ Avoid all personal contact, including inhalation.
- Wear protective clothing when risk of exposure occurs.
- Use in a well-ventilated area.
- Prevent concentration in hollows and sumps.
- ▶ DO NOT enter confined spaces until atmosphere has been checked.
- ▶ DO NOT allow material to contact humans, exposed food or food utensils.
- ▶ Avoid contact with incompatible materials.
- When handling, DO NOT eat, drink or smoke.
- ▶ Keep containers securely sealed when not in use.
- Avoid physical damage to containers.
- Always wash hands with soap and water after handling.
- ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use.
- ▶ Use good occupational work practice.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.
- Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.

Other information

Safe handling

- ▶ 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

20L 5L containers.

- ▶ Polyethylene or polypropylene container.
- Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
- Storage incompatibility

None known

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	20 ppm / 96.9 mg/m3	242 mg/m3 / 50 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
sodium dodecylbenzenesulfonate; (Dodecyl benzene sodium sulfonate)		2.1 mg/m3	23 mg/m3	87 mg/m3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
sodium metasilicate, anhydrous	Sodium silicate; (Sodium metasilicate)	3.8 mg/m3	42 mg/m3	250 mg/m3

Ingredient	Original IDLH	Revised IDLH
sodium dodecylbenzenesulfonate	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	Not Available

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sodium metasilicate, anhydrous	Not Available	Not Available
water	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
sodium dodecylbenzenesulfonate	Е	≤ 0.01 mg/m³
sodium metasilicate, anhydrous	≤ 0.01 mg/m ³	
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

MATERIAL DATA

Exposure controls

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.

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.

Appropriate engineering controls

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Personal protection









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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 lenses or restrictions on use, should be created for each workplace or task. This should Eye and face protection 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 ▶ 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 Hands/feet protection 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

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

Body protection

Other protection

The effect(s) of the following substance(s) are taken into account in the *computer-generated* selection:

non-perfumed moisturiser is recommended.

See Other protection below

Overalls.P.V.C apron.

Barrier cream.Skin cleansing cream.Eye wash unit.

Respiratory protection

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a

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

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

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Material	СРІ
BUTYL	A
NEOPRENE	В
NAT+NEOPR+NITRILE	С
NATURAL RUBBER	С
NITRILE	С
PE/EVAL/PE	С
PVA	С
PVC	С
SARANEX-23	С
VITON	С

* 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.

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	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Green non viscous liquid with characteristic odour; miscible with water.				
Physical state	Liquid	Relative density (Water = 1)	1.02-1.04 @ 25C		
Odour	Not Available	Partition coefficient n-octanol / water	Not Available		
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Applicable		
pH (as supplied)	10.5-11.5	Decomposition temperature	Not Available		
Melting point / freezing point (°C)	0	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	Not Available	Explosive properties	Not Available		
Flammability	Not Applicable	Oxidising properties	Not Available		
Upper Explosive Limit (%)	Not Applicable	Surface Tension (dyn/cm or mN/m)	Not Available		
Lower Explosive Limit (%)	Not Applicable	Volatile Component (%vol)	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

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Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Inhaled

Eye

Chronic

Information on toxicological effects	
	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as
	classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following

exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.

Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial

Not normally a hazard due to non-volatile nature of product

Ingestion Accidental ingestion of the material may be damaging to the health of the individual.

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.

The material may accentuate any pre-existing dermatitis condition

Open cuts, abraded or irritated skin should not be exposed to this material

Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.

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.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Limited evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a significant number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue,

Pulmonary sensitisation, resulting in hyperactive airway dystunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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 repeated skin contact may cause degreasing with drying, cracking and dermatitis following.

Puma Aqua Degreaser	TOXICITY	IRRITATION
	Not Available	Not Available
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 0.25 mg/24hr-SEVERE
	Inhalation (rat) LC50: 0.31 mg/l/4H ^[2]	Eye (rabbit): 1% - SEVERE
sodium	Oral (rat) LD50: 438 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
dodecylbenzenesulfonate		Skin (rabbit): 20 mg/24 hr-SEVERE
		Skin: adverse effect observed (corrosive) ^[1]
		Skin: no adverse effect observed (not irritating) ^[1]
	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
ethylene glycol monobutyl ether	Inhalation (rat) LC50: 449.48655 mg/l/4H ^[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg, open; mild
		Skin: adverse effect observed (irritating) ^[1]

Skin: no adverse effect observed (not irritating)[1]

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sodium metasilicate,	TOXICITY dermal (rat) LD50: >5000 mg/kg ^[1]	IRRITATION Skin (human): 250 mg/24h SEVERE	
anhydrous	Oral (rat) LD50: =600 mg/kg ^[2]	Skin (rabbit): 250 mg/24h SEVERE	
water	TOXICITY Oral (rat) LD50: >90000 mg/kg ^[2]	IRRITATION Not Available	
Legend:	1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances		

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.

Linear alkylbenzene sulfonic acids (LABS) are strong acids (pKa<2) are classified as corrosive (R34) Branched materials exhibit comparable toxicity to linear species.

Acute toxicity: The available data indicate minimal to moderate toxicity, with LD50 values ranging from 500 to 2000 mg/kg body weight (bw). Acute inhalation data also indicate a lack of significant toxicity. Available dermal exposure data also shows a lack of significant toxicity.

LAS are readily absorbed by the gastrointestinal tract after oral administration in animals. LAS are not readily absorbed through the skin. The bulk is metabolised 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. **Repeat dose toxicity:** 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.

Genotoxicity: 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.

Reproductive toxicity: 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)

For aromatic sulfonic acids

Aromatic sulfonic acids are very corrosive as was demonstrated in skin and eye irritation studies, in the acute oral studies, and in the single repeated dose oral study.

Health records from industrial manufacturing exposure, including manufacturing plant book of injuries and a physician report, show toluene-4-sulphonic acid (as handled in manufacturing plants; i.e., a 65% aqueous solution with < 5% free sulphuric acid) is an irritant to the eye and skin.

Sensitisation:

There is a single, key study for sensitization of the aromatic sulphonic acids. None of the tested animals showed positive responses in a, well documented, GLP guinea pig sensitization study with toluene-4-sulphonic acid (CAS No. 104-15-4). The test substance can be considered a non-sensitizer in guinea pigs as none of the test animals showed a positive response to combined intradermal and topical induction followed by topical challenge.

Repeat dose toxicity:

A GLP guideline study with p-toluenesulphonic acid (CAS No. 104-15-4) reported no adverse effects to male and female rats exposed orally for 28 days. The highest dose was 500 mg/kg bw/day (>490 mg/kg bw/day based on >98% active ingredient). Therefore the NOAEL was set at 500 mg/kg bw/day.

Toxicity to reproduction:

No fertility studies are reported for the aromatic sulphonic acids. There are however studies for the chemically related hydrotrope substances that looked at reproductive organs and development of offspring. Hydrotropes are the salt form of the sulphonic acids and therefore are used as read-across for this endpoint. The 90-day oral rat and oral mouse studies and the 2-year chronic dermal rat and mouse studies with the closely related compound sodium xylene sulfonate (CAS No. 1300-72-7) included examination of sex organs of both sexes. No treatment related effects on reproductive organs were reported at doses roughly equivalent to those in the developmental toxicity study. he NOAEL for both maternal and foetal toxicity was the highest dose tested - 3000 mg/kg bw /day which is equivalent to 936 mg active ingredient per kilogram body weight per day. The conclusion of the study was no indications of developmental toxicity including teratogenesis.

SODIUM DODECYLBENZENESULFONATE

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Genetic toxicity:

There is a fully documented, GLP Guideline (OECD 471) Ames Test and a fully documented, GLP Guideline (OECD 473) Chromosome Aberration Test for one of the aromatic sulphonic acids, p-toluenesulphonic acid (CAS No. 104-15-4). Both tests were conducted with and without metabolic activation. The Ames test exposed up to 5000 micrograms/plate and the chromosome aberration test exposed up to 1902 micrograms per liter of the test substance. These studies conclude the substance is neither mutagenic norcytotoxic.

There is an additional, published report of an Ames Test for another of the aromatic sulphonic acids, benzenesulfonic acid (CAS No. 98-11-3). Exposures up to 10,000 micrograms/plate were done with and without metabolic activation. The conclusion is the same as for the p-toluenesulphonic acid; that is, not mutagenic and not cytotoxic.

There are no in vivo mutagenicity studies for the aromatic sulphonic acids, but there are two in vivo mouse micronucleus studies for the related hydrotropes – sodium cumene sulfonate (CAS 28348-53-0) and calcium xylene sulfonate (CAS 28088-63-3). Both are GLP-compliant Guideline mouse micronucleus studies with full documentation. Both studies conclude the test substances were not mutagenic in these assays.

Disulfonic acids have not been the subject of concern.

Carcinogenicity:

There are no carcinogenicity studies for the aromatic sulphonic acids Two hydrotrope studies involve 2-year rat and mouse dermal exposures conducted under GLP. Up to 240 mg (rats) and 727 mg (mice) sodium xylenesulfonate/kg body weight in 50% ethanol were dosed 5 days per week for 104 weeks. There were no treatment related incidences of mononuclear cell leukenia, neoplasms, or nonneoplatic lesions of the skin and other organs. The increased incidence of epidermal hyperplasia may have been related to exposure to the test substance. The NOAEL was reported as 240 mg/kg bw/day for rats and 727 mg/kg bw/day for mice.

Elimination

The US EPA has evaluated the metabolism of analogs in in the sodium alkyl naphthalenesulfonate cluster (SANS), a group of sodium salts of naphthalenesulfonic acids. In a US EPA final rule for SANS, it was stated that "the 1- or 2-sulfonic acid sodium salt moieties on the naphthalene ring may provide a handle by which these compounds can be readily conjugated and eliminated."

NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS

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

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 ethylene glycol monoalkyl ethers and their acetates (EGMAEs):

Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.

EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.

Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE *in vitro* than those of rats.

Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA *in vitro* and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA *in vitro*.

Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in *S. typhimurium* strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenicity and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and in vivo micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental

ETHYLENE GLYCOL MONOBUTYL ETHER

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toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic.

The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).

Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.

At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma.

1: NTP Toxicology Program Technical report Series 484, March 2000.

For ethylene glycol:

Ethylene glycol is quickly and extensively absorbed through the gastrointestinal tract. Limited information suggests that it is also absorbed through the respiratory tract; dermal absorption is apparently slow. Following absorption, ethylene glycol is distributed throughout the body according to total body water. In most mammalian species, including humans, ethylene glycol is initially metabolised by alcohol.

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO2, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO2, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

Respiratory Effects. Respiratory system involvement occurs 12-24 hours after ingestion of sufficient amounts of ethylene glycol and is considered to be part of a second stage in ethylene glycol poisoning The symptoms include hyperventilation, shallow rapid breathing, and generalized pulmonary edema with calcium oxalate crystals occasionally present in the lung parenchyma. Respiratory system involvement appears to be dose-dependent and occurs concomitantly with cardiovascular changes. Pulmonary infiltrates and other changes compatible with adult respiratory distress syndrome (ARDS) may characterise the second stage of ethylene glycol poisoning Pulmonary oedema can be secondary to cardiac failure, ARDS, or aspiration of gastric contents. Symptoms related to acidosis such as hyperpnea and tachypnea are frequently observed; however, major respiratory morbidities such as pulmonary edema and bronchopneumonia are relatively rare and usually only observed with extreme poisoning (e.g., in only 5 of 36 severely poisoned cases).

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol.

Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

Gastrointestinal Effects. Nausea, vomiting with or without blood, pyrosis, and abdominal cramping and pain are common early effects of acute ethylene glycol ingestion. Acute effects of ethylene glycol ingestion in one patient included intermittent diarrhea and abdominal pain, which were attributed to mild colonic ischaemia; severe abdominal pain secondary to colonic stricture and perforation developed 3 months after ingestion, and histology of the resected colon showed birefringent crystals highly suggestive of oxalate deposition.

Musculoskeletal Effects. Reported musculoskeletal effects in cases of acute ethylene glycol poisoning have included diffuse muscle tenderness and myalgias associated with elevated serum creatinine phosphokinase levels, and myoclonic jerks and tetanic contractions associated with hypocalcaemia.

Hepatic Effects. Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol.

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively

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high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

Metabolic Effects. One of the major adverse effects following acute oral exposure of humans to ethylene glycol involves metabolic changes. These changes occur as early as 12 hours after ethylene glycol exposure. Ethylene glycol intoxication is accompanied by metabolic acidosis which is manifested by decreased pH and bicarbonate content of serum and other bodily fluids caused by accumulation of excess glycolic acid. Other characteristic metabolic effects of ethylene glycol poisoning are increased serum anion gap, increased osmolal gap, and hypocalcaemia. Serum anion gap is calculated from concentrations of sodium, chloride, and bicarbonate, is normally 12-16 mM, and is typically elevated after ethylene glycol ingestion due to increases in unmeasured metabolite anions (mainly glycolate).

Neurological Effects: Adverse neurological reactions are among the first symptoms to appear in humans after ethylene glycol ingestion. These early neurotoxic effects are also the only symptoms attributed to unmetabolised ethylene glycol. Together with metabolic changes, they occur during the period of 30 minutes to 12 hours after exposure and are considered to be part of the first stage in ethylene glycol intoxication. In cases of acute intoxication, in which a large amount of ethylene glycol is ingested over a very short time period, there is a progression of neurological manifestations which, if not treated, may lead to generalized seizures and coma. Ataxia, slurred speech, confusion, and somnolence are common during the initial phase of ethylene glycol intoxication as are irritation, restlessness, and disorientation. Cerebral edema and crystalline deposits of calcium oxalate in the walls of small blood vessels in the brain were found at autopsy in people who died after acute ethylene glycol ingestion.

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months. **Reproductive Effects:** Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

Developmental Effects: The developmental toxicity of ethylene glycol has been assessed in several acute-duration studies using mice, rats, and rabbits. Available studies indicate that malformations, especially skeletal malformations occur in both mice and rats exposed during gestation; mice are apparently more sensitive to the developmental effects of ethylene glycol. Other evidence of embyrotoxicity in laboratory animals exposed to ethylene glycol exposure includes reduction in foetal body weight.

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol. **Genotoxic Effects:** Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies provide consistently negative genotoxicity results for ethylene glycol.

SODIUM METASILICATE, ANHYDROUS

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

Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. Prolonged contact is unlikely, given the severity of response, but repeated exposures may produce severe ulceration.

WATER

No significant acute toxicological data identified in literature search.

SODIUM DODECYLBENZENESULFONATE & SODIUM METASILICATE, ANHYDROUS

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.

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: X − 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

Puma Aqua Degreaser	ENDPOINT	TEST DURATION (HR)	SPECIES		VALUE	SOURCE
	Not Available	Not Available	Not Available		Not Available	Not Available
	ENDPOINT	TEST DURATION (HR)	SPECIES		VALUE	SOURCE
	LC50	96	Fish	Fish		4
sodium	EC50	48	Crustacea	Crustacea 2.5m		2
dodecylbenzenesulfonate	EC50	96	Algae or other aquatic plants		1.9mg/L	5
	BCF	8	Fish		1.1mg/L	4
	NOEC	672	Fish		0.15mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	V	ALUE	SOURCE
	LC50	96	Fish	1	-700mg/L	2
ethylene glycol monobutyl ether	EC50	48	Crustacea	C	a.1-800mg/L	2
etilei	EC50	72	Algae or other aquatic plants	1	-840mg/L	2
	NOEC	24	Crustacea	>	1-mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	1	VALUE	SOURCE
sodium metasilicate,	LC50	96	Fish		2-320mg/L	2
anhydrous	EC50	48	Crustacea		1-700mg/L	2
	EC50	72	Algae or other aquatic plants		207mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	SPECIES VALUE		SOURCE
water	LC50	96	Fish	89	7.520mg/L	3
	EC50	96	Algae or other aquatic plants	87	'68.874mg/L	3
Legend:	3. EPIWIN Su	ite V3.12 (QSAR) - Aquatic Toxicit	e ECHA Registered Substances - Ecotoxic y Data (Estimated) 4. US EPA, Ecotox dat NTE (Japan) - Bioconcentration Data 7. M.	abase - Aqu	atic Toxicity D	ata 5.

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.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
water	LOW (KOC = 14.3)

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SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

- ▶ 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.
- Product / Packaging
 disposal

 ▶ Recycle wherever possible.
 ▶ Consult manufacturer for re
 - 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



HAZCHEM

Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

SODIUM DODECYLBENZENESULFONATE 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 Inventory of Chemical Substances (AICS)

ETHYLENE GLYCOL MONOBUTYL ETHER 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 6

Australia Inventory of Chemical Substances (AICS)

International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

SODIUM METASILICATE, ANHYDROUS IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Inventory of Chemical Substances (AICS)

WATER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (sodium dodecylbenzenesulfonate; ethylene glycol monobutyl ether; sodium metasilicate, anhydrous; water)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes

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Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - ARIPS	Yes
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	10/10/2017

SDS Version Summary

Version	Issue Date	Sections Updated
2.1.1.1	10/10/2017	Storage (suitable container), Supplier Information
3.1.1.1	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

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