

MICROSHIELD T TRICLOSAN CLEANSER

Schülke Australia Pty Ltd

Chemwatch: 60-3472

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Chemwatch Hazard Alert Code: 1

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L.GHS.AUS.EN

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

Product Identifier

Product name	MICROSHIELD T TRICLOSAN CLEANSER
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	SDS are intended for use in the workplace. For domestic-use products, refer to consumer labels. Hand and skin antiseptic for external use.
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Details of the supplier of the safety data sheet

Registered company name	Schülke Australia Pty Ltd
Address	2-4 Lyonpark Road Macquarie Park NSW 2113 Australia
Telephone	+61 2 8875 9300
Fax	+61 2 8875 9301
Website	www.schuelke.com.au
Email	customerservice.au@schuelke.com

Emergency telephone number

Association / Organisation	Poisons Information Centre
Emergency telephone numbers	13 11 26
Other emergency telephone numbers	Not Available

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

	Min	Max
Flammability	0	
Toxicity	0	
Body Contact	1	
Reactivity	0	
Chronic	0	

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

Poisons Schedule	Not Applicable
Classification [1]	Acute Aquatic Hazard Category 3, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI

Label elements

GHS label elements	Not Applicable
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SIGNAL WORD **NOT APPLICABLE**

Hazard statement(s)

H412	Harmful to aquatic life with long lasting effects.
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Supplementary statement(s)

Not Applicable

Precautionary statement(s) Prevention

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P273 Avoid release to the environment.

Precautionary statement(s) Response

Not Applicable

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
57-55-6	1-10	<u>propylene glycol</u>
Not Available	<10	citric acid, ethanolamine salt
3380-34-5	<10	triclosan
28348-53-0	<10	<u>sodium cumenesulfonate</u>
9004-82-4	<10	<u>sodium lauryl ether sulfate</u>
9004-62-0	<1	<u>hydroxyethylcellulose</u>
61790-81-6	<1	<u>lanolin, ethoxylated</u>
Not Available	<1	fragrance
Not Available	<1	dye
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	No adverse effects anticipated from normal use. If skin or hair contact occurs: ► 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	► For advice, contact a Poisons Information Centre or a doctor. ► 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.

Emesis is contraindicated as the product may foam. Gastric lavage may be considered.

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
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none">► Alert Fire Brigade and tell them location and nature of hazard.► Wear breathing apparatus plus protective gloves in the event of a fire.► Prevent, by any means available, spillage from entering drains or water courses.► Use fire fighting procedures suitable for surrounding area.► DO NOT approach containers suspected to be hot.► Cool fire exposed containers with water spray from a protected location.
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	<ul style="list-style-type: none">► If safe to do so, remove containers from path of fire.► Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none">► Non combustible.► Not considered to be a significant fire risk.► Expansion or decomposition on heating may lead to violent rupture of containers.► Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).► May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of: carbon dioxide (CO₂)</p>
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Slippery when spilled. Clean up all spills immediately. Wipe up. Place in clean drum then flush area with water.</p>
Major Spills	<p>Slippery when spilled. Minor hazard.</p> <ul style="list-style-type: none">► Clear area of personnel.► Alert Fire Brigade and tell them location and nature of hazard.► Control personal contact with the substance, by using protective equipment as required.► Prevent spillage from entering drains or water ways.► Contain spill with sand, earth or vermiculite.► Collect recoverable product into labelled containers for recycling.► Absorb remaining product with sand, earth or vermiculite and place in appropriate containers for disposal.► Wash area and prevent runoff into drains or waterways.► If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none">► Limit all unnecessary personal contact.► Wear protective clothing when risk of exposure occurs.► Use in a well-ventilated area.► When handling DO NOT eat, drink or smoke.► Always wash hands with soap and water after handling.► Avoid physical damage to containers.► Use good occupational work practice.► Observe manufacturer's storage and handling recommendations contained within this SDS.
Other information	<p>Keep cool. Store below 25 deg.C</p> <ul style="list-style-type: none">► Store in original containers.► Keep containers securely sealed.► Store in a cool, dry, well-ventilated area.► Store away from incompatible materials and foodstuff containers.► Protect containers against physical damage and check regularly for leaks.► Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	Plastic container
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	propylene glycol	Propane-1,2-diol total: (vapour & particulates) / Propane-1,2-diol: particulates only	474 mg/m ³ / 10 mg/m ³ / 150 ppm	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
propylene glycol	Polypropylene glycols	30 mg/m ³	330 mg/m ³	2,000 mg/m ³

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propylene glycol	Propylene glycol; (1,2-Propanediol)	30 mg/m3	1,300 mg/m3	7,900 mg/m3
Ingredient		Original IDLH		
propylene glycol		Not Available		
citric acid, ethanolamine salt		Not Available		
triclosan		Not Available		
sodium cromenesulfonate		Not Available		
sodium lauryl ether sulfate		Not Available		
hydroxyethylcellulose		Not Available		
lanolin, ethoxylated		Not Available		
fragrance		Not Available		
dye		Not Available		
water		Not Available		

MATERIAL DATA

None assigned. Refer to individual constituents.

Exposure controls

Appropriate engineering controls	None under normal operating conditions. Provide adequate ventilation in warehouse or closed storage areas.
Personal protection	   
Eye and face protection	No special equipment for minor exposure i.e. when handling small quantities. OTHERWISE: <ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	Wear general protective gloves, eg. light weight rubber gloves.
Body protection	See Other protection below
Other protection	No special equipment needed when handling small quantities. OTHERWISE: <ul style="list-style-type: none"> ▶ Overalls. ▶ Barrier cream. ▶ Eyewash unit.
Thermal hazards	Not Available

Recommended material(s)**GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:

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Material	CPI
BUTYL	C
NATURAL RUBBER	C
NEOPRENE	C
PE/EVAL/PE	C
PVA	C
VITON	C

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

C: Poor to Dangerous Choice for other than short term immersion

NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -

* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

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

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required.

Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 5 x ES	A-AUS / Class 1 P2	-	A-PAPR-AUS / Class 1 P2
up to 25 x ES	Air-line*	A-2 P2	A-PAPR-2 P2
up to 50 x ES	-	A-3 P2	-
50+ x ES	-	Air-line**	-

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

^ - 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

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Information on basic physical and chemical properties

Appearance	Clear blue-aqua viscous liquid with citrus/floral fragrance; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	1.09
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	5.5	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	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 (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	222.8

SECTION 10 STABILITY AND REACTIVITY

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

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	Not normally a hazard due to non-volatile nature of product		
Ingestion	Ingestion may result in nausea, abdominal irritation, pain and vomiting		
Skin Contact	Excessive use or prolonged contact may lead to defatting, drying and irritation of sensitive skin Not considered to cause discomfort through normal use.		
Eye	The liquid may produce eye discomfort causing transient smarting, blinking		
Chronic	No adverse effects anticipated from normal use. Long-term exposure to the product is not thought to produce chronic effects adverse to health (as classified by EC Directives using animal models); nevertheless exposure by all routes should be minimised as a matter of course.		

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	Not Available	Not Available
propylene glycol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[1] Oral (rat) LD50: 20000 mg/kg ^[2]	Eye (rabbit): 100 mg - mild Eye (rabbit): 500 mg/24h - mild Skin(human):104 mg/3d Intermit Mod Skin(human):500 mg/7days mild
triclosan	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >6000 mg/kg ^[2] Oral (rat) LD50: 3700 mg/kg ^[2]	Eye: SEVERE ** Skin (human):0.75 mg/3d-l- mild Skin (rabbit): 10% - mild
sodium cumenesulfonate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Not Available

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	Oral (rat) LD50: 5200 mg/kg ^[2]	
sodium lauryl ether sulfate	TOXICITY Oral (rat) LD50: 1600 mg/kg ^[2]	IRRITATION Skin (rabbit):25 mg/24 hr moderate
hydroxyethylcellulose	TOXICITY Not Available	IRRITATION Not Available
lanolin, ethoxylated	TOXICITY Oral (rat) LD50: >21300 mg/kg ^[2]	IRRITATION Eye (rabbit): non-irritating * Skin (rabbit): non-irritating *
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

PROPYLENE GLYCOL	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis. The acute oral toxicity of propylene glycol is very low, and large quantities are required to cause perceptible health damage in humans. Serious toxicity generally occurs only at plasma concentrations over 1 g/L, which requires extremely high intake over a relatively short period of time. It would be nearly impossible to reach toxic levels by consuming foods or supplements, which contain at most 1 g/kg of PG. Cases of propylene glycol poisoning are usually related to either inappropriate intravenous administration or accidental ingestion of large quantities by children. The potential for long-term oral toxicity is also low. Because of its low chronic oral toxicity, propylene glycol was classified by the U. S. Food and Drug Administration as "generally recognized as safe" (GRAS) for use as a direct food additive. Prolonged contact with propylene glycol is essentially non-irritating to the skin. Undiluted propylene glycol is minimally irritating to the eye, and can produce slight transient conjunctivitis (the eye recovers after the exposure is removed). Exposure to mists may cause eye irritation, as well as upper respiratory tract irritation. Inhalation of the propylene glycol vapours appears to present no significant hazard in ordinary applications. However, limited human experience indicates that inhalation of propylene glycol mists could be irritating to some individuals. It is therefore recommended that propylene glycol not be used in applications where inhalation exposure or human eye contact with the spray mists of these materials is likely, such as fogs for theatrical productions or antifreeze solutions for emergency eye wash stations. Propylene glycol is metabolised in the human body into pyruvic acid (a normal part of the glucose-metabolism process, readily converted to energy), acetic acid (handled by ethanol-metabolism), lactic acid (a normal acid generally abundant during digestion), and propionaldehyde (a potentially hazardous substance). Propylene glycol shows no evidence of being a carcinogen or of being genotoxic. Research has suggested that individuals who cannot tolerate propylene glycol probably experience a special form of irritation, but that they only rarely develop allergic contact dermatitis. Other investigators believe that the incidence of allergic contact dermatitis to propylene glycol may be greater than 2% in patients with eczema. One study strongly suggests a connection between airborne concentrations of propylene glycol in houses and development of asthma and allergic reactions, such as rhinitis or hives in children. Another study suggested that the concentrations of PGEs (counted as the sum of propylene glycol and glycol ethers) in indoor air, particularly bedroom air, is linked to increased risk of developing numerous respiratory and immune disorders in children, including asthma, hay fever, eczema, and allergies, with increased risk ranging from 50% to 180%. This concentration has been linked to use of water-based paints and water-based system cleaners. Patients with vulvodynia and interstitial cystitis may be especially sensitive to propylene glycol. Women suffering with yeast infections may also notice that some over the counter creams can cause intense burning. Post menopausal women who require the use of an estrogen cream may notice that brand name creams made with propylene glycol often create extreme, uncomfortable burning along the vulva and perianal area. Additionally, some electronic cigarette users who inhale propylene glycol vapor may experience dryness of the throat or shortness of breath. As an alternative, some suppliers will put Vegetable Glycerin in the "e-liquid" for those who are allergic (or have bad reactions) to propylene glycol. Adverse responses to intravenous administration of drugs which use PG as an excipient have been seen in a number of people, particularly with large dosages thereof. Responses may include "hypotension, bradycardia... QRS and T abnormalities on the ECG, arrhythmia, cardiac arrest, serum hyperosmolality, lactic acidosis, and haemolysis". A high percentage (12% to 42%) of directly-injected propylene glycol is eliminated/secreted in urine unaltered depending on dosage, with the remainder appearing in its glucuronide-form. The speed of renal filtration decreases as dosage increases, which may be due to propylene glycol's mild anesthetic / CNS-depressant -properties as an alcohol. In one case, intravenous administration of propylene glycol-suspended nitroglycerin to an elderly man may have induced coma and acidosis. Propylene glycol is an approved food additive for dog food under the category of animal feed and is generally recognized as safe for dogs with an LD50 of 9 mL/kg. The LD50 is higher for most laboratory animals (20 mL/kg) Similarly, propylene glycol is an approved food additive for human food as well. The exception is that it is prohibited for use in food for cats due to links to Heinz body anemia.
	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.
TRICLOSAN	Side-reactions during manufacture of the parent compound may result in the production of trace amounts of polyhalogenated aromatic hydrocarbon(s). Halogenated phenols, and especially their alkali salts, can condense above 300 deg. C. to form polyphenoxophenols or, in a very specific reaction, to form dibenz-p-dioxins
	Polyhalogenated aromatic hydrocarbons (PHAHs) comprise two major groups. The first group represented by the halogenated derivatives of dibenzodioxins (the chlorinated form is PCDD), dibenzofurans (PCDF) and biphenyls (PCB) exert their toxic effect (as hepatotoxicants, reproductive toxicants, immunotoxicants and procarcinogens) by interaction with a cytosolic protein known as the Ah receptor. In guinea pigs the Ah receptor is active in a mechanism which "pumps" PHAH into the cell whilst in humans the reverse appears to be true. This, in part, may account for species differences often cited in

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the literature. This receptor exhibits an affinity for the planar members of this group and carries these to the cellular nucleus where they bind, reversibly, to specific genomes on DNA. This results in the regulation of the production of certain proteins which elicit the toxic response. The potency of the effect is dependent on the strength of the original interaction with the Ah receptor and is influenced by the degree of substitution by the halogen and the position of such substitutions on the parent compound.

The most potent molecule is 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) while the coplanar PCBs (including mono-ortho coplanars) possess approximately 1% of this potency. Nevertheless, all are said to exhibit "dioxin-like" behaviour and in environmental and health assessments it has been the practice to assign each a TCDD-equivalence value.

The most subtle and important biological effects of the PHAHs are the effects on endocrine hormones and vitamin homeostasis. TCDD mimics the effect of thyroxin (a key metamorphosis signal during maturation) and may disrupt patterns of embryonic development at critical stages. Individuals from exposed wildlife populations have been observed to have altered sexual development, sexual dysfunction as adults and immune system suppression. Immunotoxic effects of the PHAHs (including the brominated congener, PBB) have been the subject of several studies. No clear pattern emerges in human studies however with T-cell numbers and function (a blood marker for immunological response) increasing in some and decreasing in others.

Developmental toxicity (e.g. cleft palate, hydronephrosis) occurs in relatively few species; functional alterations following TCDD exposure leads to deficits in cognitive functions in monkeys and to adverse effects in the male reproductive system of rats.

Three incidences have occurred which have introduced abnormally high levels of dioxin or dioxin-like congeners to humans. The explosion at a trichlorophenol-manufacturing plant in Seveso, Italy distributed TCDD across a large area of the country-side, whilst rice-oil contaminated with heat-transfer PCBs (and dioxin-like contaminants) has been consumed by two groups, on separate occasions (one in Yusho, Japan and another in Yu-cheng, Taiwan). The only symptom which can unequivocally be related to all these exposures is the development of chloracne, a disfiguring skin condition, following each incident. Contaminated oil poisonings also produced eye-discharge, swelling of eyelids and visual disturbances. The Babies born up to 3 years after maternal exposure (so-called "Yusho-babies") were characteristically brown skinned, coloured gums and nails and (frequently) produced eye-discharges. Delays in intellectual development have been noted. It has been estimated that Yu-cheng patients consumed an average level of 0.06 mg/kg body weight/day total PCB and 0.0002 mg/kg/day of PCDF before the onset of symptoms after 3 months. When the oil was withdrawn after 6 months they had consumed 1 gm total PCB containing 3.8 mg PCDF. Taiwanese patients consumed 10 times as much contaminated oil as the Japanese patients (because of later withdrawal); however since PCB/PCDF concentration in the Japanese oil was 10 times that consumed in Taiwan, patients from both countries consumed about the same amount of PCBs/PCDFs. Preliminary data from the Yusho cohort suggests a six-fold excess of liver cancer mortality in males and a three-fold excess in women.

Recent findings from Seveso indicate that the biological effects of low level exposure (BELLES), experienced by a cohort located at a great distance from the plant, may be hormetic, i.e. may be protective AGAINST the development of cancer. The PHAHs do not appear to be genotoxic - they do not alter the integrity of DNA. This contrasts with the effects of the many polycyclic aromatic hydrocarbons (PAHs) (or more properly, their reactive metabolites). TCDD induces carcinogenic effects in the laboratory in all species, strains and sexes tested. These effects are dose-related and occur in many organs. Exposures as low as 0.001 ug/kg body weight/day produce carcinoma. Several studies implicate PCBs in the development of liver cancer in workers as well as multi-site cancers in animals. The second major group of PHAH consists of the non-planar PCB congeners which possess two or more ortho-substituted halogens. These have been shown to produce neurotoxic effects which are thought to reduce the concentration of the brain neurotransmitter, dopamine, by inhibiting certain enzyme-mediated processes. The specific effect elicited by both classes of PHAH seems to depend on the as much on the developmental status of the organism at the time of the exposure as on the level of exposure over a lifetime.

NOTE: Some jurisdictions require that health surveillance be conducted on workers occupationally exposed to polycyclic aromatic hydrocarbons. Such surveillance should emphasise

- ▶ demography, occupational and medical history
- ▶ health advice, including recognition of photosensitivity and skin changes
- ▶ physical examination if indicated
- ▶ records of personal exposure including photosensitivity

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 triclosan:

Triclosan is readily absorbed in humans by the skin, through the oral mucous membranes (Lin 2000), through the gastrointestinal tract, and through mucosal tissues following intra-vaginal administration

Triclosan was excreted into the urine and faeces essentially unchanged with some evidence of conjugation. Triclosan has been detected in the liver and fat. In a 13-week dermal subchronic study of triclosan in rats signs of severe dermal irritation were seen in the treated groups, especially in the high-dose group. These signs were erythema, dema, desquamation, and eschar formation. Microscopically, hyperplasia of sebaceous glands, inflammation, and focal necrosis were seen on the skin of treated animals. The dermal effects were reversible during the recovery period. There were no systemic effects that could be treatment-related, although liver masses were observed in two treated animals.

Skin Sensitisation:

Subchronic dermal studies were conducted by applying 0.4 mL of a 2.5% or 5% suspension of triclosan in gum Arabic five times each week for four weeks to the shaved backs of male and female rats (5/sex). No local dermal irritation or systemic toxicity was reported.

Human Skin Irritation and Sensitisation:

Studies were conducted on the skin of human volunteers to determine the compatibility of dermal application of triclosan. The subjects were topically treated with 0.5% triclosan in 1% soap solution according to the Draize method. In the soap control, 0/50 subjects had sensitization or irritation, while 2/50 subjects receiving 0.5% triclosan had a very mild reaction. The conclusion was that triclosan was not a sensitizer or irritant.

Reproductive/Developmental Toxicity:

Reproductive studies were cited in which a NOEL of 50 mg/kg/day was reported for the dams based on effects on the pups. A NOEL based on developmental outcomes was listed as 150-300 mg/kg/day; however, no reproductive tract or fertility abnormalities were reported

Oral administration of triclosan to pregnant mice (gestation days 1-16) resulted in maternal and foetal toxicity at 50 and 100 mg/kg. The authors report no indications of teratogenesis in the mice, or in rats (50 and 100 mg/kg) or in rabbits (10, 25, 50, 100 mg/kg) following administration during gestation.

In a two-generation dose study conducted in rats at doses of 0, 300, 1000, and 3000 ppm in the diet (equivalent to 0, 15, 50, 150 mg/kg), toxicity was noted in the neonates from dams consuming the highest dose, and reductions in survival were seen in f1 and f2 populations with increased kidney dilations.

Triclosan has also been detected in human breast milk, and is probably associated with the fat due to its high lipophilicity

Cellular toxicity:

Triclosan has also been shown to inhibit cell growth in MCF-7 and SK Br-3 human breast cancer cell lines resulting in cellular apoptosis. The authors demonstrated that triclosan reversibly inhibited mammalian fatty acid synthesis (enzyme from SK Br-3 cells and goose uropygial gland). Triclosan was shown also to induce apoptosis in Smulow-Glickman human gingival epithelial cells *in vitro*

Genetic toxicity: The results of 18 mutagenicity tests were summarised, of which 13 were conducted by industry and not reported in the literature. Only one test indicated that triclosan was a mutagen (mammalian spot test), and a repeat of that study was negative.

Endocrine disruption: There have been several reports on endocrine disruptor activity of triclosan. In one study triclosan was weakly androgenic as evidenced by altered fin length and sex ratio in Japanese Medaka fish starting at age 2 days. Additional studies indicated that triclosan was toxic and had weak oestrogenic activity in Medaka. Oestrogen antagonism was induced in frogs following intraperitoneal administration of high doses of triclosan, while lower doses reduced testosterone in male frogs. Additional studies with frogs showed that triclosan bound to thyroid hormone receptor

In another study triclosan exhibited oestrogenic activity as evidenced by competitive binding with estradiol at the estrogen receptor and supported growth of the oestrogen-dependent MCF-7 cell line. The same study demonstrated triclosan bound to the rat androgen receptor, demonstrating androgenic activity.

Nomination Profile Supporting Information for Toxicological Evaluation by the National Toxicology Program July 2008
[Van Waters & Rogers] [Thompson Research]**

MICROSHIELD T TRICLOSAN CLEANSER

for alkyl sulfates; alkane sulfonates and alpha-olefin sulfonates

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

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

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

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

C10-; 290-580

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

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

C14-18, C16-18-; >5000

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

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

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

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

C12-; 200

C12-13 and C10-16->500

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

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

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

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

Under occlusive conditions:

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

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

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

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

Repeat dose toxicity: After repeated oral application of alkyl sulfates with chain lengths between C12 and C18, the liver was the only target organ for systemic toxicity. Adverse effects on this organ included an increase in liver weight, enlargement of liver cells, and elevated levels of liver enzymes. The LOAEL for liver toxicity (parenchymal hypertrophy and an increase in comparative liver weight) was 230 mg/kg/day (in a 13 week study with C16-18 alkyl sulfate, sodium). The lowest NOAEL in rats was 55 mg/kg/day (in a 13 week study with C12-alkyl sulfate, sodium).

C14- and C14-16-alpha-olefin sulfonates produced NOAELs of 100 mg/kg/day (in 6 month- and 2 year studies). A reduction in body weight gain was the only adverse effect identified in these studies.

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

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

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

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

No carcinogenicity studies were available for the alkane sulfonates.

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

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

SODIUM CUMENESULFONATE

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

For alpha-olefin sulfonates (C14-16-alpha-olefin sulfonate, sodium) the NOAEL was 600 mg/kg/day both for maternal and developmental toxicity.

No data were available for the reproductive and developmental toxicity of alkane sulfonates. Based on the available data, the similar toxicokinetic properties and a comparable metabolism of the alkyl sulfates and alkane sulfonates, alkane sulfonates are not considered to be developmental toxicants.

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

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

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

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

HERA Report (Hydrotopes) September 2005

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

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

* Nease Corporation MSDS

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

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

Acute toxicity: AES are of low acute toxicity. Neat AES are irritant to skin and eyes. The irritation potential of AES containing solutions depends on concentration. Local dermal effects due to direct or indirect skin contact with AES containing solutions in hand-washed laundry or hand dishwashing are not of concern because AES is not a contact 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 ug /kg bw/day.

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

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

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

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

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

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

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

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

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

* [CESIO]

Human beings have regular contact with alcohol ethoxylates through a variety of industrial and consumer products such as soaps, detergents, and other cleaning products. Exposure to these chemicals can occur through ingestion, inhalation, or contact with the skin or eyes. Studies of acute toxicity show that volumes well above a reasonable intake level would have to occur to produce any toxic response. Moreover, no fatal case of poisoning with alcohol ethoxylates has ever been reported. Multiple studies investigating the acute toxicity of alcohol ethoxylates have shown that the use of these compounds is of low concern in terms of oral and dermal toxicity.

Clinical animal studies indicate these chemicals may produce gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhea, and lethargy. Similarly, slight to severe irritation of the skin or eye was generated when undiluted alcohol ethoxylates were applied to the skin and eyes of rabbits and rats. The chemical shows no indication of being a genotoxin, carcinogen, or mutagen (HERA 2007). No information was available on levels at which these effects might occur, though toxicity is thought to be substantially lower than that of nonylphenol ethoxylates.

Alcohol ethoxylates are according to CESIO (2000) classified as Irritant or Harmful depending on the number of EO-units:

EO < 5 gives Irritant (Xi) with R38 (Irritating to skin) and R41 (Risk of serious damage to eyes)

EO > 5-15 gives Harmful (Xn) with R22 (Harmful if swallowed) - R38/41

EO > 15-20 gives Harmful (Xn) with R22/41

>20 EO is not classified (CESIO 2000)

Oxo-AE, C13 EO10 and C13 EO15, are Irritating (Xi) with R36/38 (Irritating to eyes and skin).

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

In general, alcohol ethoxylates (AE) are readily absorbed through the skin of guinea pigs and rats and through the gastrointestinal mucosa of rats. AE are quickly eliminated from the body through the urine, faeces, and expired air (CO₂). Orally dosed AE was absorbed rapidly and extensively in rats, and more than 75% of the dose was absorbed. When applied to the skin of humans, the doses were absorbed slowly and incompletely (50% absorbed in 72 hours). Half of the absorbed surfactant was excreted promptly in the urine and smaller amounts of AE appeared in the faeces and expired air (CO₂). The metabolism of C12 AE yields PEG, carboxylic acids, and CO₂ as metabolites. The LD₅₀ values after oral administration to rats range from about 1-15 g/kg body weight indicating a low to moderate acute toxicity.

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

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

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

* [Emery Chemical Co.]

SODIUM
CUMENESULFONATE &
SODIUM LAURYL ETHER
SULFATE &
HYDROXYETHYLCELLULOSE
& WATER

No significant acute toxicological data identified in literature search.

Acute Toxicity	<input checked="" type="checkbox"/>	Carcinogenicity	<input checked="" type="checkbox"/>
Skin Irritation/Corrosion	<input checked="" type="checkbox"/>	Reproductivity	<input checked="" type="checkbox"/>
Serious Eye Damage/Irritation	<input checked="" type="checkbox"/>	STOT - Single Exposure	<input checked="" type="checkbox"/>
Respiratory or Skin sensitisation	<input checked="" type="checkbox"/>	STOT - Repeated Exposure	<input checked="" type="checkbox"/>
Mutagenicity	<input checked="" type="checkbox"/>	Aspiration Hazard	<input checked="" type="checkbox"/>

Legend: X – Data available but does not fill the criteria for classification

✓ – Data required to make classification available

○ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
propylene glycol	LC50	96	Fish	710mg/L	4
propylene glycol	EC50	48	Crustacea	>1000mg/L	4
propylene glycol	EC50	96	Algae or other aquatic plants	10905.921mg/L	3
propylene glycol	EC50	384	Crustacea	311.145mg/L	3
propylene glycol	NOEC	168	Fish	98mg/L	4
triclosan	LC50	96	Fish	0.108mg/L	3
triclosan	EC50	48	Crustacea	0.191mg/L	2
triclosan	EC50	96	Algae or other aquatic plants	0.0007mg/L	4
triclosan	BCF	168	Fish	0.03mg/L	4
triclosan	EC25	96	Algae or other aquatic plants	0.00067mg/L	4
triclosan	NOEC	72	Algae or other aquatic plants	0.0002mg/L	4
sodium cumeresulfonate	EC50	72	Algae or other aquatic plants	>1000mg/L	1
sodium cumeresulfonate	EC50	504	Crustacea	=154mg/L	1
sodium cumeresulfonate	NOEC	504	Crustacea	<30mg/L	1
sodium lauryl ether sulfate	NOEC	48	Fish	0.26mg/L	5

Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data

Legend:

DO NOT discharge into sewer or waterways.

Continued...

MICROSHIELD T TRICLOSAN CLEANSER

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
propylene glycol	LOW	LOW
triclosan	HIGH	HIGH
hydroxyethylcellulose	LOW	LOW
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
propylene glycol	LOW (BCF = 1)
triclosan	LOW (BCF = 90)
hydroxyethylcellulose	LOW (LogKOW = -8.995)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
propylene glycol	HIGH (KOC = 1)
triclosan	LOW (KOC = 18420)
hydroxyethylcellulose	LOW (KOC = 10)
water	LOW (KOC = 14.3)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none">► Recycle wherever possible or consult manufacturer for recycling options.► Consult State Land Waste Management Authority for disposal.► Bury residue in an authorised landfill.► Recycle containers if possible, or dispose of in an authorised landfill.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION

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

PROPYLENE GLYCOL(57-55-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards

Australia Inventory of Chemical Substances (AICS)

Australia Hazardous Substances Information System - Consolidated Lists

TRICLOSAN(3380-34-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

SODIUM CUMENESULFONATE(28348-53-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

SODIUM LAURYL ETHER SULFATE(9004-82-4) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists

Australia Inventory of Chemical Substances (AICS)

HYDROXYETHYLCELLULOSE(9004-62-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

LANOLIN, ETHOXYLATED(61790-81-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

WATER(7732-18-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Continued...

MICROSHIELD T TRICLOSAN CLEANSER

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (propylene glycol; water; hydroxyethylcellulose; lanolin, ethoxylated; sodium cumeresulfonate; sodium lauryl ether sulfate; triclosan)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (hydroxyethylcellulose; lanolin, ethoxylated)
Japan - ENCS	N (water)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION**Other information****Ingredients with multiple cas numbers**

Name	CAS No
triclosan	3380-34-5, 112099-35-1, 164325-69-3, 261921-78-2, 88032-08-0
sodium cumeresulfonate	28348-53-0, 32073-22-6, 15763-76-5, 65130-69-0
sodium lauryl ether sulfate	9004-82-4, 3088-31-1, 68891-38-3, 1335-72-4, 68585-34-2, 91648-56-5, 51286-51-2, 1335-73-5, 11121-04-3, 12627-22-4, 12627-23-5, 32057-62-8, 37325-23-8, 39390-84-6, 39450-08-3, 42504-27-8, 51059-21-3, 53663-56-2, 56572-89-5, 57762-43-3, 57762-59-1, 66747-17-9, 73651-68-0, 74349-47-6, 76724-02-2, 95508-27-3, 98112-64-2, 113096-26-7, 115284-60-1, 116958-77-1
lanolin, ethoxylated	61790-81-6, 8039-09-6

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

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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