



TUB PROTECT

August Race Worldwide Limited

Part Number: 3484184

Version No: 1.2.23.11

Safety Data Sheet (Conforms to Regulation (EU) No 2020/878)

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L.REACH.GBR.EN

SECTION 1 Identification of the substance / mixture and of the company / undertaking

1.1. Product Identifier

Product name	TUB PROTECT
UFI	MTS8-K1UK-2A0X-7W3G
Synonyms	Not Available
Other means of identification	Not Available

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

Product Category Consumer	PC31	Polishes and wax blends
Relevant identified uses	UV Protector/ Sealer for RIB tubes	
Uses advised against	Not Applicable	

1.3. Details of the supplier of the safety data sheet

Registered company name	August Race Worldwide Limited
Address	Unit 7 (The Sail Loft) Singers Yards, Paignton Devon TQ32AH United Kingdom
Telephone	01803 224363
Fax	Not Available
Website	www.august-race.com
Email	info@august-race.com

1.4. Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	01803 224363
Other emergency telephone numbers	Not Available


SECTION 2 Hazards identification

2.1. Classification of the substance or mixture

Classification according to regulation (EC) No 1272/2008 [CLP] and amendments [1]	H315 - Skin Corrosion/Irritation Category 2, H319 - Serious Eye Damage/Eye Irritation Category 2
Legend:	1. Classification by vendor; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

2.2. Label elements

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Hazard pictogram(s)	
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Signal word	Warning
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Hazard statement(s)

H315	Causes skin irritation.
H319	Causes serious eye irritation.

Supplementary statement(s)

EUH208	Contains di-CG 20-568 ethoxylated, CG 20-568 ethoxylated. May produce an allergic reaction.
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Precautionary statement(s) Prevention

P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.

Precautionary statement(s) Response

P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P337+P313	If eye irritation persists: Get medical advice/attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P332+P313	If skin irritation occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

Not Applicable

2.3. Other hazards

isopropanol	Listed in the Europe Regulation (EC) No 1907/2006 - Annex XVII (Restrictions may apply)
di-CG 20-568 ethoxylated	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors
CG 20-568 ethoxylated	Listed in the Europe Regulation (EU) 2018/1881 Specific Requirements for Endocrine Disruptors

SECTION 3 Composition / information on ingredients

3.1. Substances

See 'Composition on ingredients' in Section 3.2

3.2. Mixtures

1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Nanoform Particle Characteristics
1.60828-78-6 2.Not Available 3.Not Available 4.Not Available	0.3	<u>trimethylnonyl ether ethoxylated</u>	Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 4; H302, H315, H318, H413 ^[1]	Not Available
1.26172-55-4 2.247-500-7 3.613-167-00-5 4.Not Available	0.03	<u>5-chloro-2-methyl-4-isothiazolin-3-one</u>	Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 3, Acute Toxicity (Inhalation) Category 3, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 1; H301, H311,	Not Available

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1.CAS No 2.EC No 3.Index No 4.REACH No	%[weight]	Name	Classification according to regulation (EC) No 1272/2008 [CLP] and amendments	Nanoform Particle Characteristics
			H331, H314, H317, H400, H410 [2]	
1.67-63-0 2.200-661-7 3.603-117-00-0 4.Not Available	2	<u>isopropanol</u>	Flammable Liquids Category 2, Serious Eye Damage/Eye Irritation Category 2, Specific Target Organ Toxicity - Single Exposure (Narcotic Effects) Category 3; H225, H319, H336 [2]	Not Available
1.104810-47-1 2.Not Available 3.Not Available 4.Not Available	0.5	<u>di-CG 20-568 ethoxylated</u> [e]	Sensitisation (Skin) Category 1, Hazardous to the Aquatic Environment Long-Term Hazard Category 2; H317, H411 [1]	Not Available
1.104810-48-2 2.400-830-7 3.Not Available 4.Not Available	0.5	<u>CG 20-568 ethoxylated</u> [e]	Sensitisation (Skin) Category 1; H317 [1]	Not Available
1.107-41-5 2.203-489-0 3.603-053-00-3 4.Not Available	10.5	<u>hexylene glycol</u>	Skin Corrosion/Irritation Category 2, Serious Eye Damage/Eye Irritation Category 2; H315, H319 [2]	Not Available
1.308074-31-9 2.Not Available 3.Not Available 4.Not Available	0.1	<u>tallowalkyl(ethylhexyl)dimethylammonium sulfate</u>	Corrosive to Metals Category 1, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 1B, Serious Eye Damage/Eye Irritation Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 1; H290, H302, H314, H318, H400 [1]	Not Available
Legend:		1. Classification by vendor; 2. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 3. Classification drawn from C&L; * EU IOELVs available; [e] Substance identified as having endocrine disrupting properties		

SECTION 4 First aid measures

4.1. Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ 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	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ 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	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.
Ingestion	<ul style="list-style-type: none"> ▶ Immediately give a glass of water. ▶ First aid is not generally required. If in doubt, contact a Poisons Information Centre or a doctor. ▶ If spontaneous vomiting appears imminent or occurs, hold patient's head down, lower than their hips to help avoid possible aspiration of vomitus.

4.2 Most important symptoms and effects, both acute and delayed

See Section 11

4.3. Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

To treat poisoning by the higher aliphatic alcohols (up to C7):

- ▶ Gastric lavage with copious amounts of water.
- ▶ It may be beneficial to instill 60 ml of mineral oil into the stomach.
- ▶ Oxygen and artificial respiration as needed.
- ▶ Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte

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replacement unless shock or severe acidosis threatens.

- To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 l/min.
- Monitor and treat, where necessary, for shock.
- Monitor and treat, where necessary, for pulmonary oedema.
- Anticipate and treat, where necessary, for seizures.
- **DO NOT use emetics.** Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.
- Give activated charcoal.

ADVANCED TREATMENT

- Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.
- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- If the patient is hypoglycaemic (decreased or loss of consciousness, tachycardia, pallor, dilated pupils, diaphoresis and/or dextrose strip or glucometer readings below 50 mg), give 50% dextrose.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Treat seizures with diazepam.
- Proparacaine hydrochloride should be used to assist eye irrigation.

EMERGENCY DEPARTMENT

- Laboratory analysis of complete blood count, serum electrolytes, BUN, creatinine, glucose, urinalysis, baseline for serum aminotransferases (ALT and AST), calcium, phosphorus and magnesium, may assist in establishing a treatment regime. Other useful analyses include anion and osmolar gaps, arterial blood gases (ABGs), chest radiographs and electrocardiograph.
- Positive end-expiratory pressure (PEEP)-assisted ventilation may be required for acute parenchymal injury or adult respiratory distress syndrome.
- Acidosis may respond to hyperventilation and bicarbonate therapy.
- Haemodialysis might be considered in patients with severe intoxication.
- Consult a toxicologist as necessary. BRONSTEIN, A.C. and CURRANCE, P.L. EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

SECTION 5 Firefighting measures

5.1. Extinguishing media

- Alcohol stable foam.
- Dry chemical powder.
- BCF (where regulations permit).
- Carbon dioxide.
- Water spray or fog - Large fires only.

5.2. Special hazards arising from the substrate or mixture

Fire Incompatibility	▸ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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5.3. 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. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	carbon dioxide (CO2) other pyrolysis products typical of burning organic material.

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May emit poisonous fumes.
May emit corrosive fumes.

SECTION 6 Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

See section 8

6.2. Environmental precautions

See section 12

6.3. Methods and material for containment and cleaning up

Minor Spills

Environmental hazard - contain spillage.

- 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

Environmental hazard - contain spillage.
Chemical Class: alcohols and glycols
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

cross-linked polymer - particulate	1	shovel	shovel	R, W, SS
cross-linked polymer - pillow	1	throw	pitchfork	R, DGC, RT
sorbent clay - particulate	2	shovel	shovel	R, I, P
wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT
foamed glass - pillow	4	throw	pitchfork	R, P, DGC, RT

LAND SPILL - MEDIUM

cross-linked polymer - particulate	1	blower	skiploader	R, W, SS
polypropylene - particulate	2	blower	skiploader	W, SS, DGC
sorbent clay - particulate	2	blower	skiploader	R, I, W, P, DGC
polypropylene - mat	3	throw	skiploader	DGC, RT
expanded mineral - particulate	3	blower	skiploader	R, I, W, P, DGC
polyurethane - mat	4	throw	skiploader	DGC, RT

Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT: Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

R.W Melvold et al: Pollution Technology Review No. 150: Noyes Data Corporation 1988

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.

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- ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.
- ▶ If contamination of drains or waterways occurs, advise emergency services.

6.4. Reference to other sections

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

SECTION 7 Handling and storage

7.1. Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Avoid contact with moisture. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ▶ DO NOT allow clothing wet with material to stay in contact with skin
Fire and explosion protection	See section 5
Other information	

7.2. Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<p>Hexylene glycol:</p> <ul style="list-style-type: none"> ▶ is incompatible with strong oxidisers, sulfuric acid, nitric acid, caustics, aliphatic amines, isocyanates <p>Alcohols</p> <ul style="list-style-type: none"> ▶ are incompatible with strong acids, acid chlorides, acid anhydrides, oxidising and reducing agents. ▶ reacts, possibly violently, with alkaline metals and alkaline earth metals to produce hydrogen ▶ react with strong acids, strong caustics, aliphatic amines, isocyanates, acetaldehyde, benzoyl peroxide, chromic acid, chromium oxide, dialkylzincs, dichlorine oxide, ethylene oxide, hypochlorous acid, isopropyl chlorocarbonate, lithium tetrahydroaluminate, nitrogen dioxide, pentafluoroguanidine, phosphorus halides, phosphorus pentasulfide, tangerine oil, triethylaluminium, triisobutylaluminium ▶ should not be heated above 49 deg. C. when in contact with aluminium equipment



X — Must not be stored together

O — May be stored together with specific preventions

+ — May be stored together

Note: Depending on other risk factors, compatibility assessment based on the table above may not be relevant to storage situations, particularly where large volumes of dangerous goods are stored and handled. Reference should be made to the Safety Data Sheets for each substance or article and risks assessed accordingly.

7.3. Specific end use(s)

See section 1.2

SECTION 8 Exposure controls / personal protection

8.1. Control parameters

Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
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Ingredient	DNELs Exposure Pattern Worker	PNECs Compartment
5-chloro-2-methyl-4-isothiazolin-3-one	Inhalation 0.02 mg/m ³ (Local, Chronic) Inhalation 0.04 mg/m ³ (Local, Acute) <i>Oral 0.09 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.02 mg/m³ (Local, Chronic) *</i> <i>Oral 0.11 mg/kg bw/day (Systemic, Acute) *</i> <i>Inhalation 0.04 mg/m³ (Local, Acute) *</i>	3.39 µg/L (Water (Fresh)) 3.39 µg/L (Water - Intermittent release) 3.39 µg/L (Water (Marine)) 0.027 mg/kg sediment dw (Sediment (Fresh Water)) 0.027 mg/kg sediment dw (Sediment (Marine)) 0.01 mg/kg soil dw (Soil) 0.23 mg/L (STP)
isopropanol	Dermal 888 mg/kg bw/day (Systemic, Chronic) Inhalation 500 mg/m ³ (Systemic, Chronic) <i>Dermal 319 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 89 mg/m³ (Systemic, Chronic) *</i> <i>Oral 26 mg/kg bw/day (Systemic, Chronic) *</i>	140.9 mg/L (Water (Fresh)) 140.9 mg/L (Water - Intermittent release) 140.9 mg/L (Water (Marine)) 552 mg/kg sediment dw (Sediment (Fresh Water)) 552 mg/kg sediment dw (Sediment (Marine)) 28 mg/kg soil dw (Soil) 2251 mg/L (STP) 160 mg/kg food (Oral)
CG 20-568 ethoxylated	Dermal 0.25 mg/kg bw/day (Systemic, Chronic) Inhalation 0.35 mg/m ³ (Systemic, Chronic) <i>Dermal 0.025 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 0.085 mg/m³ (Systemic, Chronic) *</i> <i>Oral 0.025 mg/kg bw/day (Systemic, Chronic) *</i>	Not Available
hexylene glycol	Dermal 42 mg/kg bw/day (Systemic, Chronic) Inhalation 44.4 mg/m ³ (Systemic, Chronic) Inhalation 49 mg/m ³ (Local, Chronic) Inhalation 98 mg/m ³ (Local, Acute) <i>Dermal 15 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 7.8 mg/m³ (Systemic, Chronic) *</i> <i>Oral 1.5 mg/kg bw/day (Systemic, Chronic) *</i> <i>Inhalation 25 mg/m³ (Local, Chronic) *</i> <i>Inhalation 49 mg/m³ (Local, Acute) *</i>	0.429 mg/L (Water (Fresh)) 0.043 mg/L (Water - Intermittent release) 4.29 mg/L (Water (Marine)) 1.59 mg/kg sediment dw (Sediment (Fresh Water)) 0.159 mg/kg sediment dw (Sediment (Marine)) 0.066 mg/kg soil dw (Soil) 20 mg/L (STP)

* Values for General Population

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
UK Workplace Exposure Limits (WELs)	isopropanol	Propan-2-ol	400 ppm / 999 mg/m ³	1250 mg/m ³ / 500 ppm	Not Available	Not Available
UK Workplace Exposure Limits (WELs)	hexylene glycol	2-Methylpentane-2,4-diol	25 ppm / 123 mg/m ³	123 mg/m ³ / 25 ppm	Not Available	Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
5-chloro-2-methyl-4-isothiazolin-3-one	0.6 mg/m ³	6.6 mg/m ³	40 mg/m ³
isopropanol	400 ppm	2000* ppm	12000** ppm
hexylene glycol	2.3 ppm	25 ppm	150 ppm

Ingredient	Original IDLH	Revised IDLH
trimethylnonyl ether ethoxylated	Not Available	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available	Not Available
isopropanol	2,000 ppm	Not Available
di-CG 20-568 ethoxylated	Not Available	Not Available
CG 20-568 ethoxylated	Not Available	Not Available
hexylene glycol	Not Available	Not Available
tallowalkyl(ethylhexyl)dimethylammonium sulfate	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
trimethylnonyl ether ethoxylated	E	≤ 0.1 ppm

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Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
5-chloro-2-methyl-4-isothiazolin-3-one	D	> 0.01 to ≤ 0.1 mg/m ³
di-CG 20-568 ethoxylated	D	> 0.1 to ≤ 1 ppm
CG 20-568 ethoxylated	D	> 0.1 to ≤ 1 ppm
tallowalkyl(ethylhexyl)dimethylammonium sulfate	E	≤ 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

For hexylene glycol:

Saturation vapour concentration is 60 ppm @ 20 C. As this is above the exposure standard it indicates atmospheres at ambient temperatures may readily exceed exposure standards.

Exposure at or below the TLV-C is recommended to prevent eye and respiratory irritation.

Odour threshold reported as 50 ppm. At 15-50 ppm most humans detected odour and some minor eye irritation. At 100 ppm for 5 minutes odour was plainly detectable and a slight nasal and respiratory discomfort was experienced by several volunteers. At 1000 ppm for 5 minutes, various degrees of eye irritation and throat and respiratory discomfort were recorded. Values of between 100 and 1000 ppm were probably measured in air saturated with a mist.

Odour Safety Factor(OSF)

OSF=0.5 (HEXYLENE GLYCOL)

Odour Threshold Value: 3.3 ppm (detection), 7.6 ppm (recognition)

Exposure at or below the recommended isopropanol TLV-TWA and STEL is thought to minimise the potential for inducing narcotic effects or significant irritation of the eyes or upper respiratory tract. It is believed, in the absence of hard evidence, that this limit also provides protection against the development of chronic health effects. The limit is intermediate to that set for ethanol, which is less toxic, and n-propyl alcohol, which is more toxic, than isopropanol

8.2. Exposure controls

8.2.1. Appropriate engineering controls

Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.

The basic types of engineering controls are:

Process controls which involve changing the way a job activity or process is done to reduce the risk.

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use. Employers may need to use multiple types of controls to prevent employee overexposure.

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


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

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	<p>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.</p>
8.2.2. Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▸ Safety glasses with side shields. ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▸ Wear chemical protective gloves, e.g. PVC. ▸ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▸ 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. <p>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.</p> <p>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.</p> <p>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.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>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.</p> <p>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.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> · 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 <p>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.</p>
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▸ Overalls. ▸ P.V.C apron. ▸ Barrier cream. ▸ Skin cleansing cream. ▸ Eye wash unit.

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Respiratory protection

Type A 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	-	A-PAPR-AUS / Class 1
up to 25 x ES	Air-line*	A-2	A-PAPR-2
up to 50 x ES	-	A-3	-
50+ x ES	-	Air-line**	-

^ - Full-face

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

- Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

8.2.3. Environmental exposure controls

See section 12

SECTION 9 Physical and chemical properties

9.1. Information on basic physical and chemical properties

Appearance	White		
Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Sweet Smelling	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	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 Available
Flash point (°C)	Not Available	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Available	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available
Nanoform Solubility	Not Available	Nanoform Particle Characteristics	Not Available
Particle Size	Not Available		

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9.2. Other information

Not Available

SECTION 10 Stability and reactivity

10.1.Reactivity	See section 7.2
10.2. Chemical stability	<ul style="list-style-type: none"> ▸ Unstable in the presence of incompatible materials. ▸ Product is considered stable. ▸ Hazardous polymerisation will not occur.
10.3. Possibility of hazardous reactions	See section 7.2
10.4. Conditions to avoid	See section 7.2
10.5. Incompatible materials	See section 7.2
10.6. Hazardous decomposition products	See section 5.3

SECTION 11 Toxicological information

11.1. Information on toxicological effects

Inhaled	<p>The material is not thought to produce adverse health effects or irritation of the respiratory tract (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols. Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>The odour of isopropanol may give some warning of exposure, but odour fatigue may occur. Inhalation of isopropanol may produce irritation of the nose and throat with sneezing, sore throat and runny nose. The effects in animals subject to a single exposure, by inhalation, included inactivity or anaesthesia and histopathological changes in the nasal canal and auditory canal.</p>
Ingestion	<p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols. Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p> <p>Symptoms of over-exposure to hexylene glycol include stimulation of the central nervous system followed by depression, drowsiness, vomiting, coma, respiratory failure, convulsions, renal damage and death. Lethal doses in animals progress to a narcosis lasting for several days; death was delayed for 1-4 days. Human subjects have ingested 5 gm daily for 5 days without apparent ill-effect or urine abnormalities. Eliminated in the urine, partly (20-25%) in conjugated forms</p>

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	<p>The material has NOT been classified by EC Directives or other classification systems as "harmful by ingestion". This is because of the lack of corroborating animal or human evidence. The material may still be damaging to the health of the individual, following ingestion, especially where pre-existing organ (e.g liver, kidney) damage is evident. Present definitions of harmful or toxic substances are generally based on doses producing mortality rather than those producing morbidity (disease, ill-health). Gastrointestinal tract discomfort may produce nausea and vomiting. In an occupational setting however, ingestion of insignificant quantities is not thought to be cause for concern.</p> <p>Following ingestion, a single exposure to isopropyl alcohol produced lethargy and non-specific effects such as weight loss and irritation. Ingestion of near-lethal doses of isopropanol produces histopathological changes of the stomach, lungs and kidneys, incoordination, lethargy, gastrointestinal tract irritation, and inactivity or anaesthesia.</p> <p>Swallowing 10 ml. of isopropanol may cause serious injury; 100 ml. may be fatal if not promptly treated. The adult single lethal doses is approximately 250 ml. The toxicity of isopropanol is twice that of ethanol and the symptoms of intoxication appear to be similar except for the absence of an initial euphoric effect; gastritis and vomiting are more prominent. Ingestion may cause nausea, vomiting, and diarrhoea.</p> <p>There is evidence that a slight tolerance to isopropanol may be acquired.</p> <p>Swallowing of the liquid may cause aspiration of vomit into the lungs with the risk of haemorrhaging, pulmonary oedema, progressing to chemical pneumonitis; serious consequences may result.</p> <p>Signs and symptoms of chemical (aspiration) pneumonitis may include coughing, gasping, choking, burning of the mouth, difficult breathing, and bluish coloured skin (cyanosis).</p>
Skin Contact	<p>Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis.</p> <p>The material may accentuate any pre-existing dermatitis condition</p> <p>Skin contact is not thought to have harmful health effects (as classified under EC Directives); the material may still produce health damage following entry through wounds, lesions or abrasions.</p> <p>When hexylene glycol was applied as a dressing to paediatric burn patients, 36 out of 483 exhibited highly variable periods of coma (hours to weeks) with almost half of the comatose group eventually dying as a result of renal failure.</p> <p>Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>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.</p> <p>511ipa</p>
Eye	<p>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.</p> <p>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.</p> <p>Isopropanol vapour may cause mild eye irritation at 400 ppm. Splashes may cause severe eye irritation, possible corneal burns and eye damage. Eye contact may cause tearing or blurring of vision.</p>
Chronic	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Animal feeding studies with hexylene glycol produce evidence of slight liver and kidney changes.</p> <p>Long term or repeated ingestion exposure of isopropanol may produce incoordination, lethargy and reduced weight gain.</p> <p>Repeated inhalation exposure to isopropanol may produce narcosis, incoordination and liver degeneration. Animal data show developmental effects only at exposure levels that produce toxic effects in the adult animals. Isopropanol does not cause genetic damage in bacterial or mammalian cell cultures or in animals.</p>

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There are inconclusive reports of human sensitisation from skin contact with isopropanol. Chronic alcoholics are more tolerant of systemic isopropanol than are persons who do not consume alcohol; alcoholics have survived as much as 500 ml. of 70% isopropanol.

Continued voluntary drinking of a 2.5% aqueous solution through two successive generations of rats produced no reproductive effects.

NOTE: Commercial isopropanol does not contain "isopropyl oil". An excess incidence of sinus and laryngeal cancers in isopropanol production workers has been shown to be caused by the byproduct "isopropyl oil". Changes in the production processes now ensure that no byproduct is formed. Production changes include use of dilute sulfuric acid at higher temperatures.

LR Sealer UV	TOXICITY	IRRITATION
	Not Available	Not Available
trimethylnonyl ether ethoxylated	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 4780 mg/kg ^[2]	Eye (rabbit): 100 mg-SEVERE
	Oral(Rat) LD50; 5650 mg/kg ^[2]	Eye (rabbit): 5 mg - SEVERE
		Skin (rabbit): 500 (open) - mild
5-chloro-2-methyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	dermal (rat) LD50: >1008 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation(Rat) LC50; 1.23 mg/l4h ^[2]	Skin: adverse effect observed (corrosive) ^[1]
	Oral(Rat) LD50; 481 mg/kg ^[2]	Skin: adverse effect observed (irritating) ^[1]
isopropanol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 12792 mg/kg ^[1]	Eye (rabbit): 10 mg - moderate
	Inhalation(Mouse) LC50; 27.2 mg/l4h ^[2]	Eye (rabbit): 100 mg - SEVERE
	Oral(Mouse) LD50; 3600 mg/kg ^[2]	Eye (rabbit): 100mg/24hr-moderate
		Skin (rabbit): 500 mg - mild
di-CG 20-568 ethoxylated	TOXICITY	IRRITATION
	Not Available	Not Available
CG 20-568 ethoxylated	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Skin (guinea pig): Strong sensit.
	Oral(Rat) LD50; >5000 mg/kg ^[2]	Skin (rabbit): non-irritant
hexylene glycol	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >5000 mg/kg ^[2]	Eye (rabbit): 93mg - SEVERE
	Oral(Guinea) LD50; 2585 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
		Skin (rabbit):465 mg open-mild
		Skin (rabbit):465mg/24hr-moderate
		Skin: no adverse effect observed (not irritating) ^[1]
tallowalkyl(ethylhexyl)dimethylammonium sulfate	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye : Severe
	Oral(Rat) LD50; 1410 mg/kg ^[2]	Skin : Moderate
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	

TRIMETHYLNONYL ETHER ETHOXYLATED

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. RTECS No.: WZ 6210000

5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE

Exposure to the material may result in a possible risk of irreversible effects. The material may produce mutagenic effects in man. This concern is raised, generally, on the basis of appropriate studies with similar materials using mammalian somatic cells in vivo. Such findings are often supported by positive results from in vitro mutagenicity studies.

In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance.

Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration.

The European Union has reclassified several formaldehyde-releasing agents (FRAs) such as methylenedimorpholine (MBM), oxazolidine (MBO) and hydroxypropylamine (HPT) as category 1B carcinogens. Previously, formaldehyde itself was classed as a carcinogen – but formaldehyde-releasing agents were not. This is no longer the case. Based on this regulation, formulations for

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which the maximum theoretical concentration of releasable formaldehyde is more than > 1000 ppm (>0.1%), have to be labelled as carcinogenic.

Water mix metalworking fluids are subject to contamination by bacteria and fungi, and the control of this is an essential part of good fluid maintenance. The use of preservatives both within the formulation and tank-side treatment plays a significant contribution in the protection of potentially harmful microbes that could cause health problems for workers.

A large proportion of bactericides on the market today are classed as formaldehyde releasing biocides which means that under specific conditions they release small amounts of formaldehyde – this is their mode of action in the presence of bacteria. Although they are effective as a biocide their use may become restricted or unfavourable due to potential changes in legislation.

A decision by the ECHA (European Chemicals Agency) was made to re-classify formaldehyde as a category 1b H350 carcinogen and category 2 mutagen in June 2015.

It has also been proposed by the ECHA Risk Assessment Committee (RAC) that formaldehyde release biocides should be classified the same as formaldehyde because formaldehyde is released when these substances come into contact under favorable conditions (i.e. interaction with microorganisms).

Formaldehyde generators (releasers) are often used as preservatives (antimicrobials, biocides, microbiocides). Formaldehyde may be generated following hydrolysis. The most widely used antimicrobial compounds function by releasing formaldehyde once inside the microbe cell. Some release detectable levels of formaldehyde into the air space, above working solutions, especially when pH has dropped.

Many countries are placing regulatory pressure on suppliers and users to replace formaldehyde generators.

Formaldehyde generators are a diverse group of chemicals that can be recognised by a small, easily detachable formaldehyde moiety, prepared by reacting an amino alcohol with formaldehyde ("formaldehyde-condensates"),

There is concern that when formaldehyde-releasing preservatives are present in a formulation that also includes amines, such as triethanolamine (TEA), diethanolamine (DEA), or monoethanolamine (MEA), nitrosamines can be formed,; nitrosamines are carcinogenic substances that can potentially penetrate skin.

One widely-discussed hypothesis states that formaldehyde-condensate biocides, such as triazines and oxazolidines, may cause an imbalance in the microbial flora of in-use metalworking fluids (MWFs). The hypothesis further asserts that this putative microbial imbalance favours the proliferation of certain nontuberculosis mycobacteria (NTM) in MWFs and that the subsequent inhalation of NTM-containing aerosols can cause hypersensitivity pneumonitis (HP), also known as extrinsic allergic alveolitis, in a small percentage of susceptible workers. Symptoms of HP include flu-like illness accompanied by chronic dyspnea, i.e., difficult or laboured respiration

According to Annex VI of the Cosmetic Directive 76/768/EC, the maximum authorised concentration of free formaldehyde is 0.2% (2000 ppm). In addition, the provisions of Annex VI state that, *All finished products containing formaldehyde or substances in this Annex and which release formaldehyde must be labelled with the warning "contains formaldehyde" where the concentration of formaldehyde in the finished product exceeds 0.05%.*

Formaldehyde-releasing preservatives have the ability to release formaldehyde in very small amounts over time. The use of formaldehyde-releasing preservatives ensures that the actual level of free formaldehyde in the products is always very low but at the same time sufficient to ensure absence of microbial growth. The formaldehyde reacts most rapidly with organic and inorganic anions, amino and sulfide groups and electron-rich groups to disrupt metabolic processes, eventually causing death of the organism.

The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.

NOTE: Substance has been shown to be mutagenic in at least one assay, or belongs to a family of chemicals producing damage or change to cellular DNA.

Considered to be the major sensitizer in Kathon CG (1) (1). Bruze et al - Contact Dermatitis 20: 219-39, 1989

ISOPROPANOL

For isopropanol (IPA):

Acute toxicity: Isopropanol has a low order of acute toxicity. It is irritating to the eyes, but not to the skin. Very high vapor concentrations are irritating to the eyes, nose, and throat, and prolonged exposure may produce central nervous system depression and narcosis. Human volunteers reported that exposure to 400 ppm isopropanol vapors for 3 to 5 min. caused mild irritation of the eyes, nose and throat.

Although isopropanol produced little irritation when tested on the skin of human volunteers, there have been reports of isolated cases of dermal irritation and/or sensitization. The use of isopropanol as a sponge treatment for the control of fever has resulted in cases of intoxication, probably the result of both dermal absorption and inhalation. There have been a number of cases of poisoning reported due to the intentional ingestion of isopropanol, particularly among alcoholics or suicide victims. These ingestions typically result in a comatose condition. Pulmonary difficulty, nausea, vomiting, and headache accompanied by various degrees of central nervous system depression are typical. In the absence of shock, recovery usually occurred.

Repeat dose studies: The systemic (non-cancer) toxicity of repeated exposure to isopropanol has been evaluated in rats and mice by the inhalation and oral routes. The only adverse effects-in addition to clinical signs identified from these studies were to the kidney.

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Reproductive toxicity: A recent two-generation reproductive study characterised the reproductive hazard for isopropanol associated with oral gavage exposure. This study found that the only reproductive parameter apparently affected by isopropanol exposure was a statistically significant decrease in male mating index of the F1 males. It is possible that the change in this reproductive parameter was treatment related and significant, although the mechanism of this effect could not be discerned from the results of the study. However, the lack of a significant effect of the female mating index in either generation, the absence of any adverse effect on litter size, and the lack of histopathological findings of the testes of the high-dose males suggest that the observed reduction in male mating index may not be biologically meaningful.

Developmental toxicity: The developmental toxicity of isopropanol has been characterized in rat and rabbit developmental toxicity studies. These studies indicate that isopropanol is not a selective developmental hazard. Isopropanol produced developmental toxicity in rats, but not in rabbits. In the rat, the developmental toxicity occurred only at maternally toxic doses and consisted of decreased foetal body weights, but no teratogenicity

Genotoxicity: All genotoxicity assays reported for isopropanol have been negative

Carcinogenicity: rodent inhalation studies were conducted to evaluate isopropanol for cancer potential. The only tumor rate increase seen was for interstitial (Leydig) cell tumors in the male rats. Interstitial cell tumors of the testis is typically the most frequently observed spontaneous tumor in aged male Fischer 344 rats. These studies demonstrate that isopropanol does not exhibit carcinogenic potential relevant to humans. Furthermore, there was no evidence from this study to indicate the development of carcinomas of the testes in the male rat, nor has isopropanol been found to be genotoxic. Thus, the testicular tumors seen in the isopropanol exposed male rats are considered of no significance in terms of human cancer risk assessment

The substance is classified by IARC as Group 3:

NOT classifiable as to its carcinogenicity to humans.

Evidence of carcinogenicity may be inadequate or limited in animal testing.

DI-CG 20-568 ETHOXYLATED

Increase in absolute liver weight observed. No effect on microsomal protein content was noted, while a dose-dependent decrease in cytosolic protein content was observed. Decreased microsomal hydrolase activity and glutathione S-transferase activity were observed at 50 and 100 mg/kg. Comparatively, increased peroxisomal fatty acid β -oxidation activity and bilirubin UDP-glucuronosyltransferase activity were observed at all tested doses. Dose-dependent increases in lauric acid 11- and 12-hydroxylase activity and decreases in morphine UDP-glucuronosyltransferase activity were noted. Ethoxyresorufin O-de-ethylase activity was significantly decreased at 100 mg/kg and pentoxyresorufin O-depentyase was increased at 50 mg/kg. Immunohistochemical studies indicated conflicting effects on various microsomal P450 isoform levels. Total number and structural changes were increased in hepatocyte organelles. Enlarged hepatic peroxisomes containing matrical plates were observed at 50 and 100 mg/kg. No clinical signs were observed at 10 mg/kg/day for F and at 10 and 50 mg/kg/day for M. Drooling was observed in M and F at 200 and 1000 mg/kg. Alopecia was observed in F at 50 and 1000 mg/kg. 4/5 M died at the highest dose. At 10, 200, and 100 mg/kg, one F died in each group. Decreased body weights were seen at 200 and 1000 mg/kg and 1000 mg/kg in M and F, respectively. Increased albumin levels were noted in all dosed F and 50 and 200 mg/kg M. β -Globulin levels were decreased in 200 mg/kg M and 200 and 1000 mg/kg F. Increased platelet counts in M and F at =200 mg/kg and decreased in lymphocytes in three animals at 1000 mg/kg were noted. Increased alkaline phosphatase activity was observed in M and F at 200 mg/kg and F at 1000 mg/kg. Reduced organ size and weight changes were observed in M and F at 200 and/or 1000 mg/kg. A dose-dependent increase in the development of liver necrosis foci was observed starting at 50 mg/kg. Renal tubular degeneration was noted in all M at 1000 mg/kg. Dam livers showed "moderate to striking peroxisome proliferation at all investigated periods of gestation." Peroxisomes were identified as "slightly increased" or "increased." No mitochondrial changes and a slight decrease in glycogen content on GD 21 were noted. Absolute liver weight was increased. Additionally, peroxisomal fatty acid β -oxidation, lauric acid 11- and 12-hydroxylase, and catalase activities were increased at all time points. Liver malondialdehyde content was increased at GD 15. Selenium-dependent and -independent glutathione peroxidase activities were decreased at GD 15, 18, and 21, and 21, respectively. Subcutaneous and skeletal muscular hemorrhages within the connective tissues noted. Peroxisome proliferation was moderately to strikingly increased at all time points in fetuses. Peroxisome size was also increased. Increased mitochondrial volume and enlarged mitochondria were noted on GD 18 and 21. Glycogen content was "marginal" on GD 18 and 21. Absolute liver weight was not affected. Peroxisomal fatty acid β -oxidation activity was increased at all time points while lauric acid 11- and 12-hydroxylase, and catalase activities were increased at GD 18 and 21. Liver malondialdehyde content was increased at GD 21. Liver total glutathione content and liver content of reduced glutathione were decreased on GD 21 while selenium-dependent glutathione peroxidase activity was increased on GD 21.

CG 20-568 ETHOXYLATED

Inhalation (rat) LC50: > 5.8 mg/l Aerosol Eye (rabbit): non-irritant Ames Test: Non Mutagenic Increase in absolute liver weight observed. No effect on microsomal protein content was noted, while a dose-dependent decrease in cytosolic protein content was observed. Decreased microsomal hydrolase activity and glutathione S-transferase activity were observed at 50 and 100 mg/kg. Comparatively, increased peroxisomal fatty acid β -oxidation activity and bilirubin UDP-glucuronosyltransferase activity were observed at all tested doses. Dose-dependent increases in lauric acid 11- and 12-hydroxylase activity and decreases in morphine UDP-glucuronosyltransferase activity were noted. Ethoxyresorufin O-de-ethylase activity was significantly decreased at 100 mg/kg and pentoxyresorufin O-depentyase was increased at 50

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	<p>mg/kg. Immunohistochemical studies indicated conflicting effects on various microsomal P450 isoform levels. Total number and structural changes were increased in hepatocyte organelles. Enlarged hepatic peroxisomes containing matrical plates were observed at 50 and 100 mg/kg Dam livers showed "moderate to striking peroxisome proliferation at all investigated periods of gestation." Peroxisomes were identified as "slightly increased" or "increased." No mitochondrial changes and a slight decrease in glycogen content on GD 21 were noted. Absolute liver weight was increased. Additionally, peroxisomal fatty acid β-oxidation, lauric acid 11- and 12-hydroxylase, and catalase activities were increased at all time points. Liver malondialdehyde content was increased at GD 15. Selenium-dependent and -independent glutathione peroxidase activities were decreased at GD 15, 18, and 21, and 21, respectively. Subcutaneous and skeletal muscular hemorrhages within the connective tissues noted. Peroxisome proliferation was moderately to strikingly increased at all time points in fetuses. Peroxisome size was also increased. Increased mitochondrial volume and enlarged mitochondria were noted on GD 18 and 21. Glycogen content was "marginal" on GD 18 and 21. Absolute liver weight was not affected. Peroxisomal fatty acid β-oxidation activity was increased at all time points while lauric acid 11- and 12-hydroxylase, and catalase activities were increased at GD 18 and 21. Liver malondialdehyde content was increased at GD 21. Liver total glutathione content and liver content of reduced glutathione were decreased on GD 21 while selenium-dependent glutathione peroxidase activity was increased on G</p>
<p>TALLOWALKYL(ETHYLHEXYL)DIMETHYLAMMONIUM SULFATE</p>	<p>No specific data describing the health effects of cationic dialkyldimethylammonium (DADMA - dimonium) salts are readily available. However, many of the properties described for alkyltrimethylammonium (ATMA) salts also apply to DADMA salts, although these are generally less irritating than the corresponding ATMA salts</p> <p>For alkyltrimethylammonium chloride (ATMAC)</p> <p>Most undiluted cationic surfactants satisfy the criteria for classification as Harmful (Xn) with R22 and as Irritant (Xi) for skin and eyes with R38 and R41. In addition, certain surfactants will satisfy the criteria for classification as Corrosive with R34 in addition to the acute toxicity.</p> <p>According to Centre Europeen des Agents de Surface et de leurs Intermediaires Organiques (CESIO), C8-18 alkyltrimethylammonium chloride (ATMAC) (i.e., lauryl, coco, soya, and tallow) are classified as Corrosive (C) with the risk phrases R22 (Harmful if swallowed) and R34 (Causes burns). C16 ATMAC is classified as Harmful (Xn) with the risk phrases R22 (Harmful if swallowed), R38 (Irritating to skin), and R41 (Risk of serious damage to eyes). C20-22 ATMAC are classified as Irritant (Xi) with R36/38 (Irritating to eyes and skin).</p> <p>Toxokinetics and Acute Toxicity: The few available absorption studies conducted with cationic surfactants indicate that absorption occurs in small amounts through the skin. Percutaneous absorption of radiolabelled C12 alkyltrimethylammonium bromide (ATMAB) in 3% aqueous solution (applied to an 8 cm² area with occlusion) in the rat was low and corresponded to 0.6% of the applied ¹⁴C activity in 72 hours. Most of the absorbed surfactant was excreted in the urine, i.e. 0.35% of the applied ¹⁴C activity within the first 24 hours, whereas 13.2% remained on the skin after rinsing. Cutaneous application of the surfactant without rinsing resulted in a greater degree of percutaneous absorption (3.15%) in 48 hours. In the rat elimination after parenteral administration was rapid and was effected primarily via the urine, - more than 80% of the radioactivity was eliminated within 24 hours of application. About 80% of the ¹⁴C activity was found in the gastrointestinal tract 8 hours after oral administration of ¹⁴C-labelled C16 ATMAB. Only small amounts of the applied radioactivity were found in the urine and in the blood plasma. This indicates poor intestinal absorption. Similar small amounts of ¹⁴C were found in the liver, kidneys, spleen, heart, lungs and skeletal muscles. Within 3 days of ingestion, 92% of the administrated radioactivity had been excreted in the faeces and 1% in the urine. No appreciable enterohepatic circulation of the radioactivity was found. The acute oral toxicity of alkyltrimethylammonium salts is somewhat higher than the toxicity of anionic and nonionic surfactants. This may be due to the strongly irritating effect which cationic surfactants exhibit on the mucous membrane of the gastrointestinal tract (SFT 1991). Cationic surfactants are generally about 10 times more toxic when administrated by the intravenous route compared to oral administration.</p> <p>Skin and Eye Irritation: Skin irritation depends on surfactant concentration. Regardless of the structure, cationic surfactants lead to serious destruction of the skin at high concentrations. Solutions of approximately 0.1% are rarely irritating, whereas irritation is usually pronounced at concentrations between 1.0 and 10.0% surfactant. C16 ATMAC was severely irritating to rabbit skin in a concentration of 2.5%. The surfactant was applied to intact and abraded sites and scored after 34 hours. Then the skin was rinsed and then scored again after 48 hours. The erythema and Eschar Index was 3.75 (maximum 4) and the edema Index was 2.0 (maximum 4).</p> <p>With regard to eye irritation, cationic surfactants are the most irritating of the surfactants. The longer chained alkyltrimethylammonium salts are less irritating to the rabbit eye than the shorter alkyl chain homologues. C10 ATMAB, C12 ATMAB, and C16 ATMAC were tested in concentrations between 0.1 and 1.0% in water and were found to be significantly irritating or injurious to the rabbit eye. A 5% solution of C18 ATMAC was instilled into the eyes of guinea pigs, and this concentration was very irritating with a total PII (The Primary Irritation Index) score of 96 (maximum 110).</p> <p>A homologous series of ATMAB produced very little swelling of the stratum corneum and some homologues produced a shrinkage of the stratum corneum after prolonged exposure.</p> <p>Many proteins in the skin are considerably more resistant to the denaturing effects of cationic surfactants compared to those of anionic surfactants. As cationic surfactants frequently have a lower critical micelle concentration than the anionic surfactants, a saturation of the surfactant/protein complex is prevented by the formation of micelles.</p> <p>Compared to a representative anionic surfactant, the cooperative binding with subsequent protein</p>

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denaturation requires about a tenfold higher concentration of a cationic surfactant. Contrary to the irreversible denaturing effect of sodium dodecyl sulfate, the adverse effects of some cationic surfactants on proteins may be reversible. Cationic surfactants can interact with proteins or peptides by polar and hydrophobic binding. Polar interactions result in electrostatic bonds between the negatively charged groups of the protein molecule and the positively charged surfactant molecule.

Sensitisation: A repeated insult patch test of C16 ATMAC was conducted with 114 volunteers. Seventeen days after the last induction of 0.25% surfactant, a challenge patch of 0.25% was applied. No sensitization was observed.

Sub-chronic toxicity: C16 ATMAC was administered at concentrations of 10, 20, and 45 mg/kg/day via the drinking water to rats for one year. The only effect observed was a decrease in body weight gain in the 45 mg/day dose group.

Reproductive Toxicity: No embryo toxic effects were seen, when C18 ATMAC was applied dermally to pregnant rats during the period of major organogenesis (day 6-15 of gestation). The concentrations of C18 ATMAC were 0.9, 1.5 and 2.5%. There was no increase in the incidence of fetal malformations. C16 ATMAC was not teratogenic in rats after oral doses. Mild embryonic effects were observed with 50 mg/kg/day, but these effects were attributed to maternal toxicity rather than to a primary embryonic effect. Lower doses of C16 ATMAC showed no embryo toxic or teratogenic effects.

Mutagenicity: C16 ATMAC was studied in in vitro short-term tests to detect potential mutagenic effects. Cultures of Syrian golden hamster embryo cells were used for an in vitro bioassay. No in vitro transformation of hamster embryo cells was induced, and C16 ATMAC was not mutagenic in *Salmonella typhimurium* (Inoue and Sunakawa 1980). No mutagenic effects or genetic damages were indicated in a survey of nine short-term genotoxicity tests with C16 and C18 ATMAC (Yam *et al.* 1984).

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

For quaternary ammonium compounds (QACs):

Quaternary ammonium compounds (QACs) are cationic surfactants. They are synthetic organically tetra-substituted ammonium compounds, where the R substituents are alkyl or heterocyclic radicals. A common characteristic of these synthetic compounds is that one of the R's is a long-chain hydrophobic aliphatic residue.

The cationic surface active compounds are in general more toxic than the anionic and non-ionic surfactants. The positively-charged cationic portion is the functional part of the molecule and the local irritation effects of QACs appear to result from the quaternary ammonium cation.

Due to their relative ability to solubilise phospholipids and cholesterol in lipid membranes, QACs affect cell permeability which may lead to cell death. Further QACs denature proteins as cationic materials precipitate protein and are accompanied by generalised tissue irritation.

It has been suggested that the experimentally determined decrease in acute toxicity of QACs with chain lengths above C16 is due to decreased water solubility.

In general it appears that QACs with a single long-chain alkyl groups are more toxic and irritating than those with two such substitutions,

The straight chain aliphatic QACs have been shown to release histamine from minced guinea pig lung tissue. However, studies with benzalkonium chloride have shown that the effect on histamine release depends on the concentration of the solution. When cell suspensions (11% mast cells) from rats were exposed to low concentrations, a decrease in histamine release was seen. When exposed to high concentrations the opposite result was obtained.

In addition, QACs may show curare-like properties (specifically benzalkonium and cetylpyridinium derivatives, a muscular paralysis with no involvement of the central nervous system. This is most often associated with lethal doses. Parenteral injections in rats, rabbits and dogs have resulted in prompt but transient limb paralysis and sometimes fatal paresis of the respiratory muscles. This effect seems to be transient.

From human testing of different QACs the generalised conclusion is obtained that all the compounds investigated to date exhibit similar toxicological properties.

Rat data based on similar material * Moderately irritating to the skin* Practically non-toxic in contact with skin * Severely irritating to eyes * Akzo Nobel MSDS*

LR Sealer UV & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & DI-CG 20-568 ETHOXYLATED & CG 20-568 ETHOXYLATED

The following information refers to contact allergens as a group and may not be specific to this product.

Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.

LR Sealer UV & HEXYLENE GLYCOL

For hexylene glycol

Acute toxicity: Hexylene glycol is of relatively low acute toxicity to mammals, the acute oral LD50 is >2000 and <5000 mg/kg="" (range="">>2000-4700 mg/kg) while the dermal LD50 is >2000 mg/kg (range >1.84-12.3 g/kg). The acute inhalational LC50 is ³ the saturated vapour concentration. Skin and eye irritation guideline studies indicate that hexylene glycol has low potential to irritate the skin

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	<p>and is slightly irritating to the eye. Skin and eye effects are reversible. Hexylene glycol is not a skin sensitiser.</p> <p>Repeat dose toxicity: Repeated exposure by oral gavage to rats at 50, 150 or 450 mg/kg/day hexylene glycol for 90 days, with additional animals at the top dose also allowed a 4 week exposure-free recovery period, resulted in hepatocellular hypertrophy and increased liver weight, male rat specific nephropathy and inflammatory changes in the forestomach and to a lesser extent the glandular stomach. The liver changes were reversible and considered an adaptive physiological response to increased metabolic demand. The male rat nephropathy was partially reversible and associated with an increased severity of acidophilic globules, subsequently identified by specific staining (Masson's trichrome) as alpha-2-microglobulins, and considered of questionable biological significance to humans. Changes in the stomach (reversible) and forestomach (partially reversible) were considered attributable to local irritation induced by the gavage procedure. The NOAEL for this local effect being 50 mg/kg/day. The systemic NOAEL for this guideline study is considered to be 450 mg/kg/day with a no effect level for local irritation to the stomach and forestomach of 50 mg/kg/day.</p> <p>Genotoxicity: Hexylene glycol is not genotoxic in either mammalian or non-mammalian cells <i>in vitro</i>.</p> <p>Reproductive and developmental toxicity: No standard fertility studies are available. No effects on the gonads were observed in a good quality 90-day oral gavage study in rats, which were, administered hexylene glycol at doses up to 450 mg/kg/day by oral gavage. In a good quality developmental toxicity study, in which rats received 30, 300 or 1000 mg/kg/day hexylene glycol by oral gavage, the LOAEL for maternal toxicity was 1000 mg/kg/day, based on slightly reduced weight gain at this top dose level. Greater pre-implantation loss observed at this dose level may be regarded of questionable biological significance. This dose level was also the LOAEL for foetotoxicity based on a, slight delay in ossification, a greater number of foetuses with extra thoraco-lumbar ribs, and a slight decrease (not statistically significant) in foetal body weight. There was no evidence of teratogenicity up to the limit dose of 1000 mg/kg.</p>
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOPROPANOL & TALLOWALKYL(ETHYLHEXYL)DIMETHYLAMMONIUM SULFATE	<p>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.</p>
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & DI-CG 20-568 ETHOXYLATED & CG 20-568 ETHOXYLATED	<p>No significant acute toxicological data identified in literature search.</p>
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE & ISOPROPANOL	<p>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.</p>
DI-CG 20-568 ETHOXYLATED & CG 20-568 ETHOXYLATED	<p>For benzotriazoles</p> <p>There are several indications that the effects of phenolic benzotriazoles described in the literature might be caused by endocrine disruption, e.g. reduced concentrations of testosterone, higher concentrations of CYP 450, or higher activity of ethoxyresorufin-O-deethylase (EROD-activity). As in these cases there are also indications for toxic effects on the liver reported, the effects might actually be only secondary effects. With the present knowledge it is not possible to attribute them unambiguously as endocrine adverse effects of an equivalent level of concern.</p> <p>Several benzotriazole UV stabilisers showed significant human aryl hydrocarbon receptor (AhR) ligand activity. The AhR has roles in regulating immunity, stem cell maintenance, and cellular differentiation. A study indicated that certain benzotriazole UV stabilisers have the potential to accumulate and exert potent physiological effects in humans, analogous to polycyclic aromatic hydrocarbons and dioxins, which are known stable and toxic ligands. The polycyclic aromatic hydrocarbon the polycyclic aromatic hydrocarbon, benzo[a]pyrene (BaP), a ligand for AhR, induces its own metabolism and bioactivation to a toxic metabolites.</p> <p>Benzotriazole is the core structure present within the phenolic benzotriazole class. In vitro metabolism with rat liver microsomes yielded formation of 5- and 4-hydroxybenzotriazole (1.6 and 0.32% of the amount added, respectively). Overall metabolism was low (<5% of the total amount added). Oral acute studies in rats and mice yielded LD50 values that ranged from 560 to 909 mg/kg. Intraperitoneal LD50 values in mice and rats ranged from 400-1000 and 500-900 mg/kg, respectively. A mouse intravenous LD50 of 238 mg/kg was identified. Dermal LD50 values were =1000 mg/kg in rats and rabbits, and inhalation LC50 values in rats were 1.5 mg/L and 1.91 mg/L/3 hours). Subchronic and short-term studies showed that oral administration to mice produced</p>

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minimal effects on body weight while dose-dependent decreases in body weight were observed in rats. Endocrine effects, normocytic anemia, and leukopenia were noted in rats dosed for 26 weeks. The TDLo was 109 mg/kg. No effects on deaths and no clinical symptoms were noted in mice or rats orally administered (in food) benzotriazole for 78 weeks. Additionally, no dose-related effects on reproductive organs were noted in either sex. Neoplastic liver nodules were observed in male Fischer rats fed 12,100 ppm benzotriazole for 78 weeks. However, historic laboratory controls incidences varied from 0 to 11% so the treatment-related effects could not be determined. Brain tumors occurred in three males and one female rat. Incidence of endometrial stromal polyps was increased significantly in female rats fed 6700 ppm for 78 weeks (22%), but not in female rats fed 12,100 ppm (16%). Significant increase in alveolar/bronchiolar carcinomas (18%) was observed female B6C3F1 fed 11,700 ppm benzotriazole for 104 weeks. Comparatively, a similar increase was not observed in female mice fed 23,500 ppm benzotriazole for the same period of time (6% increase). Historical laboratory control incidences varied from 0 to 7%. Genotoxicity studies indicate that the compound was not mutagenic to *S. typhimurium* strains TA97, TA98, or TA100 in the presence or absence of S9, or Chinese hamster ovary cells. Benzotriazole was also not mutagenic to *S. typhimurium* strain TA1535 in the absence of S9, but was mutagenic in the presence of S9. Conflicting results were obtained for effects in *S. typhimurium* strains TA1537 and TA1538 and *E. coli* WP2 uvrA. It did not produce DNA damage in *E. coli* PQ37. In Chinese hamster ovary cells, benzotriazole induced chromosomal aberrations in the presence of S9 and sister chromatid exchange in the absence of S9. Benzotriazole was not genotoxic in the mouse micronucleus assay at 800 mg/kg. Benzotriazole was identified as a non-sensitizer in the guinea pig maximization test. Benzotriazole was identified as irritating to rabbit eyes and minimally irritating to rabbit and guinea pig skin.

For phenolic benzotriazoles

Overall, oral exposure (either through gavage or in feed) of the tested chemicals to rats led to liver effects. Increased absolute and/or relative liver weights were observed in several studies. Body weight and body weight gain changes were observed after administration of several test substances. Histopathological changes (e.g., foci, hypertrophy, and cytoplasmic vacuolization) and altered liver enzyme content and activities were also noted after treatment with different phenolic benzotriazoles. Haematological effects (e.g., altered white and red blood cell counts, altered albumin levels, and packed cell volume) were observed. For those studies that calculated no observed adverse effect levels (NOAELs), the values ranged from <0.5 to ~5685 mg/kg/day.

Reproductive and teratology effects: The chemicals tested produced a variety of effects. Some chemicals were shown to affect reproductive organ weights, but no direct studies in reproduction and development were located.

Genotoxicity None of the tested compounds were identified as mutagenic in vitro in the absence or presence of a metabolic system (S9) or in vivo.

Chemical Information Review Document for Phenolic Benzotriazoles: Supporting Nomination for Toxicological Evaluation by the National Toxicology Program October 2011

http://ntp.niehs.nih.gov/ntp/noms/support_docs/phenolicbenzotriazoles_cird_oct2011_508.pdf

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved.

Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

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

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autooxidation also increases the irritation. Because of their irritating effect, it is difficult

to diagnose ACD to these compounds by patch testing.

Allergic Contact Dermatitis—Formation, Structural Requirements, and Reactivity of Skin Sensitizers.

Ann-Therese Karlberg et al; Chem. Res. Toxicol. 2008, 21, 53-69

Polyethylene glycols (PEGs) have a wide variety of PEG-derived mixtures due to their readily linkable terminal primary hydroxyl groups in combination with many possible compounds and complexes such as ethers, fatty acids, castor oils, amines, propylene glycols, among other derivatives. PEGs and their derivatives are broadly utilized in cosmetic products as surfactants, emulsifiers, cleansing agents, humectants, and skin conditioners.

PEGs and PEG derivatives were generally regulated as safe for use in cosmetics, with the conditions that impurities and by-products, such as ethylene oxides and 1,4-dioxane, which are known carcinogenic materials, should be removed before they are mixed in cosmetic formulations. Most PEGs are commonly available commercially as mixtures of different oligomer sizes in broadly- or narrowly-defined molecular weight (MW) ranges. For instance, PEG-10,000 typically designates a mixture of PEG molecules (n = 195 to 265) having an average MW of 10,000. PEG is also known as polyethylene oxide (PEO) or polyoxyethylene (POE), with the three names being chemical synonyms. However, PEGs mainly refer to oligomers and polymers with molecular masses below

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20,000 g/mol, while PEOs are polymers with molecular masses above 20,000 g/mol, and POEs are polymers of any molecular mass. Relatively small molecular weight PEGs are produced by the chemical reaction between ethylene oxide and water or ethylene glycol (or other ethylene glycol oligomers), as catalyzed by acidic or basic catalysts. To produce PEO or high-molecular weight PEGs, synthesis is performed by suspension polymerization. It is necessary to hold the growing polymer chain in solution during the course of the poly-condensation process. The reaction is catalyzed by magnesium-, aluminum-, or calcium-organoelement compounds. To prevent coagulation of polymer chains in the solution, chelating additives such as dimethylglyoxime are used

Safety Evaluation of Polyethylene Glycol (PEG) Compounds for Cosmetic Use: Toxicol Res 2015; 31:105-136 The Korean Society of Toxicology
<http://doi.org/10.5487/TR.2015.31.2.105>

Acute Toxicity	✗	Carcinogenicity	✗
Skin Irritation/Corrosion	✓	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✗
Respiratory or Skin sensitisation	✗	STOT - Repeated Exposure	✗
Mutagenicity	✗	Aspiration Hazard	✗

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

11.2.1. Endocrine Disruption Properties

Many chemicals may mimic or interfere with the body's hormones, known as the endocrine system. Endocrine disruptors are chemicals that can interfere with endocrine (or hormonal) systems. Endocrine disruptors interfere with the synthesis, secretion, transport, binding, action, or elimination of natural hormones in the body. Any system in the body controlled by hormones can be derailed by hormone disruptors. Specifically, endocrine disruptors may be associated with the development of learning disabilities, deformations of the body various cancers and sexual development problems. Endocrine disrupting chemicals cause adverse effects in animals. But limited scientific information exists on potential health problems in humans. Because people are typically exposed to multiple endocrine disruptors at the same time, assessing public health effects is difficult.

SECTION 12 Ecological information

12.1. Toxicity

LR Sealer UV	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
trimethylnonyl ether ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	0.018-0.026mg/L	4
	LC50	96h	Fish	0.13-0.31mg/L	4
	EC50	48h	Crustacea	4.71mg/l	1
	NOEC(ECx)	504h	Crustacea	0.172mg/l	1
	EC50	96h	Algae or other aquatic plants	0.03-0.13mg/L	4
isopropanol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	24h	Algae or other aquatic plants	0.011mg/L	4
	EC50	72h	Algae or other aquatic plants	>1000mg/l	1
	LC50	96h	Fish	4200mg/l	4
	EC50	48h	Crustacea	7550mg/l	4
	EC50	96h	Algae or other aquatic plants	>1000mg/l	1
di-CG 20-568 ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

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CG 20-568 ethoxylated	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
hexylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	72h	Algae or other aquatic plants	>429mg/l	2
	EC50	48h	Crustacea	2800mg/l	1
	LC50	96h	Fish	>100mg/l	4
	EC10(ECx)	72h	Algae or other aquatic plants	>429mg/l	2
tallowalkyl(ethylhexyl)dimethylammonium sulfate	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available
Legend: Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data					

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

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.

Toxic to soil organisms.

For hexylene glycol:

log Kow : -0.14

BOD 5 : <0.004-0.02

COD : 2.2-2.3

Environmental fate:

Hexylene glycol is a liquid, melting point – 50 C, boiling point 197.5 C, vapour pressure 0.07 hPa at 20 C, it is fully miscible in water and has a calculated n-octanol water partition coefficient (log Kow) of 0.58.

The calculated half-life for the photo-oxidation (reaction with hydroxyl radicals) of hexylene glycol in air is 9 hours. Hexylene glycol is not expected to undergo direct photolysis and is not susceptible to hydrolysis.

Hexylene glycol is predicted to distribute in the environment primarily to water or water and soil. Based on a calculated log Kow of 0.58 which suggests a log Koc of <1, hexylene glycol has low potential to bioaccumulate (BCF=3) and low potential for sorption to soil. In water, hydrolysis and photodegradation are not expected to occur. Hexylene glycol is at least inherently biodegradable.

Ecotoxicity:

Hexylene glycol is of low acute toxicity to aquatic organisms. The lowest valid 96h LC50 for fish was 8510 mg/l (Mosquito fish, *Gambusia affinis*) and the lowest valid 48 h EC50 for invertebrates was 2800 mg/l (*Ceriodaphnia reticulata*). Tadpoles of the frog *Rana catesbiana* were tested, with a 96 hour EC50 = 11800 mg/l. The 72 hour EC50 for the freshwater alga *Selenastrum capricornutum* is >429 mg/l (highest level tested) based on both growth rate and biomass.

DO NOT discharge into sewer or waterways.

12.2. Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	HIGH
isopropanol	LOW (Half-life = 14 days)	LOW (Half-life = 3 days)
hexylene glycol	LOW	LOW

12.3. Bioaccumulative potential

Ingredient	Bioaccumulation
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (LogKOW = 0.0444)
isopropanol	LOW (LogKOW = 0.05)
hexylene glycol	LOW (LogKOW = 0.5802)

12.4. Mobility in soil

Ingredient	Mobility
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (KOC = 45.15)
isopropanol	HIGH (KOC = 1.06)

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Ingredient	Mobility
hexylene glycol	HIGH (KOC = 1)

12.5. Results of PBT and vPvB assessment

	P	B	T
Relevant available data	Not Available	Not Available	Not Available
PBT	✗	✗	✗
vPvB	✗	✗	✗
PBT Criteria fulfilled?	No		
vPvB	No		

12.6. Endocrine Disruption Properties

The evidence linking adverse effects to endocrine disruptors is more compelling in the environment than it is in humans. Endocrine disruptors profoundly alter reproductive physiology of ecosystems and ultimately impact entire populations. Some endocrine-disrupting chemicals are slow to break-down in the environment. That characteristic makes them potentially hazardous over long periods of time. Some well established adverse effects of endocrine disruptors in various wildlife species include; eggshell-thinning, displayed of characteristics of the opposite sex and impaired reproductive development. Other adverse changes in wildlife species that have been suggested, but not proven include; reproductive abnormalities, immune dysfunction and skeletal deformities.

12.7. Other adverse effects

Not Available

SECTION 13 Disposal considerations

13.1. Waste treatment methods

Product / Packaging disposal	<ul style="list-style-type: none"> Containers may still present a chemical hazard/ danger when empty. Return to supplier for reuse/ recycling if possible. <p>Otherwise:</p> <ul style="list-style-type: none"> If container can not be cleaned sufficiently well to ensure that residuals do not remain or if the container cannot be used to store the same product, then puncture containers, to prevent re-use, and bury at an authorised landfill. Where possible retain label warnings and SDS and observe all notices pertaining to the product. <p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> Reduction Reuse Recycling Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> DO NOT allow wash water from cleaning or process equipment to enter drains. It may be necessary to collect all wash water for treatment before disposal. In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. Where in doubt contact the responsible authority. Recycle wherever possible. Consult manufacturer for recycling options or consult local or regional waste management authority for disposal if no suitable treatment or disposal facility can be identified. Dispose of by: burial in a land-fill specifically licensed to accept chemical and / or pharmaceutical wastes or incineration in a licensed apparatus (after admixture with suitable combustible material). Decontaminate empty containers. Observe all label safeguards until containers are cleaned and destroyed.
Waste treatment options	Not Available
Sewage disposal options	Not Available

SECTION 14 Transport information

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	Class	Not Applicable
	Subrisk	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Hazard identification (Kemler)	Not Applicable
	Classification code	Not Applicable
	Hazard Label	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Tunnel Restriction Code	Not Applicable

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

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	ICAO/IATA Class	Not Applicable
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Special provisions	Not Applicable
	Cargo Only Packing Instructions	Not Applicable
	Cargo Only Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Packing Instructions	Not Applicable
	Passenger and Cargo Maximum Qty / Pack	Not Applicable
	Passenger and Cargo Limited Quantity Packing Instructions	Not Applicable
	Passenger and Cargo Limited Maximum Qty / Pack	Not Applicable

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

14.1. UN number	Not Applicable	
14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	IMDG Class	Not Applicable
	IMDG Subrisk	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	EMS Number	Not Applicable
	Special provisions	Not Applicable
	Limited Quantities	Not Applicable

Inland waterways transport (ADN): NOT REGULATED FOR TRANSPORT OF DANGEROUS GOODS

14.1. UN number	Not Applicable
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14.2. UN proper shipping name	Not Applicable	
14.3. Transport hazard class(es)	Not Applicable	Not Applicable
14.4. Packing group	Not Applicable	
14.5. Environmental hazard	Not Applicable	
14.6. Special precautions for user	Classification code	Not Applicable
	Special provisions	Not Applicable
	Limited quantity	Not Applicable
	Equipment required	Not Applicable
	Fire cones number	Not Applicable

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

Not Applicable

14.8. Transport in bulk in accordance with MARPOL Annex V and the IMSBC Code

Product name	Group
trimethylnonyl ether ethoxylated	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available
isopropanol	Not Available
di-CG 20-568 ethoxylated	Not Available
CG 20-568 ethoxylated	Not Available
hexylene glycol	Not Available
tallowalkyl(ethylhexyl)dimethylammonium sulfate	Not Available

14.9. Transport in bulk in accordance with the ICG Code

Product name	Ship Type
trimethylnonyl ether ethoxylated	Not Available
5-chloro-2-methyl-4-isothiazolin-3-one	Not Available
isopropanol	Not Available
di-CG 20-568 ethoxylated	Not Available
CG 20-568 ethoxylated	Not Available
hexylene glycol	Not Available
tallowalkyl(ethylhexyl)dimethylammonium sulfate	Not Available

SECTION 15 Regulatory information

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

trimethylnonyl ether ethoxylated is found on the following regulatory lists

Not Applicable

5-chloro-2-methyl-4-isothiazolin-3-one is found on the following regulatory lists

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI

isopropanol is found on the following regulatory lists

EU REACH Regulation (EC) No 1907/2006 - Annex XVII - Restrictions on the manufacture, placing on the market and use of certain dangerous substances, mixtures and articles

Europe EC Inventory

European Union - European Inventory of Existing Commercial Chemical Substances (EINECS)

European Union (EU) Regulation (EC) No 1272/2008 on Classification, Labelling and Packaging of Substances and Mixtures - Annex VI
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

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Ingredient	CAS number	Index No	ECHA Dossier
CG 20-568 ethoxylated	104810-48-2	Not Available	01-2119472279-28-XXXX 01-0000015075-76-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Sens. 1; Aquatic Chronic 2	GHS09; GHS07; Wng	H317; H411
2	Skin Sens. 1; Aquatic Chronic 2; STOT RE 1	GHS09; GHS08; Dgr	H317; H411; H372

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

Ingredient	CAS number	Index No	ECHA Dossier
hexylene glycol	107-41-5	603-053-00-3	01-2119539582-35-XXXX

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Skin Irrit. 2; Eye Irrit. 2	GHS07; Wng	H315; H319
2	Skin Irrit. 2; Eye Irrit. 2; Acute Tox. 4; STOT RE 2	Wng; GHS05; GHS08	H315; H319; H302; H304; H332; H336; H411; H373

Harmonisation Code 1 = The most prevalent classification. Harmonisation Code 2 = The most severe classification.

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Canada - DSL	Yes
Canada - NDSL	No (trimethylnonyl ether ethoxylated; 5-chloro-2-methyl-4-isothiazolin-3-one; isopropanol; di-CG 20-568 ethoxylated; CG 20-568 ethoxylated; hexylene glycol; tallowalkyl(ethylhexyl)dimethylammonium sulfate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	No (trimethylnonyl ether ethoxylated; di-CG 20-568 ethoxylated; CG 20-568 ethoxylated; tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Japan - ENCS	No (di-CG 20-568 ethoxylated; CG 20-568 ethoxylated; tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Korea - KECI	No (tallowalkyl(ethylhexyl)dimethylammonium sulfate)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (tallowalkyl(ethylhexyl)dimethylammonium sulfate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (trimethylnonyl ether ethoxylated; di-CG 20-568 ethoxylated; CG 20-568 ethoxylated; tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Vietnam - NCI	No (tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Russia - FBEPH	No (di-CG 20-568 ethoxylated; CG 20-568 ethoxylated; tallowalkyl(ethylhexyl)dimethylammonium sulfate)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

SECTION 16 Other information

Revision Date	26/09/2021
Initial Date	26/09/2021

Full text Risk and Hazard codes

H225	Highly flammable liquid and vapour.
H290	May be corrosive to metals.
H301	Toxic if swallowed.
H302	Harmful if swallowed.
H304	May be fatal if swallowed and enters airways.
H310	Fatal in contact with skin.
H311	Toxic in contact with skin.

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H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H318	Causes serious eye damage.
H330	Fatal if inhaled.
H331	Toxic if inhaled.
H332	Harmful if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H335	May cause respiratory irritation.
H336	May cause drowsiness or dizziness.
H340	May cause genetic defects.
H370	Causes damage to organs.
H372	Causes damage to organs through prolonged or repeated exposure.
H373	May cause damage to organs through prolonged or repeated exposure.
H400	Very toxic to aquatic life.
H410	Very toxic to aquatic life with long lasting effects.
H411	Toxic to aquatic life with long lasting effects.
H413	May cause long lasting harmful effects to aquatic life.

Other information

Ingredients with multiple cas numbers

Name	CAS No
5-chloro-2-methyl-4-isothiazolin-3-one	26172-55-4, 61840-41-3, 55965-84-9, 137086-87-4, 137662-59-0
di-CG 20-568 ethoxylated	104810-47-1, 131743-50-5, 1427265-93-7, 391270-80-7
CG 20-568 ethoxylated	104810-48-2, 2081883-59-0
hexylene glycol	107-41-5, 99210-90-9

Classification of the preparation and its individual components has drawn on official and authoritative sources 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.

For detailed advice on Personal Protective Equipment, refer to the following EU CEN Standards:

EN 166 Personal eye-protection

EN 340 Protective clothing

EN 374 Protective gloves against chemicals and micro-organisms

EN 13832 Footwear protecting against chemicals

EN 133 Respiratory protective devices

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

ES: Exposure Standard

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

AIRC: Australian Inventory of Industrial Chemicals

DSL: Domestic Substances List

NDSL: Non-Domestic Substances List

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IECSC: Inventory of Existing Chemical Substance in China
EINECS: European INventory of Existing Commercial chemical Substances
ELINCS: European List of Notified Chemical Substances
NLP: No-Longer Polymers
ENCS: Existing and New Chemical Substances Inventory
KECI: Korea Existing Chemicals Inventory
NZIoC: New Zealand Inventory of Chemicals
PICCS: Philippine Inventory of Chemicals and Chemical Substances
TSCA: Toxic Substances Control Act
TCSI: Taiwan Chemical Substance Inventory
INSQ: Inventario Nacional de Sustancias Químicas
NCI: National Chemical Inventory
FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances